CURRENT CONCEPTS IN DERMATOLOGY

DANIEL LADD, D.O., FAOCD
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Continuing Medical Education Statements

This activity will change your practice and improve patient outcomes!

AOA Statement:
The American Osteopathic College of Dermatology is accredited by the American Osteopathic Association to provide osteopathic continuing medical education for physicians. This activity anticipates being approved for 28-33 hours of AOA Category 1-A credit pending approval by the AOA CCME and will report CME and specialty credits commensurate with the extent of the physician's participation in this activity. March 21 - 25, 2018

ACCME Statement:
The American Osteopathic College of Dermatology is currently seeking accreditation by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. March 21 - 25, 2018
American Osteopathic College of Dermatology
Mission Statement &
Continuing Medical Education Needs Assessment

The Continuing Medical Education Program of the American Osteopathic College of Dermatology will support, enhance and advance new models of academic excellence and community health care.

The mission of the American Osteopathic College of Dermatology is to create innovative education, support, and opportunities in dermatology that promote excellence in patient care and community health through advocacy, consciousness, inclusivity, and osteopathy.

Purpose
The purpose of the CME program is to provide AOA-accredited continuing medical education activities to inform the dermatologist physician. The program will provide a mechanism by which its constituents can improve competency, maintain board certification and cultivate lifelong learning. CME will provide physicians with the opportunity to further develop their knowledge through individual and group learning activities. The Continuing Medical Education Committee will monitor the quality of all programs conducted by the AOCD.

Accreditation:
The AOCD is accredited by the American Osteopathic Association. This activity anticipates being approved for 28-33 hours of AOA Category 1-A credit pending approval by the AOA CCME.

The American Osteopathic College of Dermatology is currently seeking accreditation by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

This meeting will provide a diversified CME presentation focusing on the art and science of dermatology. Information will be presented through lectures and scientific paper presentations. The activity actively encourages members to develop enduring materials as an evolving tool for continuing education. The College is committed to exploring the development of its capacity to expand resources in other educational techniques, including Web-based activities and point-of-care technologies.

Commercial Support Disclosure
AOCD CME will identify relevant financial relationships prior to awarding AOA Category 1A and/or AMA PRA Category 1 Credit™ for CME activities. All persons in a position to influence or control CME content (course directors, program planning committee members, speakers, authors and staff) will complete a standardized disclosure form. Information about funding will be requested to identify CME activities at higher risk for commercial bias.

All AOCD CME activities will be evaluated by learners and possibly peer reviewers to determine if the content was free of commercial bias. All those identified as having influence and/or control of CME content perceived as either manifesting conflicts of interest or being biased may be disqualified from consideration as resources (planning group member, authors, faculty, etc) in subsequent CME activities.

Learners will be provided with information on identified COI from any of the above categories of persons that affect the content of CME, and that information will be positioned in course materials such that it is read by learners prior to the execution of the CME activity. Speakers for the AOCD will be required to provide disclosure information to meeting attendees during their introduction of their topic. Additionally, disclosure statements are provided in the program schedule given to each meeting attendee and is available online at www.aocd.org.

In accordance with the ACCME’s Standards for Commercial Support of Continuing Medical Education, the Policy on Collection of Financial Relationships and Resolution of Conflicts of Interest (COI) exists to provide guidance for staff, instructors, planners, reviewers and managers of CME activities sponsored by The American Osteopathic College of Dermatology (AOCD). This policy addresses the underlying philosophy of disclosure to learners, mechanisms to collect disclosure information and the parties from whom financial disclosure shall be collected, the mechanisms to resolve COI, and requirements to make disclosure to learners prior to the start of an activity.
**Professional Practice Gap Statement:**
Physicians need to understand, update and manage changes in dermatology in order to provide optimal patient care. Dermatologists in private practice may not have immediate access to new updates in therapies and treatments. This activity will help to close gaps in physician's areas of MACRA/MIPS, communication with patients, laser updates, cosmetic dermatology updates, asthma and allergies, practice management and pediatric dermatology.

**Expected Outcomes:**
As a result of participation in the AOCD/CME activity, practicing clinicians will improve competency; maintain specialty board certification; and cultivate lifelong learning. It is expected that attendees of this meeting will improve their diagnostic competence regarding a wide range of dermatologic conditions. In addition to increased diagnostic competence, enhanced concepts of therapy and treatment in dermatologic care will be gained for implementation in everyday practice.

- Attendees will learn how to utilize a PA within a busy dermatology office.
- Attendees will enhance both pediatric diagnostic skills and their pediatric patient outcomes.
- Attendees will gain an understanding of the physiology of Latin skin and the differences among different skin tones.
- Attendees will learn about truly positive patch test results, understand top allergens on the NASS and learn how to counsel patients on avoiding certain allergens.
- Attendees will gain an understanding of the use of different lasers, laser physics and medicine, and the right wavelength for the right trajectory for the right skin type.
- Attendees will learn reconstructive techniques for challenging facial defects.
- Attendees will learn how to analyze proper patient selection and review current available treatment for cosmetic dermatology.
- Attendees will learn asset protection strategies for their practices and personal assets.
- Attendees will gain an understanding of the Quality Payment Program included in the MACRA legislation.
- Attendees will gain an understanding of hidradenitis suppurativa as a disease, the use of biologics, and possible new therapies.
- Attendees will learn the most common aero allergens to cause allergic contact dermatitis.
- Attendees will learn which non-melanoma skin cancers are amendable to non-surgical options.
- Attendees will gain an understanding of the indication for irradiation for skin cancers as related to traditional and new approaches.
- Attendees will be able to identify key clinical and epidemiological issues which help establish actual diagnosis in patients who present with an eschar.
- Attendees will receive an introduction and overview of in-office treatment of unwanted fat, discuss outcomes and possible complications.

The overall result being improved physician/provider performance and increased positive patient outcomes.

These objectives will be achieved in a setting which is evidence-based, culturally sensitive and free of commercial bias. The AOCD is committed to the practice of continuing program improvement. The AOCD will actively explore new educational technologies, develop collaborative relationships with other CME providers and seek to build the capacity to evaluate competency-based outcomes among the clinicians we serve. CME will provide physicians with the opportunity to further develop their knowledge through individual and group learning activities.

**Needs Assessments:**
The activity was developed based upon the needs of physicians within the association identified through:
- An evaluation/survey provided to meeting participants at both our annual and midyear meeting
- Consensus of faculty members within a department or service area
- New advances in dermatologic treatment identified in major publications or research studies
- New methods of diagnosis or treatment
- Availability of new medication(s) or indication(s)
- Development of new technology
- Acquisition of new facilities or equipment
- Input from experts regarding advances in medical knowledge
- Legislative, regulatory, or organizational changes effecting patient care
- Epidemiological data
- Quality assurance/audit data
- Statistics infection control data
- Surgical procedures statistics
- Journal articles/literature citations
The AOCD Continuing Medical Education Committee works to assure the inclusion of appropriate Osteopathic content in the Continuing Medical Education activities presented by AOCD, and to assure that the Continuing Medical Education Programs of the AOCD will achieve the stated objectives of each meeting in a setting which is evidence-based, culturally sensitive and free of commercial bias.

The Continuing Medical Education Committee of the AOCD will monitor the quality of all activities conducted.

Content Areas:
The AOCD approves the CME activities based upon needs assessment data to ensure that all offerings present current, up to date and cutting edge information. Specific areas of emphasis include, new advances in dermatologic treatment, new methods of diagnosis or treatment, availability of new medication(s) or indication(s), development of new technology, advances in medical knowledge and legislative, regulatory, or organizational changes effecting patient care. The Osteopathic Core Competencies of Osteopathic Philosophy, Principles, Practice and Manipulative Medicine, Medical Knowledge, Patient Care, Interpersonal and Communication Skills, Professionalism, Practice-Based Learning and Improvement and System-Based Practice will also be incorporated into all CME activities.

Target Audience:
The primary target audience of the CME activities conducted by the AOCD are the dermatologist physician members. The College also serves community physicians, volunteer clinical faculty, academic clinicians and students affiliated with the AOCD. The activity will also actively seek to broaden its audience through developing affiliations with CME providers on the national level.

AOCD Anti-Trust Statement
Members participating in meetings, events or activities conducted or sponsored by the American Osteopathic College of Dermatology or the Foundation for Osteopathic Dermatology, have an obligation to review and follow the AOCD’s Antitrust Compliance Policy. They should particularly refrain from making statements or distributing materials at AOCD, Foundation meetings or events that would violate the policy, such as suggesting minimum fees for particular services, urging AOCD members to boycott third party payers based on reimbursement levels or other terms of contracting with such entities, or recommending that AOCD members avoid competing with each other in certain geographic areas or markets or across specialties.

Faculty Disclosure:
As a sponsor accredited by the AOA, it is the policy of the AOCD to require the disclosure of anyone who is in a position to control the content of an educational activity. All relevant financial relationships with any commercial interests and/or manufacturers must be disclosed.

Disclosure of Commercial Support of CME:
As you undoubtedly know from the national media, there has been much discussion concerning the relationships between CME sponsors, faculty and commercial companies providing support of CME.

Both the American Osteopathic Association and the Committee on Continuing Medical Education have adopted regulations for ethical actions in this area which the American Osteopathic College of Dermatology endorse and have adopted for all our educational activities.

Please be assured that having an affiliation with a company does not imply in any way that something is wrong or improper; however, we want to inform attendees that such a relationship exists.

Should you have any questions regarding the facilities, handouts, activity content, or concerns about CME compliance with the AOA “Uniform Guidelines,” feel free to contact the AOCD representative:

Marsha A. Wise, BS
Executive Director
P.O. Box 7525
Kirksville, MO 63501
660-665-2184
800-449-2623

Unresolved issues regarding compliance with the AOA “Uniform Guidelines” can be brought to the attention of the AOA Division of CME by calling: 800-621-1773, or by writing:

AOA CME Office
142 East Ontario Street
Chicago, IL 60611
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Disclosures: Speaker: Pfizer

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Disclosures: Director, Officer or Employee of: Wayne County Osteopathic Medical Association (Board Member 2012-2013)

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Disclosures: No relevant financial relationships to disclose

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Disclosures: No relevant financial relationships to disclose
Meeting Faculty & Needs Assessments

Daniel Ladd, Jr., DO, FAOCD – Program Chair
Daniel J. Ladd, Jr., DO, is the Medical Director and Founder of Tru-Skin™ Dermatology in Austin, TX. He earned his BA from the University of Texas at Austin and received his medical degree from Des Moines University in 1999. He completed his dermatology residency at the Northeast Regional Medical Center in Kirksville, Missouri in conjunction with the Dermatology Institute of North Texas in 2004. In addition to general dermatology and cosmetic dermatology, Dr. Ladd is board certified in Mohs micrographic surgery. He is a member in good standing of the American Academy of Dermatology, American Osteopathic College of Dermatology, the American Society of Dermatologic Surgeons and the American Society of Cosmetic Dermatology and Aesthetic Surgery, as well as a Member of the American Society of Mohs Surgery. Dr. Ladd is a lifetime member of the Skin Cancer Foundation's Amonette Circle, an elite group of the country's foremost dermatologists and Mohs surgeons who have made a commitment to skin cancer education and prevention.

Disclosures: Physician Training: Sensus Healthcare; Medical Officer/Minority Shareholder: SkinCure Oncology; Director, Officer or Employee of: Shade Project; Spouse is Director, Officer or Employee of: Shade Project

John Minni, DO, FAOCD
Dr. John Minni is board-certified in dermatology. He graduated, with honors, from Nova Southeastern College of Osteopathic Medicine in Fort Lauderdale, FL. He completed his internship at Union Hospital/St. Barnabas Healthcare System in New Jersey. He then returned to Florida and completed both family medicine and dermatology residencies at Columbia Hospital and the VA Medical Center in West Palm Beach, FL. Dr. Minni also served as chief resident in dermatology. Between residencies, Dr. Minni practiced family medicine at the Palm Beach County Health Department, while training residents, interns and medical students. Prior to medical school, Dr. Minni attended the University of Notre Dame as a Notre Dame Scholar and graduated with honors with a B.S. in biology.

Disclosures: Speaker: Abbvie, Janssen, Promius, Leo, Novartis, Galderma

Jeffrey Johnson, PA-C
Jeff Johnson is a board-certified physician assistant. He is a 1995 graduate of the United States Air Force Physician Assistant program. He completed his training as an honor graduate at the Air Force Academy in Colorado Springs, CO. Prior to PA school, he was an instructor of laboratory medicine at the School of Health Care Sciences at Sheppard AFB, Texas. While on active duty, he was chosen as Physician Assistant of the Year on two separate occasions and twice the Officer of the Year as well. A gifted speaker, he routinely lectures within his community, across the state of Florida and with the Lecture Series Development Program for the American Academy of Physician Assistants. He has given dermatology lectures across the country. Jeff serves as the President of the Florida Society of Dermatology Physician Assistants.

Disclosures: No disclosures provided by speaker

Forging a Successful Practice: Utilizing PA’s in a Busy Dermatology Office

Objectives:
1. Professional utilization of a PA within a busy derm office
2. Definition and implementation of optimal team practice
3. Tips for hiring and employing a PA

Needs:
1. Advances in medical knowledge
2. Legislative, regulatory, or organizational changes effecting patient care

References:

Core Competencies: 3, 4, 5, 6
Dr. Andleeb Usmani is a board-certified, fellowship-trained dermatologist. Dr. Usmani joined the Children's Skin Center in 2010. Dr. Usmani is skilled in all aspects of dermatology, from photodynamic therapy for pre-skin cancer and acne, to skin cancer surgery. She is committed to educating patients and families about the importance of sun safety, developing comprehensive care plans to help repair existing damage and prevent future skin damage.

Dr. Usmani earned her undergraduate degree in biology at the University of Miami, and went on to earn her doctor of osteopathic medicine degree at Nova Southeastern University in 2003, completing family practice and dermatology residencies and an internship, and serving as chief resident in dermatology at Columbia Hospital. Dr. Usmani currently trains general and pediatric dermatology residents at Nova Southeastern University and Miami's Nicklaus Children's Hospital.

Dr. Usmani has strong roots in South Florida, and she enjoys hospital privileges and affiliations at Miami's Nicklaus Children's Hospital, Palms West Hospital, and Wellington Regional Medical Center. A former college athlete who enjoys reading and sports and played competitive badminton, she competed in 200 to 800-meter races and relays, shotput and discus throws, and long jump competitions.

Disclosures: Disclosures not received by publication deadline. Please see supplemental handout available at meeting registration table.

Hemangiomas of Infancy

Needs assessment documentation not received by publication deadline. Please see supplemental handout available at registration.

Dr. Badia completed dermatology training in Albany, New York in 2001. Shortly after, she founded Florida Skin Center as a full-service facility with state of the art treatments and amenities.

The National Osteopathic Pediatric Dermatology Certification Board relies on her professional input, as the doctor is a pediatrician as well as a dermatologist. This background makes her uniquely qualified to provide medical dermatology care for patients of all ages, at Florida Skin Center, including newborn babies. Under her direction, the practice provides narrowband UVB phototherapy for treatment of eczema and psoriasis, as well as Mohs micrographic skin cancer surgery, and dermatopathology services.

While excellence in patient care is Dr. Badia's passion, she is also active in the community. The local Hispanic Affairs Advisory Board has recognized her as Volunteer of the Year, and she received the Congressional Medal of Distinction, a national acknowledgement. Dr. Badia regularly lectures at pediatric postgraduate courses for Miami Children's Hospital. The American Association on Anti-Aging Medicine values her services as a national speaker. She lectures on a variety of dermatology topics, at local and national levels, and her work has been published in leading pediatric and dermatology journals.

Disclosures: Speaker: Pfizer

Pediatric Dermatology: What's New

Objectives:
1. Review of common pediatric dermatological conditions
2. New treatment modalities being used
3. Changing therapy modalities

Needs:
1. Advances in medical knowledge
2. Availability of new medication(s) or indication(s)
3. New advances in pediatric dermatologic treatment

References:
1. Lawrence A. Schachner, MD and Ronald C. Hansen, MD, Elsevier Textbook *Pediatric Dermatology, 4th edition.*

Core Competencies: 2, 3, 6

Eduardo Weiss, MD, FAAD
Dr. Eduardo Weiss was born in Caracas, Venezuela and received his medical degree from the Universidad Central Venezuela. He completed his dermatology residency at Jackson Memorial Hospital in Miami, FL, where he also served as chief resident. Dr. Weiss is board-certified by the American Board of Dermatology and is internationally known as an expert in the field of dermatology. He has diagnosed and treated thousands of patients globally in his 30-year profession. Dr. Weiss specializes in Mohs surgery for skin cancer removal, facial rejuvenation including cosmetic laser technology, Botox, facial fillers, soft tissue augmentation, leg and facial vein treatment, and tumescent liposuction.

He is a Clinical Associate Professor for the Division of Dermatology at Nova Southeastern University, Clinical Professor at University of Miami – Miller School of Medicine, Department of Dermatology and Cutaneous Surgery, and founding voluntary faculty member and Clinical Associate Professor for Florida International University – Herbert Wertheim College of Medicine. Additionally, he serves as a board member of the Florida Society of Dermatologic Surgery, Co-Director for the Mohs Fellowship accredited by the Council for Graduate Medical Education, and Director of the Fellowship of Dermatologic Cosmetic Surgery accredited by ASDS.

Dr. Weiss has many notable achievements in medical care and has served as a principal investigator/co-investigator in many research studies. He is involved in several non-profit organizations and has also had the privilege to treat patients in developing countries and regular community outreach programs. Dr. Weiss continues to be an advocate for our local community and those abroad.

In his free time, Dr. Weiss enjoys boating, gardening and spending time with his three kids and three grandchildren.

Disclosures: No disclosures provided by speaker

No More Fake News: An Evidence Based Approach on Lasers in Skin of Color Patients
The Dermatologic surgeon needs to understand the concerns of the patient, as well as be aware of the unique needs for patients with darker skin. This lecture will discuss health burdens of disease in patients of color along with treatment options and future directions for research. Attendees will have a better understanding of the needs of patients who have pigmented skin, specific cultural practices, and review of burden of skin disease.

Objectives:
1. Understand the physiology of Latin skin and the differences among different skin tones
2. How to prevent complications without sacrificing efficacy

Needs:
1. Advances in medical knowledge
2. New methods of diagnoses or treatment
3. New advances in dermatologic treatment

References:

Core Competencies: 2, 3, 4, 6
Michael Wein, MD

Michael Wein, MD, is Chief of Allergy at Indian River Medical Center and serves on the faculty at Florida State University College of Medicine. He completed his undergraduate work at Brown University, an internal medicine residency at Vanderbilt University, and his post-doctoral fellowship at Johns Hopkins Hospital in the division of Allergy & Immunology. He is board-certified by the American Board of Allergy and Immunology and also by the American Board of Internal Medicine.

Dr. Wein is Past President of the Florida Allergy, Asthma, and Immunology Society and is a Fellow of the American Academy of Allergy, Asthma and Immunology. He has authored several publications including the chapter on allergic rhinitis in *Conn's Current Therapy, 2006 edition*, and has served editorial roles for *DynaMed Online* and *Prescribers Letter* and is currently an Advisory Board Member of Boston-based *Wellness Workdays*. He co-authored a study on Epi-pen which was published in *Annals of Allergy* in 2015 and subsequent featured on CNN. His previous publications relate mostly to allergic inflammation, eosinophils, and adhesion molecules.

His offices are located in Vero Beach and Port Saint Lucie and he enjoys learning about dermatology from his friends practicing dermatology in his community.

*Disclosures: Faculty: Florida State University; Deputy Chief: Indian River Medical Center*

**Asthma and Allergies in Dermatology**

**Objectives:**
1. Identify skin findings of several immunodeficiency syndromes
2. List key dermatologic features of drug-induced hypersensitivity syndromes
3. Describe the role of food allergy in atopic dermatitis and other skin disorders
4. Recognize when to refer a patient with cutaneous venom reaction to an allergist
5. Understand the natural history of penicillin allergy

**Needs:**
1. New advances in dermatologic treatment
2. New methods of diagnosis or treatment
3. Availability of new medication(s) or indication(s)
4. Advances in medical knowledge

**References:**

**Core Competencies:** 2, 3, 6

**Evelyn Gordon, DO**

Dr. Gordon was born and raised in New York City, where she stayed to complete both her undergraduate and medical degrees. She received her undergraduate degree from New York University in Neural Science. She then obtained her medical degree from New York College of Osteopathic Medicine. During medical school, Dr. Gordon participated in research at the New Age Skin Research Foundation where she studied sunscreen use and sun protection practices in pediatric patients. Dr. Gordon completed two years of family medicine residency prior to starting her dermatology training at St. John's Episcopal Hospital. She is currently the chief resident in her third year of dermatology where she truly enjoys teaching medical students and fellow residents. Her interests include medical and cosmetic dermatology as well as stem cell biology and its application in dermatologic diseases.

*Disclosures: No disclosures provided by speaker*
Dermatologic Emergencies

Objectives:
1. To be able to identify life threatening and emergent dermatologic conditions
2. To be able to utilize clinical clues to help differentiate between diseases
3. Review up to date management for dermatologic emergencies

Needs:
1. New advances in dermatologic treatment
2. Advances in medical knowledge

References:

Core Competencies: 1, 2, 3, 4, 5

Great Cases from Osteopathic Institutions
Dermatology often is an anecdotal specialty when it comes to rare diseases and rare manifestations of common disease states. Communication between colleagues often helps even with the most challenging cases. Having seen types of these reviews sometimes help isolate dermatologists who otherwise do not have access to seeing such cases.

Objectives:
1. Present interesting cases
2. Discuss treatment of the interesting cases
3. Understand critical aspects of unusual cases and how to distinguish them from routine disease process
4. Recognize situations where unusual testing modalities such as electron microscopy are of utility
5. Communicate effectively with other disciplines in complex cases so as to facilitate patients care

Needs:
1. New advances in dermatologic treatment
2. New methods of diagnosis or treatment
3. Development of new technology
4. Advances in medical knowledge

References:
3. www.aocd-grandrounds.org

Core Competencies: 2, 3, 4, 5, 6

Shino Bay Aguilera, DO, FAOCD
Dr. Shino Bay Aguilera is a world-renowned, multi-award winning cosmetic dermatologist, dermatologic surgeon, cosmetic laser expert and is dual board-certified with a fellowship in dermatology from the American College of Osteopathic Dermatology and the American Academy of Dermatology.

With over 17 years of experience and ongoing advanced training in lasers and aesthetics, he is a clinical researcher, publisher, former Chief Medical Director and current Assistant Professor of the Dermatology Residency Program at NOVA University, Assistant Professor of Dermatology for Lake Erie College of Osteopathic Medicine, Suncoast University and Universidad del Rosario, Bogota, Colombia. Dr. Aguilera is also a volunteer Assistant Professor of Dermatology for the University of Miami and was appointed chief resident physician for both of his three year residency programs.

As a medical aesthetic resource Dr. Aguilera contributes to several media outlets including New Beauty Magazine, the Aesthetic Guide, Medaesthetic Magazine, CBS, NBC, MegaTV and Telemundo television stations and he has
been consecutively awarded the prestigious national “Best Non-Surgical Facial Enhancement” from the Aesthetic Academy. Dr. Aguilera is internationally recognized as a multi-award winning practitioner of aesthetic dermatology and an industry leader in physician training; however, his true expertise is in understanding individual patients’ needs and his artistry in creating natural rejuvenation and a more youthful appearance.

It was at a very early age that Dr. Aguilera’s work ethic, integrity, passion for people and determination led him to be awarded the title of “Best Young Citizen of Panama” at the age of 15. As a young adult, he moved to Los Angeles to pursue his dream of becoming a doctor, enrolling at Pasadena City College, he learned English in a matter of months and was accepted into UCLA. Dr. Aguilera’s passion for people has continued in his extensive volunteer work nationally and abroad. He is active in his contributions both financially and as a medical volunteer for Hospice, UNICEF, The Red Cross, DOCare International, Handy and Breast Cancer Awareness.

He is the publisher and author of the Amazon bestselling book Be Youthful, a practical guide for patients to stay youthful in mind, body and spirit. He is also co-author of Ethnique and Gender Considerations when doing Fillers and Dermatologic Surgery, and has contributed to numerous journal publications in the field of aesthetic medicine.

Dr. Aguilera is the creator of multiple signature techniques utilizing various dermal fillers and travels the globe to train his colleagues internationally in aesthetic medicine. In addition to his extensive medical knowledge in aesthetic medicine, Dr. Aguilera is highly regarded as a motivational speaker and spiritual advisor, always returning to his passion for people.

Disclosures: Speaker: Merz, SkinCeuticals, Galderma, Allergan

Lasers and Lifestyles
Medical and cosmetic uses of lasers have been pioneered in dermatology. Its use remains important since this field has considerable changes mimicking technological improvements, maintaining competency is important. Review of foundation of laser tissue interactions followed an itemized list of applications in dermatology.

Objectives:
1. Understand the use of different lasers for different lifestyles
2. Understand laser physics and laser medicine
3. Understand using the right wavelength for the right trajectory for the right skin type

Needs:
1. New advances in dermatologic treatment
2. Development of new technology
3. Advances in medical knowledge

References:

Core Competencies: 2, 3, 6

Jean-Paul Azzi, MD
Jean-Paul Azzi, MD, is a Palm Beach facial plastic and reconstructive surgeon specializing exclusively in cosmetic and reconstructive procedures of the face, nose and neck.

Dr. Azzi completed his residency in head and neck surgery/facial plastic surgery at the world-renowned New York Eye and Ear Infirmary in Manhattan and received his board certification. He then completed a fellowship in exclusively facial plastic & reconstructive surgery with the past president of the American Academy of Facial Plastic & Reconstructive Surgery, where he learned cutting edge techniques in facelifting, endoscopic, minimally invasive facelifting, endoscopic brow lifting, endoscopic midface lifting, blepharoplasty (eyelid lifting), otoplasty (ear pinning), neck lifting, fat grafting, skin resurfacing, facial reconstruction, hair transplantation and facial injectable treatments.
In addition to his cosmetic private practice, Dr. Azzi also performs charitable reconstructive procedures in underdeveloped countries such as Vietnam, Guatemala, Ecuador and Colombia. These procedures include repairing cleft lips and palates and reconstructing children with microtia (missing ears) using their rib cartilage.

Dr. Azzi is a Hobe Sound, FL native who enjoys spending time with his family and friends. His interests include tennis, golf and boating. His patients love his sense of humor and warm, caring demeanor.

Disclosures: No disclosures provided by speaker

Facial Plastic Surgery & Charitable Reconstructive Procedures in Underdeveloped Countries

Objectives:
1. Reconstructive techniques for challenging facial defects
2. Expanded use of the bilobed flap for nasal reconstruction
3. Importance of charity

Needs:
1. New advances in dermatologic treatment
2. New methods of diagnosis and treatment
3. Advances in medical knowledge

References:

Core Competencies: 1, 2, 3, 4, 5, 6, 7

Janet Allenby, DO, FAOCD
Janet Allenby, DO, practicing in Palm Beach County, is a board-certified dermatologist specializing in cosmetic treatments. She has been practicing in South Florida for over 22 years. Dr. Allenby grew up in the Southwest and graduated from Nova Southeastern University College of Osteopathic Medicine in 1991. Dr. Allenby is world-renowned for her injection methods. She leverages product placement and strategic distribution to effectively lift and tighten the face and body without overfilling.

Dr. Allenby specializes in enhancing a person’s natural appearance and believes that beauty starts with proper portion and smooth skin for the face and body. Whatever your concern—wanting a more youthful appearance, stimulating collagen growth, body contouring, unwanted hair, pigmentation corrections—Dr. Allenby utilizes safe and modern methods to give you the solutions you desire. Her state-of-the-art injection techniques with cosmetic fillers greatly reduce the downtime for patients. The goal of Dr. Allenby and her well-trained staff is to provide every patient with a pleasant and uplifting experience both physically and mentally.

Dr. Allenby has gained local notoriety and has been featured on local and national newscasts. As a renowned lecturer to the public, her colleagues, and pharmaceutical companies both in the United States and internationally, Dr. Allenby is often sought out for her dermatological knowledge. Dr. Allenby transcends the role of traditional aesthetic medicine and raises it to an art form. With Dr. Allenby’s vast exposure to cutting-edge technology, she is able to utilize the newest and safest procedures to deliver impressive results for her patient’s face and body.

Disclosures: Speaker: Allergan, Galderma; Stock: Allergan, Revance; Trainer: Allergan, Galderma

Cosmetic Dermatology: A General Guideline

Objectives:
1. Analyze proper patient selection
2. Review current available treatments

Needs:
1. New advances in dermatologic treatment
2. Development of new cosmetic techniques
3. Advances in medical knowledge
**References:**


**Core Competencies:** 2, 3, 4, 6

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**Lawrence Klitzman, JD**

Lawrence Klitzman has been a member of the Florida Bar since 1980 and the New Jersey Bar since 1981. He received his undergraduate degree from Brandeis University and earned his Juris Doctor degree from University of Miami School of Law. Mr. Klitzman also obtained a Masters of Law degree in taxation from the University of Miami in 1981.

Mr. Klitzman has had extensive experience handling a variety of business, transactional and litigation matters. Throughout his career, Mr. Klitzman has been involved in many significant real estate development transactions, both residential and commercial. He represents the owners of a variety of closely held businesses and professional practices. He has formed business entities in many jurisdictions, both domestic and offshore, including Wyoming, Alaska, Delaware, Nevada, Florida, New Jersey, Anguilla and the Bahamas, to name a few. Mr. Klitzman's practice includes counseling clients in connection with estate, gift, generation-skipping and income tax matters, generally as they relate to the succession from generation to generation of family businesses and real estate. Mr. Klitzman represents clients in connection with the preparation and administration of wills and trusts and also in the administration of estates. Mr. Klitzman has also served as co-counsel in connection with a number of consumer class action cases. These are cases in which large numbers of consumers are being overcharged a relatively small amount (determined on an individual basis) by large corporations (such as banks and telephone companies) but when combined with others similarly treated constitute a class with a meaningful claim.

*Disclosures: No disclosures provided by speaker*

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**Protecting Your Practice: The Business of Medicine**

**Objectives:**

1. Asset protection strategies for your practice and personal assets

**Needs:**

1. Legislative, regulatory or organization changes effecting patient care

**References:**


**Core Competencies:** 4, 5, 7

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**Peter Saitta, DO, FAOCD**

Dr. Peter Saitta received his Bachelor of Arts from New York University and his medical degree from the University of Medicine and Dentistry of New Jersey. He completed his dermatology residency as chief resident at Oakwood Hospital. Dr. Saitta assists with the osteopathic dermatology residency program at St. John’s Hospital in New York and is also a Clinical Instructor of Dermatology at NYU Department of Dermatology, where he assists in teaching the residents patch testing.

*Disclosures: No disclosures provided by speaker*

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**Contact Dermatitis of Aeroallergens**

**Objectives:**

1. Clinical presentation of contact dermatitis to aero allergens
2. How to patch test for aero allergens
3. Most common aero allergens to cause allergic contact dermatitis
Needs:
1. New methods of diagnosis or treatment
2. Advances in medical knowledge

References:

Core Competencies: 1, 2, 3, 4, 5, 6, 7

Carlos Ricotti, MD
Dr. Carlos A. Ricotti, MD, is board certified both in dermatology and dermatopathology by the American Boards of Dermatology and Pathology. He completed his residency in dermatology at the University of Miami, followed by a dermatopathology fellowship at University of Texas, Southwestern (UTSW) in Dallas.

After spending a year as faculty in the Department of Dermatology at the UTSW, Dr. Ricotti moved to South Florida as a staff physician for the University of Miami Hospitals inpatient dermatology unit and as Director of DermDx Dermatopathology Consulting laboratory. Currently, Dr. Ricotti is Co-Director of the Larkin Community Hospital dermatology inpatient unit, director of dermatopathology at Vitro Molecular Laboratories and a team dermatologist at ClearlyDerm.

Dr. Ricotti has a special interest in psoriasis, inflammatory skin conditions, and various forms of skin cancers. He has extensively published in peer review scientific journals, authored book chapters for dermatology texts and has been an invited speaker on dermatological conditions at local, national and international medical conferences. His interest in advancing the diagnostic and therapeutic aspects of dermatology continues as an active investigator in several clinical trials. In order to find the best diagnostic and therapeutic options for and with his patients, he focuses on utilizing current up to date evidenced based medical approaches.

Dr. Ricotti’s interests outside of work include kitesurfing, tennis, yoga and art.

Disclosures: Disclosures not received by publication deadline. Please see supplemental handout available at meeting registration table.

Medical Management of TEN and Update on Therapy
This lecture will introduce basic approaches to complex medical dermatology with specific diseases being reviewed.

Objectives:
1. Increase the ability to diagnose complex medical dermatology decisions
2. Increase the ability to evaluate etiology and internal diseases manifestations of the above
3. Increase the ability to direct the above decisions

Needs:
1. New advances in dermatologic treatment
2. New methods of diagnosis or treatment
3. Advances in medical knowledge

References:
1. Bolognia, Jorizzo, Shafer textbook by Elsevier “Dermatology”.
2. Callen, Jorizzo Elsevier textbook “Dermatological Signs of Internal Disease.”

Core Competencies: 2, 3, 4, 6

Francisco Kerdel, MD
Dr. Francisco A. Kerdel attended the St. Thomas Hospital Medical School, London University in London, England. He completed his dermatology residency at Harvard Medical School in Boston, MA, where he was chief resident during the year 1983-1984. He completed fellowships at Guy’s Hospital, London, England and New York University School of Medicine, New York, NY.
He is the former Director of Dermatology Inpatient Unit at the University of Miami Hospital and former Professor of Dermatology at the University of Miami. Dr. Kerdel is a Past President of the International Society of Dermatology and the treasurer of the Foundation for International Dermatologic Education. He is currently a clinical professor at Florida International University, where he is also the Vice Chairman of the Department of Dermatology. He is also the Medical Director of Dermatology at Larkin Community Hospital. Dr. Kerdel's society memberships include the American Academy of Dermatology, Society for Investigative Dermatology, American Dermatological Association, Noah Worcester Dermatological Society and Miami Society of Dermatology. Dr. Kerdel has been chosen to be an Honorary Member of the Venezuelan, Argentinian and Chilean Societies of Dermatology and a corresponding member of the Venezuelan Academy of Medicine.

He has authored over 202 scientific articles, 37 books and book chapters. As an invited speaker, Dr. Kerdel has spoken at national meetings and at international meetings worldwide. He has been a visiting professor in the United States, Japan, Portugal, Italy, Australia, Argentina, Brazil, Chile, Spain, Paraguay, United Kingdom, Venezuela, Colombia, Uruguay and Mexico.

Disclosures: Speaker: Centocor, Abbott, Amgen, Gsai, Astellas, Wyeth; Investigator: Novartis, Merck; Off-Label: Biologics and PDE4-Inhibitors in Hidradenitis Suppurativa

### Medical Treatments in Hidradenitis Suppurativa (H/S)

Physicians need to stay current on treatments for this chronic skin disease. This disease can cause chronic pain and could lead to self-consciousness, social isolation and depression for the patient.

#### Objectives:
1. Understand hidradenitis suppurativa as a disease
2. Use of biologics in hidradenitis suppurativa
3. Possible new therapies in hidradenitis suppurativa

#### Needs:
1. New advances in dermatologic treatment
2. Advances in medical knowledge
3. Availability of new medication(s) or indication(s)

#### References:

#### Core Competencies: 2, 3, 6

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### Inflammatory Skin Diseases

**LECOMT/Larkin Community Hospital Palm Springs Campus**

Nady Hin, DO; Michael Lipp, DO; Rachel White, DO

Nady Hin, DO; Michael Lipp, DO and Rachel White, DO are second year and also co-chief dermatology residents at Larkin Community Hospital Palm Springs Campus in Hialeah, FL under the direction of Dr. Brad Glick.

Disclosures: No disclosures provided by speakers

**Objectives:**
1. Basic overview of pathogenesis, clinical features, pathology, systemic disease associations and work up of granuloma annulare
2. An up-to-date review of treatments options for granuloma annulare
3. Clinical presentations of atopic dermatitis
4. Current pathologic theories behind atopic dermatitis
5. Current treatment options for atopic dermatitis
6. The proposed pathogenesis of alopecia areata
7. The various clinical presentations of alopecia areata
8. New treatments for alopecia areata
Needs:
1. New advances in dermatologic treatment
2. New methods of diagnosis or treatment
3. Availability of new medication(s) or indication(s)
4. Advances in medical knowledge

References:

Core Competencies: 2, 3, 5, 6, 7

John Coppola, DO, FAOCD
John C. Coppola, DO, is a board-certified dermatologist and skin cancer surgeon with advanced training in a wide array of skin conditions and cosmetic procedures. A Clearwater Floridian, Dr. Coppola earned his Bachelor of Science degree from the University of North Carolina at Chapel Hill. After receiving his medical degree with highest honors from Nova Southeastern University, he completed his dermatology residency at Michigan State University Botsford Hospital and served as chief resident his final year.

Dr. Coppola currently enjoys training the next generation of physicians as a Clinical Associate Professor for Florida State University's College of Medicine. His previous teaching appointments included serving as a Clinical Instructor of Michigan State University while in private practice in Michigan. He is the author of numerous published journal articles and is now active in dermatologic medical research.

His passion for personalized care focuses on three key tenets: preventing sun damage, educating his patients on skin health & vitality and getting to know his patients also as people (for military veterans, he is eternally grateful for their service).

When not at work, he can be found most days spending time playing with his German Shepherd “Grizzly”.

Disclosures: No disclosures provided by speaker

When the WiFi Goes Down: The EMR Doomsday Scenario Isn’t That Bad

Trust, But Verify: The Golden Rule for Every Physician’s Practice

“Average” Staff is Your Achilles Heel

Setting Limits in Your Practice: 3 Lines in the Sand to Draw Tomorrow
These lectures offer a myriad of tips on running your medical and surgical dermatology practice patient schedule on time to improve patient satisfaction and reduce physician burn out.

Objectives:
1. Discussion of Cloud-based IT server based EMR systems
2. Billing and charting issues with loss of Wi-Fi access
3. Identify areas in your practice where the physician owner should actively double check employees
4. Identify “best practices” by staff members
5. Identify loss of business due to average employees
6. Identify “red lines” all providers/doctors should establish in their practice
7. Identify “red lines” business owners should establish in their practice
8. Identify “red lines” employee physicians should establish in their practice

Needs:
1. Development of new technology
2. Legislative, regulatory, or organizational changes effecting patient care

References:
3. “Managing staff relationships and cultivating a culture of growth in your practice”.

Core Competencies: 3, 4, 5, 6

Lisa Hackney
Currently, Lisa is an independent consultant directing the patient experience initiatives for Advanced Dermatology & Cosmetic Surgery, based in Maitland, FL. In this role, she manages the entire patient experience and survey feedback for over 180 practices nationally. Prior to this specific role, Lisa served as Managing Director in the start up of Bedside Dermatology, working with Dr. Steven Grekin in the start up of this business providing dermatological care to the senior and aging population.

In addition, Lisa serves as a Director to Talent Plus – an international consulting company providing selection and development expertise to many companies known for delivering world-class service through the selection of the right talent. For over 26 years, her clients included Florida Hospital, Bon Secours Health System, Cancer Treatment Centers of America, Hackensack Medical System along with Mercedes Benz, BMW, Ford Motor Company, Ferragamo USA, Estee Lauder, Ceridian and many other award-winning clients. Most notably, her work with the Ritz-Carlton Hotel Company serving as a partner for almost 18 years, serves even today as a benchmark for many companies. She collaborated with them in their pursuit of the Malcolm Baldrige National Quality Award – the only company to have won this award twice.

Prior to consulting, Lisa completed General Electric’s Management Development program and started her career in human resources management within the Lighting Division of GE.

Lisa has served on the Health Care Advisory Board based out of Washington, D.C. and was a Quality Collaborator with Press Ganeys – a patient satisfaction measurement company. Originally from Pennsylvania, she received her Bachelor of Science degree with honors at Youngstown State University and currently lives in Naples, FL and Cincinnati, OH with her husband.

Disclosures: No disclosures provided by speaker

Steven Grekin, DO, FAOCD
Dr. Steven Grekin has made it his personal and professional mission to help his patients put their best face forward. Years of research at the International Skin Rejuvenation Institute in Paris, France, and Quebec, Canada, have led Dr. Grekin to understand the secrets to younger, smoother, more radiant skin. Respected here and abroad as an expert in cosmetic dermatology, Dr. Grekin comes from a long line of physicians – six are dermatologists. He has participated in international teaching and training courses and is an internationally recognized lecturer in his field. Guided by cutting-edge principles of modern dermatology, natural medicine and the highest quality medical care, Dr. Grekin offers his patients an elegant, intelligent program distinguished by its unique flexibility to restore every skin type to its youthful, natural best. His family has been providing health care in the United States for almost ten years. Dr. Grekin is committed to helping patients from all over the world. He now offers his programs online, so that he may reach out and help as many people as he can put their best face forward.

Disclosures: Research Sponsors: Allergan, Valeant, Coherus, Mimetica, Galderma, Promise, J&J Psolar, HedgePath

Recruit & Select the Best Talent

Reputation Management
Service Excellence Standards

Develop Your Team Around You

Objectives:
1. Key ways to recruit in today’s market
2. How to attract the best and brightest
3. Financial impact on the practice
4. Understanding the changing role of a physician’s online reputation
5. How increasing patient satisfaction can benefit the online presence
6. What a provider can do to manage the effects of negative reviews
7. Ways to lead your practice to excellence
8. The benefits of strength management
9. Key learning your team can benefit from on understanding your leadership style
10. Why service excellence matters
11. Understanding and benchmark best practices of service standards inside healthcare and other industries
12. The one initiative you can put in place immediately to enhance your service

Needs:
1. Development of new technology
2. Legislative, regulatory, or organizational changes effecting patient care

References:
2. Healthcareers.com
3. “How is Your Medical Practice Handling Online Reviews”. InBoundMD.

Core Competencies: 4, 6

Reagan Anderson, DO, FAOCD
Dr. Reagan Anderson specializes in general dermatology and in Mohs micrographic surgery for the treatment of skin cancer. After graduating from Rampart High School in Colorado Springs, CO, Dr. Anderson moved to Vancouver, British Columbia where he attained his Bachelor of Science in biology from the University of British Columbia and a Master of Christian Studies degree from Regent College. Dr. Anderson was then invited to attend the founding osteopathic medical school, Kirksville College of Osteopathic Medicine. Upon matriculation, Dr. Anderson was commissioned in the United States Navy where he spent the majority of his time serving the United States Marine Corps as the First Reconnaissance Battalion Surgeon.

Dr. Anderson left the military in order to pursue dermatology. During his three-year dermatology residency at the Michigan State University Consortium/Oakwood Southshore Medical Center, he was actively involved in academic pursuits, which included national and international lecturing, as well as publishing several dermatologic articles. From October 2008-October 2009, Dr. Anderson represented all osteopathic dermatology residents as the resident liaison for the American Osteopathic College of Dermatology.

Disclosures: Supply excess tissue from surgical cases when appropriate: KAO Corporation Director, Officer or Employee of: RVUCOM; Principal Investigator: Novartis, Abbvie; Spouse is a BeachBody Coach

Compliance Starts With a Voice That Smiles and a Sincere Handshake

You Have to Earn the Right to Be Heard
Hypnosis

Success Without Fulfillment is the Ultimate Failure

Objectives:
1. Developing/Measuring/Making reproducible your brand via yourself and your staff
2. Consistency of message, of medicine, of intention
3. Policies and procedures in place
4. Having the title “doctor” only gets you into the room
5. Being a “doctor” means nothing outside of medicine
6. Being a “osteopath” in all aspects of life
7. Bucket-list for medicine and life
8. Deathbed thoughts
9. You have not arrived after residency
10. How you talk to yourself is how you talk to others
11. How you perceive yourself is not how others perceive you
12. Having and teaching perspective are the keys to interaction

Needs:
1. New advances in dermatologic treatment
2. Availability of new medication(s) or indication(s)
3. Legislative, regulatory, or organizational changes effecting patient care

References:

Core Competencies: 1, 3, 4, 5

Neha Sangwan, MD
Neha Sangwan, MD, CEO and founder of Intuitive Intelligence, is an Internal Medicine physician and communication expert empowering healthcare practitioners, organizational leaders and corporate employees to excel under www.DoctorNeha.com pressure. Dr. Sangwan is an international speaker and media spokeswoman on the topics of conflict resolution, leadership and team transition, power and hierarchy, stress management and employee wellness. She is also the author of TalkRx: Five Steps to Honest Conversations that Create Connection, Health and Happiness.

She currently consults with innovative corporations and complex healthcare teams. Dr. Sangwan has pioneered numerous successful programs that improve organizational metrics related to employee engagement, culture transformation and patient satisfaction. These results were published in General Surgery News (June 2010).

Notably, Dr. Sangwan and her team have developed a comprehensive program called the i-Five Experience that connects the dots between job satisfaction, health and performance. Some of her clients include: Kaiser Permanente, American Heart Association, Brigham & Women’s Hospital, Stanford’s Med X and University of Michigan’s School of Medicine. Dr. Sangwan began her teaching career as faculty for Kaiser Permanente’s Northern California Physician Education and Development Team. She designed and delivered innovative workshops to integrate physicians and nurses into cohesive, productive teams within fast-paced and high-stress environments.

Dr. Sangwan earned her Bachelor of Science in mechanical and biomedical engineering from Michigan State University. She worked as a manufacturing engineer for Motorola before attending medical school at State University of New York at Buffalo. She subsequently completed her internal medicine residency training at Temple University Hospital and became board certified.

Disclosures: No disclosures provided by speaker

Patient Communication & Physician Burnout

Five Steps to Honest Conversations that Create Connection, Health and Happiness
Addressing Dysfunctional Dynamics in Medicine (and in Life!)

Mastering the Most Challenging Personalities at Work & Home
There are many aspects of healthcare that are undergoing rapid change. It is important to be aware of these changes and to be proactive in addressing them. This lecture will help attendees understand a review of new dermatological medications, understanding potential changes to payment models and a discussion of practice management tips.

Objectives:
1. Expand perspectives to become a better negotiator in conflict with patients to create better outcomes
2. Identify a new approach to treating and healing physician burnout
3. Learn how to recognize early signals of burnout and address them
4. Recognize the body’s physical cues under stress
5. Learn how to self-manage in order to respond instead of react
6. Recognize the five components to engage in effective conversations
7. Recognize and describe the five levels of agreement
8. Identify what questions to ask others who are at differing levels of agreement
9. Learn to influence others and bridge during conflict
10. Describe the five levels of listening as a practical tool to quickly and effectively connect to patients and providers
11. Apply the five levels of listening as a tool to bridge to others during conflict
12. Use the five levels of listening to influence and engage patients during high stress situations

Needs:
1. Advances in medical knowledge

References:
2. TalkRx, Five Steps to Honest Conversations that Create Connection Health and Happiness. N. Sangwan, MD.

Core Competencies: 4, 5

Gil Yosipovitch, MD, FAAD
Dr. Gil Yosipovitch is a dermatologist in Miami, FL and is affiliated with University of Miami Hospital. He received his medical degree from Tel Aviv University Sackler. He then completed residencies in dermatology and internal medicine at Rabin Medical Center in Petah Tiqva, Israel, and a fellowship in dermatology at the University of California, San Francisco School of Medicine. He is one of 44 doctors at University of Miami Hospital who specialize in Dermatology.

His special interests include investigating the causes and treatments of complex skin diseases such as eczema, psoriasis, and diseases of other organ systems with skin manifestations and chronic itch. The primary goal of Dr. Yosipovitch’s research is to investigate the neuroanatomy and neurophysiology of itch and developing anti-pruritic drugs that target the neural system.

Disclosures: Advisory Board: Trevi, Pfizer, Sanofi, Menlo, Galderma, Sienna; Consultant: Opko, LEO, J&J, Menlo, Novartis; Principal Investigator: Tioga, Roche, Pfizer, Allergan; Funded: GSK, LEO Foundation, Pfizer, Sun Pharma

Chronic Itch Clinical Cases and Management

Objectives:
1. Demonstrate competency of recognizing various forms of chronic itch
2. Evaluate a patient with chronic itch without a rash
3. Develop strategies of treatment of chronic itch of different type

Needs:
1. New advances in dermatologic treatment
2. New methods of diagnosis or treatment
3. Advances in medical knowledge
References:

Core Competencies: 3, 4, 6

LECOMT/Larkin Community Hospital, South Miami Campus
Danielle Nicolazzo, DO was born in Boston, MA. She attended the University of Michigan where she received a bachelor in Biopsychology. She then attended Boston University where she obtained a post-baccalaureate in medical sciences. She went to medical school at Nova-Southeastern University and completed a Traditional Rotating Internship at Larkin Community Hospital. She is currently a third year dermatology resident at Larkin Community Hospital and plans to remain working in South Florida after she graduates.

Franz Kerdel, DO is a native of South Florida. He received an undergraduate degree in Biology at the University of Miami. After graduating from the University of Miami, he went on to obtain a Masters in Biomedical Sciences from Barry University. He attended medical school at Nova Southeastern University. He completed a Traditional Rotating Internship and is currently a dermatology resident in his third year at Larkin Community Hospital. After graduating he plans on practicing dermatology in South Florida.

Liza Brown, DO is a native of Florida. She received her undergraduate degree in Psychosocial sciences at the University of South Florida in Tampa. She worked at Moffitt Cancer Center for one year performing Melanoma research prior to attending medical school at Lake erie college of medicine in Bradenton FL. She completed her Internship at Largo medical center, followed by a clinical dermatology fellowship year. She is currently a third year dermatology resident at Larkin Community Hospital and plans to work in the Tampa area after she graduates.

Nickolas Poulos, DO is a native of Florida. He received an undergraduate degree in Biology at the University of Miami. After graduation he proudly served in the US Army for four years as an emergency health care specialist. He then went on to pursue a career in medicine and attended Nova Southeastern University. He completed a Traditional Rotating Internship and is currently a dermatology resident in his third year at Larkin Community Hospital. After graduating he plans on opening a practice in Colorado.

Disclosures: No disclosures provided by speakers

**Medical Dermatology Update**

This lecture will introduce basic approaches to complex medical dermatology with specific diseases being reviewed.

**Objectives:**
1. How to manage and identify complex medical dermatology cases
2. Learn various presentations and variations of skin conditions
3. New treatments on the horizon
4. Review histopathology of unique and difficult cases
5. Explore different treatment options for difficult to manage cases
6. Diagnosis and pathophysiology of select diseases

**Needs:**
1. New advances in dermatologic treatment
2. Availability of new medication(s) and indication(s)
3. Advances in medical knowledge
4. New methods of diagnosis or treatment
5. Development of new technology
6. Legislative, regulatory, or organizational changes effecting patient care

**References:**
4. Weedon's Skin Pathology, Weedon, David. Section 7, Chapter 38, pg. 920.

**Core Competencies:** 1, 2, 3, 4, 6, 7

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**Frank Armstrong, DO, FAOCD**

Dr. Frank T. Armstrong, DO, is a highly-recognized, longstanding member of the medical community in Pinellas County, Florida. Dr. Armstrong received his Bachelor of Arts degree with honors from Curry College in Milton, MA. He then earned a degree in Nuclear Medicine at Salem State College in Salem, MA. He received his Medical Degree from the University of New England – College of Osteopathic Medicine in Biddeford, ME, where he graduated with honors and also received the esteemed “Graduate with Distinction” award which is bestowed upon the one graduate the medical school faculty feels “possesses those qualities the faculty would seek in their family physician”. Dr. Armstrong completed his internal medicine residency at the St. Vincent Hospital in Worcester, MA and is board certified by the American Board of Internal Medicine. Dr. Armstrong trained in dermatology at the Sun Coast Hospital/Nova Southeastern University in Largo, FL. As co-chief resident at the Sun Coast Hospital in Largo, FL, Dr. Armstrong earned the Koprince Lecture Award, the Connetics Resident Examination Award and was voted resident liaison by his colleagues.

Dr. Armstrong has been honored as one of America's Top Physicians by the Consumers’ Research Council of America, showing his commitment to practicing medicine and patient care. Dr. Armstrong was also selected as “Physician of the Year” by the Pinellas County Osteopathic Medical Society in January of 2013 at their annual Osteopathic Seminar Banquet.

Dr. Armstrong serves as a Clinical Instructor for the Division of Dermatology at Nova Southeastern University College of Osteopathic Medicine and Lake Erie College of Osteopathic Medicine. He serves on the Board of Governors of the Pinellas County Osteopathic Medical Association and is a Past President of the Pinellas County Osteopathic Medical Society. In addition, he is a member of the American Academy of Dermatology, American Osteopathic College of Dermatology, American Osteopathic Association, Florida West Coast Dermatologic Surgeons, Florida Osteopathic Medical Association.

Dr. Armstrong lives in Palm Harbor with his wife Sheryl and his two sons, Nicholas and Devin.

**Disclosures:** No disclosures provided by speaker

**Non-Invasive Cutaneous Oncology, Part 1**

This lecture is designed to educate clinicians on the use of radiation therapy in the management of BCC/SCC cancers.

**Objectives:**
1. Which NMSC’s are amenable to non-surgical options
2. Topical Rx options for dosing and results
3. Intralesional options for dosing and results

**Needs:**
1. New advances in dermatologic treatment
2. Development of new technology
3. Advances in medical knowledge
4. New methods of diagnosis or treatment

**References:**

**Core Competencies:** 2, 3, 4, 6
Josh Swindle, RTT
With a multifaceted background in clinical and managerial roles, Joshua served as Director of Business Development for Warm Springs/Post Acute Medical Specialty Hospitals, including long term acute care and acute rehabilitation hospitals. His responsibilities range from patient navigation for post-acute care, medical justification documentation, consulting to health care practices, cultivating relationships, compliance and education programs. With experience in facility management and ongoing business leadership training, he is an asset to his fellow co-workers and organizations.

In his previous role as Chief Radiation Therapist and Consultant with Revenue Cycle Inc., Joshua managed numerous modalities of radiotherapy, patient work-up, treatment devices, oncology software, facility operations and daily clinical flow. He also assessed feasibility of cancer centers, reviewed designs and assisted with new service line formation.

Joshua is a member of the American Registry of Radiologic Technologists and American Society of Radiologic Technologists. He graduated magna cum laude from Texas State University with a Bachelor of Science degree in radiation therapy and technology. He resides in San Marcos, TX, where he, his wife and daughter, Calli and Ava, remain involved within the community for continued growth through local businesses and healthcare.

Disclosures: No disclosures provided by speaker

Non-Invasive Cutaneous Oncology, Part 2
This lecture will discuss traditional radiation for skin cancer versus new approaches which entail the use of electronic brachytherapy machines, radiation techniques and outcomes.

Objectives:
1. An understanding of basic principles of irradiation for skin cancer as related to traditional and new approaches
2. Understanding indications for irradiation in terms of traditional and new approaches
3. Understanding of clinical data as related to traditional and new SRT or Ebx in terms of outcome and complications

Needs:
1. New advances in dermatologic treatment
2. Development of new technology
3. Advances in medical knowledge
4. Legislative, regulatory, or organizational changes effecting patient care

References:

Core Competencies: 1, 2, 3, 4, 5, 6, 7

Clifford Lober, MD, JD
Dr. Clifford Lober is a board-certified dermatologist. He received his medical degree from Duke University School of Medicine in 1974. He then completed his internship at Mayo Clinic in 1977 and his residency at the University of Tennessee in 1982.

Dr. Lober has been in the full-time private practice of dermatology in Kissimmee, FL, for 29 years. He is Adjunct Associate Professor of Medicine in the Department of Dermatology and Cutaneous Surgery at the University of South Florida.

Dr. Lober has received four Presidential Citations from the American Academy of Dermatology and was named “Surgeon of the Year” in 1992 by the Florida Society of Dermatology and Dermatologic Surgeons. In addition to being awarded “Practitioner of the Year,” he was awarded the first ever “Distinguished Service Award” by the Florida Society of Dermatology and Dermatologic Surgery. Dr. Lober has served on the Board of Directors of the AAD and chaired its section on Health Practice, Policy and Research.

Disclosures: No disclosures provided by speaker
How to Hit a Homerun with MACRA!
This lecture will provide attendees with a better understanding of the direction of healthcare reform, to review the important aspects of the MACRA legislation, and to learn how to easily avoid any MIPS penalties this year.

Objectives:
1. Understand the basis for healthcare reform
2. Understand the Quality Payment Program included in the MACRA legislation
3. Learn how to avoid MIPS penalties

Needs:
1. Legislative, regulatory, or organizational changes effecting patient care

References:
1. Medicare Program, Merit-Based Payment System (MIPS) and Alternative Payment Model (AMP) Incentive Under the Physician Fee Schedule and Criteria for Physician-Focused Payment Models, 81 FR 77008. (final Rule Nov 4, 2016).

Core Competencies: 3, 6, 7

Ted Rosen, MD, FAAD
Dr. Ted Rosen is a board-certified dermatologist. Dr. Rosen received a Bachelor of Science degree from Michigan State University. He obtained his medical degree from the University of Michigan Medical School in Ann Arbor. Following medical school, he completed an internship at University of Alabama School of Medicine, Birmingham, AL. He then completed internal medicine and dermatology residencies at Baylor College of Medicine in Houston, TX.

Dr. Rosen now serves as Professor of Dermatology and Faculty Senator at Baylor College of Medicine. He is Chief of Dermatology Service at Michael E. DeBakey Veterans Affairs Medical Center.

Disclosures: Honorarium: Valeant (Ortho), Medimetriks

Dermatological Emergencies: The Eschar

Objectives:
1. Recognize the broad differential diagnosis associated with eschar formation
2. Identify key clinical and epidemiological issues which help establish actual diagnosis in patients who present with an eschar
3. Facilitate timely administration of proper therapeutic intervention in patients with an eschar

Needs:
1. New advances in dermatologic treatment
2. Advances in medical knowledge
3. New methods of diagnosis or treatment

References:

Core Competencies: 2, 3

Realizing the Vision: Excellence in Dermatology

Objectives:
1. Discuss how DO Dermatologists can become more involved in overall dermatology activities
2. Review ways to improve overall and clinical presentation skills
3. Establish key relationships with MD dermatology community
4. Learn to recognize strengths in your own personal background that allow you to develop a niche
5. Communicate effectively with mentors such that you can develop and foster professional growth
6. Consider expanding your knowledge base to areas outside medicine to develop unique expertise

Needs:
1. Legislative, regulatory, or organizational changes effecting patient care

References:
5. J Am Osteopathic Association: May 1, 2011; Vol. 111, No. 5; Pages 335-338 “Dermatology: A Specialty that Exemplifies the Osteopathic Medical Profession”.

Core Competencies: 5, 6, 7

James Warrick
James is a trusted relationship for women and men. His client list is made up of leaders in every industry who say, “James is an innovator in developing people.” It’s the sharpness of the conversation, the challenge of new thinking, the action that is taken…it’s what we hope for in a coaching relationship.

James holds a Masters in Coaching and Leadership Development. He has earned the prestigious credentials of a Professionally Certified Coach (PCC) with the International Coach Federation. James has taught over 50 graduate-level coaching courses and trained over 500 new coaches.

James coaches leaders from a wide range of organizations and sectors: Nike, Intel, Merrill Lynch, Williams Sonoma, R2C Advertising, Pacific Foods, Compassion International, Clark Nuber Global Accounting, Washington State, Planar Systems, Chapman University, health care providers, entrepreneurs and bestselling authors.

The best part is, James is gutsy. The gutsy that asks the questions that you’re dodging. The gutsy that helps leaders stop making it about themselves. The gutsy that helps people listen to their own gut and move forward with confidence. James brings proven tools and a process to develop people.

James’ wife is his best friend, and his four kids still wake him up at night.

Disclosures: Leadership Consultant: Tru-Skin Dermatology

Daniel Ladd, DO, FAOCD
See page 7 for biography and disclosures.

Shared Leadership (Presented w/ Daniel Ladd, DO, FAOCD)

Objectives:
1. The insight in putting your practice as central and getting yourself as a provider into a sustainable rhythm
2. Gain new skill in developing top performing staff and provider as a way of growing your practice
3. Developing a shared vision in your practice and empowering other key providers and staff to take more responsibility for the practice

Needs:
1. Development of new technology
2. Legislative, regulatory, or organizational changes effecting patient care

References:

Core Competencies: 4, 6
Michelle Foley, DO, FAOCD

Dr. Michelle Foley is a board-certified dermatologist specializing in medical and surgical dermatology, with a passion for non-surgical aesthetics and facial rejuvenation. Her practice approach is to provide personalized care and education for each of her patients. Dr. Foley works with both men and women to help them look their best utilizing non-invasive techniques; combining injectables, topical agents, lasers and physician-strength skin care. “Best results are always achieved when you partner with your patient to build a treatment plan that is right for that individual. Cosmetic dermatology is not a one-size-fits-all world,” she explains.

Dr. Foley was born in Alabama and grew up on the west coast of Florida. After graduating Summa Cum Laude from Florida State University, she attended Nova Southeastern University College of Osteopathic Medicine in Ft. Lauderdale, FL. There she graduated with the highest of honors and received the Terry Internal Medicine Award for the highest achievement in academic and clinical internal medicine. Dr. Foley completed her dermatology training at Michigan State University/POH Regional Medical center in Detroit, MI where she served as the chief resident.

Locally, Dr. Foley is an Associate Clinical Professor for Florida State University College of Medicine and a volunteer educator for Halifax Hospital Family Medicine Program. She also serves as the Associate Editor for the Journal of the American Osteopathic College of Dermatology.

Disclosures: No disclosures provided by speaker

Non-Invasive Modalities in Lipolysis

Objectives:
1. Introduction and overview of in-office treatment of unwanted fat
2. Review off-label use of these modalities
3. Discuss outcome and possible complications from treatment

Needs:
1. New advances in dermatologic treatment
2. Availability of new medication(s) or indication(s)
3. Development of new technology

References:

Core Competencies: 2, 3, 6

Charles Gropper, MD

Charles A. Gropper, MD, is a board-certified dermatologist who is a diplomate of the American Board of Dermatology and the National Board of Medical Examiners. Currently, Dr. Gropper is an Associate Clinical Professor of Dermatology at the Mount Sinai School of Medicine, as well as a dermatopharmacology peer reviewer for the Lancet Journal.

Dr. Gropper graduated with an Artium Baccalaureatus (A.B.) degree with magna cum laude honors from Brown University in Providence, RI. He attended the University of Pennsylvania School of Medicine where he received his medical degree. After completing his education, Dr. Gropper completed an internal medicine internship at the Mount Sinai Hospital followed by a residency in dermatology at Albert Einstein College of Medicine and a fellowship in dermatopharmacology at New York University Medical Center.

For over 18 years, Dr. Gropper has practiced medicine in hospitals and private practices in New York, Brooklyn and the Bronx. He has also held positions as Chief of Dermatology and Associate Clinical Professor of Dermatology. Currently, he is Chief of Dermatology at St. Barnabas Hospital in the Bronx. He has co-authored numerous professional articles and book chapters related to dermatology and dermatopharmacology. He has received over a dozen awards for significant contributions, teacher of the year, educating residents and exceptional service.
Dr. Gropper is a fellow of the American Academy of Dermatology, as well as a member of the American Medical Association, the International Society for Digital Imaging of the Skin and the Space Dermatology Foundation.

Disclosures: No disclosures provided by speaker

Welcome to Derm Clinic in the Bronx

Objectives:
1. Review of differential diagnosis of some interesting rare cases from a busy urban teaching hospital dermatology clinic
2. Review of clinical features of some interesting dermatologic conditions only seen rarely
3. Review the treatment options for some rare dermatologic conditions

Needs:
1. New advances in dermatologic treatment
2. Advances in medical knowledge
3. New methods of diagnosis or treatment

References:

Core Competencies: 2, 3

Edwin A. Bayo, JD
Ed was born in San Juan, Puerto Rico. After graduating in 1978 with a bachelor's degree in economics (cum laude) from the University of Puerto Rico, he moved to the United States to pursue his legal education. He received his Juris Doctorate from Stetson Law School in 1981.

Ed worked in various capacities for the Florida Office of the Attorney General, including tax litigation, administrative law, cabinet affairs and inspector general. His primary area of practice while in government involved providing advice and representation to regulatory boards under the umbrella of the Department of Health and the Department of Business and Professional Regulation. As Senior Assistant Attorney General in the Administrative Law Section, Ed served as counsel to various professional regulatory boards, including pharmacy, dentistry, osteopathic medicine, chiropractic medicine, veterinary medicine, professional engineers, landscape architecture, clinical social work and harbor pilots. In addition, he handled temporary duties or litigation for several other boards including medicine, psychology, nursing, architecture and accountancy. In 2002, Ed served as the Attorney General Representative to the Pedigree Paper Task Force, created by the Legislature to review and reform Florida’s regulation of the prescription drug wholesale industry.

Ed’s current practice at Grossman, Furlow and Bayo includes the representation of professional licensees, regulated entities and interested parties before regulatory agencies, Florida Courts and the Division of Administrative Hearings. He concentrates his practice in the areas of administrative and regulatory law with emphasis on the laws and regulations affecting pharmacies, drug manufacturers and drug wholesalers. He is a frequent speaker before local, state and national professional organizations on licensure and regulatory issues. He has published several articles on these topics.

Disclosures: No disclosures provided by speaker

Prescribing Laws and Rules for Florida Licensed Healthcare Professionals
This course addresses the mandatory content for Physicians in Florida registered to prescribe controlled substances as allowed by Florida law, effective January 1, 2017.

Objectives:
Upon completing this course and reviewing the resources, participants should be able to:
1. Explain why the issue of prescribing issue is so important and illustrate best practices
2. Identify the extent of problems that may be encountered in prescribing controlled substances
3. Identify substance abuse screening tools that you can use in your practice
4. Illuminate where to find substance abuse treatment resources in your area
5. Identify the criteria for substance use disorders
6. Highlight the legal requirements for prescribing controlled substances in your practice
7. Describe different prescribing practices that will help keep you out of trouble while prescribing controlled substances

References:

Jason Winn, PA, Attorney at Law
Jason D. Winn, Esquire, is a 1996 graduate of the University of Maryland and received his Juris Doctorate from Nova Southeastern University - Shepard Broad Law in Ft. Lauderdale, FL. Mr. Winn was admitted to the Florida Bar in September 2001.

From 2001 until 2004, Mr. Winn worked for the Assistant Public Defender in the Fifth Judicial Circuit where he conducted over 15 jury trials, numerous non-jury trials, and many hearings including, violations of probation, restitution and early termination motions for defendants in juvenile, misdemeanor and felony court. Mr. Winn was also an adjunct professor at Lake Sumter Community College teaching business law during this time. In 2003, Mr. Winn was appointed by Governor Bush to serve a one-year term on the Judicial Nominating Commission for Judicial Compensation Judges. From 2004-2006, Mr. Winn worked for the law office of Clyde M. Taylor, Jr. focusing on both state and federal criminal defense and parole violation hearings. In 2006, he opened his own practice, where he is managing partner and continues to focus on criminal, administrative, governmental, civil, wills and trusts.

Mr. Winn currently serves as general counsel for the Florida Osteopathic Medical Association (FOMA), the Florida Podiatric Medical Association (FPMA) and the Florida Society of Hearing Healthcare Professionals (FSHHP). Mr. Winn lectures throughout Florida on the laws and rules that affect health care practitioners, including osteopathic, allopathic, podiatric, and various other licensed health care providers.

He is a member of the Florida Bar, Tallahassee Bar, Legal Services of North Florida, a lifetime member of the state Florida Association of Criminal Defense Lawyers (FACDL), and the local FACDL chapter. As a member of the Tallahassee Bar, Mr. Winn volunteers his legal services to the Wakulla County Senior Citizens Center and Legal Services of North Florida. Mr. Winn is a devoted husband and father to three boys. During his downtime, he enjoys hunting, fishing, golfing and the great outdoors.

Disclosures: No disclosures provided by speaker

Florida Laws and Rules Osteopathic Medicine

Objectives:
This course addresses the mandatory content for Physicians in Florida. Upon completing this course and reviewing the resources, participants should be able to:
1. Understand the CME requirements for continued Florida licensure
2. Be aware of any necessary office signage that must be posted
3. Understanding of applicable laws & rules for licensed osteopathic physicians
4. Knowledge of the disciplinary process
5. Learning of rights afforded to physicians in licensure disciplinary cases
6. Ability to locate applicable statutes and rules through online resources
7. How to protect their right to practice

References:
Ray Moseley, Ph.D.
Dr. Moseley received his Master's Degree in Philosophy and Medical Ethics at the University of Tennessee and completed his Doctorate in Bioethics at the Kennedy Institute of Ethics at Georgetown University in Washington, D.C.

Dr. Moseley teaches Bioethics in the Program in Bioethics, Law and Medical Professionalism in the UF College of Medicine where he is the Grace H. Osborn Professor in Bioethics. He is also an active clinical bioethics consultant at the UF/Shands Medical Center as well as at several other Florida Hospitals.

He is the founder and Board Member of the Florida Bioethics Network (FBN), and has played a key role in the development of the FBN as a significant statewide resource. Dr. Moseley is an expert on hospital ethics committees and research ethics, and he serves as vice chair of the UF IRB and is the author of CITI Hospital Ethics Committees training program. He regularly consults with national and international governments/institutions on the development of health care ethics committees and human subject's protections programs.

Currently, he is the Principal Investigator on a major NIH research grant developing and evaluating an innovative web-based informed consent process with “information on demand” features. He was recently the PI on a grant which created an iPad App which assists with developing and documenting advance directives, including video advance directives. His publications include articles on “Withdrawal of Life-Sustaining Medical Treatment”, “Advance Medical Directives”, “Genetic Testing”, and “Ethics Committees”.

Disclosures: No disclosures provided by speaker

Professional Medical Ethics

Objectives:
This course addresses the mandatory content for Physicians in Florida. Upon completing this course and reviewing the resources, participants should be able to:
1. Fulfill the requirements of the Florida Mandatory CME course on professional medical ethics
2. Address current ethical issues regarding medical ethics in Florida
3. Describe the meaning and importance of medical professionalism
4. Discuss how conflict of interest may compromise professionalism
5. Identify three significant boundary issues and proper limits in relation to each
6. Describe two important ethical issues with respect to operating a practice

References:

Arnold Mackles, MD, MBA, LHRM
Arnold Mackles, MD, MBA, LHRM is a nationally-recognized expert on patient safety. The mission of Dr. Mackles’ 35-year healthcare career has been to provide access to the highest quality care; first, during his 23 years of practice as a neonatal physician and then, for the past 12 years as a National Patient Safety Expert in all areas of medical practice. Dr. Mackles’ specific areas of expertise include: proactive prevention of medical errors, risk management, healthcare performance improvement strategies, creating/maintaining a culture of patient safety excellence, benchmarking industry leading communication protocols, safe and accurate medication delivery, root cause analysis, healthcare technology, medical documentation and emerging leadership strategies in patient safety.

Dr. Mackles is an industry thought leader who brings a refreshing, no-nonsense, and solutions-based approach to all areas of patient safety. He plays many roles within the healthcare space that include expert witness, continuing medical and nursing education program developer and facilitator, keynote speaker, and industry consultant. As an expert witness and consultant, Dr. Mackles’ fundamental philosophy is based on identifying root causation in the event of adverse patient safety circumstances and medical errors. He leads the field in his commitment to improving patient safety, reducing medical errors and enhancing the overall reputation of the healthcare industry. Program evaluations from physicians, nurses, CME directors, senior management and attorneys are outstanding and verifiable. Dr. Mackles is an industry change agent, a problem solver and a creator of a positive organizational culture.
His background as a neonatologist, patient safety expert witness, physician reviewer, author and presenter position Dr. Mackles as one of the nation’s top experts on the subjects of patient safety and risk management. He teaches and promotes new, cutting-edge subject matter, while researching emerging trends to ensure full compliance with healthcare protocols and policies to meet stringent client mandates and objectives.

Dr. Mackles is a former hospital-based physician with more than 23 years of experience, specializing in neonatology. He was a neonatologist/pediatrician for Pediatrix Medical Group, Florida Regional Neonatal Associates, and St. Mary’s Hospital (all located in South Florida), between 1983 and 2005. His experience and background as a physician clearly exposed him to the challenges the medical profession faced and the need to significantly improve patient care to reduce medical errors. In 2006, Dr. Mackles made the decision to become a full time advocate for patient safety.

He now travels the country as a speaker, trainer, expert witness, and consultant to improve patient safety and reduce or eliminate avoidable medical errors. Dr. Mackles’ credentials include a neonatology fellowship from Cornell University Medical Center, a pediatric residency from Lenox Hill Hospital in New York, a medical degree (MD) from the University of Bologna in Italy, an MBA Degree from Nova Southeastern University, and he received his bachelor’s degree (pre-med) from Syracuse University. Dr. Mackles attained his healthcare risk management licensure through studies at the University of South Florida. He is a Certified Professional Compliance Officer (CPCO) awarded by Healthcare Compliance Resources (currently certified by the AAPC, Salt Lake City, UT). He has served as an Instructor and Program Developer for the Risk Management and Patient Safety Program at the University of Florida, and participated as an instructor and faculty member of the University of South Florida Distance Education Risk Management Licensure Program. Dr. Mackles is currently a physician reviewer for the monthly newsletter “Healthcare Risk Management,” published by Relias Learning (formerly AHC Media). He is also a member of the Publication’s Editorial Board. In addition, Dr. Mackles is the author of 10 accredited online continuing medical and nursing education courses on patient safety topics offered by the Sullivan Group. Dr. Mackles is an active member of the Florida Society of Healthcare Risk Management and Patient Safety (FSHRMPS) and was elected to the Board of Directors from 2006 to 2008, and again between 2014 and 2015. He is a member of the American Society of Healthcare Risk Management (ASHRM) and has presented at their national meetings. In addition, Dr. Mackles is affiliated with the American Society of Professionals in Patient Safety.


Prevention of Medical Errors

Objectives:
This course addresses the mandatory content for Physicians in Florida. The purpose of this educational activity is to provide physicians with the most current information regarding the prevention of common performance and diagnostic errors. This monograph is specific to Florida statutes. After completing this activity, learners will be able to:
1. Identify the two most common qualities of care violations
2. Name four of the most prevalent diagnostic and performance errors
3. Cite two necessary elements of a root cause analysis
4. Create two risk management measures designed to prevent medical errors and increase patient safety

References:
2. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2723204/
Wednesday, March 21, 2018

8:00 a.m. - 12:00 p.m.  Exhibitor Set Up

12:30 p.m. - 1:15 p.m.  *Forging a Successful Practice: Utilizing PA's in a Busy Dermatology Office*
  John Minni, DO, FAOCD & Jeffrey Johnson, PA-C

1:15 p.m. - 2:15 p.m.  *Hemangiomas of Infancy*
  Andleeb Usmani, DO, FAOCD

2:15 p.m. - 3:15 p.m.  *Pediatric Dermatology: What's New*
  Anais Badia, DO, FAOCD

3:15 p.m. - 3:30 p.m.  Break with Exhibitors

3:30 p.m. - 4:30 p.m.  *No More Fake News: An Evidence Based Approach on Lasers in Skin of Color Patients*
  Eduardo Weiss, MD, FAAD

4:30 p.m. - 5:30 p.m.  *Asthma and Allergies in Dermatology*
  Michael Wein, MD

5:30 p.m. - 5:45 p.m.  *Dermatologic Emergencies*
  Evelyn Gordon, DO
  LECOMT/St. John’s Episcopal Hospital, South Shore
Utilizing PAs in a Busy Dermatology Office

Jeffrey Johnson, PA-C, DFAAPA

Disclosure Statement
I Have No Financial Information to Disclose

Agenda
- Brief Look at the PA profession
- Why Has it Worked for 50 Years
- Why Should You Consider Hiring a PA
- “Optimal Team Practice”
- Hiring and Retaining a Physician Assistant

What is a Physician Assistant?
- Physician assistants (PAs) are medical providers who are licensed to diagnose, treat and prescribe medication for patients. PAs work in offices, hospitals and clinics in collaboration with a licensed physician.
- At their core, PAs are Dependent Practitioners
- Our daily function within the Physician-led health-care team is directed solely by delegation from our supervising Physician.

Physician Assistants
AKA:
- Physician Extenders
- Mid-level Provider
- Advanced Practice Providers
- Allied Health Providers
- Limited License Provider
- Non-Physician Provider
- Physician Associate

PA Profession: Brief History
- Take advantage of military trained combat medics
- Training modeled the fast track for Physicians WWII
- Design was for PAs to “Think like a doctor”
- Work closely with physician
- Duke University 1965 – First Class of PAs
PA Education
- 27 Continuous Months equates to 3 academic years
- 75 Hours Pharmacology
- 175 Hours Behavioral Sciences
- 400 Hours Basic Science
- 580 Hours Clinical Medicine
- 100 hours Category I CME every 2 years
- Pass National Certifying Exam every 10 years
- Master’s Degree by 2020 or Lose Accreditation

PA Education
• Modeled on Physician Education
  One Year Basic Medical Sciences
  Anatomy, Pathophysiology, Pharmacology, Biochemistry
  Clinical Phase Training
  Family Medicine, Internal Med, OB/Gyn, ER, Peds, General Surgery, Psychiatry, ENT, Dermatology, Orthopedics, etc.
  • 2000 Hours of Supervised Clinical Practice

“Scope of Practice”
A PA’s scope of practice is determined by their training and experience, state law, facility policy and agreed upon with their supervising physician.

Where PAs can Practice Medicine:
All states, the District of Columbia and all US territories except Puerto Rico authorize PAs to practice medicine. This is also true for prescribing privileges. Ability to prescribe controlled substances varies by state and PAs must obtain own DEA number.

PA-"C"
- Physician Assistant is “Certified”
- Pass National Certifying Exam
- 100 Hours of CME every Two Years
- Pass Recertification Exam every 10 years
- No Dermatology Specialty Exam Exists

Current Status of PA Profession
- Approximately 130,000 Practicing PAs
- 539 living abroad
- 2800 in the field of Dermatology
- 225 Accredited PA Programs (270 by 2020)
- 8900 New PAs every Year
What Do Applicants Look Like?
- Greater than 3,000 hours patient contact experience
  - Paramedics
  - Medical Assistants
  - Athletic Trainers
- 27 years of age on average (24 y/o med. school)
- 66% are female

PAs in the Daily Clinic
- Allows the physician to focus on the items you want
  - PAs can play a supervisory role
  - Education of staff
- Patients offered appointment with physician first
  - Told they are seeing a PA when apt made, at confirmation
    and when the patient is roomed
- Mohs: More Patients Seen = More Cancers Treated

Physician Assistant: Added Benefits
- Patient Waiting Times are Decreased
- Readily Available for Follow-ups/Wound Checks
- Education Programs for Community
- Minimize Amount of Time On Call
- Most Importantly: Quality Patient Care
- Assist in Hiring, Training and Managing the Staff

Patients Acceptance of PAs
- Kaiser Permanente research shows patient satisfaction
  with PAs approaches 96%.
  - Understanding of the Patient’s Problems
  - Quality of Personal Care
  - Confidence in the Provider

Comparable Level of Care
- Berkeley Healthcare Forum Report a systematic
  review of 16 different studies revealed “no
  significant differences in patient satisfaction
  between NPPs versus physicians”
- Kaiser Permanente Center for Health Studies has
  also shown NPPs score equally with physicians in
  terms of patient satisfaction

Physician Assistants by Specialty
- Family Medicine: 25.9%
- Emergency Medicine: 10.5%
- Internal Medicine: 15.6%
- Dermatology: 3.6%
- Pediatrics: 4.3%
- Occupational Med.: 2.3%
- Surgery Subspecialty: 25.1%
- Other: 10.4%
How Did We Get to This Point?

The number of dermatologists emerging from residency programs each year is thought to be insufficient to meet growing patient demand. Aging Baby-Boomers and increased number of insured patients through the ACA worsens that shortage.

Physician Shortage

In 2015 the Association of American Medical Colleges (AAMC) forecasted the US will have 29,800 fewer primary care physicians than it needs which equates to 135 million ambulatory visits annually.

Demands Will Likely Increase

1. AAMC projects a shortage of 130,600 physicians by 2025.
2. AAMC also found in a separate study that 60% of patients would prefer an NPP rather than having to wait even a few days for a physician.

Why Even Consider Adding a PA?

1. PAs allow doctors to adjust their roles to meet the needs of the clinic
2. Flexibility in dealing with emergencies
3. Excessive workloads
4. Offer off peak (nights and weekends) appointments.
5. Help to train and manage the staff

Understanding the Risks

- Obviously NPPs are Not the Cure-all
- Strict Guidelines Outlining Scope of Authority
- Writing Prescriptions
- Signing Charts
- NPPs sued for Malpractice at Lower Rate

How Great is the Risk?

- Incorporating NPPs can Increase Liability Risks
- SPs often named as co-defendants in suits
- Since physicians often own practice, suits that exclude the physician are rare.
**How “We” Do It?**

- Employ: 48 Practitioners
  - MD: 13
  - DO: 11
  - PA: 16
  - NP: 8
- Patient Offered Appointment with Physician First
- Patient Informed Clearly the Credentials of Provider
  - At the Time the Appointment is Made
  - At Confirmation of the Appointment
  - Upon Rooming the Patient

**Why Does it Work?**

- **Variety of Procedures**
  - General Dermatology (Most Will See 35 – 38 Patients)
  - Surgical Dermatology
  - Cosmetics
  - Laser Treatment
  - Assist with Mohs Closures
- **All Connected via EMR**
- **Physician Only Minutes Away – same day evaluation**

**Salary**

AVERAGE: $105,000 Annual Salary

Cost to Employ a PA:
- 30 cents on the Dollar Collected

Example: $400,000 x .30 = $120,000

**Example of Salary Breakdown**

- **Base Salary:** $65,000
- **To The House:** $250,000
- **10%** $350,000
- **15%** $450,000
- **20%** $750,000
- **25%** Over

$600,000 = $120,000 Annual Salary

Total Cost of Employment = $180,000

**Benefits Package**

- "Competitive" Salary
- 401K
- CME Allowance ($1500 - $2000 annually)
- State License
- Professional Fees (NCCPA)
- Insurance (Medical, Dental, Life, Malpractice)
- Uniform (Scrubs)
- Professional Organizations
- Maternity Leave / Holidays
- Vacation/Personal Days

**Third-Party Coverage**

Nearly all private payers cover medical and surgical services provided by PAs. However, private health insurance companies do not necessarily follow Medicare’s coverage policy rules.
Medicare Reimbursement

- Medicare pays the PA’s employer for medical and surgical services provided by PAs in all settings at 85 percent of the physician’s fee schedule.

Hiring a Crucial Member of Your Team

- If you are considering hiring a PA, the success of the hire likely rests on a few simple questions:
  - What do you want the person to do?
  - What are you willing to let them do?
  - What amount of support will they receive?

The Hiring Process: Physician Assistants

- You need to be clear on how you’ll incorporate that person into the practice and really understand how you want them to perform.
- Defining the parameters of the job, especially during the interview, may eliminate future problems.
- The main reasons physician assistants leave is not because of the money, it’s the relationship with their supervising physician, the practice as a whole or the opportunity to grow.

Consider Training PA Students

- AAPA’s Data Services and Statistics Division reports that more than 1/3 of all PAs say they met their first employer through clinical rotations while attending PA school.

The Hiring Process: Physician Assistants

- Background checks are vital for promising applicants.
- Include a license check.
- Ask applicant if they are under investigation.
- Ask if they are under a Medicare Audit.
- Ask about pending liability litigation.
- Ask about convictions.

Hired a PA ... What Next!

- Notify your malpractice carrier
- Nominal premium increase
- Verify credentials
- Have written protocols – update regularly
- Supervise appropriately
- Be aware of state laws
- Be approachable – encourage questions
  - Meet or talk regularly
  - Foster an Environment of Learning
  - Take an active role in development
How to Avoid These Liability Pitfalls
- Hire Experienced, well-trained PAs
- Ensure One on One Training
- Establish Guidelines for Practice
- Be a Collaborator Not Just a Boss

Set the Parameters of the Job
- Formalize a Job Description
- Additional Duties Beyond Patient Care?
- Will the PA be on call and if so, how often?
- Will the PA be allowed to see new patients?
- What is the level of supervision that will take place?
- How Independent they be?
- Will the PA perform procedures; Assist with Mohs?
- Determine how the PA reacts to constructive tips

No Surprise Here!!
Solo Physicians Who Employ PAs Experience:
- Increased Patient Satisfaction
- Improved Patient Care
- Greater Access to Care
- Greater Efficiency
- Improved Quality of Life

“Optimal Team Practice”
- Originally known as “Full Practice Authority”
- The newest name for a political movement underway in the PA profession.
- Just in its infancy; but discussions are heating up
- There's A LOT to be worked out before legislation occurs
- May be asked for your professional input

Are Laws Regarding PAs Outdated?
State law requirements to have a supervisory agreement with a physician in order to practice were included in early PA practice acts. Fifty years ago when the PA profession was new, these requirements were intended to ensure strict oversight of an untested profession.
**OTP: What is it?**

- Member of a larger team of Healthcare Professionals
- Would recognize limits of their knowledge and skill
- Would understand when condition requires consultation or referral to other qualified healthcare providers.
- PAs would accept liability for the care they provide.
- Establish Autonomous State Board
- Reimbursed directly from Public/Private Insurance

**OTP: What it is Not!**

- **Independent Practice**: practice without the benefits of physicians or other qualified medical providers for collaboration, consultation, referral or team-based care.
- **OTP**: practice with access to physicians and other qualified medical professionals for collaboration, consultation and referral, as indicated by the patient’s condition and standard of care in accordance with the PA’s education, training and experience. Eliminates the requirement for assignment to a specific physician.

**Advantages for the PA Profession**

- The creation of an autonomous medical board of PAs which oversee the licensing and discipline of the professional.
- Allow PAs more flexibility in the workplace
- Eliminates regulations that PAs have to report to a specific supervising physician
- Following the lead of the NP profession’s success

**There’s A Lot to Work Out**

- Dependent practitioner is the hallmark of who we are
- This would require legislative action in all 50 states
- 54% of respondents to AAPA said they do not have the time or are opposed to lobbying activities
- What happens if some states pass and others do not resulting in a patchwork of differing PA practice acts

**What Drove OTPs Development?**

- Competition for jobs with Nurse Practitioners
- Changing requirements of employers
- What got us here ... Won’t get us there!

**Changing Landscape for Everyone**

- 76.1% of Physicians were Practice Owners in 1983
- 47.1% of Physicians were Practice Owners in 2016
- 38% Decrease from 1983 to 2016
Is OTP Better for Everyone?

- PAs must fulfill strict licensing requirements with includes 100 hours of CME every 2 years.
- The PA profession is so well established, highly trusted and essential to the US healthcare workforce.
- Study after study confirms PAs provide quality health care.
- Nevertheless, PAs are still required to enter into a supervisory, collaborating agreement with a specific physician.
- The PA profession remains fiercely committed to team practice with physicians.

Fiercely Committed to Collaborative Practice

Some have suggested the profession is seeking independent practice – that PAs wish to work alone, without collaborating physicians. That is not the case. OTP policy includes two important parts that distinguish it from independent practice:

Commitment Remains Unchanged

1. Optimal team practice reinforces PA’s commitment to team practice with physicians and explicitly states the PA/Physician team model continues to be relevant, applicable and patient-centered.

2. OTP calls for a decision about the degree of collaboration between PAs and physicians to be made at the practice level, in accordance with the practice type and the education and experience of the practicing PA. It puts more control in the hands of the physician as the leader of the healthcare team.

Times are Changing

When the PA profession began over 50 years ago, physicians were likely to be solo or joint practice owners. The increase in potential liability was offset by the financial and practice benefits of working with a PA. The day to day burdens of providing patient care and coverage of call were reduced, but the practice could also care for a greater number of patients at a lower cost than if another physician were added.
Is OTP Beneficial for You?

1. Today, however, physicians are more likely to be employees rather than practice owners and don’t realize the financial benefits of supervising a PA. They only take on the increased potential liability.

2. Also, in larger groups as providers come and go it becomes increasingly more difficult to maintain the strict supervisory mandates.

So, What’s the Bottom Line Again?

In addition to helping you deliver quality care to your patients …

Questions? Need More Information?

Feel free to contact me.

Email: fairways2@comcast.net
FSDPA: www.fsdpda.org
SDPA: www.dermpa.org
AAPA: www.aapa.org
NO MORE NEWS
AN EVIDENCE-BASED APPROACH ON LASERS IN SKIN OF COLOR PATIENTS
DR. EDUARDO WEISS, M.D., FAAD

KEY POINTS
• Hispanics/Latinos are the fastest growing segment of the skin of color population
• Use of lasers in persons with skin of color requires an understanding of laser physics and laser tissue interactions
• Epidermal melanin acts as a competing chromosphere which can decrease the effect of the laser treatment and cause nonselective thermal injury to the epidermis
• Proper selection of device, wavelength, and treatment parameters are essential for safety and efficacy

SKIN OF COLOR
• Defining skin of color in the Hispanic/Latino population can be particularly challenging
• Encompasses not only cultural aspects but historical ones as well
• Composed of various ancestries, which include white, Native Indian, African, and European descent
• Skin hues range from white to black
• Skin of color has distinct reaction patterns to cutaneous disease and this must be taken into consideration when approach in treatment options

HISPANIC SKIN OF COLOR
• The Hispanic population encompasses the range of phototypes and therefore one rule cannot apply to all Latinos/Hispanics
• Dr. Leal of Monterey, Mexico deciphered a way to predict the propensity of white phototype Latinos to experience PIH based on palmar and digital creases
• Rates the color diversity of linear creases from 0 to 3, with the high number indicating a darker skin tissue response despite skin phototype
LASERS IN SKIN OF COLOR

- When treating skin phototypes IV to VI the challenge is to deliver efficacious and reproducible results and minimize unwanted adverse reactions.
- Targeted chromospheres (water, hemoglobin, melanin).
- Selective photothermolysis:
  - If the target is heated for longer than its thermal relaxation time, there is ensuing diffusion of the thermal energy to the surrounding structures, causing unwanted nontargeted tissue damage.
  - By choosing a pulse duration equal to or less than the thermal relaxation time of the target chromophore, one ensures that the heat delivered is confined to the target chromophore.

LASERS IN SKIN OF COLOR

- Although there is no difference in the melanocyte density between Latino skin and lighter skin types, in darker-skinned individuals there is an increase in the number of melanin granules within the basal layer.
- This large amount of melanin within the epidermis of Latino skin and darker skin types competitively absorbs laser light targeted for other chromophores.
- With the absorption spectrum of melanin ranging from 250 to 1200 nm, great care and diligence must be taken when using laser light on Latino skin.
- Laser parameters that suit skin of color in regards to safety include longer wavelengths, a long pulse duration, and efficient cooling.

VASCULAR LESIONS

- The vascular lasers when used at appropriate settings can be used for the light and dark skin tones in the Latino population.
- The main vascular chromophore is oxyhemoglobin.
- Phototypes IV to VI have epidermal melanin that acts as a competitive chromophore against hemoglobin and oxyhemoglobin.
- Lasers that target vasculature:
  - Pulsed dye laser (585, 590, 595, 600 nm)
  - Intense pulsed light (IPL) (400 to 1200 nm)
  - Neodymium:yttrium aluminum garnet (Nd:YAG) (532, 1064 nm)
  - Long-pulsed alexandrite (755 nm); long-pulsed diode laser (800 nm).

LASER HAIR REMOVAL

- With the advent of lasers with longer wavelengths, longer pulse durations, and efficient cooling devices, all skin types can be treated with lasers for hair removal without side effects.
- The longer wavelengths penetrate deeper into the dermis where the hair follicle resides.
- Two lasers that are appropriate for use in Latino skin:
  - Diode laser 810nm (up to phototypes V)
  - Nd:YAG 1064 nm (up to phototypes VI)
  - Intense pulsed light (IPL).

KELOIDS AND ACNE SCARS

- Keloids are a prevalent problem, particularly in individuals of African, Hispanic, and Asian descent.
- While keloids do occur in all skin types there is a higher incidence in the Black and Hispanic populations.
- Several reports state that non-ablative lasers (585 nm, 1320 nm, 1064 nm, 1450 nm, 1550 nm) used to treat acne scars also provide an improvement in active acne, as a result of their collagen remodelling effect.
SKIN REJUVENATION

- Skin rejuvenation principally includes collagen stimulation and remodeling to improve the texture of scarred or wrinkled skin, decrease pore and acne scars and tighten skin laxity.
- The objective of collagen stimulation and remodeling is to form new dermal collagen and to tighten up pre-existing collagen through dermal heating.
- Traditionally, ablative lasers, such as the carbon dioxide (CO2) and Erbium:YAG, have been the gold standard.
- Delayed onset hypopigmentation, hyperpigmentation and transient erythema lasting months are common side effects.
- The increase in adverse effects when resurfacing patients with skin of color makes these lasers mostly contraindicated in these patients.

SKIN REJUVENATION

- Laser skin rejuvenation procedures are challenging in patients with higher Fitzpatrick skin types due to potential dyschromia. In a retrospective review of fractional laser treatments.
- More appropriate treatment options to improve laxity for darker skin types would be non-ablative infrared and radio frequency lasers.
- The newer category of micro-ablative resurfacing lasers (fractional CO2, fractional Erbium, and the 2790 nm Yttrium Scandium Gallium Garnet [YSAG]), offers a safer modality with which to treat Fitzpatrick skin type IV but until more studies are done, remain contraindicated in darker skin types (Fitzpatrick skin types V–VI).
- Compared to the older generation resurfacing lasers the micro-ablative lasers minimize the amount and duration of erythema and edema, which can last just three to four days.

SPECIFIC LASERS AND THEIR CLINICAL APPLICATIONS

PULSED DYE LASER (585, 595 NM)

- The 585-nm wavelength pulsed dye laser penetrates to a depth of 1.2 mm.
- The longer 595-nm wavelength allows for a slightly deeper penetration; however, the absorption coefficient of oxyhemoglobin is 3 times higher at 585 nm than 590 nm.
- 585-nm pulsed dye laser is superior in treating the vascular lesions such as port wine stains.
- Both suitable for fair-complexioned phototype IV skin.
- For darker phototypes V and VI, longer wavelengths should be used. In addition, longer pulse durations are safer in darker-skinned Latinos.
- Treatment of choice for vascular lesions such as port wine stains, facial telangiectasias, and some superficial hemangiomas.
DIODE LASER (800-810 NM)

- 800-nm diode laser has a fluence range of 10 to 100 J/cm² and a pulse duration range of 5 to 400 milliseconds
- Targets vascular structures such as leg veins or can be used for hair removal
- Because hemoglobin has a small absorption peak in the 700-nm to 900-nm range the long-pulsed diode laser can be used to treat larger-caliber veins
- Diode laser can be used to target the follicular melanin chromophore for use in hair removal
- High fluences can be achieved when using long pulse durations
- In darker phenotypes 75% to 90% hair reduction was reported after 8 to 10 treatments at 10 J/cm² and 30 milliseconds pulse duration

INTENSE PULSED LIGHT (IPL)

- A large number of IPL devices are available
- As IPL devices emit a spectrum of wavelengths, the three key chromophores can be activated with one single light exposure (reduced selectivity)
- The patient's skin type and the skin condition present determine the choice of suitable cut-off filters and therefore the spectrum of wavelengths to be emitted
- Pulse duration can be set in relatively wide ranges (depending on the particular device) in the millisecond range.
- Similar to laser devices, pulse duration should be lower than the thermal relaxation time of the target structure to prevent unselective damage to the surrounding tissue
- Conditions treated with IPL: acne, pigmented and vascular lesions, unwanted hair growth, photodamaged skin, scars
**ND:YAG (1064 NM)**

- Long-pulsed 1064-nm Nd:YAG laser has a multitude of uses in the Latino population with skin of color
- Produces less interference with epidermal melanin because the longer wavelength penetrates deeper into the skin; however, the absorption by hemoglobin is less than that of other lasers
- The long-pulsed 1064-nm Nd:YAG can effectively photocoagulate superficial and deep vessels up to a diameter of 3 mm
- This laser can be used to target venulactasias, such as spider veins and blue reticular veins, in the deep reticular dermis

**TABLE 1. Advantages and Disadvantages of IPLs**

<table>
<thead>
<tr>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lower purchase price</td>
<td>Inconsistency of emitted spectrum and fluence</td>
</tr>
<tr>
<td>Larger spot size</td>
<td>Weight of machine</td>
</tr>
<tr>
<td>High skin coverage rate</td>
<td>Larger spot size</td>
</tr>
<tr>
<td>High versatility</td>
<td>Light can not be focused</td>
</tr>
<tr>
<td>Robust technology</td>
<td>Gel application required*</td>
</tr>
<tr>
<td></td>
<td>Direct contact of handpiece to the skin required*</td>
</tr>
</tbody>
</table>

*Dual use observation of immediate local response.

**TABLE 2. Factors Influencing the Susceptibility of the Skin to IPL Treatment**

<table>
<thead>
<tr>
<th>Skin type</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin thickness</td>
</tr>
<tr>
<td>Individual skin resistance</td>
</tr>
<tr>
<td>Skin temperature</td>
</tr>
<tr>
<td>Blood perfusion</td>
</tr>
<tr>
<td>Frequency of sebaceous glands</td>
</tr>
<tr>
<td>Presence of hair follicles</td>
</tr>
<tr>
<td>Presence of a tattoo</td>
</tr>
<tr>
<td>Presence of melanocytic nevi</td>
</tr>
<tr>
<td>Sunburn</td>
</tr>
</tbody>
</table>

**ND:YAG (1064 NM)**

- Considered to be the safest for hair removal in the darker pigmented population
- It is suitable for use in all skin types I to VI
- For phototypes V and VI the pulse duration is safest at 30 milliseconds or greater
- Recent studies also show efficacy and safety in treating scars in darker skin types in addition to skin rejuvenation when used in combination with other treatment modalities


**Nd:YAG Laser Hair Removal in Fitzpatrick Skin Types IV to VI**

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*Department of Dermatology, Yale University School of Medicine, New Haven, CT

**Abstract**

Safe and effective laser treatments are crucial, especially in darker-skinned individuals. Herein, we report our experience treating Fitzpatrick skin types IV to VI with varying results. In 1,064-nm neodymium-doped yttrium aluminum garnet laser treatment, the right treatment settings, darker pigmented individuals can undergo laser hair removal effectively.

**Figure 1. a) Before treatment. b) After laser hair removal treatments of the beard with a neodymium-doped yttrium aluminum garnet device.**
The Use of the 300 Microsecond 1064nm Nd:YAG Laser in the Treatment of Keloids

Abstract

METHODS & MATERIALS: A retrospective analysis of treatment efficacy was conducted on a patient with keloids. The patient underwent treatment with the 300 microsecond Nd:YAG laser. The treatment was performed by a dermatologist in a clinical setting. The laser was set at a fluence of 15-30 J/cm² and a pulse duration of 300 microseconds. The treatment was repeated every 3-4 weeks until the lesions were no longer visible. The patient tolerated the treatment well and experienced minimal discomfort. The lesions showed significant improvement with complete resolution in all cases.

TABLE 1

<table>
<thead>
<tr>
<th>Treatment Group</th>
<th>Treatment Type</th>
<th>Treatment Duration</th>
<th>Lesion Size Reduction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Laser alone</td>
<td>20 treatments</td>
<td>4 weeks</td>
<td>75%</td>
</tr>
<tr>
<td>Combination</td>
<td>10 treatments</td>
<td>2 weeks</td>
<td>85%</td>
</tr>
</tbody>
</table>

FIGURE 1: Treatment with the Nd:YAG Laser – Skin Type IV. Fluence: 15-18 J/cm², 0.3 mm, 2000 pulses. a) Hispanic patient s/p trauma and multiple excisions; b) After treatment protocol; c) 5 year follow up.

FIGURE 2: Treatment with the Nd:YAG and ILX – Skin Type IV. Fluence 13-18 J/cm², 0.3 mm, 2000 pulses. a) Hispanic Patient 24+ years of a keloid s/p vaccination; b) After Treatment Protocol; c) 5 year follow up.

FIGURE 3: Treatment with the Nd:YAG plus ILX – Skin Type IV Fluence: 13-18 J/cm², 0.3 mm, 2000 pulses. a) Hispanic patient s/p trauma; b) After treatment protocol; c) 5 year follow up.

FIGURE 4: Journal of Pigmentary Disorders

Clinical Evaluation of Intense Pulsed Light vs. Combined Treatment of Intense Pulsed Light and Nd:YAG Laser for Facial Rejuvenation in Latin American Women

Abstract: A prospective, randomized, controlled trial comparing the efficacy of intense pulsed light (IPL) and a combination of IPL and Nd:YAG laser for facial rejuvenation in Latin American women. The primary outcome was the percent improvement in skin texture and the secondary outcomes included patient satisfaction and adverse events.

Patients were randomized to one of two groups: Group A received IPL alone and Group B received IPL followed by Nd:YAG laser treatment. Both groups received treatments at 4-week intervals for a total of 6 treatments. The percentage improvement in skin texture was calculated using a blinded, independent evaluator.

RESULTS: A total of 80 patients were enrolled, with 40 in each group. The mean age of the patients was 40.5 years. The percentage improvement in skin texture was significantly higher in Group B compared to Group A (p<0.05). Patient satisfaction was also higher in Group B, with 85% of patients reporting satisfaction compared to 70% in Group A. Adverse events were reported in 17.5% of the patients in Group B and 27.5% in Group A (p=0.05).

COMMENTS: The combination of IPL and Nd:YAG laser treatment showed superior efficacy compared to IPL alone for facial rejuvenation in Latin American women. Further studies are needed to elucidate the optimal parameters for this combined treatment.
ER-YAG (29040 NM, 1550 NM)

- Technologies include ablative, fractional ablative (Er:YAG 2940 nm fractional) and fractional non-ablative (1,550 nm Er:glass laser)
- A recent retrospective study of Chinese patients treated with the 1,550 nm erbium-doped fractional laser (Fraxel 1550, Solta Medical) found that using fewer passes per treatment, but increasing the total number of treatments was associated with a lower risk of postinflammatory hyperpigmentation without compromising efficacy.
CO2 LASERS (10,600 NM)

- CO2 resurfacing and CO2 microfractionated laser systems are reliable tools to improve different facial pathologic skin conditions but are associated with a high rate of complications specially in Fitzpatrick III, IV, and V skin phototypes, predominant in the Latin population.

- CO2 lasers are associated with hyperpigmentation in 31% of all skin types increasing to 50% in type III Fitzpatrick skin phototypes.

- Fractionated use offers a quicker recovery time for patients and has a significant reduction in side effects compared to conventional resurfacing procedures.
Laser Resurfacing for Latin Skins: The Experience with 665 Cases

Lim Reiyan1,2,3, César Rubins1,2,3, Carlos Varas1,2,3, Mero Zambudio1

*Presenting author.

**Corresponding author.

Abstract

BACKGROUND: CO2 resurfacing and CO2 microfracionated laser systems are reliable tools to improve facial skin appearance and can offer a wide range of treatment options for a variety of skin conditions. The aim of this study was to evaluate the use of these two techniques and to provide an overview of the results and outcomes.

METHODS: We conducted a retrospective study of patients who underwent laser skin resurfacing or microfracionated CO2 laser treatments from 2010 to 2018 at our center. The patients' medical records were reviewed to collect demographic data and surgical outcomes.

RESULTS: A total of 665 patients were included in the study. The mean age of the patients was 43 years (range, 20 to 65 years). Most patients were women (85%). The majority of patients had Fitzpatrick skin type IV (60%). The procedure was well tolerated, with a low incidence of complications (5%). The most common adverse effects were transient erythema, edema, and pain. All patients were satisfied with their results and returned for follow-up visits.

CONCLUSIONS: CO2 resurfacing and microfracionated CO2 laser treatments are effective and safe for improving facial skin appearance in Latin skin types. Further research is needed to compare these techniques with other laser resurfacing modalities.

Table 1: Demographics of patients treated with CO2 laser

<table>
<thead>
<tr>
<th>Age</th>
<th>CO2 resurfacing</th>
<th>Microfraccionated CO2</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;30</td>
<td>40</td>
<td>30</td>
</tr>
<tr>
<td>30-40</td>
<td>240</td>
<td>180</td>
</tr>
<tr>
<td>41-60</td>
<td>300</td>
<td>260</td>
</tr>
<tr>
<td>&gt;60</td>
<td>50</td>
<td>40</td>
</tr>
</tbody>
</table>

Table 2: Phototype distribution.

<table>
<thead>
<tr>
<th>Phototype</th>
<th>Type I</th>
<th>Type II</th>
<th>Type III</th>
<th>Type IV</th>
<th>Type V</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO2</td>
<td>44 (92%)</td>
<td>30 (62%)</td>
<td>20 (39%)</td>
<td>31 (58%)</td>
<td>4 (8%)</td>
</tr>
<tr>
<td>Microfraccionated CO2</td>
<td>9 (19%)</td>
<td>10 (20%)</td>
<td>8 (16%)</td>
<td>9 (18%)</td>
<td>1 (2%)</td>
</tr>
</tbody>
</table>

Table 3: Complications related to laser system applied.

<table>
<thead>
<tr>
<th>Complication</th>
<th>CO2 resurfacing (%)</th>
<th>Microfraccionated CO2 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Erythema</td>
<td>0.5</td>
<td>0.3</td>
</tr>
<tr>
<td>Hypertension</td>
<td>5.3</td>
<td>12.2</td>
</tr>
<tr>
<td>Hypertension</td>
<td>0.7</td>
<td>0.5</td>
</tr>
<tr>
<td>Edema</td>
<td>12.3</td>
<td>15.8</td>
</tr>
<tr>
<td>Drying</td>
<td>20</td>
<td>15</td>
</tr>
<tr>
<td>Hypertrophic scar</td>
<td>0.3</td>
<td>0.2</td>
</tr>
<tr>
<td>Ectropion</td>
<td>0.5</td>
<td>0.3</td>
</tr>
<tr>
<td>Milia</td>
<td>0.3</td>
<td>0.5</td>
</tr>
<tr>
<td>Ocular complaints</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Allergic reaction</td>
<td>0.3</td>
<td>0.1</td>
</tr>
</tbody>
</table>

Table 4: Hypertension rates according to Fitzpatrick’s skin type.

<table>
<thead>
<tr>
<th>Phototype</th>
<th>Type II</th>
<th>Type III</th>
<th>Type IV</th>
<th>Type V</th>
<th>Average</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resurfacing CO2 (%)</td>
<td>19.1</td>
<td>26.3</td>
<td>39.1</td>
<td>42.4</td>
<td>30.4</td>
</tr>
<tr>
<td>Microfraccionated CO2 (%)</td>
<td>0</td>
<td>13</td>
<td>15</td>
<td>16.2</td>
<td>11</td>
</tr>
<tr>
<td>Mixed CO2 (%)</td>
<td>0</td>
<td>9</td>
<td>17</td>
<td>23</td>
<td>16.3</td>
</tr>
</tbody>
</table>
SUMMARY

• Use of lasers in persons with skin of color requires an understanding of laser physics and laser tissue interactions

• The Hispanic population encompasses the range of phototypes and therefore one rule cannot apply to all Latinos/Hispanics

• Proper selection of device, wavelength, and treatment parameters are essential for safety and efficacy
Dermatologic Emergencies

Objectives

• Identify life threatening and emergent dermatologic conditions
• Discuss clinical clues to help distinguish different diseases
• Review up to date management

Outline

• Introduction
• Emergent conditions:
  • SJS/TEN
  • EM
  • MIRM
• Conclusion

Introduction

• In the United States, about 34% of all Emergency Department visits are due to dermatologic complaints
• It is imperative to recognize life-threatening and almost life-threatening conditions that require immediate attention to improve overall prognosis

Steven Johnsons Syndrome/Toxic Epidermal Necrolysis

• Life threatening mucocutaneous eruption
• Both SJS and TEN are regarded as variants on a continuous spectrum of adverse drug reactions
• Mortality rate for SJS is 1% to 5%
• Mortality rate for TEN is 25% to 35%
Surface area of Epidermal Detachment

SJS/TEN
- Pathophysiology: immune dysregulation resulting in apoptotic keratinocytes
- Etiology: >100 causative agents
  - Most common include:
    - Alcohol
    - NSAIDs
    - Sulfonamides
    - Anticonvulsants
  - May sometimes be precipitated by viral illness

Clinical Features
- Timing: Occurs 1 to 3 weeks after starting causative drug (aromatic anticonvulsants may take up to 2 months)
- Initially fever and URI symptoms, painful skin → dusky, atypical targetoid skin lesions → painful mucosal erosions → progresses to epidermal detachment → bullae and sloughing

TEN

Histology
- Full thickness epidermal necrosis with subepidermal bulla formation

Diagnosis
- Clinicopathological correlation
  - Nikolsky Sign: epidermal detachment with pressure on area adjacent to blister
  - Asboe-Hansen sign: pressure on bulla causes spreading to uninvolved skin
- Skin biopsy: full thickness epidermal necrosis
**Prognosis**
- Depends on EARLY Diagnosis

**Management**
- STOP offending agent
- Transfer to IV/Radius Unit
- Supportive therapy, wound care, nutrition, fluids
- Consult Ophthalmology, Otolaryngology
- Adjunct therapy options remain controversial
- No established guidelines
- Systemic steroids: recent literature showing INCREASE in mortality with use of systemic steroids as sole therapy
- Promising data with IVIG and cyclosporine
- Etanercept

**Clinical Features**
- **EM Major**
  - Targeted lesions, typical targets, "butterfly" pattern
  - Extremities, face
  - Severe mucosal involvement, systemic symptoms
- **EM Minor**
  - Targeted lesions, extremities—elbows, knees, wrists, hands, face
  - Minimal mucosal involvement, no systemic symptoms

**Erythema Multiforme**
- Acute, self-limited eruption
- Seen in 1% and 0.1%, primarily in young adults and children
- EM Major (mucosal) and EM Minor (no mucosal involvement)
- Etiology
  - Most common cause: HSV
  - Other infections: Mycoplasma, rarely drugs
  - Does NOT progress to TEN vs SJS
  - NOW CONSIDERED SEPARATE ENTITY FROM SJS/TEN

**Erythema Multiforme**
- [Image of clinical features]
Histology

- Vacuolar interface dermatitis with a perivascular lymphocytic infiltrate and necrotic keratinocytes

Management

- Check Mycoplasma serology
- Treat precipitating factor if identified
- If recurrent HSV associated EM, consider prophylaxis with Acyclovir or Valacyclovir x 6 months
- EM Major: prednisone, dapsone, mycophenolate mofetil
- EM Minor: symptomatic rx, oral antihistamines
- Apremilast

Mycoplasma Induced Rash and Mucositis (MIRM)

- Previously thought to be a variant of EM/SJS
- Etiology: *Mycoplasma pneumoniae*
- 25% patients experience extrapulmonary complications

Clinical Features

- Young patients
- M&F
- Prodrome fever, cough preceding rash x 1 week
- Prominent mucositis: oral mucosa > urogenital, conjunctival
- Cutaneous involvement less common, usually acral distribution
- Polymorphic lesions: Vesiculobullous, targetoid, macules, papules
- Mortality 3%

- Recurrent EM defined as at least two episodes, 6 episodes in one year over a course of 6>10 years
- Apremilast has been tried in 3 patients (≥60mg daily)
- Has also been used to treat Behcet's disease

- Authors performed literature search, reviewed 202 cases, to characterize morphology and disease course with M. pneumoniae and associated mucocutaneous disease.
How to Differentiate from SJS/TEN

- Negative Nikolsky sign
- Cutaneous lesions usually acrally distributed
- <10% BSA affected
- Absence of drug exposure
- Evidence of atypical pneumonia i.e. symptoms, CXR, M. Pneumonias serology
- Milder clinical course

Management

- Serology for M. Pneumonias, CXR
- Skin biopsy; lesser degree of epidermal necrosis vs SJS/TEN
- Supportive care, magic mouthwash
- Ophthalmology, Gyn/Urology consults
- Treatment is controversial, no evidence based guidelines
- Anecdotally, treatment with steroids, antibiotics or both IV Ig in some cases
- Antibiotic therapy helps prevent pulmonary/neurologic complications, unclear if helps with mucocutaneous eruption

Conclusion

- Early identification of dermatologic emergencies is critical
- Steroids are NOT always the answer
- Time is key in order to improve prognosis
- If clinical suspicion is high, treat empirically
  - do not wait for diagnostic testing

Thank You!
References

Thursday, March 22, 2018

6:00 a.m. - 7:00 a.m.  Continental Breakfast with Exhibitors

7:00 a.m. - 8:00 a.m.  Great Cases from Osteopathic Institutions

8:00 a.m. - 9:00 a.m.  Lasers and Lifestyles
Shino Bay Aguilera, DO, FAOCD

9:00 a.m. - 10:00 a.m.  Facial Plastic Surgery & Charitable Reconstructive Procedures in Underdeveloped Countries
Jean-Paul Azzi, MD

10:00 a.m. - 11:00 a.m.  Cosmetic Dermatology: A General Guideline
Janet Allenby, DO, FAOCD

11:00 a.m. - 11:30 a.m.  Break with Exhibitors

11:30 a.m. - 12:30 p.m.  A Closer Look at Taltz
Eugene Conte, DO, FAOCD
Lilly USA, LLC Product Theater (No CME Awarded)
Located in Coral D & E

12:30 p.m. - 1:00 p.m.  Break with Exhibitors

1:00 p.m. - 2:00 p.m.  Protecting Your Practice: Not Usually Part of Your Medical School Curriculum
Lawrence Klitzman, JD

2:00 p.m. - 3:00 p.m.  Contact Dermatitis of Aeroallergens
Peter Saitta, DO, FAOCD

3:00 p.m. - 3:30 p.m.  Break with Exhibitors

3:30 p.m. - 4:30 p.m.  Medical Management of TEN and Update on Therapy
Carlos Ricotti, MD

4:30 p.m. - 5:30 p.m.  Medical Treatments in Hidradenitis Suppurativa (H/S)
Francisco Kerdel, MD

5:30 p.m. - 6:30 p.m.  SkinCure Oncology Product Theater
(No CME Awarded)
Located in Coral D & E
Market Opportunity

- Skin rejuvenation is one of the leading growth drivers in aesthetic medicine.
  - Over nine million procedures are performed each year, generating more than four billion dollars in fees for physicians.
  - The category is forecasted to grow to 11 million annual procedures within five years.
  - Baby Boomers account for 51% of the US population with an ample disposable income available.

HISTORY OF LASERS FOR SKIN REJUVENATION

- Ablative lasers: Early 1900s, CO₂, Er:YAG, 1064nm, 1320nm, 1550nm Fractional
- Non-ablative lasers: IPL, PDL, 1064nm
- Fractional LASER
- Intense Pulsed Light (IPL)
- CO₂ Laser
- Er:YAG 1064nm
- 1320nm IPL
- Skin Laxity
- RF, IPL
- 21st century

Depth of Penetration by Wavelength (not to scale)

- 1064 nm Nd:YAG
- 755 nm Alexandrite
- 1540 nm Er:Glass
- 1550 nm Fiber Laser
- 694 nm Ruby
- 585-595 nm Pulse Dye
- 1440 nm Nd:YAG
- 2940 nm Er:YAG
- 1320 nm Nd:YAG

Intensity Pulsed Light

CO₂ Laser

Er:YAG 1064nm

IPL

CO₂ Laser

Er:YAG 1064nm

Market Opportunity

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  - Baby Boomers account for 51% of the US population with an ample disposable income available.
Trends are favorable ...

- Large number of aging baby boomers in the U.S., Europe and Japan
- Skin rejuvenation is now the most popular energy-based aesthetic procedure
- Skin rejuvenation is the second most common procedure after LHR
- Skin rejuvenation will experience market-beating growth in procedure volume (4% CAGR) and will be roughly equivalent to LHR by 2017
- Skin rejuvenation has very good procedure fees and account for roughly half of all worldwide procedure fees for energy based aesthetic treatments

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Who Else Purchases Aesthetic Procedures?

- Generation X has a population of 50 million, ages 31-45.
- Though their numbers are fewer than Boomers, they lead in cosmetic procedures.
- They account for almost 4 million or 42.9% of procedures performed in 2011.
- Gen X is more accepting of cosmetic procedures than past generations.
- Many consider these procedures simply part of their budget, for personal ‘maintenance’= EASY RETENTION

---

Fitzpatrick Skin Types

I II III IV V VI
Dermatoheliosis

Solar Elastosis

SmartSkin CO₂ LaserSkinRenewal®

Documented Efficacy and Safety in Multiple Publications
Combination of CO₂ and micro-ablative technologies

How does it work?

- **Spot pitch** – influences how much tissue is ablated
- **Power** – determines the ablation depth
- **Dwell time** – defines lateral thermal effect

Clinical Results
• 1440nm wavelength is absorbed ~2-2.5x better than 1540nm in water
• 1440nm confines effect to the zone of photodamage ~300 mm thick
• Less energy is required for effective treatment
• Less pain
• Greater safety in peri-orbital area

Non-ablative skin rejuvenation

Service Offerings
• Wrinkle reduction
• Coagulation resulting in tissue tightening
• Scar and Striae treatment
• Treatment of pigmentation and redness

Powered By MultiPlex™

CAP Technology

• CAP technology produces varying levels of heat distribution in the skin:
  – Apex pulses create high intensity lesions remodeling collagens
  – Low level heating surrounds the apex pulses creating collagen stimulation
Dermal Tissue Histology
E. Tanghetti, MD / R. Weiss, MD

**Columns**
Width = 100 µm

**Acute Inflammation**
Depth = ~1.0 µm

Fibrosis creates inflammation

Inflammation in simple terms does the following:
- Inflammatory mediators induce fibroblast and cytokines activity
- Fibroblasts start tissue repair and enhance new collagen production

So the fibrosis causes a wound healing response that leads to delayed tissue contraction because of the inflammation created.

---

**Multiplex Tx**

**Skin Laxity**

Before | Immediately Post | 2 Days Post

**Micro-thermal Rejuvenation**

![Baseline](3.0 J/cm²)

1 Month Post 3 Tx

3.0 J/cm²

---

**Acne Scarring**

Baseline | 1 Month Post 5 Tx

Courtesy A. Myers, RN

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**Histology Outcomes**

- Fibrosis creates inflammation

Inflammation in simple terms does the following:
- Inflammatory mediators induce fibroblast and cytokines activity
- Fibroblasts start tissue repair and enhance new collagen production

So the fibrosis causes a wound healing response that leads to delayed tissue contraction because of the inflammation created.
1540 Fractional Clinical Results

- Well tolerated treatment
- Mild tolerated pain, no need for anesthetic
- Effective collagen shrinkage
- FDA-cleared for soft tissue coagulation
- Long term collagen growth

- Rapid healing
- 3 to 5 Tx., intervals of 14 days apart or more
- No infections, bleeding, oozing
- No down time – can shave or wash face immediately
- Mild erythema
- Mild edema 1-2 days

1540 Fractional Laser Microlenses

**XF™ Microlens**
- Extra Fast
- High speed with increased surface coverage
- Allows for a full face, one pass treatment in under 15 minutes

**XD™ Microlens**
- Extra Deep
- Uses unique lens design to allow for maximum depth of penetration up to 2mm depth

1540 post 3 TX; dr. sinclair, FL

1540 Fractional Laser Handpiece

Photos courtesy of Dwight Scarborough, MD
1540 post 3 TX

Fraxel® DUAL 1550/1927

Treatment Overview

- Predictable, reproducible results
- Safe on
  - All skin types
  - Anywhere on the body
- Advanced continuous motion handpiece for consistent, even treatment

The Fraxel DUAL 1550/1927 laser is safe and easy to delegate to a trained professional*

*Please check your local regulations

Skin Reaction Progression

1550 nm - Post 1 Treatment

Before 1550 Treatment Immediately Post 2 Days Post 1 Day Post

3 Days Post 1 Month Post 5 Days Post 4 Days Post

All before and after photos are un-retouched. Results may vary. Courtesy of Solta Medical Aesthetic Clinic

Skin Reaction Progression

1927 nm - Post 1 Treatment

Clear + Brilliant® Overview

Integrated Skincare +

Patient Profiles

Shelly, 26

- Uses skincare products and sunscreen, gets facials
- Has tried microdermabrasion and chemical peels
- Doesn’t have textural irregularities… yet!

“What else can I do for my skin?”

Shelly needs to be proactive in her skin care:

- Continue current skin care regimen
- Add Clear + Brilliant® treatments to microdermabrasion

Skin Care Rx

Clear + Brilliant:
New treatment option for younger patients

SkinCeuticals available in U.S. and Canada Markets only
Patient Profiles

Jeanine, 31
• Wants radiant skin with minimal downtime
• Doesn’t want to use injectables
• Getting all she can from chemical peels

“I want more but don’t have a lot of free time. What can I get for a radiant glow?”

Jeanine needs elevated continuum of care:
• Continue current skincare regimen
• Add Clear + Brilliant® treatments to chemical peels
Clear + Brilliant:
Incremental treatment for radiant skin

Sarah, 37
• Takes care of her skin, but has years of photo damage
• Is new to laser procedures
• Wants long term results with little downtime

“I have some sun damage. Is there anything I can do to even out my tone?”

Sarah needs a comprehensive regimen:
• Continue at home skin care
• Clear + Brilliant® Perméa® treatments

Diane, 43
• Uses professional skincare products
• Started Botox®/fillers a few years ago
• May benefit from a more aggressive treatment

“I’m not ready for a big laser treatment just yet – is there something else you can offer me?”

Diane needs a treatment to bridge the gap:
• Continue current skin care regimen
• Supplement Botox®, fillers with Clear + Brilliant®
Clear + brilliant:
A stepping stone to transformative treatments

Caroline, 50
• Uses professional skin care products, Botox®/fillers and peels
• Received a series of Fraxel® laser treatments in the past

“I need a refresher for my skin. Is there an affordable option?”

Caroline needs maintenance care:
• Continue current skin care regimen
• Offer Clear + Brilliant® to maintain results from Fraxel
Clear + Brilliant:
Affordable treatment to protect “skin” investment

Managing Patient Expectations

Treatment Experience
• Multiple treatments are ideal
  – Spaced 2-4 weeks apart
• Maintenance treatments, as desired
• Appointment will take approximately 35-60 minutes. Depends on treatment area(s)
  – Topical anesthetic agent: 15-30 min
  – Procedure time: 20-30 min

Most patients describe an increased sensation of heat or “prickling” during treatment

Managing Patient Expectations

Results
• Uniform skin tone
• Improved texture
• More radiant skin
• Minimized pore size appearance
• Skin that looks and feels clear and brilliant
• Immediate and progressive – improves over multiple treatments

In a clinical study, “100%” of patients reported improvements in their skin

*Data on file. N=28; 100% reported improvements after 5 treatments
Clear + Brilliant® Results
Healing Progression Photos

Before and after photos have not been retouched. Individual results may vary.

Immediately Post Treatment
*Felt redness very next day and felt dryness for a while*
*A little puffy around my eyes*
*A little red and rough to the touch*

1 Day Post Treatment
*Skin peeling and no dryness*
*Skin has a rough texture*
*Skin feels a bit scaly*

2 Days Post Treatment
*Skin is much smoother*
*Rough skin starting to flake off*
*Complexion is starting to look better*

3 Days Post Treatment
*Brightened skin tone*
*Make-up goes on so much smoother*
*Skin just looks better*

4 Days Post Treatment
*Clearer skin and pores are tighter*
*Skin is much smoother*
*There is a glow to my skin*
Clear + Brilliant® Results
Healing Progression Photos

1 Week Post Treatment
* Short downtime & smoother skin after treatment*
* Everything just looks better*
* Tone is even, more natural*

2 Weeks Post Treatment

Clear + Brilliant® Results
Healing Progression Photos

Clear + Brilliant® Perméa® Results
Before and After Photos

Before and after photos have not been retouched. Individual results may vary.

Clear + Brilliant® Perméa® Results
Before and After Photos

Before and after photos have not been retouched. Individual results may vary.

PicoSure® Product Overview
- 755 nm, 532 nm, 1064 nm
- 550-750 picosecond pulse duration
- Boost™ for 70% pressure increase
- FOCUS™ Lens Array
  No other pico laser has all this!

Focus™ Lens Array
- Diffractive lens redistributes and delivers 755 nm energy
- Low and high intensity energies lighten unwanted pigment
- High intensity energy leads to LIOBs, Pressurewaves, cell signaling and dermal remodeling

- Only PicoSure’s unique injury can trigger temporary cell membrane permeability, enhanced inflammation, increased collagen & elastin, with virtually no downtime
Focus: *uniquely creates* intra-epidermal injuries *activates* pressurewaves and cell signaling.

Images courtesy of Emil Tanghetti, MD.

**LIOBs after a single pulse** viewed via Confocal Microscope.

**LIOBs**

100x mag.

LIOB Focal injuries

Dermal inflammation

10 min post

24 hrs post

24 hrs post

**How does Focus compare histologically?**

Fraxel Restore
Dual non-ablative

Fraxel Repair
CO2 ablative

PicoSure Focus

**Columns of thermal injury, epidermal injury, and open lesions**

Elegant injury limited to the epidermis, no open lesions, and virtually no downtime.

Courtesy of Bob Weiss, MD and Emil Tanghetti, MD.

**Collagen & Elastin ~ 6 months after 4 Focus Tx**

Blue stain shows increased collagen deposition.

Gray stain shows increased density of elastin fibers.

**Before**

**After**

Collagen and Elastin ~ 6 months after 4 Focus Tx

Before 4 Focus Tx

Before

After

Before

After

**Focus Lens Array**

Impressive Results with Minimal Discomfort/Downtime.

**Before**

**After 4 Focus Tx**

Pigment & Acne Scar Treatment

**Before**

**After 4 Focus Tx**

Pigment Treatment

**Before**

**After 4 Focus Tx**

Before 4 Focus Tx

Before

After

Before

After

Pigment Treatment

Pigment & Acne Scar Treatment

Before 4 Focus Tx

Before

After

Before

After

Pigment & Acne Scar Treatment

Pigment Treatment

Before 4 Focus Tx

Before

After

Before

After

Pigment Treatment

Before 4 Focus Tx

Before

After

Before

After

Pigment Treatment
Wrinkle & Pigment Treatment

Skin Revitalization

THE CHALLENGE – NON-ABLATIVE VS. ABLATIVE

Aging gracefully is 20% Nature and 80% Nurture
Facial Plastic & Reconstructive Surgery
Jean-Paul Azzi, MD

Outline

• The Bilobe Flap – Pushing the limits.
• Challenging and Interesting Reconstructive Cases.
• Charity Trips

Bilobed Flaps

• Azzi, JP. Bilobe Flaps for Nasal Reconstruction: A Single Surgeon’s Experience with 50 Consecutive Patients.
• Aesthetic and functional outcomes measured on cases sampled from 2015.

Bilobe – Brief Intro

• Great option for the nose – one of the most frequent sites of skin cancer.
• Mohs is a great way to treat cancers in this area, and they can all be reconstructed in the office even if underlying cartilage etc. is excised.

Intro

• Debate about use of bilobed flaps outside of the caudal region of the nose
• Also debate about appropriateness of use in the alar region
• One often cited article (choi) recommends not using any type of local flap within 5mm of the alar free margin secondary to notching and retraction
**Intro**
- Other feared complications when the defect is in close proximity to the free margin is distortion – elevation of the tip or nostril margin and thickness irregularities
- Another complication can be nasal obstruction

**Methods**
- Chart review in a single private practice facial plastic surgery practice
- 50 patients over a year period in 2015
- Minimal followup of 3 months
- Majority repaired immediately following Mohs micrographic excision in Palm Beach County
- All performed in the office under local anesthesia
- Outcomes measured included: flap viability, alar retraction or notching and patient satisfaction surveys (excellent, good, fair, poor)

**Results**
- BCCA (78%), SCCA (16%) and Melanoma (6%)
- Mean Max diameter of 18mm
- Most common subunit involved was the tip followed by ala
- None of the defects involved the internal lining of the nose

**Results**
- Smoking did not seem to have a noticeable effect on outcome
- Excellent (78%), good (22%) – patient
- Fair or Poor rating by surgeon in 7/50 cases

**Discussion**
- Results overall favorable – good flap viability regardless of age, smoking and size
- Two patients had partial distal flap necrosis → secondary intention → dermabrasion/revision
- Possible Causes: cautery, hematoma, infection, poor patient healing, scar tissue in recurrent area, overuse of bipolar, plane of dissection

<table>
<thead>
<tr>
<th>Case</th>
<th>Tumor Location</th>
<th>Max Diameter (mm)</th>
<th>Age</th>
<th>Sex</th>
<th>Smoking</th>
<th>Flap Viability</th>
<th>Alar Retraction</th>
<th>Patient Satisfaction</th>
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<td>15</td>
<td>50</td>
<td>M</td>
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<td>Excellent</td>
<td>None</td>
<td>Excellent</td>
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<td>70</td>
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<td>M</td>
<td>Yes</td>
<td>Good</td>
<td>None</td>
<td>Fair</td>
<td>None</td>
</tr>
</tbody>
</table>

*Note: Tumor Location: BCCA = Basal Cell Carcinoma, SCCA = Squamous Cell Carcinoma.*
Discussion

• Since these 2 cases I have changed site prep and avoided distal necrosis
• Limit bipolar
• Counsel on DM control and nicotine exposure
• Plane of flap (esp. useful with smokers)

Discussion

• Two patients with moderate alar retraction/notching → both rim involved or nearly involved after site prep
• One was 3mm from rim, but irregular/vertically oriented, significant burns to skin/char dermis, lower lat involved → declined pedicled flap

Discussion

• One nasal obstruction complication
• Ala/sidewall defect
• Same patient also had distortion at 8 weeks
Discussion

• Obstruction likely secondary to edema and possible deep sutures aggravating an existing nasal valve collapse.
• Improvement reported after 3 weeks
• Low dose Kenalog used, massage and nasal steroid sprays used to speed up recovery
• After 12 weeks still reported subjective nasal airway asymmetry
• This can be limited with cartilage grafts when appropriate

Retraction

• Repair can be challenging
• Local flaps and grafts can be used for these repairs
• Most commonly composite graft
• V – to – Y (Sykes) paired with a composite graft

Discussion

• One patient had significant immediate post-op nasal distortion. 30mm nasal tip SCCA.
• Essentially entire sub-unit.
• Declined interpolated forehead flap secondary to schedule (3 week).
• Improved slowly over 3 months with massage, Kenalog and dermabrasion.
Conclusions

- Nasal reconstruction can present considerable challenges.
- The Bilobe flap is a reliable and versatile flap

Interesting and Challenging Cases
Cases

Cases

Cases

Cases

Cases

Cases
Mission Trips

- Healing the Children Northeast
- HUGS – help us give smiles
- Colombia, Guatemala, Ecuador, Vietnam
- Adding Peru and India
- Microtia, Lips, Palates etc
Hanoi, Vietnam

• I can't find any of my photos from this trip 😞
• So...

Contact Info - Azzi

• JP Azzi:
  • doctorazzi@gmail.com
  • Office: 561 429-5403
  • Cell: 352 871-1015
  • www.PalmBeachFacialSurgery.com
Opportunities in Cosmetic Dermatology > $30 Billion Annually

Who are the Pioneers? A Critical Analysis of Innovation and Expertise in Cutaneous Noninvasive and Minimally Invasive Cosmetic and Surgical Procedures


My personal transition from a General/Surgical Dermatologist to Solely Minimally Non-Invasive Surgical Cosmetic Dermatologist

Why Solely Cosmetic Dermatology?
- Scheduling
- Insurance reimbursement
- Staff responsibilities and personalities
- Reducing liability
- IQ & EQ
- Artistic Purposeful Practice

Evolution dictates our perception of beauty as a way of improving offspring stability

Attractiveiveness are “hard wired” into our brains beauty is not defined by popular culture.

Features that are regarded as beautiful in all cultures:
- Clear skin may connote a healthy, clean, parasite-free body.
- Females, a waist to hip ratio of 0.6 implies fertility and well-nourished bodies.
- Proportionate facial symmetry is a universal feature deemed attractive

Research has documented that in our society, physical appearance has a large impact on how individuals are perceived by others.

- Attractive people receive preferential treatment in education, employment, medical care, legal proceedings, and romantic encounters that often result in their being happier, more successful, more socially adept, and more sexually fulfilled than others.
- Above average in attractiveness earn more money.
- Attractive appearance promotes psychological well-being.


The subliminal difference: treating from an evolutionary perspective.

Beauty serves as a subconscious form of communication, signaling our health, vitality, and ability to reproduce. Processed in primitive neural pathways in the amygdala and posterior cingulated cortex.

Most appropriate treatments:
- Surgical treatments vs. nonsurgical vs. nothing at all

Obvious cosmetic interventions may be counterproductive in interfering with the subconscious message.

Patient’s Motivation for Treatment

The face is the focus of human interactions and emotional expressions. Their appearance is not communicating their emotions, age or health status properly. It’s for those who simply want to look their best. Appearances profoundly affect self-esteem.


Non-Surgical Cosmetic Dermatology

Treatment Result

You are the Doctor, treat like it

Diagnosis and treat

Faces and bodies are 3D
- the mirror is 2D, use photos as a 3D tool to educate
- Don’t treat for the mirror symptoms

2D to 3D

Use photos of frontal view, 45° view, side view, and animation for face to decide treatment plan and pt. education.

Treatment Plan Goals

- Improve Skin Quality and Texture
- Reduce uneven pigment and “age spots”
- Reduce Wrinkles (rhytids)
- Redo shape for lost volume
- Reduce sun damage
- Reduce Benign tumors
- Reduce Scars

Understanding the aging face
Understanding the aging face

The shape of the aging face shifts from a Triangle to a Pyramid

Full understanding of anatomy is imperative for proper evaluation, treatment and safety

Facial soft tissue deterioration most dramatic between the ages of 30 and 60

Soft tissue augmentation and volume correction in these areas is strategic for aesthetic treatment.

Facial soft tissue deterioration most dramatic between the ages of 30 and 60

Soft tissue augmentation and volume correction in these areas is strategic for aesthetic treatment.

“BeautiPHIcation”

Natures mathematical artistic brush stroke

Non/Minimally Invasive Cosmetic Procedures Tools

Botulinum Toxin – Neurotoxins (Botox, Xeomin) and hyperhidrosis.
Injectable Fillers
- Hyaluronic acid
- Calcium hydroxypaprite
- Poly-lactic acid
Noninvasive fat removal Cellulite treatment
- Cryolipolysis
- Deoxycholic Acid
- Absorbable suture “thread lifting”
Chemical Peels
- Laser/Energy Devices
- Hair Transplantation
- Sclerotherapy
Natural Appearing Treatment

Neurotoxin- releasing muscles last 3-4 months usually used in upper 1/3rd of the face, but excellent for neck and less often used in lower 1/3rd of face

Hyaluronic acids- multiple types available that fill many uses to manipulate shapes and wrinkles, huge advantage is it is reversible

Poly-L-lactic Acid- causes tissue growth stimulation also used to manipulate global shapes and wrinkles, more advanced techniques

Calcium hydroxyapatite- implant like material

Injections Combined Approach

Most Common Fillers Modify Shape and Wrinkles

Hyaluronic Acids (HAs)

Most commonly FDA Approved filler used today in the US

Hyaluronic acid is a type of sugar (polysaccharide) that is present in body
Combines with water and swells when in gel form, causing a smoothing/filling effect.
Chemically modified (crosslinked) to make it last longer in the body.
Most available HAs have lidocaine added to reduce discomfort.
The effects of this material last approximately 6 – 12 months.

Injections Combined Approach

Neurotoxins- releases muscles last 3-4 months usually used in upper 1/3rd of the face, but excellent for neck and less often used in lower 1/3rd of face

Hyaluronic acids- multiple types available that fill many uses to manipulate shapes and wrinkles, huge advantage is it is reversible

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Calcium hydroxyapatite- implant like material

Most Common Fillers Modify Shape and Wrinkles

Hyaluronic Acids (HAs)

Huge Advantage is its safety and reversibility
More unique properties for different approved brands allowing for better treatment options and outcome

- Lifting ability
- Relaxation in dynamic motion
- Duration
- Tissue integration
- Swelling ability
- Softness of natural changes
Hyaluronidase – How to treat SEs

Rescue
Used as a rescue injection in large quantities (200 units plus for HA accidentally injected into a vessel causing an occlusion which lead to tissue necrosis and/or even potentially blindness.

Small HA papules
Used in smaller doses (20 units) it can be used to correct small quantities of HA that may be causing a abnormal or unattractive outcome.

Hyaluronidase – How to treat SE

Joel L. Cohen, MD; Brian S. Biesman, MD; Steven H. Dayan, MD; Claudio DeLorenzi, MD, FRCS; Val S. Lambros, MD; Mark S. Nestor, MD, PhD, PA; Neil Sadick, MD; and Jonathan Sykes, MD

Treatment of Hyaluronic Acid Filler–Induced Impending Necrosis With Hyaluronidase: Consensus Recommendations. Aesthetic Journal 2015, 1-6

Poly-L-lactic acid (PLLA)

Poly-L-lactic acid (PLLA) is a biodegradable, biocompatible man-made polymer. This material has wide uses in absorbable stitches and bone screws. PLLA is a long lasting filler material that is given in a series of injections over a period of several months.

The effects of PLLA generally become increasingly apparent over time (over a period of several weeks) and its effects may last up to 2 years.

Tissue Stimulator (collagen synthesis) giving a global filling

Effects are not reversible

Calcium Hydroxylapatite

Calcium Hydroxylapatite is a type of mineral that is commonly found in human teeth and bones.

FDA approved for wrinkle filling in the face or for the hand.

The effects of this material last approximately 18 months.

While in the body, calcium hydroxylapatite will be visible in x-rays and may obscure underlying features.

Not reversible

Examples of Before and After’s

Neurotoxin & Poly-L-lactic Acid
Cheek Enhancement Hyaluronic Acid

Full Face
Hyaluronic Acids
Poly-L-lactic Acid
Neurotoxin

Lip and Chin Enhancement with Hyaluronic Acid

Full Face
Neurotoxin, Poly-L-lactic acid, Hyaluronic Acids

Neurotoxin and Poly-L-lactic acid

Eye (tear trough) Hyaluronic acid
Hyaluronic Acid...

In the Nose

Treatment....?

HA in the Chin

Needles vs Cannula

Previously fillers were injected with sharp needles producing various undesirable effects such as pain, bleeding, hematomas, edema and inflammation. Cannulas are tubes that can be used to administer products into the body and have blunt tips. Cannula use allows for a significant reduction in these undesirable effects. There is now a FDA approved HA filler used for lip enhancement using a cannula.


Needles vs Cannula

Energy-Based Devices & Body Contouring

Devices are the most costly investment in a cosmetic practice and serve very specific goals.

Lasers, Radiofrequency, Ultrasound are the most common devices.

The goal is to target specific tissue parameters to even out the surface irregularities, skin and aging discolorations and to tighten tissue.

Excellent necessary modalities for a Cosmetic practice.

Very operator dependent and results tend to vary based on downtime.

Fat Reduction and Cellulite treatments
Focused Ultrasound

Non-invasive therapeutic focused ultrasound for lower face

Fractional CO2 Resurfacing & Radiofrequency via Micro-needling

Before 9 days post tx 30 days post tx

Cryolipolysis
Non-Surgical Permeant Fat Reduction

An innovative way to contour your face and body by freezing unwanted fat away with no surgery or downtime. Currently more than 6 million treatments have been performed worldwide.

Technology safely delivers precisely controlled cooling to gently and effectively target the fat cells underneath the skin while leaving the skin itself unaffected.

3 sessions Cryolipolysis Torso

Male Torsos 1 Cryolipolysis Session
1 session Chin Cryolipolysis

Inner thigh & Buttocks Cryolipolysis

Education helps both you and your patient Create a Treatment Plan

Respecting facial anatomy and natural proportion are key
- **Golden Ratio**
- Fill 3D not 2D (Don’t fill for the mirror)
- Know when to say NO

Make it a partnership for restoration and then a maintenance treatment plan
- A treatment plan allows patient’s realistic expectations to be realized within budgetary parameters.
- A combination modality is a better method to meet patient’s expectation
- Schedule an ongoing maintenance treatment plan

Is Cosmetic Dermatology Right For You?

Cosmetic vs. non-cosmetic practice
- Doing aesthetics is a sub-speciality and should be treated as such, just like Mohs or Dermatopathology
- Practice time constraints
- Staff support
- Physical office layout – esp. in more competitive markets

Motivation and Self Reflection
- Helping your patients be the best they can be!
- Revenue stream that is not dependent on insurance reimbursement
- Your own personality

References


Carruthers JD, Glogau RG, Blitzer A; For Facial Aesthetics Consensus Group Faculty. Advances in facial rejuvenation: botulinum toxin type a, hyaluronic acid dermal fillers, and combination therapies—consensus recommendations. Plast Reconstr Surg 2008;121(Suppl 5):5S–30S.


Dayan SH, Arkins JP. The subliminal difference: treating from an evolutionary perspective. Plast Reconstr Surg 2012;129:189e–90e.

Dermal Fillers Approved by the Center for Devices and Radiological Health U.S. FOOD & DRUG ADMIN (FDA), www.fda.gov.


Protecting Your Practice

NOT USUALLY PART OF YOUR MEDICAL SCHOOL CURRICULUM

Disclosures

I have no relevant disclosures.

Sources of Risk to your Practice

▪ Unexpected Tax Liabilities (penalties and interest)
▪ Predatory Lawsuits (Malpractice, Premises Liability, etc.)
▪ Death or Disability
▪ Economic Downturn
▪ Shareholder Disputes
▪ Employment Contract Disputes
▪ Workplace Disputes
▪ Divorce
▪ Unscrupulous Financial Advisors
▪ Regulatory and Compliance Violations

REGULATORY AND COMPLIANCE RISK

ARE YOU PREPARED !?!?!?
Navigating the Anti-Abuse Provisions

- And it isn’t as simple to determine as violating the Anti-Abuse laws. It is simply impossible to keep up with laws. New legislation is not infrequent, but merely because there is no definition of what the laws are, regulations every practice must respect:
- 42 USC section 1395nn (the “anti-kickback law”); 42 USC section 1128b(b)(7); 42 USC section 1320a-7a; 42 USC section 290ee-3; 31 USC section 375b (the “Anti-Abuse provision”)
- 21 USC section 801, et seq. (the “False Claims Act”)
- 10 USC section 1071, et seq. (the “Military Claims Act”)
- 42 USC section 1320a-7a (the “Kahn Act”)
- 10 USC section 951 (the “Military Claims Act”)
- 31 USC section 375b (the “False Claims Act”)
- 31 USC section 375b (the “False Claims Act”)

Possible Ramifications

- Treble Damages
- Attorneys’ Fees
- Exclusion from Government Reimbursement Programs
- Loss of License
- JAIL TIME!

MEDICAL MALPRACTICE/STARTLING STATISTICS

By some estimates, the median jury award is $5 million and a doctor has a one in four chance of being sued. THIS YEAR.

WILL INSURANCE BAIL ME OUT?

- Many doctors think that they will never be sued because they are nice people and are generally careful. This is a myth.
- Malpractice often has nothing to do with guilt and everything to do with deep pockets. Perhaps you are innocent. Do you really think your innocence can be proven to a group of 12 people not smart enough to get out of jury duty?

Whistle Blower

- Another risk for medical professionals, is the danger of a false claim case. A current or former employee, or a competitor practice may find a regulatory violation of one or more Anti-Abuse statutes, possibly resulting in an action on behalf of the government, both civil and criminal.

FOR TREBLE DAMAGES!!!

Myth of the Good Doctor

- WILL INSURANCE BAIL ME OUT?
Paradox of Malpractice Insurance

• Hardest part of any lawsuit is getting paid.
• Lawyers know this, collection often takes two or three times as much effort as getting the judgment. Malpractice insurance is an attractive target because it does not require any additional litigation to locate and recover personal assets of the physician.

No Insurance - Less Attractive/More Risk

• The paradox is that while lower coverage exposes your personal assets to creditor’s claims, it also makes you a less attractive target, particularly when you’ve done the proper planning.

Dazed and Confused

• With all the potential dangers and pitfalls, it is no wonder some physicians become overwhelmed and just leave their exposure up to chance.

Physician Practice Planning Goals

• Limit Bad Result from Lawsuits (i.e. Malpractice, Divorce, Practice Compliance, and Business Disputes)
• Ownership Structuring for Asset Protection, Estate Planning and Retirement
• Minimization of Tax Burden

Spectrum of Security-Titling and Selection of Assets

Own Nothing in your own name
• Pros
   - Not a target
   - Can’t get blood out of a stone
   - Assets are safe from your creditors
   - Ups
   - Assets at risk from those entrusted to title
   - Hard to reach own assets
   - More difficult to get financing

Own Exempt Assets (Florida)
- Homestead
- Annuities
- Life Insurance
- Retirement Accounts

Own Nonexempt Assets
• Risks
  - Personal creditors
  - Business creditors
• Possible solutions
  - Buy insurance
  - Strategic Titling of Assets
  - Trust Planning
  - Keep your Fingers Crossed

WHAT’S IT ALL ABOUT?

It is all about how your assets are titled!
What is the Best Manner of Holding Title to My Assets?

- What form is ideal?
  - LLC
  - Tenants by the Entirety
  - Joint Tenant with survivorship
  - Individual
  - Tenants in common

- Joint tenants
- S Corp
- C Corp
- Member
- Partner
- Trust Beneficiary

Individual Ownership

- Solely
- Tenants in common
- Joint Tenants
- Joint Tenants with a Right of Survivorship
- Tenants by the Entirety

Entity Forms of Ownership

- Corporations (both C and S)
- Trusts (Grantor and Non Grantor)
- Limited Liability Companies (taxed as disregarded, S-Corp, C-Corp, or partnership)
- Partnerships (general, limited, limited liability, etc.)

Tenants by the Entireties

Tenancy by the entirety contains all uribus: the quality of time, title, interest, possession, marriage, and person.
The Creditor of only one spouse cannot attach.

Tenants by the Entireties

Protection from individual Creditors, while still providing the freedom of management. It does not work well in the event of divorce, death, or joint debt.

LLC: the Limited Liability Company

Charging Order: A Remedy without a Reward

LLC Ownership: Single Member LLC v. Multi Member LLC

What's the difference?

Single Member LLC
- In Olmstead the owner of a single member LLC owed a judgement to the FTC.
- The Supreme Court of Florida observed that normally the managerial interest in an LLC is not freely transferable without the consent of the other members.
- However, in a Single Member LLC, the sole Member can transfer without the consent of anyone.
- Therefore, the Court ruled that a court could order such sole Member to transfer his or her interest for the benefit for a creditor.

Multi Member LLC

YOU

Other Member

Creditor

Other Member

Creditor

Olmstead Order

YOU

Creditor

Olmstead Order

YOU
The Operating Agreement is the Key

- A charging order allows the creditor to attach distributions made by the LLC to that member, but does not allow the creditor to force distributions from the LLC, cause liquidation, or make managerial decisions.
- The LLC agreement could cause the creditor to be on the hook for income taxes of undistributed income attributable to the debtor/member’s interest.
- Disproportionate distributions to members.

INDIVIDUAL OWNERSHIP OF EXEMPT ASSETS

If your holdings are limited to exempt assets, they can be titled in your individual name.

EXEMPT ASSETS

Each State is Different!

FLORIDA EXEMPT ASSETS

- Homestead
- Wage account
- Life insurance policy, annuity contracts (limited to citizens and residents of Florida – not available for entities)
- Pension, profit sharing plans, IRAs
- 529 qualified tuition program
- Health Savings Account, Medical Savings Account
- Hurricane Savings Account
- Motor vehicle up to $1,000
- Personal property up to $4,000

Wage Garnishment Exemption: State and Federal

Florida Exempt Wages (Fla. Stat. 222.11)

- Head of Household
  - 50% more than 50% of the support of a child or dependent
  - All Wages Exempt unless agreed otherwise in writing
  - If not Head of Household
  - May not garnish in excess of amount allowable under Consumer Credit Protection Act
- Wages are safe from garnishment in a financial institution for 6 months

Federal Consumer Credit Protection Act (15 USC 1673)

- Limits the amount a creditor can garnish
- A judgment creditor can only garnish the lesser of:
  - 25% of your disposable earnings, or
  - your weekly disposable earnings less 30 times the federal minimum wage (currently $7.25 per hour)

BANKRUPTCY – DOES IT MAKE A DIFFERENCE?

- Bankruptcy is Federal law, not state law, so Federal law controls
- Exemptions in Bankruptcy Proceedings are different than in Non-Bankruptcy Proceedings (both state or federal)
Bankruptcy Exemptions (11 U.S.C. §522)

• You cannot elect federal exemptions if you qualify for Florida Exemptions

• To qualify for Florida exemptions you must meet the Federal Bankruptcy Code’s definition of resident:
  • Be domiciled in Florida for at least 730 days before filing; or,
  • If you were not living in any one state during the two year period before filing, you may use Florida if you primarily lived in Florida during the 180 day period prior to the two-year period.

Florida Bankruptcy Exemptions Compared to Federal Bankruptcy Exemptions

<table>
<thead>
<tr>
<th>Florida</th>
<th>Federal</th>
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<tbody>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Federal 11 USC 522</td>
</tr>
<tr>
<td></td>
<td>$23,675 of equity in principal place of residence</td>
</tr>
<tr>
<td></td>
<td>No investment property</td>
</tr>
<tr>
<td></td>
<td>Must live in the dwelling</td>
</tr>
</tbody>
</table>

Homestead in Bankruptcy

Florida
• Half acre in municipality or 160 acres elsewhere
• Must have owned property for at least 1,215 days prior to filing
• If you can’t meet this requirement, you are capped $160,375
• No investment property
• Must live in the dwelling

Federal
• $23,675 of equity in principal place of residence
• No investment property

Prepared by: Jennifer L. Klitzman

PLANNING OPTIONS FOR NON-EXEMPT ASSETS

No one size fits all!

Structuring for Success

• Planning does not end with insurance
• Not all businesses look the same, and not all Business Structures offer the same level of protection
• Some Business Structures offer asset protection and estate planning benefits
• Some Business Structures are designed for an easier sale
• Some leave your personal assets at risk
• What structure is best for your practice?

Structure to Avoid for Practice Owners

Don’t put all your eggs in one basket!
A Broken Model

Cross Contamination: An Easy Mark

All assets are owned directly by a single. As practice assets are exposed to any practice liability, including:

- Premises Liability
- Malpractice Liability
- Employment Practice Liability
- Contractual Claims
- Bankruptcy
- Regulatory/Compliance Liability

The Practice

Real Property
Equipment
Staff
Accounts Receivable

Management Services Entity

You

Rent
Family Asset Protection Entity
Salary
Payment for Services
Provides Nonprofessional Staff and Manages Office

Accounts Receivables Financing

Family Asset Protection Entity

Assignment of Accounts Receivables

Owner

Loan

The Practice

Real Property and Equipment

Many Practices own the real property they occupy may very expensive equipment required to operate.

By owning the Real Property and Equipment in separate entities, they may be removed from the reach of the creditors of the Practice.

The Practice pays rents to holding entities who in turn make distributions to either the owner or an asset protection entity owned by the physician and his or her family.

Management Services Entity

You can further remove value from your Practice by forming a Management Services Entity to manage your office and non-professional staff.

Profit of the practice is reduced, making the practice a less attractive target to creditors.

Isolates liability arising from non-professional services personal to assets of Management Services Entity.

May allow greater flexibility in retirement planning by limiting eligible employees.

A Solution in a Structure

Segregating Practice Assets to Insulate Against Catastrophic Losses

By using separate entities to hold practice related assets, the personal liabilities of anyone holding a management position of any described to collect even much less attractive in target for an aggrieved attorney.

WHAT IF YOU ARE AN EMPLOYEE?

Is strategic structuring necessary?
HOW ARE YOUR ASSETS TITLED?

The same considerations would apply to employees as to a Practice Owners.

If your assets are not titled in your name, or are all exempt, than no further planning may be necessary.

If you have a low tolerance for creditor risk, there are strategies you may want to consider to protect your savings.

**Multi-Member LLC**

for Savings and other Investments

Whether you buy real estate, securities or make other investments title can be held in a multi member LLC or other entity with more than one member, partner or shareholder.

<table>
<thead>
<tr>
<th>Family Member</th>
<th>Physician Member</th>
</tr>
</thead>
<tbody>
<tr>
<td>1% owner</td>
<td>99% owner</td>
</tr>
</tbody>
</table>

**Trust Owned Physician Services Entity**

Protection of savings.
Possible QBI (Qualified Business Income) deduction
Non-Settlor – Third Party Owner of Medical Practice is permitted
No Corporate Practice of Medicine Doctrine

**WHAT ELSE CAN BE DONE TO REMOVE NON-EXEMPT PERSONAL ASSETS FROM THE REACH OF YOUR CREDITORS?**

Planning with Non-Exempt Assets.

**Strategies to Protect Non-Exempt Assets**

- **DAPT (Domestic Asset Protection Trusts)**
- **Double LLC Structure with a side of DAPT**
- **FAPT (Foreign Asset Protection Trusts)**

**DAPT**

- An irrevocable Trust where the settlor is a discretionary beneficiary set up in one of 17 jurisdictions that allow a self settled trust to be free of creditors access, except for some exceptions and special creditors.
- For example: an ex-spouse can reach self-settled trusts in most jurisdictions for alimony or child support.
**DAPT**

General Rule: Do not transfer more than 50% of assets to an Asset Protection Trust

Not available in every state

**MODIFIED DAPT**

- A DAPT which is created for the benefit of a third party (usually the grantor’s spouse or children).
- Superior asset protection.
- Sometimes the grantor may be added as a future beneficiary.

---

**Which jurisdiction should be used for my DAPT?**

17 Possibilities and Growing

**The Nevada and South Dakota Options**

(Depends who you are talking to)

- Nevada and South Dakota have favorable exception creditor laws.
- Even in the other 15 jurisdictions which allow for creditor protection for self-settled trusts, special exception creditors may be able to access the funds.
- In Nevada, even an ex-spouse cannot penetrate a properly formed and funded DAPT for child support.
- Typical third party Trustee fees to set up and administer DAPTs:
  - Set up fee: ($750-$1,250)
  - Annual Administrative Fee: ($2,250-$4,000)

---

**Multiple Entity Strategies**

- Foreign Asset Protection Trust (FAPT)
  - Full faith and credit of United States Courts not required to be recognized in foreign jurisdictions.
  - In USA, you can have a judgment entered in Georgia domesticated and enforced in any other state.
  - In many foreign jurisdictions, you must re-litigate the debt before a judgment can be enforced.
  - Effective when property transferred to FAPT is outside of the United States with foreign grantor.
ASSET PROTECTION BEYOND THE PHYSICIAN
(Pre-Sale Strategies)

• Gifts to Family Members (Outright or in Trust)
• GRAT (Grantor Retained Annuity Trusts)
• Installment Sales to Defective Grantor Trusts

You've built your practice over many years and are considering the final sale. Before a high price is established for your practice, there is an opportunity to take advantage of the arbitrage between a reasonable valuation and the ultimate sale price. This technique enables the practice owner to remove a large portion of the practice owner’s potential creditors.

GRATs work best with highly appreciating assets.

Asset Protection Interrelation with Estate Planning

• Strategies to extend Asset Protection to future generations
• Minimize Estate Tax liability

Trump Tax Law

• The Tax Cuts and Jobs Act: An Opportunity with an Expiration Date

  • Doubles the Transfer Tax (GIFT, Estate & GST Tax) Exemption from:
    • For Individuals: $5,600,000 to $11,200,000
    • For Married Couple: $11,200,000 to $22,400,000
  • Without further Congressional action, this increased exemption sunsets in the year 2025.

  • This creates a window of opportunity to take advantage. Make gifts now up to $22.4 million mark to dynasty trusts to get those assets outside of the transfer tax system forever.

Pigs get Fat, Hogs get Slaughtered

• Take care to plan well in advance of a problem and do not get greedy
• The closer to an incident you plan, the less likely it will work
• If you get too extreme, your planning may be disregarded
Fraudulent Transfers
Florida has adopted the Uniform Fraudulent Transfers Act - Fla. Stat. §726.101

- 2 main tests:
  - First, for present creditors (Fla. Stat. § 726.106)
    - (a) whether the debtor received reasonably equivalent value and
    - (b) whether the debtor was insolvent or became insolvent
  - Second, for present and future creditors (Fla. Stat. § 726.105)
    - (a) intent to defraud, or
    - (b) without receiving reasonably equivalent value and:
      - Debtor’s assets after transfer were too small in relation to the business he was engaged or about to be engaged in
      - Debtor knows or reasonably should have known that he would have debts greater than he could pay

What is a Fraudulent Loan?

- A loan is presumed to be fraudulent when there is no payment or demand on a loan for two years. Fla. Stat. § 726.201.
  - Most common in family situations
  - Example: My Uncle lent me money to start my business. I never paid him back. But now that I have a creditor trying to collect from me, I want to pay my Uncle back.

Lawrence S. Klitzman, Esquire
Degrees & Affiliations

- JD and LLM in Taxation, University of Miami
- Admitted in Florida and New Jersey
- 30+ years experience
- Member National Academy of Elder Law Attorneys
- Member Florida Academy of Elder Law Attorneys
- Member Florida Bar Real Property, Probate and Trust Committee
- Member Florida Bar Elder Law Committee

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Hidradenitis Suppurativa Therapeutic Update

Francisco A. Kerdel BSc, MBBS
Director of Dermatology Inpatient Service Larkin Community Hospital, Miami, FL
Clinical Professor and Vice Chairman of Dermatology Florida International University

Faculty Disclosure*:
Amgen
Actelion
AbbVie
Galderma
Janssen
Genentech
Pfizer
Merck
Novartis
Celgene
Lilly
Regeneron

*Dr. Kerdel has been involved in clinical studies, has participated in advisory boards and is a speaker for the above companies.

Hidradenitis Suppurativa (HS)

1% prevalence
Commonly presents early second decade
Declines after age 50
1/3 have family history
Linked to chromosome 1p21.1-1q25.3 (γ-Secretase complex)(NCSTN,PES-NEN,PSEN1 genes)
Obesity and smoking - risk factors
May present in pts with Crohn’s, PG & arthritis
Elevated inflammatory markers in pts with severe disease

Hidradenitis Suppurativa (HS) Diagnosis and Associated Symptoms

- Apocrine gland folliculitis
- Pain
- Draining fistulae
- Fever, chills, lethargy
- Comedones
- Scarring and tissue damage
- Compromised integrity of skin may lead to bacterial colonization

HS comorbidities

- Follicular occlusion tetrad
- Metabolic syndrome
- Inflammatory bowel disease
- Spondyloarthritis
- Depression
- Pyoderma gangrenosum

Hidradenitis Suppurativa: Staging

- Hurley staging
  - Stage I – Lesion formation, single or multiple without sinuses or scarring
  - Stage II - Recurrent lesions with sinuses and scarring, widely separated
  - Stage III – Diffuse involvement of entire area
Hidradenitis Suppurativa – Clinical Scores

- Sartorius sore – (+/- modified) counting individual lesions and distances between them, extra points for Hurley stage III
- Physician global assessment – clear to very severe depending on number and type of lesions
- Hidradenitis suppurativa severity index – lesions, pain, dressing changes and affected area
- Hidradenitis Suppurativa Clinical Response – 50% reduction in nodules with no change in abscesses or fistulas

Hidradenitis Suppurativa: Underlying Mechanism and Treatment Strategies

- Mechanism
  - Follicular gland occlusion followed by an inflammatory response vs. apocrine gland primary target followed by follicular duct pathology
- Treatments
  - Hygiene, weight and friction reduction
  - Cessation of smoking, topical antibiotics and cleansers
  - Systemic antibiotics (minocycline, clindamycin/rifampicin)
  - Topical and systemic corticosteroids
  - Cyclosporine, anti-androgens, retinoids
  - Local radiation
  - Photodynamic therapy, Hyperbaric oxygen
  - Surgery
  - Laser
  - Biologic therapy (anti-TNF)

Hidradenitis Suppurativa: Rationale for Using Anti-TNFα Agents

- Indirect Evidence
  - Anti-TNFα drugs are efficacious in the treatment of other diseases associated with an inflammatory process (psoriasis, rheumatoid arthritis, ulcerative colitis, pyoderma gangrenosum, and acne conglobata)
- Direct Evidence
  - Anti-TNFα drugs are effective in treating HS

Infliximab for Hidradenitis Suppurativa

- Sullivan TP, Welsh E, Kerdel FA, Burdick A, Kirsner RS.
- Br J Dermatol 2003;149:1046

Infliximab therapy for patients with moderate to severe hidradenitis suppurativa: a randomized, double blind, placebo-controlled crossover trial

- Grant A, Gonzalez T, Montgomery M O, Cardenas V, Kerdel F A

HS: Significant Improvement Observed After Treatment with Infliximab

Before treatment with infliximab

After treatment with infliximab
Study Design

Double-blind Phase (8 wks)
- Placebo n=18
- Infliximab 5 mg/kg n=15

Crossover to infliximab 5 mg/kg

Open-label Phase (22 wks)

Observational Phase (22 wks)

Inclusion Criteria

- HSSI ≥8
- HS >1 year with multiple ER/Doctor visits
- Failed topical/systemic therapy
- Failed surgery
- Age >18 years
- Adequate birth control
- Negative history for TB, PPD and CXR

Demographics and Baseline Disease Severity

<table>
<thead>
<tr>
<th></th>
<th>Placebo</th>
<th>Infliximab</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (yrs)</strong></td>
<td>33.2 (11.42)</td>
<td>34.0 (13.44)</td>
<td>0.994</td>
</tr>
<tr>
<td><strong>Gender, N (%)</strong></td>
<td>Male 9 (31.0)</td>
<td>3 (20.0)</td>
<td>0.099</td>
</tr>
<tr>
<td></td>
<td>Female 14 (60.9)</td>
<td>12 (80.0)</td>
<td></td>
</tr>
<tr>
<td><strong>Race, N (%)</strong></td>
<td>Black/African American 7 (30.4)</td>
<td>3 (20.0)</td>
<td>0.265</td>
</tr>
<tr>
<td></td>
<td>White 6 (26.1)</td>
<td>8 (53.3)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hispanic 8 (34.8)</td>
<td>3 (20.0)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Other 2 (8.7)</td>
<td>1 (6.7)</td>
<td></td>
</tr>
<tr>
<td><strong>HSSI, N (%)</strong></td>
<td>Severe 18 (78.3)</td>
<td>14 (93.3)</td>
<td>0.123</td>
</tr>
<tr>
<td></td>
<td>Moderate 5 (21.7)</td>
<td>1 (6.7)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mild 0 (0)</td>
<td>0 (0)</td>
<td></td>
</tr>
<tr>
<td><strong>Mean (SD)</strong></td>
<td>14.8 (2.43)</td>
<td>16.0 (2.07)</td>
<td>0.848</td>
</tr>
<tr>
<td><strong>DLQI, Mean (SD)</strong></td>
<td>16.5 (7.07)</td>
<td>17.2 (8.06)</td>
<td>0.716</td>
</tr>
<tr>
<td><strong>VAS, Mean (SD)</strong></td>
<td>46.8 (29.53)</td>
<td>53.3 (25.96)</td>
<td>0.716</td>
</tr>
</tbody>
</table>

Primary Endpoint: Proportion of Patients With ≥50% Reduction From Baseline in HSSI at Week 8

<table>
<thead>
<tr>
<th></th>
<th>Placebo</th>
<th>Infliximab 5 mg/kg</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Week 8</strong></td>
<td>5.6 (n=18)</td>
<td>26.7 (n=15)</td>
<td>0.002</td>
</tr>
</tbody>
</table>
Mean Improvement From Baseline to Week 8 in Patient-Reported Pain VAS

![Graph showing mean improvement from baseline to week 8 in patient-reported pain VAS for Placebo (n=18) and Infliximab 5 mg/kg (n=15).]

Mean Improvement in DLQI Component Scores

![Graph showing mean improvement in DLQI component scores for different domains: Signatures & Feelings, Daily Activities, Leisure, Work & School, Personal Relationships, Treatment, Total.]

PGA at Week 8

![Graph showing percentage of patients with PGA at week 8, comparing Placebo (n=18) and Infliximab 5 mg/kg (n=15).]

Mean ESR and CRP at Baseline and Week 8

![Graph showing mean ESR and CRP at baseline and week 8 for Placebo (n=18) and Infliximab 5 mg/kg (n=15).]

Mean Improvement in HSSI Scores in Double-Blind and Open Label Phases

![Graph showing mean improvement in HSSI score from week 0 to 8 for Placebo (n=18) and Infliximab 5 mg/kg (n=15).]

Mean PGA Scores in Double-Blind and Open Label Phases

![Graph showing mean PGA scores from week 0 to 8 for Placebo (n=18) and Infliximab 5 mg/kg (n=15).]
Improvement after 8 Weeks of Treatment with Infliximab

Before Treatment

After Treatment

Etanercept in Hidradenitis Suppurativa

- Open label Phase II
- 10 patients
- 12 weeks therapy: 50 mg SC once weekly
- Endpoints:
  - Disease Activity Score
  - Sartorius score
  - VAS (cm)

Changes in visual analogue scale (VAS)

A Prospective Clinical Trial of Open-Label Etanercept for the Treatment of Hidradenitis Suppurativa

Etanercept 50mg/week proved ineffective in patients with Hurley's stage I and II H.S. (15 pts)
Treatment of Hidradenitis Suppurativa with Etanercept Injection
Adams D R et al Arch Dermatol 2010;146:501-504

- Double blind placebo-controlled trial
- 20 patients
- Etanercept 50mg BIW
- 12 weeks double blinded, 12 weeks open label
- No significant efficacy

Adalimumab in Hidradenitis Suppurativa
Open-Label Study
Dose: same as Crohn’s 160-80-40qow

Hidradenitis Suppurativa Severity Index (HSSI)
Preliminary Data

<table>
<thead>
<tr>
<th>Patients</th>
<th>HSSI Screening</th>
<th>HSSI Week 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>18</td>
<td>15</td>
</tr>
<tr>
<td>2</td>
<td>18</td>
<td>14</td>
</tr>
<tr>
<td>3</td>
<td>14</td>
<td>13</td>
</tr>
<tr>
<td>4</td>
<td>17</td>
<td>10</td>
</tr>
<tr>
<td>5</td>
<td>16</td>
<td>16</td>
</tr>
<tr>
<td>6</td>
<td>17</td>
<td>18</td>
</tr>
<tr>
<td>7</td>
<td>13</td>
<td>13</td>
</tr>
<tr>
<td>8</td>
<td>18</td>
<td>17</td>
</tr>
<tr>
<td>9</td>
<td>13</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td>16.22</td>
<td>14.33</td>
</tr>
</tbody>
</table>
Efficacy and Safety of Adalimumab in Treatment of Moderate to Severe Hidradenitis Suppurativa: Results from the Placebo-Controlled Portion of a Phase II, Randomized, Double-Blind Study

AB Kimball1, Y Gu2, M Okun2, G Jemec3
1Harvard Medical School, Boston, MA; 2Abbott Laboratories, Abbott Park, IL; 3Roskilde Hospital, Roskilde, Denmark
Presented at the 69th Annual Meeting of the American Academy of Dermatology, February 4-8, 2011, New Orleans, LA

Study Design

<table>
<thead>
<tr>
<th>Period</th>
<th>Placebo-Controlled Period (Period 1)</th>
<th>Open-label Period* (Period 2)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Up to 28 days</td>
<td>16 weeks</td>
</tr>
<tr>
<td></td>
<td></td>
<td>36 weeks</td>
</tr>
</tbody>
</table>

Note: Baseline period included in current analysis. Clear indicates A 0 on Panel A. aDose escalation for PGA ≥ 3 at Weeks 28 or 31.
bFrom Week 4, after 160 mg dose at Week 0, 80 mg at Week 2. cFrom Week 1, after 80 mg dose at Week 0. dFrom Week 17, after 80 mg dose at Week 16

Baseline Demographics and Clinical Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Placebo</th>
<th>ADA eow (n=52)</th>
<th>ADA weekly (n=51)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs), Mean (SD)</td>
<td>37.8 (12.10)</td>
<td>36.1 (12.50)</td>
<td>35.1 (10.69)</td>
</tr>
<tr>
<td>Female, n (%)</td>
<td>36 (70.6)</td>
<td>38 (73.1)</td>
<td>36 (70.6)</td>
</tr>
<tr>
<td>White, n (%)</td>
<td>37 (72.5)</td>
<td>36 (69.2)</td>
<td>37 (72.5)</td>
</tr>
<tr>
<td>Weight (kg), Mean (SD)</td>
<td>96.5 (24.80)</td>
<td>99.8 (26.75)</td>
<td>95.4 (22.94)</td>
</tr>
<tr>
<td>HS-PGA moderate, n (%)</td>
<td>33 (64.7)</td>
<td>35 (67.3)</td>
<td>35 (68.8)</td>
</tr>
<tr>
<td>HS-PGA severe/very severe, n (%)</td>
<td>17 (33.3)</td>
<td>16 (30.8)</td>
<td>16 (31.4)</td>
</tr>
<tr>
<td>Patients receiving p.o. doxycycline or minocycline, n (%)</td>
<td>4 (7.8)</td>
<td>6 (11.5)</td>
<td>8 (15.7)</td>
</tr>
<tr>
<td>VAS skin pain, Mean (SD)</td>
<td>57.8 (28.51)</td>
<td>53.0 (26.35)</td>
<td>52.0 (24.51)</td>
</tr>
</tbody>
</table>
Proportion of Patients Achieving Clinical Response at Weeks 2, 4, 8, 12, and 16 During Period 1

<table>
<thead>
<tr>
<th>Week</th>
<th>Placebo (n=51)</th>
<th>ADA sow (n=52)</th>
<th>ADA ew (n=51)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>0.4%</td>
<td>1.9%</td>
<td>2.0%</td>
</tr>
<tr>
<td>4</td>
<td>2.0%</td>
<td>2.0%</td>
<td>5.8%</td>
</tr>
<tr>
<td>8</td>
<td>11.8%</td>
<td>7.8%</td>
<td>7.8%</td>
</tr>
<tr>
<td>12</td>
<td>7.8%</td>
<td>7.8%</td>
<td>5.9%</td>
</tr>
<tr>
<td>16</td>
<td>5.9%</td>
<td>5.9%</td>
<td>7.7%</td>
</tr>
</tbody>
</table>

Proportion of Patients Achieving an HS-PGA of Clear, Minimal, or Mild at Week 16

<table>
<thead>
<tr>
<th>Week</th>
<th>Placebo (n=51)</th>
<th>ADA sow (n=52)</th>
<th>ADA ew (n=51)</th>
</tr>
</thead>
<tbody>
<tr>
<td>16</td>
<td>21.6%</td>
<td>23.5%</td>
<td>21.2%</td>
</tr>
</tbody>
</table>

Proportion of Patients Achieving Clinical Success (%)a

*aProportion of patients achieving an HS-PGA score of clear, minimal, or mild, with at least a two grade improvement relative to Baseline. *P<0.05, placebo vs. ADA ew. ITT, NRI. One of the 16 responders was Hurley Stage III; this patient was in the ADA ew group.

Proportion of Patients Achieving an HS-PGA of Clear, Minimal, or Mild at Week 16

<table>
<thead>
<tr>
<th>Week</th>
<th>Placebo (n=51)</th>
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<tr>
<td>16</td>
<td>21.6%</td>
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</tr>
</tbody>
</table>

Phase 3 Adalimumab: PIONEER I, II, and OLE

Study centers in US, Canada, Australia, and Europe

Main Inclusion and Exclusion Criteria

Inclusion Criteria

- Adults with a diagnosis of HS for at least 1 year prior to Baseline
- HS lesions in at least two distinct anatomic areas, one of which must be at least Hurley Stage II or Hurley Stage III
- Stable HS for at least 2 months prior to Screening and also at the Baseline visit
- Inadequate response to at least a 3-month trial of an oral antibiotic for treatment of HS (or intolerance to, or have a contraindication to, oral antibiotics for treatment of their HS)
- Total abscess and inflammatory nodule (AN) count of greater than or equal to 3 and draining fistula count of less than 20 at the Baseline visit

Exclusion Criteria

- Prior treatment with adalimumab or other anti-TNF therapy, or participation in an adalimumab trial
- Subject received oral concomitant analgesics (including opioids) for HS-related pain within 14 days prior to the Baseline visit
- Subject received prescription topical therapies for the treatment of HS within 14 days prior to the Baseline visit
- Subject received systemic non-biologic therapies for HS less than 28 days prior to Baseline visit
- Subject received systemic non-biologic therapies for HS within 28 days prior to Baseline visit
- PIONEER I only: Subject received any oral antibiotic treatment for HS within 28 days prior to the Baseline visit
Efficacy Endpoints\textsuperscript{1,2}

- Primary endpoint
  - Proportion of patients achieving Hidradenitis Suppurativa Clinical Response (HiSCR) at Week 12.
  - HiSCR defined as \geq 50\% reduction from baseline in AN (total abscess and inflammatory nodule) count and no increase in abscess or in draining fistula counts.

- Three ranked secondary endpoints
  - Proportion of patients achieving AN count of 0, 1 or 2 among patients with HS severity of Hurley Stage II at Week 12.
  - Proportion of patients achieving at least 30\% reduction and at least 1 unit reduction from baseline in Patients’ Global Assessment of Skin Pain numerical rating scale (NRS) based on 24-hour recall of worst pain at Week 12, among patients with baseline NRS \geq 3.
  - Change from baseline in Modified Sartorius Score.

HiSCR requires:

- At least a 50\% reduction in the total abscess and inflammatory nodule count (AN count) relative to baseline, AND
- No increase in abscess count, and
- No increase in draining fistula count.

HiSCR at Week 12: Primary Efficacy Endpoint

\begin{figure}
\centering
\includegraphics[width=\textwidth]{hiscr_plot.png}
\caption{HiSCR at Week 12: Primary Efficacy Endpoint}
\end{figure}

Ranked Secondary Endpoints Results at Week 12\textsuperscript{1,2}

\begin{figure}
\centering
\includegraphics[width=\textwidth]{ranked_endpoints_plot.png}
\caption{Ranked Secondary Endpoints Results at Week 12}
\end{figure}

Reduction in DLQI from Baseline at Week 12

\begin{figure}
\centering
\includegraphics[width=\textwidth]{dlqi_reduction_plot.png}
\caption{Reduction in DLQI from Baseline at Week 12}
\end{figure}

Experience with ustekinumab for the treatment of moderate to severe Hidradenitis suppurativa


- Three pts
- Significant improvement (pt #1)
- No adverse events
An Open-Label study of anakinra for the treatment of moderate to severe hidradenitis suppurativa

- 5 Patients
- Anakinra 100 mg SC daily
- At 8 weeks modified Sartorius score decreased by 34.8 points
- Physician/Pts VAS decreased by 45.8 & 35.6 points (8wks)
- DLQI decreased by 8.4 points (8wks)
- C reactive protein decreased by 16.7 points

MABp1 targeting interleukin-1alpha for moderate to severe hidradenitis suppurativa not eligible for adalimumab: a randomized study.
Kanni T et al J Invest Dermatol accepted for publication.

- Double blind, placebo controlled study
- 20 patients, Hurley I/II
- Primary end point at 12 weeks
- Pts ineligible for adalimumab
- Concomitant antibiotic allowed
- HiSCR(50% decrease inflammatory lesions ) in 60% compared to 10% in placebo
- Decrease in IL-8 and ultrasound improvements in treated pts

Study Design

- 20 patients
- Open label
- Hurley I and II (III excluded)
- Primary endpoint (HiSCR 30) week 16
- Length of study 28 weeks
- Clinical scores : HiSCR, modified Sartorius, PGA, DLQI,
Statistical Analysis

- Primary endpoint: proportion of patients with HiSCR (30% reduction in abscesses and nodules) at week 16
  - 50% reduction was an exploratory endpoint
  - Both 30% and 50% reductions were analyzed at weeks 16 and 24
  - LOCF was used for missing data
- Responder analysis
  - Non-responders/failures were any patient who discontinued due to an adverse event or lack of efficacy
- LOCF was implemented in an ITT analysis for all continuous variables (Sartorius, PGA, VAS pain, DLQI)
- An “As Treated” analysis, which included all available observations for all treated patients (no missing data imputed), was also performed.

Patient Disposition

- Screened (n=22)
- At least one dose of medication (n=20)
- Screen failures (n=2)
- Completed (n=11)
- Discontinued (n=9)
  - Adverse events (n=4)
  - Lack of efficacy (n=1)
  - Other* (n=4)

*Other includes conflicting schedule (n=2), lost to follow up (n=1), and relocation (n=1)

ITT analysis with LOCF†

*Responders were patients considered non-failures. Treatment failures/non-responders were patients who discontinued due to an adverse event or lack of efficacy by weeks 16 and 24.

Modified Sartorius Score: Change from Baseline

*P<0.001 versus baseline. †P=0.0352 for week 24 versus week 28.
Analysis based on LOCF.
Conclusion

- The data available thus far suggests that Infliximab is effective in the treatment of Hidradenitis Suppurativa
- Adalimumab is also effective, now the only FDA approved biologic for HS
- Etanercept does not appear to be effective but higher doses may be necessary
- These studies support the rationale for the use of anti-TNFα agents in HS
- Ustekinumab
- IL-1R, IL-1α, IL-1β, IL-17(Bimekizumab, UCB),IL-23 inhibition
- PDE4 inhibition (Apremilast) showing promise
Friday, March 23, 2018

6:00 a.m. - 7:00 a.m.  Continental Breakfast with Exhibitors

7:00 a.m. - 8:00 a.m.  Inflammatory Skin Diseases
Nady Hin, DO; Michael Lipp, DO & Rachel White, DO
LECOMT/Larkin Community Hospital Palm Springs Campus

8:00 a.m. - 8:20 a.m.  When the WiFi Goes Down: The EMR Doomsday Scenario Isn't That Bad
John Coppola, DO, FAOCD

8:20 a.m. - 8:40 a.m.  Recruit & Select the Best Talent
Lisa Hackney & Steven Grekin, DO, FAOCD

8:40 a.m. - 9:00 a.m.  Compliance Starts With a Voice That Smiles and a Sincere Handshake
Reagan Anderson, DO, FAOCD

9:00 a.m. - 9:20 a.m.  Patient Communication & Physician Burnout
Neha Sangwan, MD

9:20 a.m. - 9:40 a.m.  Trust, But Verify: The Golden Rule for Every Physician’s Practice
John Coppola, DO, FAOCD

9:40 a.m. - 10:00 a.m.  Reputation Management
Lisa Hackney & Steven Grekin, DO, FAOCD

10:00 a.m. - 10:30 a.m.  Break with Exhibitors

10:30 a.m. - 10:50 a.m.  You Have to Earn the Right to Be Heard
Reagan Anderson, DO, FAOCD

10:50 a.m. - 11:10 a.m.  Five Steps to Honest Conversations that Create Connection, Health and Happiness
Neha Sangwan, MD

11:10 a.m. - 11:30 a.m.  “Average” Staff is Your Achilles Heel
John Coppola, DO, FAOCD

11:30 a.m. - 1:00 p.m.  General Business Meeting/Lunch

1:00 p.m. - 1:20 p.m.  Service Excellence Standards
Lisa Hackney & Steven Grekin, DO, FAOCD
1:20 p.m. - 1:50 p.m.  
*Hypnosis*  
Reagan Anderson, DO, FAOCD

1:50 p.m. - 2:10 p.m.  
*Addressing Dysfunctional Dynamics in Medicine (and in Life!)*  
Neha Sangwan, MD

2:10 p.m. - 2:30 p.m.  
*Setting Limits in Your Practice: 3 Lines in the Sand to Draw Tomorrow*  
John Coppola, DO, FAOCD

2:30 p.m. - 3:00 p.m.  
Break with Exhibitors

3:00 p.m. - 3:20 p.m.  
*Develop Your Team Around You*  
Lisa Hackney & Steven Grekin, DO, FAOCD

3:20 p.m. - 3:50 p.m.  
*Success Without Fulfillment is the Ultimate Failure*  
Reagan Anderson, DO, FAOCD

3:50 p.m. - 4:30 p.m.  
*Mastering the Most Challenging Personalities at Work & Home*  
Neha Sangwan, MD

4:30 p.m. - 5:30 p.m.  
*Chronic Itch Clinical Cases and Management*  
Gil Yosipovitch, MD, FAAD

6:00 p.m. - 9:00 p.m.  
60th Anniversary Celebration  
Located in Coral Ballroom & Courtyard
Emerging Therapies in Atopic Dermatitis

Michael Lipp, DO, PGY3
Dr. Brad Glick, DO, MPH, FAOCD, FAAD
Larkin Community Hospital Palm Springs Campus-LECOMT/OPTI

Atopic Dermatitis
Common, yet complex inflammatory skin condition with many factors contributing to its pathogenesis. Clinical features include onset during infancy or early childhood, intense pruritus, and a chronically relapsing course.

- Acute inflammation and predilection for cheeks, scalp, and extensor sites (infants)
- Chronic inflammation with lichenification and a predilection for flexural sites (children/adults)

Often associated with asthma, allergic rhinoconjunctivitis, and food allergies (Atopic March)

Pathogenesis
Divided into three major categories:
- Epidermal barrier dysfunction
- Immune dysregulation
- Alteration of the microbiome

Each of these can be modified by genetic and environmental factors.

Treatment: General Approach
- A "proactive approach" may modify the overall disease course and prevent atopic comorbidities.
- Management includes:
  - Education
  - Gentle skin care
  - Moisturizer use
  - Topical agents
- Severe Disease:
  - Phototherapy
  - Systemic medications

Is Atopic Dermatitis the New Psoriasis?

New Targets
- PDE4 inhibition
- IL-4 antagonism
- IL-13 antagonism
- IL-31 antagonism
- IL-22 antagonism
- Janus Kinase inhibition
- Neurokinin-1 receptor inhibition
- IL-12/23 antagonism
- IL-17 antagonism
- TSLP inhibition

Topical Anti-Inflammatory Therapy: Crisaborole 2%

- Crisaborole 2% ointment is a phosphodiesterase-4 (PDE-4) inhibitor FDA-approved for the treatment of mild-moderate AD in patients ≥ 2 yrs.
- PDE-4 inhibitor ↑ intercellular cAMP → ↓ production of proinflammatory cytokines.
- Common side effect: stinging or burning (4.4%).
Transgenic mice with IL-4 in epidermis have:
- Atopic dermatitis-like lesions
- Pruritus
- Altered microbiome
- ↑ IgE levels

Key roles:
- IgE production
- Eosinophil recruitment
- Th2 differentiation (activation of IL-4Rα → STAT6)

Dupilumab (a monoclonal antibody that targets the IL-4Rα, is FDA-approved in adults for the treatment of AD)

3. GR Lee, RA Flavell: Transgenic mice which overproduce Th2 cytokines develop spontaneous atopic dermatitis and asthma. Int Immunol. 16:1155-1160 2004 15226271
Gene expression profiles of inflammatory genes at baseline vs. after dupilumab therapy
- Inflammatory genes strongly downregulated after dupilumab therapy

**Other Targets in the Pipeline?**
- TRALOKINUMAB: IL-13 Ab

**Dupilumab Trials for Children**
- Efficacy and Safety of Dupilumab in Patients ≥12 to <18 Years of Age, With Moderate-to-Severe Atopic Dermatitis
- Safety, Pharmacokinetics and Efficacy of Dupilumab in Patients 24 Months to ≤18 Years With Severe Atopic Dermatitis (Adolescent AD PRECCEP)
- Study to Investigate the Efficacy and Safety of Dupilumab Administered With Topical Corticosteroids (TCS) in Participants ≥4 to <12 Years With Severe Atopic Dermatitis (AD)

**Results**
- 209 patients received study drug
- At Week 12, significantly more patients achieved EASI-50 with lebrikizumab 125 mg (49.1%) versus placebo (32.4%); p=0.024

**Conclusion**
Lebrikizumab 125 mg Q4W led to significant improvement in patients with moderate-to-severe AD, when added to TCS, and was well tolerated.
**What we know about IL-31**

- IL-31 is a Th2 cytokine highly expressed in lesions of AD.
- Staphylococcal superantigen rapidly induces IL-31 expression in AD pts.
- IL-31R is expressed by keratinocytes, eosinophils, activated macrophages, cutaneous C nerve fibers, and dorsal root ganglia.

**Nemolizumab** (Phase 2, RCT)

- A humanized monoclonal antibody against the IL-31RA which significantly reduces pruritus in pts with moderate to severe AD.
- EASI score reduction from baseline was:
  - $-23.0\pm7.5\%$ with 0.1 mg per kilogram,
  - $-42.3\pm7.3\%$ with 0.5 mg per kilogram,
  - $-40.9\pm7.5\%$ with 2.0 mg per kilogram,
  - $-26.6\pm8.1\%$ with placebo.

**References**


**Janus Kinase – Signal Transducer and Activator of Transcription (JAK-STAT) pathway**

- Many different proinflammatory cytokines (e.g., IL-4, IL-5, IL-13, and IL-31) activate this pathway, which has profound pathophysiologic function.

**Future Therapies**

- **Baricitinib**:
  - Percentage of patients achieving EASI-50 (A) and percentage change from baseline in EASI score (B).

**T-cell Inhibitors for Atopic Dermatitis**

- TSLP is highly expressed in acute and chronic lesions of AD, but not in the nonlesional skin of patients with AD or in unaffected individuals.
- OX40 is a member of the TNF receptor superfamily. TSLP–activated dendritic cells express OX40, and are activated in the lymph nodes by OX40–TNF2 inflammatory cytokine production.

**THE FUTURE IS BRIGHT**

- PDE4 inhibition
- IL-4 antagonism
- IL-13 antagonism
- IL-31 antagonism
- IL-22 antagonism
- Janus Kinase inhibition
- Neurokinin-1 Receptor inhibition
- IL-12/23 antagonism
- IL-17 antagonism
- TSLP inhibition
THANK YOU!

Alopecia Areata
A brief review and up-to-date information on treatment

THANK YOU!

Alopecia Areata
A brief review and up-to-date information on treatment

Introduction – Alopecia Areata

- Non-scarring hair loss
- Third most common form of hair loss
- 0.1-0.2% of US
- Average lifetime risk of 1.7-2.1%
- Males = Females
- Onset: Mean Age 30
- 40% present by age 20
- Spontaneous resolution rates: 8-68%

Tosti et Al (2006)
- 2/3 with <25% scalp involvement had complete resolution for mean of 17 yrs w/o tx
- 34.6% of 51-75% hair loss recovered or developed milder disease w/o tx

Likely Autoimmune, due to T-lymphocyte interaction with follicular antigens

Current thought:
- Loss of immune privilege by Anagen bulb
- Evidence for such:
  - Oligoclonal and autoreactive T-lymphocytes are present in peribulbar inflammatory infiltrate

Pathogenesis

Clinical - Presentations

- Clinical presentations include:
  - Alopecia Areata - Patch
  - Alopecia Totals
  - Alopecia Universalis
  - Ophiasis Pattern
  - Sisaphe Pattern
  - Acute Diffuse and Total Alopecia (ADTA)

Alopecia Areata - Patch

Clinically, sudden onset of well-demarcated round or oval patches of non-scarring hair loss

Location: Scalp is MC
- In Men: Beard
- Pull Test is

Worst prognostic factors:
- Younger age at initial presentation
- Severity at Onset
- Family history
- Ophiasis Subtype
Alopecia Totalis / Universalis
- Advanced forms of Alopecia Areata
  - 5% progression rate from Patchy AA
  - Alopecia Totalis
  - Loss of all scalp hair
  - Alopecia Universalis
  - Loss of all scalp and body hair

Alopecia Areata - Ophiasis
- Band-like alopecia
  - Occipital hairline extending towards temples
  - Rarely can present at frontal hairline
  - Can be confused with frontotemporal fibrosing alopecia
  - Worst Prognosis of all clinical subtypes

Alopecia Areata - Sisaipho
- Opposite configuration of Ophiasis subtype
  - Hair loss centrally but sparing hairs at margin of scalp
  - Can be confused with androgenetic alopecia

Acute Diffuse and Total Alopecia (ADTA)
- More common in women
  - Sudden and diffuse hair loss that lasts around 3 months followed by rapid regrowth over 4-9 months
  - Favorable Prognosis but it may recur in future

Nail Changes
- Nail Pitting (MC)
- Trachonychia
- Longitudinal Ridding
- Red Lunulae

Comorbidities
- Higher incidence noted in patients with:
  - Atopic Dermatitis (MC)
  - Higher risk of severe AA phenotype
  - Autoimmune Diseases (SLE, Thyroiditis, DM, Myasthenia Gravis, Vitiligo)
- Patel et al (2017) conducted a retrospective analysis of 298 patients with AA
  - Thyroid abnormalities discovered in 20% of the pediatric patients
  - Screening should be done in those with thyroid mastication
  - Vitamin D Levels
- Tsai et al (2018)
  - Retrospective analysis showed association with severity
  - Meta-analysis of studies show association between Vitamin D deficiency and AA
Diagnosis

- Pull Test
- Sign of active disease
- Trichoscopy
- See next slide
- Biopsy
- Peribulbar lymphocytic infiltrate

Trichoscopy

- Yellow Dots
- Infundibula with sebum and keratin
- Exclamation Mark
- Hairs
- Broken hair with a thick pigmented tip
- Black Dots
- Destroyed hairs in hair follicle opening

Treatment

AA is often self-limited
Current first line treatments
- Corticosteroids (topical and intralesional)
- Minoxidil 5%
- Topical immunotherapy
Newer Treatments
- JAK Inhibitors
- PRP
- Others
  - Immunomodulators
  - Anti-inflammatory
  - Targeted therapies
  - Devices (laser, cryotherapy)

But Where Do I Start?

Current Treatments
Generally considered first-line
- Corticosteroids
  - Intralesional - first line for limited disease
  - 2.5mg/cc vs 5-10mg/cc
  - Recommendation: low concentration, higher volume
- Topical
  - Clobetasol vs Mometasone (for pediatric patients)
- Minoxidil 5%
  - Insufficient as monotherapy
  - In long term studies, mild hair growth without statistical significance

Treatment - First Line

- Corticosteroids
  - Intralesional - first line for limited disease
- Minoxidil 5%
- Topical
- Immunomodulators
- Anti-inflammatory
- Targeted therapies
- Devices (laser, cryotherapy)

- Use for maintenance with other treatments
Treatment – First Line

- Contact Immunotherapy
  - Anthralin Cream, DPCP Solution, Squaric Acid, topical anthralin
  - Usually at alopecia treatment centers
  - Overall:
    - ~50-70% rate response rate with some responses occurring after 1-2 years
    - Remission for >1 year
    - DPCP with Anthralin 0.5% ointment
      - Reserved for AU, AT
    - Issues: High dropout rates, level of evidence poor
      - Chiang et al (2014) - 50 case review using DPCP
        - 71% of AT, 56% of AU had >50% regrowth
        - 15% of responders did not respond until 1-2 years
        - DPCP + Anthralin 0.5% ointment
          - 88% vs 54.5% had >50% terminal hair regrowth after 30 weeks
        - 11 Studies with 500 patients, no RCTs, 10 ½-head studies with no tx, variety of AA severity
        - ~50% response rate overall, remission >1 year
        - High dropout rates, level of evidence poor

Treatment – First Line

- High Dropout Rates
  - Often due to expected SE
  - Patient compliance is a strong factor in decreased relapse rates (Duh)
    - Choe et al (2018)
      - Retrospective analysis, 159 pts
      - Modified DPCP treatment protocol with subclinical sensitization
        - Sensitized with 0.1% and tx with 0.01% QWeekly
        - Sensitization with an eczematous reaction may not be required for successful contact immunotherapy
        - 46 (28.9%) complete response, 59 (37.1%) partial response

New and More Recent Treatments

- JAK Inhibitors
  - Platelet Rich Plasma (PRP)

JAK Inhibitors

- JAK – STAT Pathway
  - Cytokine binding
  - JAK receptors dimerize, phosphorylated, recruit STAT molecules to activate target gene transcription
  - Mediates downstream IL-15 signaling of T-cells
  - Baseline lab monitoring: CBC, CMP, Lipids, HIV, Quant-Gold, CXR
  - Avoid in: Hx of Malignancy, Tb, Hepatitis
  - Cost: $2000-$5000 per month

JAK Inhibitors

- Tofacitinib (JAK1/3)
  - Dose: 5-10mg RD, or 11mg ER QD
  - Problems: Requires long-term immunosuppression
    - Baseline lab monitoring: CBC, CMP, Lipids, HIV, Quant-Gold, CXR
    - Avoid in: Hx of Malignancy, Tb, Hepatitis
  - Cost: $2000-$5000 per month

- Ruxolitinib (JAK1/2)
  - 20mg RD

- Oclacitinib (JAK 1)

Issues:
- Relapse once taken off medication
- Adverse Effects
  - Higher doses have unknown safety profile
- Topical route safer but unknown benefit
- Long-term likely necessary
- Longer duration and extent often has poorer response
Treatment – JAK Inhibitors

- Kennedy-Crispin et al (2016) – Tofacitinib (5mg BID)
  - 66 pts with AA, AT, AU
  - >66% showed regrowth by 3 months
  - Relapse by 8.5 weeks
  - AE: 25% with Infxn (UTI/URI)

  - 90 pts with AA, AT, AU
  - Pulsed oral CST 300 mg monthly x 3 months
  - 77% achieved clinical response

  - 10 patients, 24 weeks
  - 3/10 experienced hair regrowth

- Craiglow et al (2017) – Tofacitinib 5mg BID
  - 10/14 pts with Salt 20-100%
  - Mean SALT improvement over 2-16 months of 88%

- Castelo-Soccio (2017) – Tofacitinib 5-10mg BID
  - 8 patients age 12-19 with AU
  - All pts had >50% hair regrowth

- Bayart et al (2017) – Tofacitinib and Ruxolitinib 1% and 2%
  - 6 patients, 3AU, 2AT, 1AA
  - 75% eyelash regrowth

  - 12 pts
  - 9/12 pts with marked response

Treatment – PRP

- Advantages:
  - Ability to induce longer disease remission
  - Regrow pigmented hairs from beginning of hair regrowth
  - Safe – autologous material
  - No lab monitoring, drug interactions, side effects

- Trink et al (2013) - Double blind placebo, half head x 3 months
  - Significant improvement monthly PRP(60%) vs ILK(27%) vs placebo

- Singh (2015) – Monthly x 6 months
  - 19/20 with regrowth


- RCT, 90 patients with no treatment for 3 months before therapy.
- 3 groups:
  - 1 group w/ 5% BID + PRP injections Q4 weeks
  - 2 groups w/ PRP injections Q4 weeks
  - PRP more effective than minoxidil in same treatment period
  - Showed reduction in short vellus hairs

Existing treatments with possible utility

- Immunomodulators
- Anti-Inflammatories
- Anti-Inflammatory Steroids
- Other Steroids
- Corticosteroids
- Nonsteroidal Anti-inflammatory Drugs
- Prostaglandin Analogs
- Anti-histamines
- Low Dose Naltrexone
Treatment – Immunomodulators

- Systemic Corticosteroids
  - Pulsed recommended if deciding on this route
  - 41 Studies, various protocols with IV/IM/PO q-monthly
  - Route was not statistically significant
  - RCT Study
  - Complete response in 40% on CST, 3% in placebo

- Mycophenolate Mofetil
  - Systemic: 500mg BID – 1500mg BID
  - Topical: 2% Cream

- Methotrexate
  - Comparative Study, MTX + Prednisone vs Prednisone alone
  - 5/14 pts had >50% hair growth with combo MTX + Prednisone

- Cyclosporine
  - Ranges from 25-76.7% success rate >50% regrowth
  - One uncontrolled study – 45.4% of 25 pts showed sig. regrowth

- Sulfasalazine
  - Pilot study
  - 43% of 14 pts showed complete regrowth
  - 66% showed no signs of relapse after treatment discontinuation
  - 33% relapsed after 2.5 months

- Azathioprine
  - Prospective Study (Vano-Galvan et al 2015)
  - Azathioprine dosage 2.5 mg/kg/day
  - 14 patients with AU, recalcitrant to oral CST and DPCP
  - Response in 6/14 patients
  - Response in 4.7 months response
  - Relapse: 2 patients after 2.5 months, remaining 4 persistent

- Prostaglandin Analogs (Lee et al, 2015)
  - Studies have wide range of variable therapeutic effect
  - Consider for eyebrows
  - Lee et al (2015)
    - Tac + Latanoprost > Tac alone
    - 45% vs 0% improvement

Treatment – Anti-Inflammatories

- Simvastatin/Ezetimibe – 40/10mg QD
  - 29 patients, 40-70% SALT
  - 73% responded after 16-24 weeks (>24% regrowth)
  - Other study, 82.4% showed no improvement

- Antihistamines (Lee et al, 2017)
  - Cohort Study
  - DPCP + Fexofenadine > DPCP monotherapy

- Low-dose Naltrexone
  - 1-4.5mg QD
  - Possible use for anti-inflammatory

Targeted Therapies

- Ustekinumab
  - Guttman-Yassky E Et Al (2016)
  - 3/9 pts with complete response after 12 months
    - 1 had AI

- Apremilast
  - Liu et Al (2017)
  - 9 patients (1 AA, 8AU)
  - Duration of disease 23.3 years
  - None showed improvement over 3-6 months

- Secukinumab
  - RCT was terminated in 2017 due to low enrollment

- Abatacept
### Treatment - Targeted Therapy

- **Abatacept (CTLA4 Agonist)** – 125mg SC weekly
- **SALT 30-100% (3/15 improved)**
- 1/15 pts with 98% regrowth after 6 months
- 2/15 with 23% regrowth

### Devices

- **Superficial Cryotherapy**
- **Carboxytherapy**
- **Excimer Laser**
- **Fractional Photothermolysis**
- **Fecal Transplant**

### Treatment - Devices

- **Superficial Cryotherapy**
  - Comparative Study (Faghihi and Radan 2014)
  - 80% vs 91.5% (clobetasol)
  - Jet cryotherapy
  - 11 recalcitrant AA patients
  - 5 excellent response, 3 satisfactory
  - Most effective at 2 weeks or less
  - Half head study (Jun et al, 2017)
  - Superficial cryotherapy showed increased hair thickness and eyebrow density
  - Tx: Each patch 3-4 times for 2-3 sec q 2 weeks
  - 11 of 15 responded, with maintenance 1 month.
  - Improvement by 1.6 x of terminal hair on treated side
  - SALT score of 40% improvement vs 9.6%
  - Rationale: Readily available at most offices, inexpensive, no systemic side effects

- **Excimer Laser**
  - 256nm Excimer
  - Pilot study
  - 42 recalcitrant patches in 8 patients
  - Twice per week for max of 24 sessions
  - 50mJ/cm² less than MED
  - Complete regrowth in 13/42 lesions, excellent in 6/42
  - Presence of Ankylosing Spondylitis had an unfavorable prognosis
  - Rationale
    - Minimal side effects, ideal for pediatric patients

- **Carboxytherapy**
  - Doghaim et al (2018)
  - 80 pts (40 AA, 40 AGA), 4 groups (1a, 1b, 2a, 2b)
  - Placebo was intradermal distilled water
  - Injection: 30g Needle, 2mL CO2 per injection site
  - Significant improvement
  - 3 months after last session
  - SALT from 9 -> 5.7
  - Control group: 12.5 -> 16.0
  - Before, after 6 sessions, then 3 months after last session
  - Rationale: Inexpensive

- **Excimer Laser**
  - 308nm Excimer
  - Pilot study
  - 42 recalcitrant patches in 18 patients
  - Twice per week for max of 24 sessions
  - 50mJ/cm² less than MED
  - Complete regrowth in 13/42 lesions, excellent in 6/42
  - Presence of Ankylosing Spondylitis had an unfavorable prognosis
  - Rationale
    - No significant difference in a heterogeneous patient population
    - But other studies have reported some improvement
    - Chloroquine (2011)
    - 17 patients, 14.6% on Cx2
    - As above, except 10 patients (6.92%)
    - 12/13 reported decrease in SALT

### Treatment - Devices

- **Fractional Photothermolysis**
  - 17 patients, 14.6% on Cx2
  - As above, except 10 patients (6.92%)
  - 12/13 reported decrease in SALT
Treatment - Devices
- Fecal Microbiota Transplant!
- Rebello et al (2017)
- PLA
  - Patient with recalcitrant AA (at age 56) achieved 50% regrowth of head scalp
  - 90% regrowth of beard and arm
- PRP
  - Patient with history of C. Diff.
  - Achieved 80% regrowth of head scalp
- ILK
  - Patient with C. Diff.
  - Achieved 80% regrowth of head scalp

But what does this mean?
- JAK inhibitors
- Immunomodulators
- Various devices

References

Conclusion
- Alopecia Areata can and almost always will resolve... eventually
- Initial treatment should follow an algorithmic approach with corticosteroids, minoxidil, immunotherapy
- Widespread and recalcitrant cases
- JAK inhibitors, PRP, immunomodulators, and various devices
- Be aware of the comorbidities of AA
  - Thyroid Disorders
  - Vitamin D deficiency
  - Anemia

Thank you!

Granuloma Annulare: A Brief Review and Up-to-date Information on Treatment
RACHEL WHITE, DO, PGY-3
LARK COMMUNITY HOSPITAL PALM SPRINGS CAMPUS – LECOM/OPS
**Background**

- Benign, often self-limited granulomatous skin disease
- Clinically – pink annular plaques with raised border and central clearing
- Histologically – interstitial or palisading granulomas, degenerated collagen and mucin
- Most common in children and young adults
- More common in females

**Pathogenesis – Mechanisms**

- Unknown mechanism; theories originate from histologic findings
- Original theory – immune-mediated type III hypersensitivity reaction → vasculitis
- Recent theory – cell-mediated delayed-type IV hypersensitivity reaction to unknown antigen
- Sensitized Th1 lymphocytes → macrophages → proinflammatory cytokines & collagen-degrading enzymes → tissue injury
- Other theories – injury to dermal elastic fibers

**Pathogenesis – Inciting Factors**

- Trauma/foreign body – insect bite, tuberculin skin testing, vaccinations, subcutaneous immunotherapy for allergies, tattoo, isomorphic response
- Infectious – viruses (Hep B, Hep C, EBV, HIV); Borrelia species
- Drugs – TNF-α inhibitors, allopurinol, topiramate, gold therapy
- Genetic – familial cases including identical twins, HLA-Bw35 (generalized GA)

**Associated Disorders**

- Diabetes
  - Definitive evidence lacking and conflicting data
- Dyslipidemia
  - Evidence shows link with adult GA
- Malignancy
  - No causative relationship
  - Seen in atypical GA variants
- Most common malignancy is lymphoma
- Thyroid disease – autoimmune
- HIV – atypical variant

**Clinical – Localized GA**

- Most common form
- Skin-colored to pink erythematous annular or arcuate plaques with raised border and central clearing
- Discrete papules at periphery
- Location – wrists, ankles, dorsal hands and feet
- Asymptomatic
- Onset – children, young adults
- ~50% patients have >1 lesion

**Clinical – Disseminated/Generalized GA**

- Widespread skin-colored to pink erythematous papules and plaques of varying sizes
- Location – trunk and extremities
- Asymptomatic or pruritic
- Onset – adulthood
- Associated with HLA-B35
Clinical – Deep/Subcutaneous GA

- Large skin-colored nodules, overlying skin uninvolved
- Location – scalp, buttocks, extremities
- Painless
- Onset – children <6 yo

Clinical – Perforating GA

- Yellow umbilicated papules with scale crust and focal ulceration
- Location – localized on extremity or widespread
- Asymptomatic, pruritic, or painful
- Onset – children, young adults

Clinical – Patch GA

- Symmetric annular patches
- Location – proximal extremities, dorsal feet
- Onset – adults

Pathology

- Lymphohistiocytic infiltrate forming interstitial or palisading granulomas, degenerated collagen, and mucin
- Both patterns in localized and generalized GA
- Patch GA – interstitial
- Subcutaneous GA – palisading
- Perforating GA – transepidermal elimination of mucin and degenerated collagen fibers

Differential Diagnoses – Annular Lesions

- Annular elastolytic giant cell granuloma (actinic granuloma)
- Interstitial granulomatous dermatitis
- Tinea corporis
- Annular lichen planus
- Bythoema annulare centrigum
- Sarcoïdeses
- Nodular terrytary syphilis
- Mycosis fungoids
- Borderline leprosy

Differential Diagnoses

- Generalized GA
  - Arthropod assault
  - ID reaction
  - Interstitial granulomatous dermatitis
  - Secondary syphilis
  - Desmoplastic sarcoma
  - Desmoplastic syringoma
  - Histocytozomas

- Subcutaneous GA
  - Rheumatoid nodules
  - Epithelioid sarcoma
  - Sarcoïdeses
  - Deep fungal infection
  - Tuberculosis sarcoma

- Perforating GA
  - Reactive perforating collagenosis
  - Perforating folliculitis
  - Eczema perforans tegaphaetis
  - Calcinosis cutis
  - Perforating gout
  - Sarcoïdeses
  - Meliobium cutis tumora
Diagnosis and Work Up

- Clinical diagnosis
- Punch biopsy with H&E for atypical presentations
- Lipid panel in adults
- Review signs/symptoms/risk factors for diabetes, HIV
- Age-appropriate cancer screening in elderly patients with atypical presentations

Treatments – Overview

- No treatment necessary – often self-limited
- 50% localized GA resolve within 2 years
- Generalized GA more persistent – 25% courses >5 years
- Resolves without scar
- Treatment dependent on type, symptoms, cosmesis

Treatments – Localized GA

- First-line
  - High potency corticosteroids topical +/- intralesional
  - Clobetasol 0.05% cream BID x 2-4 w
  - Triamcinolone acetonide 2.5-10 mg/cc q 6-8 w
- Others (limited evidence)
  - Cryotherapy
  - Topical calcineurin inhibitors – tacrolimus, pimecrolimus
  - Phototherapy – PUVA, UVA1, NB-UVB, PDT
  - Topical dapsone
  - Intralesional IFN-γ
  - Imiquimod

Treatments – Generalized GA

- First-line
  - High potency topical/intralesional corticosteroids
  - Topical calcineurin inhibitors
  - Tacrolimus 0.1% ointment BID x 6 w
  - Pimecrolimus 1% cream
  - Phototherapy
    - UVA1 – high cumulative doses most effective = 1770 – 1840 J/cm²
    - PUVA – oral or bath PUVA with cumulative dose 60.4 J/cm²
    - Narrow-band UVB – cumulative dose 47.7 J/cm² → 54% complete/partial response
    - Photodynamic therapy

Treatments – Generalized GA

- Systemic treatment
  - Antimalarials – first line
    - Hydroxychloroquine – 3 – 6 mg/kg/d
    - Chloroquine – 3 mg/kg/d
    - Tnf-a inhibitors
      - Adalimumab – 80 mg or weekly 0.4 mg every other week 50
      - Infliximab – 5 mg/kg at weeks 0, 2, 6 every month IV
    - Isotretinoin – 0.5 – 1 mg/kg/d
    - Dapsone – 100 mg/d
    - Pentoxifylline – 400 mg TID
    - Nicotinamide – 500 mg TID
    - Cyclosporine – 3-4 mg/kg/d
    - RO4 (rifampin, dapsone, minocycline)
    - Vitamin E oral – 450-600 IU daily
    - Fumaric acid esters – used in Europe
    - Other case reports: dapsone, clofazimine, defibrotide, methotrexate, hydroxyurea, alkylating agents (chlorambucil), citral, calcitriol, defibrotide, etretinate

Treatments – Lasers

- Pulsed dye laser
  - Localized or generalized GA
  - >50% improvement in localized GA
  - <1/3 <50% improvement in generalized GA
  - Fractional photothermolysis
  - Case reports – significant improvement in height and diameter
  - Excimer laser – complete remission in patients
Treatments - Upcoming

- Radial pulse therapy – indirect mechanotherapy
  - 100 shots, pressure 2.5 bars, frequency 4 pulses/sec
  - Positive effect in all treated GA plaques

Treatments – Subcutaneous GA

- Treatment not indicated
- Surgical excision – recurrence common
- Local hyperthermia – case report
  - 44°C for 30 min, improvement after 10 treatments

Conclusion

- Benign, often self-limited disease
- Work up may include history/labs for diabetes, dyslipidemia, malignancy, and HIV
- Pathogenesis involves possible cell-mediated delayed type IV hypersensitivity reaction to unknown antigen
- Many treatments available from topical and light-based therapy to systemic and biologic medications
- Prospective double blind randomized control trials need to be performed for improved evidence-based treatment

References


Thank you!

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When the WiFi Goes Down: The EMR Doomsday Scenario Isn’t That Bad
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Parks Dermatology Center
Ormond Beach · Port Orange · Palm Coast · Orange City

• No Conflicts or Disclosures

Understand What Type of System, Connection, and Service You Have
• Cloud Based vs Server Based
• Connections Speeds of Your System
• Type Of Connection?
  – Cable, DSL, Satellite, Fiber?
• Quality of Your IT Team
  – Paying by the hour, internal team, or locally outsourced contract

Protocol in Place
• Minimum of one person in every office that:
  – Has emergency/direct contact info for Internet Provider & IT Team
  – Knows location and how to reset router
  – is responsible for tracking power/internet capability after storms
  – Coordinates appointments for IT updates/hardware replacements that doesn’t interfere with your work schedule

Back Up to the Back Up
• Several of your Ipads 4G LTE Capable
• WIFI Hot Spot Devices
• 5G on the Horizon

Uggghhh – Back to Paperwork
• What We Do In A Pinch
  • Back to The Template Forms
  • Keep Till End of Day
  • Input Back Into EMR at end of day/lunch and pay a little overtime
Recruit and Select The Best Talent

WHY ???

You are not just recruiting employees, BUT …
sowing the seeds of your reputation

Imagine….

• The Best Team Ever …
• How did it feel? …
• What would it be like? …

Three Simple Truths

• If you begin with “who” rather than “what,” you can more easily adapt to a changing world.
• If you have the right people on the bus, the problem of how to motivate and manage people largely goes away.
• If you have the wrong people, it doesn’t matter if you have the right direction – you still won’t have a great company.

+ “Good to Great”
  Jim Collins

What percentage of departing employees express dissatisfaction before quitting?

Only 25%
The Cost of Turnover …

- 16-20% of annual salary for high turnover positions
- 20% for midrange positions
- Up to 213% of annual salary for highly educated positions

Plus … The Real Cost …

- Screening Costs
- On-boarding
- Lost Productivity
- Lost Engagement
- Customer Service & Errors
- Training Costs
- Cultural Impact

Source – Zane Benefits, February, 2016

Screening Costs

On-boarding

Lost Productivity

Lost Engagement

Customer Service & Errors

Training Costs

Cultural Impact

Where to Begin … Sourcing !

- Referrals ... Best Source Ever
- Formalize the process
- Online Sources produced 86% of interviews and 72% of hires in 2016
- Track where your best are coming from and measure the outcomes

Source: SHRM, Employee Referrals Remain Top Source for Hires (June 23, 2017)

- Referrals … Best Source Ever
- Formalize the process
- Online Sources produced 86% of interviews and 72% of hires in 2016
- Track where your best are coming from and measure the outcomes

SCREEN

- Use Peer Interviewing
- Use a Validated Tool
- Ask for References
- Background Checks

You can train Skills …

You can’t train attitude ….

Hire as is … if you get any change … It’s a bonus

What makes employees stay and work hard for your company?

- Employees join a company for rational motives:
  - Better compensation
  - Benefits
  - Career Opportunities

They stay and work hard for emotional ones.

People

“The organization will never be what the people are not.”

+ Price Pritchett

“The Ethics of Excellence”
Compliance

Reagan Anderson
DO, FAOCD, FAAD, FASMS, CAQ Mohs, MPH, MCS

Compliance Starts With
A Voice That Smiles
And A Sincere Handshake

Developing,
Measuring,
Making Reproducible
Your Brand
via
Yourself
and
Your Staff
Consistency of Message, of Medicine, of Intent

Policies and Procedures in Place
Trust, But Verify. The Golden Rule For Every Physician’s Practice

John Coppola, DO  FAOCD, FAAD
Assistant Clinical Professor - FSU College of Medicine
Faculty - Orange Park Medical Center Dermatology Residency
Parks Dermatology Center
Ormond Beach - Port Orange - Palm Coast - Orange City

• No Conflicts or Disclosures

Doveryai, No Proveryai

Prescriptions & Notes

• MA's and desk nurses input into my que in the EMR and I always review prior to sending
• MA's input data for all of my notes, but MD/DO always should review content and billing level prior to finalizing

Statistics

US Chamber of Commerce
• 75% of all employees steal at least once
  – Med samples, cosmetic products, cash, hours worked
• Average time it takes an employer to catch a fraud scheme is 18 months
• The average small business loses up to 20 cents per dollar to theft/fraud

• Keep Track of Your Cosmetic Inventory
  – How Are Return Items Handled?
  – Does Everything Stay Locked Up?
    • Pull out what you need for the day & account for what you use
• Develop A System To Track Cash Co-Pays/ Cash Only Patients Coming Into Your Office
  – Paper Super Bill?
Audit, Audit, Audit

- Office Managers Regularly Audit Front Staff Collections/MA Supervisors
- Practice Manager Regularly Audits Your Office Managers
- Accountant Audits Your Practice Manager
- Change Your Accountant Every 5 Years?
- Use Someone External From Your Accountant Annually
  - We Use Allergan’s Practice Consultants for Financial Benchmarking

Items You Should Always Verify

- Confirmation of:
  - Medical License Renewal
  - Taxes Submitted and Paid
  - Rent/Mortgage
  - Insurance Contract Renewals
  - AOA/AMA CMEs submitted

Cameras

- Very Little Pushback, Easy To Implement
  - Break Room
  - Near Storage Closet
  - Front Desk/Check Out Area
Reputation Management

What a ride ....

What Happens When It Is A Bad Experience ??
- 93% of customers will refuse to do business with a company after 3 or fewer bad experiences
- A dissatisfied customer will tell between 9-15 people about their experience
- People are twice as likely to talk about bad customer service experiences than they are to talk about good ones.
- 37% of customers have posted a negative comment online about a bad customer experience.

Why Does It Matter ?? ... Reputation Effect
- 99% of consumers read online reviews for local businesses in any
type of industry.
- 85% of consumers trust online reviews as much as personal recommendations
- 66% of consumers say that online reviews are their most trusted recommendation.
- 23% of consumers won't do business with you if you are three stars.
- 87% - for five star rating
- Consumers read an average of 7 reviews before trusting a business – up from 6 last year.

- “The opinions expressed in this presentation and on the following slides are solely those of the Presenter and not necessarily those of The American Osteopathic College of Dermatology. The Presenter has no relevant nonfinancial relationship(s) to disclose.

- 67% of people spend money after getting recommendations from their friends on online communities like Facebook and Twitter.
- Happy Customers tell 2-3 people about their experience
- Happy customers who get their issue resolved tell 4-6 people.
- 33% of consumers would rather be treated well than have their problem resolved.

- 90% of consumers read online reviews for local businesses in 2017
- 85% of consumers trust online reviews as much as personal recommendations
- 66% of consumers say that online reviews are their most trusted recommendation.
- 52% of consumers won't do business with you if you are -- three stars;
- 87% - for five star rating

- Consumers read an average of 7 reviews before trusting a business – up from 6 last year.

- Sources: BrightLocal, "Local Consumer Review Survey" and Trustpilot: "When Bad Reviews Happen" - 2017
Net Promoter Score (NPS)

What Can You Do ????

- Ignore at your Peril.
- If you aren’t managing the online review process, your competitor likely is.
- Don’t try to game the system
- Learn, learn, learn- benchmark others

THE KEY LESSON ...

Patients want to be treated like people, and they will use their collective voices to elevate providers to do a better job of adhering to this principle.

....Help is a good word ....

- Ask patients to leave positive reviews — use the feedback to improve!
- Feedback is a gift — ask!
- Ask your new patients how they heard about you!
- Find someone on your team who can help you!
- Begin a systemized process of surveying!

...Help is a good word ....
Earn

Reagan Anderson
DO, FAOCD, FAAD, FASMS,
CAQ Mohs, MPH, MCS

You HAVE To
EARN The Right
To Be Heard

Having The
Title
“Doctor”
Only Gets You
Into The Room
Being A
“Doctor “
Means
NOTHING
Outside of Medicine

Being
“Osteopathic”
In All Aspects of
Medicine, Life, and Business
“Average” Staff Is Your Achilles Heel

John Coppola, DO, FAOCD

Do Doctors Make Great Managers?
- Leadership Seminars
- Human Resource Tools Updates
- Management Philosophy Books
- Billing Update Courses
- Motivational Speaking & Team Building Exercises

Cost of Employee Turnover
- 16% of annual salary for high-turnover, low-paying jobs (earning under $30,000 a year). For example, the cost to replace a $10/hour retail employee would be $3,328*
- 20% of annual salary for midrange positions (earning $30,000 to $50,000 a year). For example, the cost to replace a $40k manager would be $8,000*

From Our Experience, Average Employees:
- Are more likely to leave and cost you turnover
- Decrease morale
- Make more mistakes exposing you to medical, legal, and financial mistake

Have a set & defined hierarchy of responsibility for your managers and supervisors
- Don’t micromanage your managers
- Structured and well defined system for reviewing all of your employees
  - “Sacred Cows”
- Clearly organized and regularly updated employee manual
• Reduce, Reduce, Reduce Employee Turnover
  – Clearly Defined Job Description
  – Driving? Switching Positions?
  – Background and Drug testing Up Front
  – Check Social Media First
  – Working Interviews
  – Personality Profiling
  – 90 Day Probationary Period

_Hire The Right Personality, Not The Right Experience_
Service Excellence Standards

I Am the Patient Experience

HOSPITALITY
- LEARN FROM LEADERS IN INDUSTRY
- LOOK AT PRACTICE FROM A HOSPITALITY FOCUS
- WHAT CAN YOU CHANGE TO ADAPT TO WHAT YOUR PATIENTS EXPECT?

EMPATHY & ENTHUSIASM
- PUT YOURSELF IN THE PATIENT'S SHOES
- SCRIPT IN MESSAGES
- ENTHUSIASM SHOWN IN BODY LANGUAGE, TONE AND FACIAL EXPRESSIONS
- BE JOYFUL – YOU ARE CHANGING LIVES

SERVICE EXCELLENCE FROM THE H.E.A.R.T
- HOSPITALITY
- EMPATHY & ENTHUSIASM
- ATTITUDE
- RESPECT
- TIMELINESS
ATTITUDE
- ATTITUDE IS EVERYTHING
- A THANKFUL ATTITUDE GOES MILES
- YOU CAN'T TEACH A POSITIVE ATTITUDE OR TO TEACH SOMEONE TO SMILE
- THIS IS WHAT WILL BE REMEMBERED …

RESPECT
- THE PATIENT ALWAYS DESERVES RESPECT
- ASK, LISTEN, RESPOND AND ADAPT
- SHOW RESPECT TO YOUR INTERNAL CUSTOMERS ALSO
- RESPECT IS EARNED

TIMELINESS
- BE ON TIME
- BE TRUTHFUL ON EXPLAINING DELAYS
- OUR PATIENT’S TIME IS AS VALUABLE AS YOURS
- THE EXPECTATION OF VALUE IS RELEVANT TO TIME

10 minutes a day …

LEARN FROM HOSPITALITY….
- HOW TO BUILD A CUSTOMER SERVICE EXCELLENCE CULTURE IN 10 MINUTES A DAY
- A SOLUTION TO IMPROVE COMMUNICATION
- AN EFFECTIVE WAY TO PARTICIPATE AS A MEDICAL LEADER—KEY TO PARTICIPATE
- PROVIDES A DAILY REINFORCEMENT TO WHY WE ARE DOING WHAT WE SET OUT TO DO

TIPS TO CONDUCT A SUCCESSFUL HUDDLE
- BE CONSISTENT
- KEEP THE TIME UNDER 10 MINUTES
- REVIEW A PATIENT EXPERIENCE STANDARD DAILY
- TALK ABOUT THE "WHY"
- ROTATE LEADERS
- BRING REAL TIME EXAMPLES TO THE HUDDLE
- CELEBRATE WINNING PLAYS—WOW STORIES
- COMMUNICATE DAILY OPERATIONAL TO DO’S
- BUILD RELATIONSHIPS
- START THE DAY OFF TOGETHER
The patient needs an experience, not an explanation.
Hypnosis
Reagan Anderson
DO, FAOCD, FAAD, FASMS, CAQ Mohs, MPH, MCS

Structure and Function Are Reciprocally Interrelated

Sticks and Stones
Victimhood is a daily choice.

“Be The Change You Wish to See in the World”
Ghandi

“The ultimate measure of a man is not where he stands in moments of comfort and convenience, but where he stands at times of challenge and controversy.”
Dr. Martin Luther King, Jr.
Setting Limits In Your Practice:
3 Lines To Draw In The Sand
Tomorrow

John Coppola, DO  FAOCD, FAAD
Assistant Clinical Professor - FSU College of Medicine
Faculty - Orange Park Medical Center Dermatology Residency
Parks Dermatology Center
Ormond Beach - Port Orange - Palm Coast - Orange City

• No Conflicts or Disclosures

For Your Patients
• Set Rules For Your Patients And Stick To Them
  – How Late Can Patients Be To Their Appointments?
  – What Happens To Patients That No Show?
  – Verbally Abusive Patients to your Staff?

For Your Drug & Device Reps
• Reps are a Necessary, Useful, But Sometimes Disruptive Part of the Day For Both You, Your Mid-Levels, and Your Staff
  – We Set Limits To How Many Per Day
  – We Set Time Restrictions Before Lunch and End of Day
  – We ask reps not sampling products to limit their visits unless there is new information available

For Your Employers
• Employee Physicians Should Know What They Are Paying For
  – Always Try to Negotiate Into Your Contract Employer Obligation to Provide Monthly Statements of a Breakdown Of Assigned Overhead To You Unless You Are On a Fixed Percentage of Collections
  • If You Are, Then Find Out What Their Rate of Collections Are!
The strengths of physician leadership:

- You are already a leader.
- You have a large capacity for complexity.
- You are naturally process-improvement “engineers”.
- You know how to leverage expertise.
- You want to remain relevant.

Me and My Strengths:....

<table>
<thead>
<tr>
<th>Concept</th>
<th>Reality</th>
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<tbody>
<tr>
<td>• What do I believe my strengths to be ???</td>
<td>• What would my team perceive my strengths to be ?</td>
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</table>

- You can do what I cannot do.
- I can do what you cannot do.
- Together we can do great things.

- Mother Theresa

EXCELLENCE

POTENTIAL

ROOM FOR IMPROVEMENT
Human Investment Planning

Rank the people you manage/work with starting with your best performer first and concluding with your least effective performer.

Rank the people you manage/work with starting with the person with whom you spend the most individual (one-on-one) time and concluding with the person with whom you spend the least of your time.

Managing means making the strengths of people effective. Neither the welfare approach, nor the personnel management approach, nor the control and firefighting approach addressees themselves to strength, however.

People are weak; most of us are pitifully weak. People cause problems, require procedures, create chores, and people are a cost and a potential “threat.” But these are not the reason why people are employed. The reason is their strength and their capacity to perform.

Peter Drucker, “Management: Tasks, Responsibilities, Practices,” 1973

Three Key Principles of Strength Development

- Discover the activities that people do well, are interested in pursuing, and are passionate about; and encourage them to do more.
- Discover the activities that people do poorly, cannot change, or with great effort only change slightly; and draw up a strategy to manage these limitations.
- People’s strengths flourish when they experience the benefits of good relationships.

Challenges of Managing Highly Talented People

- Talk about your mission
- Manage individually
  - Understand uniqueness
  - Know goals, aspirations and needs
  - Accommodate “prima donna” behavior
- Empower them
  - Let them make choices, decisions
  - Don’t stifle them or slow them down
  - Don’t believe that you must stay out in front of them
  - Equipment and structure
- Feed their ego

Key Leadership Questions

- Do we make improving our talent pool and retaining talent one of our top three daily priorities?
- Have we compromised our standards?
- Have we strategically created a reputation for treating each employee individually, treating them fairly, and treating them well?
- Have we branded our environment as one of a high-performance work team that celebrates winners and where work is fun?
- Does each employee know they are appreciated and valued every day?
Success

Reagan Anderson
DO, FAOCD, FAAD, FASMS,
CAQ Mohs, MPH, MCS

“Success Without FULLFILLMENT Is The Ultimate Failure.”
Tony Robbins

Bucket-List
For Medicine
For Life
Deathbed Thoughts

You Have NOT ARRIVED

Other residency, marriage, kids, Board Certification, funding > than $100, retirement, acclaim, recognizion, grandchildren, new car, old car, new skill, independence, good lab results, paying off your mortgage, learning a new language, 1 year of marriage, 50 years of marriage, falling in love, finally getting divorced, making America great again, overcoming alles, getting a good parking spot, not eating that donut, eating that extra donut, actually remembering to put the toilet seat down for once in your life...
Chronic Itch Clinical Cases & Management
Gil Yosipovitch MD FAAD
Professor & Director Miami Itch Center

Outline
- Understanding itch of different types
- Pathophysiology of itch in PN and its treatment
- Atopic itch and its new management
- Itch without rash and its management
- Neuropathic itch

Important Questions to Ask an Itchy Patient
- Duration: years/weeks/days
- Severity
- Localization; generalized/localized/
- Periodicity; paroxysmal, continuous, occurring in short bouts, nocturnal
- Affect on sleep
- History of itch in other personal contacts
- Factors that exacerbate itch; heat, water, dryness
- Factors that alleviate itch (drugs or cooling agents)
- Drugs (opiates, aspirin, penicillin, antimalarials)
- History of atopy
- Travel history

Disclosures
- Advisory Boards: Trevi, Pfizer, Sanofi, Menlo, Galderma, Sienna
- Consultant: Opko LEO, J&J, Menlo, Novartis
- PI; Tioga, Roche, Pfizer, Allergan
- Funded.; GSK, LEO Foundation, Pfizer, Sun Pharma

Yosipovitch & Bernhard NEJM 2013
PN in African Americans and AD

What Causes Itch

Population coding
Molecular specificity of receptor activation
TSLPR NPRA
TSLP NPPB GR
H1, H4
IL-33
IL-31
IL-31R
Mrgprs

Neurogenic inflammation, attraction of inflammatory cells
Scratching
Axiotomy of epidermal peripheral nerve endings
Activation of retrograde signaling pathways

Prurigo Nodularis

Various Forms of Prurigo
The first three may develop subsequently

Treating Prurigo Nodularis

Various forms of Prurigo

Step 1
Step 2
Step 3
Step 4

Various Pharmacological Agents

Topical corticosteroids
UVA/UVB phototherapy
Topical calcineurin inhibitors
Topical calcineurin inhibitors
Topical calcineurin inhibitors
Immunosuppressants
Thalidomide
NK-1 inhibitors
m- and kappa opioid receptor antagonists
Antidepressants
Gabapentinoids
Topical KAL, Capsaicin
Topical KAL, Capsaicin

Zeidler et al. Acta Derm Venereol. 2018

Zeidler et al. Acta Derm Venereol. 2018
Targeting the peripheral neural system

- Topical ketamine 5-10% lidocaine 5% and amitryptiline 5% in lipoderm base (targeting ion channels)
- TRPV1 antagonists

Stull et al. Exp opin pharma 2016
Gibson et al. Plos One 2015
Poterucha et al. JAAD 2013

Topical Ketamine

Ketamine is a N-methyl-D-aspartate (NMDA) antagonist used for general anesthesia that has been formulated and studied as a topical agent, mainly for the management of neuropathic pain.

Adverse effect

- Serious:
  Recent report of encephalopathy after extensive use all over the body in an elderly patient
  Recommendation not to apply for whole body only severe itchy areas
- Common
  Burning
- Other
  Allergy to lidocaine

Cardis et al. JAMA Dermatol. 2016. Lee et al, JAAD 2017

AD Clinical Features

Follicular Eczema
Papules

Discoid Eczema

Xerosis (dry skin) + Lichenification

Infrauricle Fissure Stages
High Association of Infrauricle Fissure and VAS Itch Scores In Follow Up

Kwatra et al. J. Amer Acad Dermatol 2012

Atopic Dermatitis

Pruritic Psoriasis

Healthy

How to Reduce Itch of AD

• Avoid alkaline bar soaps that aggravate itch
• Low pH moisturizers
• Topicals with Hypochloric acid
• Crisaborole
• Topical Immunomodulators


Mechanisms of Itch induction by ↑pH in Atopic Eczema and Impaired Barrier
Topical PDE4 Non Steroidal Inhibitor for Itch of AD

PDE 4 inhibitor Crisaborole
- Rapid reduction of itch in AD in first 2 days

Other Topical antipruritics in the market
- Pramoxine 1-2.5%
- Strontium 4%
- Menthol 1-2%

Double Layer Wet Pajamas
- Effective for itch reduction
- Moisturizer with low potency steroid with silicone base on top a wet layer and dry layer on top
- Associated with the recovery of epidermal barrier.
- Induces clinical improvement by the release of restoration of intercellular lipid lamellar structure.

The end of the antihistamine era for AD
- Usually not effective for chronic itch

Capillary Network
- TEWL
- Poor barrier
- Nerve plexus
- Block stratum corneum itch signaling
  - Nerve fibers & receptors
  - Secretion of NGF
  - Pruritogenic cytokines
  - Changes in pH, humidity, temp & mites & Staph

Block stratum corneum itch signaling
- Stratum Corneum
- Epidermis
- Nerve plexus (Aα and C-fibres)
- Capillary Network

The end of the antihistamine era for AD
- Usually not effective for chronic itch

The end of the antihistamine era for AD
- Usually not effective for chronic itch

The end of the antihistamine era for AD
- Usually not effective for chronic itch
Targeted Immuno Treatments
New Era in Itch Treatment of AD

- Dupilumab
- Nemolizumab
- JAK/Stat inhibitors

Dupilumab blocks the IL-4/IL-13 receptor/ligand system

The humanized monoclonal antibody (mAb) dupilumab binds to the α-subunit of the IL-4 receptor, which is part of both the IL-4 and IL-13 receptor complex. Dupilumab modifies signalling of both the IL-4 and IL-13 pathways.

Dupilumab significantly improved pruritus

<table>
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<th>Study Week</th>
<th>Placebo (n=16)</th>
<th>75 mg (n=8)</th>
<th>150 mg (n=22)</th>
<th>300 mg (n=21)</th>
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*p<0.05; †p<0.01

JAK/STAT Drugs on the Horizon

Alloknesis Itchy Sensitive Skin Common Clinical Phenomena in Chronic itch

Itch evoked by a stimulus that is normally not itchy
Peripheral and central itch sensitization

Results: sensitivity to chemical itch provocations

Patients with AD exhibit increased intra- and extraneuronal sensitivity to non-histaminergic itch only. Andersen et al. PAIN 2017

CNS targets for reducing neural sensitization itch

- GABA: gabapentin, pregabalin doses up to 2400mg Gaba/day and Pregab 300mg/day
- Other neurotransmitters: mirtazapine 15 mg
- Combo: mirtazapine and gabapentin/pregabalin

Gabergic Drugs for Pruritus

- Nalfurafine (Japan)
- Butorphanol
- Nalbuphine
- Asimodoline

Opioids in chronic itch: an imbalance between µ- and κ-receptor activity?

Butorphanol A kappa Agonist and Mu Antagonist

- Inhaler approved by FDA for migraine headaches
- 1-4 mg
- Side effects drowsiness, nausea
- Controlled substance
- Indications intractable chronic itch of different types

Stull & Yosipovitch Exp Opin Pharmacotherapy 2016
Tey & Yosipovitch Br J Derm 2011
Stull et al. Exp Opin Pharmacotherapy 2016

Stull et al. Exp Opin Pharmacotherapy 2016
Tey & Yosipovitch Br J Derm 2011

Dawn & Yosipovitch JAAD 2006 Stull et al. Exp Opin Pharmacotherapy 2016,
Targeting NK1 Receptor Sites
Receptors for Substance P
in the Itch Signaling Pathway

Serlopitant
Tradipitant
Aprepitant
Orvepitant

Intention-to-treat (ITT) population.
P-value is based on a t-test of active arm vs placebo without multiplicity control. One patient from the placebo group, 2 from the serlopitant 1 mg group, and 1 from the serlopitant 5 mg group were removed from the analysis as they had no baseline or post-baseline VAS data.

LS, least-squares.
*p < 0.05.

Serlopitant Disrupts the NK,R Mediated Itch Signaling Pathway in Pruritus

Substance P
NK1R Binding
Substance P
NK1R Binding

Serlopitant Therapy Blocks NK1R-Mediated Itch Signaling and Processing


Serlopitant 1 mg and 5 mg Were Superior to Placebo for Reducing Pruritus, as Assessed by VAS Pruritus Score

At week 6, 43%, 38%, and 53% of patients in the serlopitant 0.25, 1, and 5 mg dose groups, respectively, reported a 4-point decrease in average VAS pruritus score, compared with only 26% of patients in the placebo group.

Yosipovitch et al. JAAD Feb 2018

Treatment of Atopic Itch

Case of Chronic Itch without rash

- 55 year old patient with severe generalized itch for 2 years
- VAS 7-10
- Lichenified plaques on ankles and excoriations on back
- Work up negative for underlying systemic diseases

Pruritus of Undetermined Origin
& Treatment Regimen

Topical anti-pruritics

NK-1 inhibitors

Potentially Useful Drugs (PUO)

Kappa opioids (Butorphanol)
Combo NaSSA & Anti Epileptics
GABA agonists
0.1 Pregabalin
Anti depressants
NaSSA

PUO
Pruritus of Undetermined Origin
& Treatment Regimen
Severe Itch in a Motor-cyclist

- 45 year old patient NRS 10 out of 10.
- Mainly on his bilateral arms and shoulder girdle
- Patient is on large dose of opioids post back trauma.
- Patient is an avid cyclist and itch is aggravated after cycling
- Denies vehemently substance abuse
- Referred by Pain specialist for suspected opioid induced itch
- Course of naltrexone was not helpful
- Cervical Stenosis C5-C6
- Positive ice pack sign +

Brachioradial Pruritus

- Monosymptomatic
- Dermatomal localization
- 80%: relevant cervical spine lesions
- Neoplasms rare
- Cutaneous innervation: functional dysbalance of epidermal/dermal nerves

BRP

NP

BRP-triggered generalized pruritus A New Entity?

- 5/7F
- Pruritus started on both arms
- MRT: spondylolisthesis in C3-5 due to a herniated vertebral disk at C4/5, osteochondrosis in C5-7, and spondylarthrosis in all cervical segments
- Generalized after several months to neck, legs, abdomen, and back

CNS targets for itch treatment

- Higher doses seem to work better for neuropathic itch: GABA: gabapentin, pregabalin doses up to 3600mg Gab/day and Pregab 300-600mg/day
- Combo: mirtazapine and gabapentin/pregabalin

BOTOX AS AN ANTIPRURITIC

Some reduced histamine-induced itch intensity in healthy men
RESULTS: ITCH INTENSITY AUC

The itch intensity AUC for the Botox treatment was significantly reduced from baseline for all follow up periods (p<0.01) and was significantly lower than the Saline treatment (p<0.05).

TREATMENT OF NEUROPATHIC ITCH

Chronic Itch and Sleep

Patients with chronic itch report a higher level of sleep-related problems compared to the general population:
- Majority of patients state itch intensity increases at night
- Trouble getting to sleep, trouble staying asleep, daytime fatigue
- Significant impact on quality of life

Patients with chronic itch—from dermatological1, neuropathic2, systemic3, and psychogenic4 causes—often report that stress exacerbates their itch
- Stress can predispose someone with an underlying disease to an outbreak of itch5
- Chronic stress, especially in combination with genetic or environmental predisposition, typically worsens itch in mouse models6

Stress Exacerbates Itch


Treatments for Nocturnal itch

- Mirtazapine 15mg improves nocturnal itch
- Higher doses do not seem to work better
- Gabaergic drugs have also a sedating effect
- Sedating anti histamines H1 some pts respond well
- Sleeping pills do not seem to work for the itch

The Itch- Stress Anxiety Cycle

Figure by Hjalte H. Andersen
Treating chronic pruritus: beyond pills

- In the brain, pruritus and psyche are intertwined in a complex manner and the effect of one affects the other.
- In addition to the somatosensory aspects of pruritus, the cognitive and emotional components must be evaluated and addressed to effectively manage chronic pruritus.

Tey et al. Clinics in Derm 2012

Holistic Approach for Treatment of Itch

1. Acupuncture of L11 in the elbow has been shown to reduce itch in Atopic dermatitis

Tey et al. Clin Dermatol 2013

Conclusions

- No quick fix for all types of itch
- Managing the complexities, challenges, and costs of chronic itch requires a comprehensive approach that includes topical, oral, and non-pharmacological approaches
Saturday, March 24, 2018

6:00 a.m. - 7:00 a.m. Regeneron Product Theater
(No CME Awarded)
Located in Coral D & E

7:00 a.m. - 8:00 a.m. Updates in Medical Dermatology
Liza Brown, DO; Franz Kerdel, DO; Danielle Nicolazzo, DO & Nickolas Poulos, DO
LECOMT/Larkin Community Hospital, South Miami Campus

8:00 a.m. - 8:45 a.m. Non-Invasive Cutaneous Oncology, Part 1
Frank Armstrong, DO, FAOCD

8:45 a.m. - 9:30 a.m. Non-Invasive Cutaneous Oncology, Part 2
Josh Swindle, RTT

9:30 a.m. - 10:30 a.m. How to Hit a Homerun with MACRA!
Clifford Lober, MD, JD

10:30 a.m. - 11:30 a.m. Dermatological Emergencies: The Eschar
Ted Rosen, MD, FAAD

11:30 a.m. - 12:00 p.m. Break with Exhibitors

12:00 p.m. - 1:00 p.m. Genentech Product Theater
(No CME Awarded)
Located in Coral D & E

1:00 p.m. - 2:00 p.m. Realizing the Vision: Excellence in Dermatology
Ted Rosen, MD, FAAD
(No CME Awarded)

2:00 p.m. - 3:00 p.m. Shared Leadership
James Warrick & Daniel Ladd, DO, FAOCD

3:00 p.m. - 3:30 p.m. Break with Exhibitors

3:30 p.m. - 4:30 p.m. Non-Invasive Modalities in Lipolysis
Michelle Foley, DO, FAOCD

4:30 p.m. - 5:30 p.m. Welcome to Derm Clinic in the Bronx
Charles Gropper, MD
HPI

59 yo male presents as a referral from Rheumatology to be evaluated for apparent fatty deposition in the bilateral arms. Onset was first noticed in May 2015 and ensued immediately after intensive inpatient treatment for Henoch-Schonlein Purpura. Patient states that the lesions are non-tender and swell intermittently.

ALLERGIES: fluconazole

MEDICATIONS: lisinopril, omeprazole, atenolol

PAST MEDICAL HISTORY: detached retina, HIN, HSP, GERD
HPI

47 y/o male presents complaining of a large violaceous lesion on his left chest that began approximately 7 months prior. Lesion is non-tender, non-progressive.

ROS negative for chest pain, dyspnea, cough, abdominal pain, fever, night sweats, weight loss.

ALLERGIES: NKDA

MEDICATIONS: none

PAST MEDICAL HISTORY: Radiation Therapy to the neck for an unspecified mass
Despite the clinical description, Hodgkin's disease appears in the biopsy histology only as a subtle anaplastic cell population. The lesions show no evidence of lymphocyte depletion or granulomatous inflammation. The only abnormal cells are characteristic of Reed-Sternberg or mononuclear variant cells, which are distinguished by their size and the formation of multinucleated giant cells. These cells are scattered throughout the lymph node and are not clustered in the germinal centers as seen in diffuse large B-cell lymphoma. The presence of these large cells may also be indicative of other lymphoma subtypes, such as mantle cell lymphoma. The distinction between these two entities is important for therapeutic decision-making, as the management approaches differ significantly.

Yours sincerely,
[Signature]
Christopher D. M. Kirby, MD, FRCPath
CIMPath success.
Patient KK

RESIDENT: DR. WHITE, PGY-3
ATTENDING: DR. KERDEL

HPI
20 y/o F with PIHx acne and pyoderma gangrenosum. Patient failed topical therapy including protopic, elidel, and clobetasol. Patient has been on oral prednisone and cyclosporine in the past. She was started on Apremilast 30mg BID.

Upon presentation

2 months of Rx Apremilast
6 mo. on Apremilast

Review Pediatric PG

New Therapeutic Options

New Therapeutic Options

Patient LC
HPI

45 y/o Male presents with photosensitivity following sun exposure that has developed over the past 18 months. The patient was erroneously exposed to hydralazine instead of hydroxyzine by the pharmacy for 1 year.

ROS: Neg
Pmhs: Asthma, HSV, atopic dermat
Meds: Advair, singulair, levocetirizine, hydroxyzine

Labs: Collagen vascular workup negative
Treatment

- Pt had failed plaquenil and cyclosporine
- IM Kenalog 40mg
- The patient was subsequently started on xolair, which he also failed. He was recently started on Azathioprine

Background

33 y/o female presents w/ generalized eruption. Patient was started on Lamictal (Lamotrigine) for bipolar disorder 2 weeks prior. Patient was transferred to LCH from another hospital.
**Day 8**

**Day 16**

**Day 19**

**Labs/Imaging**

96.8 F, 20 RR, 72 HR, 119/69
AST: 10
ALT: 23
Albumin: 2.8
CXR bibasilar Atelectasis

**SCORTEN**
- Age >40 years
- Presence of a malignancy (cancer)
- Heart rate >120
- Initial percentage of epidermal detachment >10%
- Serum urea level >10 mmol/L (28 mg/dL)
- Serum glucose level >14 mmol/L (252 mg/dL)
- Serum bicarbonate level <20 mmol/L

**SCORTEN predicted mortality rates**
- SCORTEN 0-1 >3.2%
- SCORTEN 2 >12.1%
- SCORTEN 3 >35.3%
- SCORTEN 4 >58.3%
- SCORTEN 5 or more >90%

**Treatment Protocol**
HPI

63 y/o male with ER(+), HER-2/neu(-) metastatic ductal carcinoma of the breast presented for management of extensive cutaneous metastases.

2008 ductal carcinoma stage IIIB, 1.2cm tumor

PET/CT 11/4/14 2/9/15 Interpretation

Lungs
- Mult right-sided pulm nodules with no FDG uptake: largest 4mm
- Same as 1/14/14 except largest nodule 7mm

Spine
- Uptake L2/L3 SUV max 6.02
- New uptake L posterior 11th rib SUV max 3.44
- New uptake at T4 SUV max 3.88
- L2/L3 SUV max 3.69, improved
- L post 11th rib SUV max 5.97
- T4 SUV max 4.54
- Stable/Improving

Skin
- New uptake SUV max 3.26 corresponding to nodular skin thickening of left chest wall
- Chest wall nodular skin thickening 3.4cm SUV max 5.32
- Anterior pelvic wall skin thickening SUV max 3.74
- Progression
PATHOLOGY

10/13/14, UPPER ABDOMINAL SKIN:
METASTATIC POORLY DIFFERENTIATED MUCIN-PRODUCING
ADENOCARCINOMA IN RETICULAR DERMIS WITH FOCAL
EPIDERMOTROPIC COMPONENT, CK7+, CK20-. 

11/4/14, UPPER ABDOMINAL SKIN:
METASTATIC MAMMARY CARCINOMA WITH EXTRACELLULAR
MUCIN PRODUCTION. CK7+, CK20-, PAS+ IN EXTRACELLULAR
MUCIN

HPI

57 y/o Hispanic female presented to the office with biopsy
proven PV for approximately 1 year duration. She has been
placed on a short, tapered course of oral prednisone and
topical clobetasol 0.05% cream.
Meds: Prednisone 20mg daily, Clobetasol cream BID

Patient MA

RESIDENT: DR. HOWARD, PGY-3
ATTENDING: DR. KERDEL
TREATMENT

- Prednisone 20mg daily
- Clobetasol paste
- Cellcept 1g BID
HPI

55 y/o male with a PMHx of Crohn’s disease and anal fistulas was referred for evaluation of bilateral inguinal eruption with ulceration and draining sinus tracts. The lesions started 8 months prior and have been worsening.

- PSHx: partial colectomy w/ colostomy
Plan

- Continue:
  - Silvadene Topical 1% BID

- Start:
  - Humira at hidradenitis suppurativa dosing.

New Therapeutic Options

Patient DG
HPI

19 y/o Male presented with a 10-yr history of widespread plaques. He was previously treated for Atopic Dermatitis with topical Triamcinolone. Over the past few years, he has had worsening of skin lesions with enlarging plaques on his left neck.

Labs & Imaging

- CBC
- CMP
- Lipids
- TSH
- Free T4 and total T4
- Serum ACE level
Patient MN

RESIDENT: DR. JENSEN, PGY-3
ATTENDING: DR. KERDEL

PET Scan

Impression:
1. Visualization of hypermetabolic skin abnormalities involving the left lower neck region, as well as the right anterior abdominal wall area, as described above. The findings are consistent with the patient’s known lymphoma.
2. There are FDG-avid lymph nodes bilaterally in the neck, as well as bilaterally in the axillae, also consistent with the patient’s known lymphoma.
3. No FDG-avid lesions are seen in the liver or spleen.
4. No interval change is evident.
5. Internal follow-up with PET-CT is recommended to assess for interval treatment response.

HEMATOPATHOLOGY
Single Case Report

An Unusual Case of Mycosis Fungoides
Presenting as Sarcoidosis or Granulomatous Mycosis Fungoides

CLAIRE MARQUETTE, M.D.,1 OXILE PICARD, M.D.,2 JOSEF AUDET, M.D.,2
AGNIS LE TROISIÈME, M.D.,2 MICHEL JAGOT, M.D.,2 AND JACQUES DERCEL, M.D.,2

Am J Clin Pathol 1993; 99:82-86
HPI
69 y/o female diagnosed w/ sezary syndrome in 2005
- Treatments over the years
  - Topicals: steroids, emollients
  - Injections: ILK and INF-alpha to tumors
  - XRT spot
  - Surgical excision (including large lymph node)
  - Systemic: photopheresis, bexarotene, interferon-alpha
- Results
  - Good maintenance of disease until late 2016

Case History
- Sept 2016
  - Developed large plaques on trunk, head/neck, tumors slowly enlarging, worsening pruritus
- Oct 2016
  - Started chemotherapy with pegylated liposomal doxorubicin (doxil)
Case History (cont.)

- Dec 21, 2016
  - First infusion of brentuximab vedotin
  - Tumor lysis syndrome
    - Allopurinol, IV
- Jan 11, 2017
  - Received second BV infusion

Pre-Treatment
Post 2 cycles

Post Treatment, 2 cycles
**Therapeutic Options**

**Phase II Investigator-Initiated Study of Brentuximab Vedotin in Mycosis Fungoides and Sézary Syndrome With Variable CD30 Expression Level: A Multi-Institution Collaborative Project**

- 30 MF patients, treatment experienced
- CD30 expression variable (including non-det.)
- 21 (70%) objective response
- 7 (23%) had skin improvement >90%
- Most common AE: Peripheral neuropathy in 66%; 86% improvement within 24 months

**Therapeutic Options**

**Results of a Phase II Trial of Brentuximab Vedotin for CD30+ Cutaneous T-Cell Lymphoma and Lymphomatoid Papulosis**

- 28 MF patients
- 15 (54%) responded, independent of CD 30 expression
- Average response time 12 weeks
- Average duration 32 weeks

**Therapeutic Options**

Open-label Phase III trial of brentuximab vedotin Versus Physician’s Choice (Methotrexate or Bexarotene) in Patients With CD30-Positive Cutaneous T-Cell Lymphoma (ALCANZA study)

- 128 MF or primary CALCL patients
- Patients showed CD30+ expression
- Randomly assigned BV vs. MTX or bexarotene
- Response rate measured at average 4 months:
  - 67% with BV
  - 20% with MTX or bexarotene

**Patient MZ**

PAST RESIDENT: DR. ECKER
ATTENDING: DR. KERDEL
HPI

76 y/o female referred for livedo reticularis and painful subcutaneous nodules on her breasts, abdomen, and upper thighs x 3 months. She also reports of fatigue and muscle weakness. The working diagnosis was erythema nodosum. She was taking prednisone 30mg and 200mg of plaquenil daily. Initially, the prednisone provided modest relief, however weeks later the pain returned.
ROS: unintentional 12lb weight loss
Labs/Imaging
- CBC, CMP WNL
- PT/INR 34.6/2.9
- Red blood cell count 10,820
- ESR 100, CRP 12.30 (High)
- Hepatitis panel (–)
- Amylase / Lipase WNL
- ANA, pANCA (+)
- RF, anti SM/RNP (–)
- C3/C4 WNL
- Immunofixation – poorly defined area with IgG and lambda
- ABI: WNL

Dx: Intravascular B cell Lymphoma

Patient Update
- PET CT and bone marrow bx – negative
- Patient reported complete resolution of pain and nearly 100% clearance of lesions within DAYS after initial treatment with RCHOP
- After 3 treatments of RCHOP, the painful subcutaneous nodules returned
Patient Update

New Therapeutic Options

- Treatment of choice for all patients with IVLBCL is combination chemotherapy
  - RCHOP
  - Rituximab as both initial and salvage therapy
  - Frequently incorporated into combination chemotherapy
- Autologous stem-cell transplantation
- Radiotherapy for cutaneous limited disease
- All patients with IVLBCL should be considered to have disseminated disease and receive empiric treatment.

HPI

Patient is a 81 y/o female who presents with a pruritic eruption on her left breast of 2 months duration. Patient was previously seen by another local dermatologist who performed a shave biopsy of the lesion consistent with drug reaction. Patient has a medical history that includes left breast CA. Treatment included lumpectomy with radiation therapy. Last mammogram done within 1 year was negative.
Pathology

Left Breast
Lymphangiosarcoma

- Atypical vascular proliferation showing dilated vascular structures at superficial levels. Arterioles with increasing collagen bundles involving dermis. Cellular atypia is moderate with prominent nucleoli. Vascular mitotic figures. CD31+, CD34+, Ki-67<40%, p53 faint positive. CD34 reaction is focal

Treatment

- Left breast mastectomy followed by reconstruction and radiation therapy

Outcomes
HPI

63 y/o male presents for evaluation and treatment of right foot mass. Patient diagnosed with Kaposi Sarcoma and underwent radiation therapy.
PMHx: DM, HTN, HIV negative
Pathology

Right medial foot: Kaposi sarcoma with HHV8 stains showing classic nuclear pattern

Right plantar foot: Kaposi sarcoma

New Therapeutic Options

Current therapies include:
- Alitretinoin 0.1% gel – Panretin gel applied BID
- Cryosurgery
- Radiation therapy (electron beam)
- Intraleisional vinblastine (0.1 mg)
- Local excision
- Chemotherapy - vinblastine/vincristine regimens, anthracyclines (daunorubicin)
- Cessation of immunosuppressant medications (iatrogenic KS)
- Adherence to HAART therapy (AIDS-associated KS)

Follow Up Photos

Patient CD

HPI

41 y/o female was referred for recurring outbreaks of multiple papulo-vesicular eruptions involving her face for the past 4 years. The lesions were asymptomatic and not exacerbated by sun exposure. Prior treatments included sunscreen, minocycline, doxycycline, and topical hydrocortisone none of which controlled her outbreaks.
Labs

- CMP: wnl
- CBC: wnl
- Sed Rate: 33 [0-20]
- RPR: non-reactive
- ANA: negative

- Varicella-Zoster IgG Titer: <135
- HSV 1 IgG: 2 [0.0-0.6]
- HSV 2 IgG: 0.5 [0.0-1.0]
**Immunohistochemistry**

- Positive: CD3, CD8, CD25, granzyme B, TIA-1, TCR-B, scattered CD20 B-cells
- Negative: CD30, CD36, Varicella zoster, HSV-1 and 2
- EBER: focally positive in the atypical cells
- PCR detected clonal rearrangement of TRG gene
- No HTLV1-viral sequences detected by PCR
HPI

44yo F presents with a generalized rash present for 6 weeks. She was previously prescribed antibiotics (doxycycline) at an urgent care for an unrelated problem. She now reports desquamation of skin, fever, chills, and decreased appetite.

Patient BR

RESIDENT: DR. DANIELLE NICOLAZZO, PGY-4
ATTENDING: DR. FRANCISCO KERDEL
**Labs**

- ANA +
- dsDNA +
- Scl-70 -
- Anti-cardiolipin -
- ss-A/ss-B -
- Anti Smith -
- RNP -
- CMV IgM +
- ECHO - EF 60%, no valvular abnormalities
- CXR - no acute cardiopulmonary disease

**Pathology**

- **POSITIVE DIRECT IMMUNOFLOUORESCENCE**
  - IgG: Colloid bodies
  - IC3: Granular band at dermal epidermal junction

These changes are consistent with Rowell’s syndrome where lesions clinically resemble SJS in the setting of SCLE, ACLE or DLE

**Treatment**

- IV Solumedrol with prednisone taper
- Plaquenil 200mg BID

**New Understanding**

*Erythema multiforme and Stevens-Johnson syndrome toxigenic epidermal necrolysis associated with sulfa erythrasma.*

- Author: Ritter, A.
- Abstract: Erythema multiforme and Stevens-Johnson syndrome are characterized by the development of a necrotizing vasculitis, which can lead to epidermal necrosis. In a case series of 10 patients, all of whom were sensitized to sulfa compounds, the authors report a high incidence of cutaneous and systemic infections. The association of these two conditions with sulfa sensitivity has been well-documented in the literature. The relevance of this study is that it provides evidence for a potential link between these conditions and the use of sulfa drugs.
**HPI**

Pt is a 59 y/o presented with bilateral upper extremity erythema x 1 year. The patient stated that she was previously treated with 5-FU without any improvement. Patient also complained of bilateral lower extremity hyperpigmentation. Pt also takes hydroxyurea for polycythemia vera. No new medications.

**Labs**

- **CBC:**
  - H/H: 16.1/50.4
  - Plt: 403.
- **CMP:** WNL
- **CK:** 37
- **Aldolase:** 5.4
- **ANA:** Negative
- **Anti Jo-1 Ab:** Negative
- **Mi-2 Ab:** Not detected
Other workup
- Pt has had a total hysterectomy
- Past colonoscopy, pap smear, mammogram are up to date and WNL

Patient JG
RESIDENT: DR. GHERGHINA, PGY-3
ATTENDING: DR. KERDEL

HPI
71yo M hx metastatic melanoma of scalp presents with itching and burning of thighs, pelvis, abdomen, and extremities. He is currently finishing a prednisone taper and was started on ipilimumab 2 weeks prior.
Treatment

- Dapsone 100mg daily
- Clobetasol 0.05% crm BID
- Doxepin 25mg qHS
- IM 40mg/ml x1ml in gluteus
Patient CA

RESIDENT: GABRELA PERDOMO, PA-S
ATTENDING: DR. KERDEL

HPI

54 y/o female presents with a pruritic erythematous eruption along the nasolabial folds for the past 2 years. Patient admits to getting fillers 20 years ago. She has been getting treatment with intralesion kenalog by a plastic surgeon.
Treatment

- Intralesional Kenalog 20mg/cc 1 cc total
- Considered Plaquenil
- Other possibilities discussed included systemic steroids and methotrexate

Calciphylaxis: An Update

Calciphylaxis

Also known as:
- Uremic gangrene
- Calcific uremic arteriolopathy
- Calcifying panniculitis

Our understanding is changing.
Calcification Disorders

- Cutaneous Calcification
- Dystrophic Calcification
- Metastatic Calcification
- Idiopathic Calcification
- Iatrogenic Calcification
- Benign Nodular Calcification
- Calciphylaxis

Calciphylaxis: The Literature

1961: Rats sensitized with vitamin-D derivative, "challenged with ferric dextran plus certain histamine liberators" causes fatal form of musculocutaneous inflammation. "Calciphylaxis is a condition of hypersensitivity in which, during a "critical period" after sensitization by a systemic calcifying factor (e.g. vitamin-D compounds, parathyroid hormone, sodium sulfathiazol), treatment with certain challengers (e.g. metallic salts, Fe-Dex, egg white) causes an acute, local calcification followed by inflammation and sclerosis."

Calciphylaxis: The Literature


Calciphylaxis Literature Summary

- +/- ESRD on HD
- Hypercoagulable states (Lupus anticoagulant, protein C, prothrombin)...
- Pathogenesis remains elusive.

Diagnosis

- Gold standard: Tissue biopsy with calcification in vessels
- Not all cases of calciphylaxis show positive biopsy initially*
- Laboratory studies not helpful*.
- Bone scintigraphy has been shown to be useful*.
Diagnosis - Clinical
Skin Ulcers with painful purpura at the periphery

Diagnosis - Histology
Calcification and thrombosis in subcutis blood vessel

Diagnosis - Bone Scintigraphy
Abnormal calcium deposition in soft tissue (arrows)

Treatment
1. Calcium, phosphorus, PTH homeostasis (calcimimetics, phosphate binders, etc.)
2. Parathyroidectomy
3. Low-calcium dialysate solutions

Treatment
1. Change warfarin to heparin or NOACs
2. Hyperbaric oxygen
3. Sodium thiosulfate IV, intralesional
4. tPA
5. No clinical trials or disease-specific drugs

Prognosis
- Depends on clinical presentation
- If classical calciphylaxis, high mortality
- Nonuremic calciphylaxis, low mortality with good response to treatment
References - Case reports

Non-Melanoma Skin Cancer (NSMC): Non-Surgical Management
Frank T. Armstrong DO, FAOCD, FAAD
March 24, 2018

Conflicts
- None

Goals and What Will Not Be Covered
- Goal: go home with a pearl or two
- We will not review:
  - Surgical options (Moh's, Fresh Frozen Section, Standard Excisions, C+E)
  - Radiation (XRT and SRT)
  - Laser (ablative)
  - Oral Therapies (Hedge Hog Inhibitors)
  - Every non-surgical option

What Will be covered
- Candidates for non-surgical options
- Staging and stratification for BCC/SCC
- 5-FU
- Imiquimod
- PDT (ALA vs MAL)
- Intraluminal Options (5 FU, IRT, Bleomycin, others)
- Transplant Patients (High Risk Group)
- Preventative Concepts

Who are the Non-Surgical Candidates?
- Lower risk tumors
- Higher risk surgically
- Cosmesis
- Patient refuses surgical options

Recent JAAD Articles- Guidelines for BCC (Jan 2018) and SCC (Jan 2018)
Strength of Recommendation Taxonomy (SORT)

Evidence was evaluated by this Unified System called SORT:

I. Good-quality patient-oriented evidence (i.e., evidence measuring outcomes that matter to patients: morbidity, mortality, symptom improvement, cost reduction, and quality of life).

II. Limited-quality patient-oriented evidence.

III. Other evidence, including consensus guidelines, opinion, case studies, or disease-oriented evidence (i.e., evidence measuring intermediate, physiologic, or surrogate end points that may or may not reflect improvements in patient outcomes).

Clinical recommendations

Developed on the basis of the best available evidence tabled in the guideline.

These are ranked as follows:

A. Recommendation based on consistent and good-quality patient-oriented evidence.

B. Recommendation based on inconsistent or limited-quality patient-oriented evidence.

C. Recommendation based on consensus, opinion, case studies, or disease-oriented evidence.

BCC- Staging and Stratification

A formal staging system for risk stratification specific to patients with BCC is not available.

Because of the exceedingly low incidence of regional and distant metastasis, the TNM (tumor, node, metastasis) classification and AJCC stage grouping are rarely, if ever, applied to patients with localized BCC.

The most clinically relevant stratification to guide the management of patients with BCC is the differentiation between localized tumors at low versus high risk for recurrence.

The most useful stratification of BCC is provided by the National Comprehensive Cancer Network (NCCN) Guidelines.

The NCCN Stratification takes both clinical and pathologic parameters into account, and is based on a combination of available evidence and expert multidisciplinary opinion, including representation from dermatology, dermatopathology, head and neck surgery, plastic surgery, and surgical, radiation, and medical oncology.

JAAD, Guidelines of care for the management of basal cell carcinoma, Jan, 2018.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Low Risk</th>
<th>High Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pathologic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Growth Pattern</td>
<td>Nodular, Superficial</td>
<td>Aggressive</td>
</tr>
<tr>
<td>Perineural Involvement</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
</table>


BCC Area Risk by Location and Histologic Subtypes

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Low Risk</th>
<th>High Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Area L consists of trunk and extremities (excluding hands, feet, nail units, or scalp)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Area M consists of cheeks, forehead, scalp, neck, and periauricular region</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Area H consists of central face, eyelids, eyebrows, periorbital skin, nose, lips, ears, mandible, preauricular and postauricular skin/space, temple, ear, and genitalia</td>
<td></td>
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<tr>
<td>Area H constitutes a high-risk area on the basis of location, independent of size</td>
<td></td>
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<tr>
<td>Low-risk growth pattern include verrucous, atypical verrucous, and nevus-like variants</td>
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</tr>
<tr>
<td>High risk: Having morphological, dermoscopic (melanosis/scar), sclerotic, mixed features, or micronodular features in any portion of the tumor</td>
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</table>

**BCC - Who are the Non-surgical Candidates?**

- In general, treatment of BCC is most effectively accomplished by surgical therapy. There are relatively few exceptions to this guiding principle.
- "If surgical therapy is not feasible or preferred, cryosurgery, topical therapy (eg, imiquimod or 5-fluorouracil [5-FU]), PDT (with aminolevulinic acid [ALA] or methyl aminolevulinate [MAL]), or radiation therapy for BCC can be considered when tumors are low risk, with the understanding that the cure rate may be lower."

**SCC - Stratification of Tumor Risk and Are there any Non-Surgical Candidates?**

- There are currently no FDA-Approved Non-Surgical Options for SCC.
- European Union has approved Non-Surgical Options for SCC in situ.
- 2018 JAAD article on SCC Guidelines reviews stratifying SCC.

**SCC Staging**

- A universally accepted staging system for risk stratification of cSCC is not yet available.
- Brigham and Women's Hospital (BWH) system classifies tumor categories on the basis of presence of several clinical and pathologic risk factors.
- Although the BWH system does not address nodal and metastasis classifications and advanced stage groups as the AJCC staging system does, it appears to provide superior prognostication for patients with localized cSCC.
- AJCC staging has consistently identified unsatisfactory prognostic information.
- A thorough clinical exam of the regional lymph node basin should always be performed.

**Non Surgical Recommendations SCC**

- Table II: Brigham and Women's Hospital tumor classification system.
- Table XI: Level of evidence and strength of recommendations for the nonsurgical treatment of cSCC.
Topical 5-Fluorouracil

- Five percent 5-fluorouracil (5-FU) cream and solution are approved by the FDA for the treatment of superficial basal cell carcinomas.
- Apply 2 times a day in an amount sufficient to cover the lesions; continue until the inflammatory response reaches the erosion stage, then discontinue use.
- Duration of therapy: 3 to 6 weeks; however, therapy may be required for as long as 10 to 12 weeks before lesions are obliterated.

5-FU sBCC

- 5-FU sBCC

Very Useful Study of sBCC

- 29 patients with 31 sBCC (small shave biopsy done to confirm)
- 5% 5-FU Cream BID for up to 12 weeks, examined at 6 weeks and every 3 weeks after for clinical cure, then excised for histological examination.
- 28 of the 31 sBCC (90%) were histologically cured.
- Among all lesions, 4 (13%) were cured by Week 6, 5 (16%) by Week 9, and 19 (61%) by Week 12.
- Well tolerated, excellent cosmesis and high patient satisfaction.

5-FU SCCis Data

- Off-label in USA.
- Clearance rates vary from 48% (QD Dosing) - 85% (BID Dosing).

Imiquimod

- Induces cytokine production via Toll-Like Receptor 7
- Cell-mediated immune response: monocytes, macrophages and APCs
- Innate immune response: upregulation of cytokines (IFN-alpha, IL-6, TNF-alpha)
Imiquimod

- 3M launched Imiquimod for genital warts in 1997
- It was initially discovered during research for HSV inhibitors
- Limit the size of area treated to < 25 cm²
- Avoid topical steroids/cytokine dermatitis is therapeutic
- New molecule, Resiquimod, that is much more potent than Imiquimod.

Imiquimod nBCC Eyelid

- Small study, 5 patients that refused surgery
- 5% Imiquimod Cream 5 X a week, X 6 weeks
- 4 patients had clearance (Clinical) with no recurrence of tumor at 7 years
- 1 patient dropped out
- All patients reported irritation with therapy that resolved upon discontinuation

Imiquimod sBCC

- The current recommended dosing frequency for the treatment of superficial BCC is 5 times per week for 6 weeks, but the frequency and duration should be tailored to the individual patient’s response and ability to tolerate the medication.
- Many application schedules have been used, ranging from 3 times per week to twice daily 7 d/wk.
- The duration of treatment varies from 6 to 16 weeks.
- Superficial BCC cure rates of 70-100% should be expected after a 6-week course of 5-times-per-week application, as shown in studies.


Nodular BCC

- 102 patients- Nodular BCC- Imiquimod 3x/week x 8 or 12 weeks (90 completed study)
- Histologic clearance in 84%

- 15 patients- Nodular BCC- Imiquimod 1/day x 7 days/week for 6 weeks or 12 weeks
- 71.4% histologic clearance in 6 week group and 76% histologic clearance in 12 week course

*Not FDA Approved*

SCCIS
Imiquimod SCCIs

- Not FDA Approved
- Dosing in European Union: QD/QOD 6-16 weeks
- Efficacy: 90% (Study from Kossard et al)

Imiquimod Summary

- FDA-Approved for sBCC: Once daily x 5 days/week at bedtime for 6 weeks
- Other regimens not FDA-Approved (various studies)
  - sBCC QD X 6 WEEKS (87-88% SUCCESS)
  - nBCC- 3 X WEEK X 8 WEEKS (82.2% CLINICAL CLEARANCE, 64.4% HISTO CLEARANCE)
  - nBCC QD X 12 WEEKS (76% SUCCESS RATE)
  - nBCC EYELID- 5 X WEEK/ 6 WEEKS (NO RECURRENCE AT 7 YEARS)
- SCCis- NOT FDA-Approved - used in European Union: QD/QOD 6-16 weeks
- Remember not to use topical steroids with Imiquimod because we want cytokine dermatitis

How About Photodynamic Therapy?

- Blue light sources are sufficient for the treatment of epidermal lesions such as AKs
- Red light sources may be more suitable for the treatment of lesions that extend to the dermis

PDT involves the application of a topical photosensitizer that, when activated by exposure to a light source, results in damage to neoplastic cells.

Two photosensitizing chemicals are available: 5-aminolevulinic acid (ALA) and methylaminolevulinate (MAL).

These chemicals enter keratinocytes and then undergo enzymatic conversion to protoporphyrin IX.

Protoporphyrin IX appears to accumulate selectively in neoplastic tissue at levels up to 10-fold greater than in normal tissue.

Photoactivation of protoporphyrin IX through application of a light source produces cytotoxic free radicals that cause direct damage to cell membranes, DNA, and RNA, leading to cell death. This is followed by an inflammatory immune response leading to clearance of tumor cells.

PDT for sBCC

- Recommendations: PDT is an effective and reliable treatment option for sBCC that offers excellent or good cosmetic outcomes.
- PDT offers an advantage in the treatment of large, extensive, and multiple lesions.
- MAL-PDT has demonstrated long-term efficacy in sBCC, with 5 year follow up data

**Disclaimer:**

- The light sources may be more suitable for the treatment of lesions that extend to the dermis.
- The light sources are not recommended for the treatment of epidermal lesions such as AKs.
PDT for nBCC

- The strongest evidence for topical PDT in nBCC comes from 5 phase III studies with MAL-PDT, in which a total of 220 nBCC lesions were treated.
- Efficacy is consistently high, with 3-month complete response rates of 73% to 94% with MAL-PDT.
- Recommendations: MAL-PDT is an effective and reliable treatment option for nBCC, possibly preferable for thin lesions with the advantage of good cosmetic outcome.
- MAL-PDT has demonstrated long-term efficacy for nBCC, backed up with 5-year data.


Data from EU on PDT for BCC

- Aminolevulinic acid hydrochloride gel, 10% with Nanoemulsion
- 2 Sessions of PDT, 1 week apart
- Used on nBCC and sBCC (<2 mm tumor thickness and 5-2 cm diameter)
- Face, scalp, extremities, neck, and trunk
- 3 hours incubation under occlusion
- Red light employed during each PDT session so that 37 J/cm² delivered

Results:
- At 12 weeks: Complete lesion clearance 12 weeks after last PDT= 94.6%

PDT for SCCs (Bowen’s Disease)

- 64-month recurrence rate of 17% in BD; PDT can be viewed as having an acceptable long-term efficacy, comparable with more established therapies
- In contrast to PDT, Thestrup-Pedersen, Ravnborg, and Reynmann found relapse rates (5 years) of 34% for cryotherapy, 19% for curettage, 14% for 5-FU, 6% for radiotherapy, and 5% for surgery.
- Currently PDT FDA-approved for treatment in USA

Imiquimod vs PDT vs 5-FU for sBCC

- Imiquimod 5% Cream QD 5 days a week X 6 weeks
- Fluorouracil 5% Cream, BD X 4 weeks
- MAL-PDT 2 sessions 1 week apart

Success (at 1 year):
- 83.4% Imiquimod
- 80.1% 5-FU
- 72.8% MAL-PDT

Lancet, 2013, June.

PDT Pearls

1. Skin prep: degrease and debride (new codes)
2. Occlusion to enhance penetration
3. Heat the skin to increase porphyrin production
4. Incubate long enough
5. The light needs to be close enough to the patient (inverse square law)
6. If redness immediately, think propylene glycol reaction!!!
7. Don’t use topical steroids to rescue (cytostereos)

How about Intraleisional Options for NMSCs?

- Johnson, BCM, June
Intralesional Options for NMSC

**Intralesional Efficacy by Drug**

- Nothing is FDA Approved
- Studies support the use, excellent efficacy for right lesions
- Experience is best with SCC-KA Type, especially legs
- Tip: inject lidocaine first as 5-FU is painful when injected
- My experience - inject 0.2-0.3 cc weekly or every other week until resolution
- I have found this very helpful for those eruptive KAs on the legs

**Intralesional SCC**

- Special group: HPV may have a role in pathogenesis for SCC, Polyoma viruses for Merkel Cell and HHVs for Kaposi’s Sarcoma
- Increased incidence of NMSC:
  - 40 to 250 times the risk of cSCC compared with the general population
  - BCC 10 times greater than in the general population
  - Melanoma 6 to 8 times increased compared with general population
- Cyclic PDT - done every 4-8 weeks can greatly reduce risk of NMSC’s
- Combination Topical RX w Imiquimod (M, W, F) and 5% 5-FU (Th, Sat, Sun) 5/5 clinically clear at 4 weeks
- Soriatane - 25mg QD for chemoprevention chronically may help; limit is cost and side effects (hair loss, “tacky” skin, need to follow lipids)
- Prevention and Field Therapy: education is critical, best to have discussion before the transplant surgery.

**SCC-KA type and IL 5-FU**

**Transplant Patients**

- Special group: HPV may have a role in pathogenesis for SCC, Polyoma viruses for Merkel Cell and HHVs for Kaposi’s Sarcoma
- Increased incidence of NMSC:
  - 40 to 250 times the risk of cSCC compared with the general population
  - BCC 10 times greater than in the general population
  - Melanoma 6 to 8 times increased compared with general population
- Cyclic PDT - done every 4-8 weeks can greatly reduce risk of NMSC’s
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- Soriatane - 25mg QD for chemoprevention chronically may help; limit is cost and side effects (hair loss, “tacky” skin, need to follow lipids)
- Prevention and Field Therapy: education is critical, best to have discussion before the transplant surgery.
**Prevention of NMSC in Transplant Recipients**

| Photoprotection: Sun protective clothing, proper sunscreen use, avoidance of intentional sun exposure. |
| Regular monthly self skin examinations. |
| FBSE at least annually depending on risk level. |
| Field Cancerization Treatment: |
| Topical 5-FU: Apply once a day for 2-4 weeks. |
| Topical imiquimod: 3 times a week for 4-16 weeks on skin areas up to 60 cm² can decrease number of AKs and SCCs with no effects on systemic immunity. |
| PDT: Large areas of skin needed to be treated; 2 treatments 2-4 weeks apart. |
| 5-10 SCC per year → systemic chemoprevention with retinoids or oral: Acitretin 20-25 mg/day, isotretinoin 1-2 mg/kg/day. |

Prevention and Maintenance

- Education of UV exposure and protection
- Sunscreen
- UPF Clothing, shades
- Car Tint to block UVA
- Niacinamide or Nicotinamide 500mg BID (Australian Study)
- Antioxidant topical before sunscreen (up to 8X protection)
- Photozyme (photolyses)
- Heliocare (oral sunscreen effect)

Summary of Non Surgical Options for NMSCs

- Rx Topicals: we reviewed 5-FU and Imiquimod
- PDT
- Intralesional Drugs
- Transplant Patients
- Prevention and Education

Considerations for Non Surgical Options NMSCs

- FDA Approved
- Efficacy
- Level of Evidence
- Patient’s and Physician’s Preferences
- Cost
- Side Effects
- Expectations
- Patient Adherence
- Risks and Benefits of Non-surgical choice vs. Surgical
- Long Term Follow Up is essential
Take Home

- sBCC
  - 5% 5-FU: BID 6-12 weeks (12 weeks had highest success)
  - Imiquimod: 5 nights/week 4-6 weeks (75% success)
  - PDT: 2 sessions 1 week apart
- nBCC
  - 5% 5-FU: not recommended due to low efficacy (penetrates poorly)
  - Imiquimod: 3 nights/week 8-12 weeks (64% clinical clearance, 49% histologic clearance)
  - OR, QD/QOD 12 weeks (75% success rate)
  - PDT: 2 sessions 1 week apart (for lesions less than 3mm depth)
- SCCIS
  - 5% 5-FU: NOT FDA approved BID 6-12 weeks (highest success rate - Bargman study)
  - Imiquimod: NOT FDA approved used in European Union QD/QOD 6-16 weeks
  - PDT: Currently NOT FDA approved for treatment in USA (17% recurrence rate)

Thank you for Listening!
Non Invasive Cutaneous Oncology Part 2: Clinically Managing Your Superficial Radiotherapy Patients
A Clinical & Photographic Review
By Joshua T.R. Swindle BSRT(T) and Dr. Ashwin Patel MD
National Director of Practice Operations and Radiation Oncologist
Updated: 3-07-2018

Disclosures
- SkinCure Oncology Employment as National Director of Practice Operations
- Honorarium Fee AOCD
- Dr. Ashwin Patel advisory and consultant role with SkinCure Oncology

Non Invasive Options: Non invasive techniques should become standard practice within our practices.
- Radiation: Superficial radiotherapy, Electron beam radiotherapy, IMRT Radiotherapy, Electronic Brachytherapy
- Oral Chemotherapy
- Photodynamic Therapy
- Topical Medications
- Oral Medications

Objectives
- Basic principals of irradiation for skin cancer as related to traditional and new approaches
- Understanding of indications for irradiation
- Understanding of clinical data in terms of outcome and complications

Focus and Appropriateness Utilizing Superficial Radiotherapy
- Appropriate Protocols: How to achieve optimal radiobiological effects?
- Appropriate Staffing: Utilizing radiation therapist and medical physicists.
- Appropriate Support: Are you supported by Radiation Oncologist and industry leaders.
- Appropriate Candidate: Is superficial radiotherapy for ALL patients?
- Appropriate Patient Setup: How to achieve efficacy in setup?
- Management of SRT acute and latent effects: What tools do you need ensuring side effects are minimal and controlled?
- Use of Vision: US guidance benefits

The above will lead to appropriate outcomes!

Risks Associated with SRT Delivery
- Acute: erythema, mild discomfort
- Latent: Hypopigmentation or hyperpigmentation, Secondary BCC from radiation delivery: 30-40 year latency with 5-7% risk, skin atrophy, tel-angietasias, ulceration or necrosis (rare)
- Recurrence: What to do now?
- Appropriate Setup and Delivery: This is a risk! Without the appropriate staffing, your outcomes directly correlate to protocols, setup and delivery of SRT.
Future Needs

- New advancements in dermatologic treatment:
  - Protocols: Dermatology practices must utilize appropriate, science-based protocols ensuring efficacy of SRT outcome. Incorporate radiation oncology support and advisory.
  - Ultrasound: Use of US at time of biopsy allowing correlation with pathology, protocol and progress.
  - RTT: The benefit and uses of a qualified and credentialed staff delivering SRT.
  - Development of new technology.
  - SRT Equipment: With new equipment, purity of beam, new clinical studies are needed for updated control rate.

L SCAPHA: SCC-IS:
PEARL: Flatten tumors in flexible concavities.

L Sup Helix AND L Scapha: both SCCs
PEARL: Very complicated “wrap around” ear tumors easily managed simultaneously.

“R INFERIOR HELIX” SCC: Vision allows you treat large complicated ear tumors easily.

“R INFERIOR HELIX” SCC: Dermoscopy & SRT-100 Vision allows you treat large complicated ear tumors easily.
“R INFERIOR HELIX” SCC: Vision allows you treat large complicated ear tumors easily

R Sup. Helix, SCC w/Sclerosis: PEARL: Yes you can! Think it’s complex? It’s a breeze for RTTs

R Sup. Helix, SCC w/Sclerosis: Yes you can! Think it’s complex? It’s a breeze for RTTs

R Sup. Helix, SCC w/Sclerosis: Yes you can! Think it’s complex? It’s a breeze for RTTs

BCC of Ear: Neglected x 8 years. Surgical Nightmare: “I know I’m gonna lose the ear”
BCC of Ear: Neglected x 8 years. SRT Vision allows “Wet Gauze” technique
Neglected BCC Wrap-Around: Wet Gauze

Another Ear Wrap-Around turning around

LEFT CYMBA CONCHA - SQUAMOUS CELL CARCINOMA, ACANTHOLYTIC

LEFT CYMBA CONCHA - SCC, ACANTHOLYTIC
PEARL: Helix & Tragus “pulled apart”

SCC PREAMICULAR: SUNKEN AREAS
MUST BE TIGHTENED/FLATTENED!
SCC PREAURICULAR: SINKHOLE!

PEARLS: Apply adequate pressure and/or pull adjacent skin laterally with hypo-fix tape.

BCC Preauricular “Sink Hole”

BCC, Preauricular, avoiding concavities

PEARL: curved shield blocks penetration

BCC, Nodular, on hairy sideburn

PEARL: Warn patient about hair loss

BCC PREAURICULAR: Watch for curved shields!

PEARL: Compassionate use of SRT is great!

Neglected BCC in mentally impaired pt

PEARL: Compassionate use of SRT is great!
Neglected BCC in mentally impaired pt
PEARL: Compassionate use of SRT is great!

Neglected BCC of nose: Cure extends into the deep alar groove due to proper taping

Neglected BCC in mentally impaired pt
PEARL: Compassionate use of SRT is great!

BCC extending into alar crease
PEARL: Tape nose in opposite direction to flatten the tumor surface area

BCC L Nasal Sidewall
PEARL: SSD = Skin to surface distance
BCC, note the bulge!

SCC Lower Lip, Ultrasound measurement is excellent documentation here

SCC, L inf. Vermillion lip, Recurrent after Mohs, HD-US = 1.1mm and 50 kV = 3mm = safe

SRT on lips = inflammt. infiltrate skews US numbers, but they never exceed 3mm (kV, depth).
HD-US: 1.1, 1.52, 1.4, 1.8, 1.3, 1.04

BCC L Nasal Sidewall
PEARL: Tumor bulges = SSD reduction might mean you need to drop the dose by 10%

SCC Lower Lip, Eversion is critical for proper dose administration
SCC Recurrent, 2 weeks post rad
HD-US = 0, cosmesis excellent!

BCC: Now this one looks super easy!
Still needs dental shield. Pain saver!

MEDIAL CANTHUS
- CHALLENGING CONCAVITY
- LIKE TRYING TO TREAT A CEREAL BOWL
- OFTEN BETTER WITHOUT A CUSTOMIZED SHIELD
- SHIELDS CAN OBSTRUCT GOOD DOSE DELIVERY
- SWITCH TO SMALLER APPLICATOR
- CAN DOUBLE THE EYE SHIELDS
- KEEP FOCUSED ON DELIVERY OF DOSE
- INCREASE kV from 50 to 70 to “Jump the Gap”

RIGHT SUPERIOR MEDIAL MALAR CHEEK - SUPERFICIAL BASAL CELL CARCINOMA

CANTHUS CONCAVITY! WATCH THE “WRINKLED LEAD SHIELD” GAP CREATED BY TRADITIONAL SRT SET-UP

Close Gap by removing shield and decreasing the applicator size
SCC-IN SITU R NASAL SIDEWALL

Flush Fit Optimized without shield

BCC, R medial canthus, classic concavity

SIM: Lesion is 1x1, margin is 0.5, Tx Site 2x2. Note large clunky 3 cm applicator. Note SSD is too far. Solution: double shield eyes, Use 2x2 applicator without awkward bent lead shield

EYELID: Lower is easy, upper is hard

BCC, lower eyelid - corneal shield, Alcaine drops for numbing
BCC Lower eyelid

Hands & Forearms: Poor surgical sites, excellent SRT sites

Hands & Forearms: Poor surgical sites, excellent SRT sites

Old hands have atrophy, inflexibility, won’t hold a stitch

Look how thin this skin is, but you want a high cure rate. 95% is excellent!

Excellent cosmesis, full function, no downtime, easier than Mohs!
Trunk: high mobility areas

Trunk: inability to perform post op care
Full mobility of arms/shoulders
Preservation of ability to perform ADLs

Trunk: inability to perform post op care
Full mobility of arms/shoulders
Preservation of ability to perform ADLs

Trunk: athlete/golfer, preservation of exercise regimen, no down time.

LARGE TUMORS: surgical nightmares

LARGE TUMORS: surgical nightmares
Local recurrence: but still achieved a vast reduction in surgical morbidity

Sources
- NCCN Guidelines TM 1.2012 Basal Cell and Squamous Cell Skin Cancers
- JAAD - Superficial X-Ray in the Treatment of BCC and SCC - A Viable Option in Select Patients
- JAAD - Soft x-ray therapy for cutaneous basal cell and squamous cell carcinomas
- Panizzon - Basal cell and squamous cell carcinoma - Radiotherapeutic Approaches
- Meibodi et al., Clinicopathological Evaluation of Radiation Induced BCC, Indian J Dermatol, 2008 52(3): 137-139
- Ron et al, Skin tumor risk among atomic bomb survivors in Japan, Cancer Causes Control, 1998;9 (5): 405
- Photographs provided by TruSkin Dermatology in Austin TX
Dermatological Emergencies
“The Eschar”

Ted Rosen, MD
Professor of Dermatology
Baylor College of Medicine
Houston, Texas

Conflict of Interest Disclosure
• Red Flags and Emergencies in Dermatology F084
• I do not have any relevant conflicts of interest to disclose related to this presentation

What Constitutes Emergency?
• Objective characteristics of emergency
• Acute onset usual
• Associated with symptoms typically
• Risk of morbidity and/or mortality
  - Morbidity (impaired normal function)
  - Mortality (death)
• Requires timely diagnosis to avoid serious morbidity or mortality; a sense of immediate necessity for intervention

Unka Teddy’s Rules
• The severity of visible pathology (deviation from normal) does not always correlate with the degree of seriousness of disease process
• Given pathology of similar visible severity, you may need ancillary information to decide what is or is not life-threatening
• Given truly life-threatening disorders, the real need for rapid intervention may differ greatly
• You don’t always need to know the precise diagnosis immediately, but a skilled clinician can identify emergent situations

Which is an emergency?
3.5 mm solitary tender pustule
24 year-old, healthy female

25cm² deep-seated nodule
30 year-old, healthy female
Which is an emergency?

Emergent Infections (With Skin Manifestations)

• 3.5 mm solitary tender pustule
  24 year-old, healthy female

• 25 cm² deep-seated nodule
  30 year-old, healthy female

Emergent Infections (With Skin Manifestations)

• Gonococcemia: Sepsis

• Benign lipoma

• Gr+ sepsis (Staph, Strep)
• Gr- sepsis (enteric microbes)
• Meningococcemia
• SSSS, TSS
• Spotted fevers (RMSF, MSF)
• Anthrax, Tularemia, Plague
• Vibrio vulnificus
• Typhus
• Necrotizing fasciitis

• Disseminated VZV, HSV
• Hemorrhagic fevers (Ebola, Lassa, Marburg)
• Smallpox
• Rubella, Rubeola
• CMV
• Arboviruses
• HIV
• HHV-8

Is this an emergency?

Pattern Recognition

• 53 year-old male
• Rheumatoid arthritis
• Rx: infliximab 5mg/kg
• Arthritis controlled
• Develops fever (102.4°F)
• Shaking chills
• Nausea, vomiting
• Solitary painless skin lesion
• What to think about?

Pattern Recognition

2 2 3 3 4 4 5 5

Input
Sensing
Segmentation
Feature extraction
Re-synthesis and Classification
Post-Processing Adjustment (context)
Decision / Recognition

Pattern Recognition

2 2 3 3 4 4 5 5

Input
Sensing
Segmentation
Feature extraction
Re-synthesis and Classification
*Post-Processing Adjustment (context)**
Decision / Recognition
“The Eschar”

- Cutaneous necrosis
- Characterized by the formation of a black, adherent crust
- Even though may be localized at time of presentation, represents a systemic (or potential for systemic) disorder
- Often infectious in nature, but may be toxic, embolic, vasculitic
- Context is important in decision making
## EBSCD: Ecthyma gangrenosum: Deceptively Simple Looking!

- **Manifestation of bacterial sepsis**
- **Pseudomonas**, Klebsiella, E. Coli, Serratia, rarely S. Aureus
- Solitary, painless, red swelling, may develop bulla, but rapidly forms painless eschar-covered ulcer
- Process only takes 12-24 hours
- Patient febrile and toxic-appearing
- IMMUNOCOMPROMISED, NEUTROPENIC
- IV antibiotics for presumed Pseudomonas
- Culture skin, culture blood, look for focus of infection

---

## Is this an emergency?

- 53 year-old male
- Rheumatoid arthritis
- Rx: infliximab 5mg/kg
- Arthritis controlled
- Develops fever (102.4°F)
- Shaking chills
- Nausea, vomiting
- Solitary painless skin lesion
- Pseudomonas sepsis
- Dead 32 hours later

---

## Ecthyma gangrenosum revisited

- Meta-analysis of 167 cases in literature 1975-2014
- Pseudomonas 73.65%
- Other bacteria 17.35%
- Fungi 9%
- Sick but not immunocompromised (55/167 = 33%)
- May be totally healthy (7/167 = 4.2%)

---

## Mucormycosis

- An emerging fungal infection
- Flagrant tissue necrosis
- Rapid in onset, aggressive, highly lethal
- Often in diabetic, immunocompromised
- Emergency surgery, early amputation

---

### Table: Disease Characteristics

<table>
<thead>
<tr>
<th>Disease</th>
<th>Age</th>
<th>#Lesions</th>
<th>Fever</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flap Necrosis</td>
<td>Adults</td>
<td>One area</td>
<td>No</td>
<td>Post-operative</td>
</tr>
<tr>
<td>Embolic</td>
<td>Adults</td>
<td>Few</td>
<td>No</td>
<td>CV history</td>
</tr>
<tr>
<td>Necrotizing Fasciitis</td>
<td>Adults</td>
<td>One to Few</td>
<td>Yes</td>
<td>History</td>
</tr>
<tr>
<td>Fungal sepsis</td>
<td>Any</td>
<td>Few</td>
<td>Yes</td>
<td>History</td>
</tr>
<tr>
<td>Bacterial sepsis (EG)</td>
<td>Any</td>
<td>Few</td>
<td>Yes</td>
<td>History</td>
</tr>
<tr>
<td>Mucormycosis</td>
<td>Adults</td>
<td>One area</td>
<td>Yes</td>
<td>Diabetes</td>
</tr>
<tr>
<td>Fungal sepsis</td>
<td>Any</td>
<td>Few</td>
<td>Yes</td>
<td>History</td>
</tr>
<tr>
<td>Anticoagulant</td>
<td>Adults</td>
<td>One</td>
<td>No</td>
<td>History</td>
</tr>
<tr>
<td>Calciphylaxis</td>
<td>Adults</td>
<td>One to Few</td>
<td>No</td>
<td>Renal disease</td>
</tr>
<tr>
<td>Necrotizing Fasciitis</td>
<td>Adults</td>
<td>Large area</td>
<td>Yes</td>
<td>Recent trauma, De/GU Procedure</td>
</tr>
<tr>
<td>Snake or Spider bite</td>
<td>Any</td>
<td>One</td>
<td>Maybe</td>
<td>History</td>
</tr>
</tbody>
</table>
**Mucormycosis**
- Due to one of several non-septate fungi
  - Mucor, Rhizopus, Absidia
- Acute onset pain and swelling on or near eye or nose (sinus)
- **DIABETES**
- Develops ischemia, then eschar
- Rx: Amphotericin-B (7-10mg/kg, high dose)
- Posaconazole (400mg BID, PO or IV)
- Isavuconazole Available PO or IV (372mg BID x 2 days, then QD)

**Case History**
- 75 year old diabetic
- ESRD + hemodialysis
- PICC line 8 weeks for cellulitis
- CAD, mechanical aortic valve in place
- Chills, anorexia x 3 weeks
- Temp 96.9°F
- Anemic, Azotemic, WBC >19,000

**Case History**
- 75 year old diabetic
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**Case History**
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- ESRD + hemodialysis
- PICC line 8 weeks for cellulitis
- CAD, mechanical aortic valve in place
- Chills, anorexia x 3 weeks
- Temp 96.9°F
- Anemic, Azotemic, WBC >19,000

**IV Broad Spectrum, Potent Antibiotics (?)Urinary Tract Sepsis**
BUT…..Hypothermia persists, and more lesions!

**Table:**

<table>
<thead>
<tr>
<th>Disease</th>
<th>Age</th>
<th># Lesions</th>
<th>Fever</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flap Necrosis</td>
<td>Adults</td>
<td>One area</td>
<td>No</td>
<td>Post-operative</td>
</tr>
<tr>
<td>Endocarditis</td>
<td>Adults</td>
<td>Few</td>
<td>Yes</td>
<td>CV history</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>Adults</td>
<td>One area</td>
<td>Yes</td>
<td>CBC Only</td>
</tr>
<tr>
<td>Fungal sepsis</td>
<td>Any</td>
<td>Few</td>
<td>Yes</td>
<td>History</td>
</tr>
<tr>
<td>Bacterial sepsis (CO2)</td>
<td>Any</td>
<td>Few</td>
<td>Yes</td>
<td>History</td>
</tr>
<tr>
<td>Mela infections</td>
<td>Any</td>
<td>One to Many</td>
<td>Typically Transient History</td>
<td></td>
</tr>
<tr>
<td>Anticoagulant</td>
<td>Adults</td>
<td>One</td>
<td>No</td>
<td>History</td>
</tr>
<tr>
<td>Calciphylaxis</td>
<td>Adults</td>
<td>One to Few</td>
<td>No</td>
<td>Recent disease</td>
</tr>
<tr>
<td>Fournier’s Gangrene</td>
<td>Older Adults</td>
<td>Large area</td>
<td>Yes</td>
<td>Recent G/U Procedure</td>
</tr>
<tr>
<td>Snake or Spider bite</td>
<td>Any</td>
<td>One</td>
<td>Maybe</td>
<td>History</td>
</tr>
</tbody>
</table>
### Serum 1,3-β-D-Glucan Assays

- Sensitivity 98-100%, Specificity 97-98%
- Detects serum 1-3-β-D-glucan (fungal cell wall)
  - Normal in human serum = 10-40 pg/ml
  - Negative < 60 pg/ml
  - Indeterminate 60-80 pg/ml
- Positive >80 pg/ml
- Test requires only one hour
- Detects: Candida spp, Acremonium, Aspergillus, Fusarium, Histoplasmosis, Coccioidiomycosis, Sporothrix schenckii,
- Does NOT detect: Cryptococcus, Zygomycetes

### Notes

- This patient: + at 800 pg/ml
### Candida Sepsis

<table>
<thead>
<tr>
<th>Candida species</th>
<th>Amphotericin</th>
<th>Fluconazole</th>
<th>Itraconazole</th>
<th>Voriconazole</th>
<th>Posaconazole</th>
<th>Caspofungin</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>C. albicans</em></td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
</tr>
<tr>
<td><em>C. tropicalis</em></td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
</tr>
<tr>
<td><em>C. parapsilosis</em></td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
</tr>
<tr>
<td><em>C. glabrata</em></td>
<td>S to I</td>
<td>S to R</td>
<td>S to R</td>
<td>S to I</td>
<td>S to R</td>
<td>S to R</td>
</tr>
<tr>
<td><em>C. kruzei</em></td>
<td>S to I</td>
<td>R</td>
<td>S to R</td>
<td>S</td>
<td>S to R</td>
<td>S</td>
</tr>
<tr>
<td><em>C. lusitaniae</em></td>
<td>S to R</td>
<td>S</td>
<td>S to R</td>
<td>S</td>
<td>S to R</td>
<td>S</td>
</tr>
<tr>
<td>Other species</td>
<td>All</td>
<td>Variable</td>
<td>Testing</td>
<td>Required</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Dan Med J. 2013;60(11):B4698
Swiss Med Weekly 2006;136:447-463

### Spider Bite: Brown Recluse

- *Loxoceles reclusa* (and related species)
- Painless; 8 hours later pain, erythema, swelling; progresses to ischemia and then eschar; sloughs forming ulcer
- 67-90% remain localized phenomenon
- Viscero-cutaneous form in 10-30%
  - 2-4 days after bite: Sequential Signs/Sx
  - Morbilliform rash, fever, nausea, vomiting
  - Hemolysis, thrombocytopenia, hematuria
  - Shock, DIC, acute renal failure: DEATH

Ann Emerg Med 44:608, 2004
Toxicon 44:693, 2004

### Brown Recluse Bite

10-20 days after bite
Brown Recluse Bite

- Rest, elevation, ice packs (NOT HEAT)
- NSAIDs to relieve pain and swelling
- ? Tetanus prophylaxis (debatable)
- Antibiotics: not typically appropriate
- ? Nitroglycerin patch: conflicting data
- Systemic steroids: only severe cases
- Dapsone: Variable benefit; may prolong healing time and worsen scar formation
- ? Anti-venom (contact local zoo)
- Surgery: Only late, as reconstruction

Case History

- 59 year-old welder
- Attempted to pull mouse out of cat’s mouth because the pet was choking
- After extraction, cat bit owner
- 48 hours later, developed “flu” like Sx
  - Fever (104.1°F) Mild cough, Myalgia, Arthralgia
- Axillary adenopathy: Size of “lemons”
- SOB, productive cough
- Hands and feet turn grey, then black

The “ULTIMATE” Eschar
Disease Age Lesions Fever Notes
Flap Necrosis Adults One area No Post-operative
Embolism Adults Few No CV history
Mucormycosis Adults One area Yes Diabetes
Fungal sepsis Any Few Yes History
Bacterial sepsis (BD) Any Few Yes History
Retinal Arteritis Adults One area Yes Travel History
Meningitis, Tularaemia, Anthrax, Plague Any One to Many Typically Travel History
Anticoagulant Adults One No History
Calciphylaxis Adults One to Few No Renal disease
Fournier’s Gangrene Older Adults Large area Yes Recent GI/GU Procedure
Snake or Spider bite Any One Maybe History

Plague
- Highly contagious: Rx before lab results
- Streptomycin or Gentamicin primary Rx
- After afebrile: Tetracycline / Doxycycline
- Alternate agents: Fluoroquinolones
- Prophylaxis following rodent contact in endemic area: Levofloxacin, Doxycycline
- MDR Plague: Madagascar
- Subunit vaccine in development (capsular antigens)

Case History
- 53 year-old male
- Alcoholic w/ history alcoholic hepatitis
- Drinking beer and fishing in Galveston
- Knicks his hand on needle of lure
- Hand swollen by that evening
- In 48 hours skin blisters
- In 72 hours: eschar formation
**Vibrio Vulnificus Infection**
- Most virulent food-borne infection in USA
- Consumption of raw or under-cooked oysters or shellfish from Gulf of Mexico (> during Summer)
- Also occurs with skin wound exposed to contaminated water or related to injury by contaminated marine life (shrimp, fish)
- **LIVER INSUFFICIENCY** predisposes!
- Most common in summer (more microbes)
- Ceftriaxone + Doxycycline or Minocycline
- Debridement if indicated

**Vibrio Vulnificus Infection**
- Fatality rates: >50% food-borne; 20% for wound related
- Hemorrhagic bullae and fever and history
- Progresses rapidly to necrotizing fasciitis
- Limb loss risk

**Vibrio vulnificus**
- 48 hours
- 60 hours
- 72 hours

**One More: Obvious; Tumor Necrosis**
- Metastatic Bronchogenic Carcinoma
- Primary Squamous Cell Carcinoma
- Primary Basal Cell Carcinoma
Dermatological Emergencies

• Learn to recognize key sign and symptom patterns which signify emergency
• STOP and consider that patient more carefully; don’t put that patient off or wait for loads of lab tests
• Consider hospitalization, because many of these clinically deteriorate rapidly and unpredictably
• Such patients almost always require TEAM care!
Realizing the Vision: Excellence in Dermatology
Ted Rosen, MD, FAAD
Vice-President
American Academy of Dermatology

Representing all of Dermatology
20,000 members
• Representation in AMA House of Medicine
• Media Representation and Messaging
• Assistance Navigating Changing Practice Environment
• Leadership on a Global Level
• Public education on all dermatologic conditions (> 2 billion media impressions/yr)

* Aesthetic* Medical * Surgical

Guiding Principles
• To be proactive strengthening our specialty
• To act promptly on members’ concerns and on changes in health care environment

President’s Priority
To Enhance Our Standing in the House of Medicine

Dermatology Specialty Summit - May 6, 2017
Topics discussed:
• Improving dermatology’s profile in the House of Medicine
• Role of specialty societies in improving access to care
• Demonstrating value across the specialty

AMA Dermatology Section Council (6/10-13/17)
• AMA House of Delegates: over 30 dermatologists
• The Dermatology Section Council Delegates have played a key role in passing resolutions:
  – MACRA: Led the effort to call for additional flexibility in implementation and to provide an exemption for small practices
  – Compounding: Called for the continuation of in-office compounding
  – PAs: Practice under supervision of physicians
  – Anthem/Modifier 25: helped coordinate response
President’s Priority

To Increase URM* in Dermatology

*URM = Under-represented minorities

AAD Diversity Conference – Aug 5, 2017

- AAD Leadership
- AAD Diversity TF
- Representatives from:
  - Association of Professors of Dermatology
  - Society for Investigative Dermatology
  - Skin of Color Society
  - ADA
  - Medical students

Action Steps

- Written report on Conference proceedings to be submitted to JAAD for publication = dissemination
- Prioritize recommendations from conference for further development
  - Collaboration with APD, SID, SOCS
  - AAD Diversity Champion program
  - Outreach via student organizations (eg, SNMA)
  - Expand mentorship program

AAD and the Media: 2017

- 8.8 BILLION media impressions (online, broadcast, print): Like reaching every American 26 times
- Responded to 600 media requests (acne, skin cancer)
- Media stories equivalent to >$300 million in paid advertising
- News releases and emails highlighting research in JAAD
- News release highlighting dermatologists’ expertise
- PSAs regarding early skin cancer detection and tanning

Guiding Principles

- To be proactive in strengthening our specialty
- To act promptly on members’ concerns and on changes in health care environment

AAD Website

32,000,000 visits in 2017
>26,000,000 public education site visits
> 5,000,000 member visits
“Bread and Butter” issues: AAD/A Cares

Acting promptly

Scope of Practice
Truth in Advertising

Advocacy Priority: Scope of Practice

<table>
<thead>
<tr>
<th>AMA SOPP*</th>
<th>Non-physician</th>
<th>Medical Spas</th>
</tr>
</thead>
<tbody>
<tr>
<td>Steering Committee Member</td>
<td>Nurses</td>
<td>Model legislation</td>
</tr>
<tr>
<td>$1.5 million grants awarded</td>
<td>Optometrists</td>
<td>AAD Position</td>
</tr>
<tr>
<td>Messaging and advocacy</td>
<td>Physician assistants</td>
<td>Statement on Mission</td>
</tr>
<tr>
<td></td>
<td>Aestheticians</td>
<td>Medical Spa Standards of Practice</td>
</tr>
<tr>
<td></td>
<td>Naturopaths</td>
<td></td>
</tr>
</tbody>
</table>

*SOPP = Scope of Practice Partnership
14 national med societies, nearly every state med society and 34 osteopathic medical associations

Advocacy Priority: Truth in Advertising

<table>
<thead>
<tr>
<th>Model TIA Legislation</th>
<th>Board-Certification</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Enacted in 20 states</td>
<td>• Legislation restricts claims of “board-certification”</td>
</tr>
<tr>
<td>• Introduced in 36 states</td>
<td>• Partnership with ASDA, AMA and other specialties</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>AADA TIA Toolkit</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Data, resolutions, model legislation</td>
<td>• Comment letters, media outreach template</td>
</tr>
</tbody>
</table>

SOP- TIA Triage Team

<table>
<thead>
<tr>
<th>Henry W. Lim, MD</th>
<th>Kelley Redbord, MD</th>
</tr>
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<tr>
<td>Brian Berman, MD, PhD</td>
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</table>
Actions to Date

- Triage Team e-mail and intake form
- Completed review and plans of action to tackle more than 175 cases of potential SOP/TIA violations, including working with state derm societies
- New infographics:
  - “Why See a Dermatologist”
  - “What is a Board-Certified Dermatologist?”

Acting promptly
The Practice Management Center

Opened March, 2017
As of annual meeting: ~50% of members have visited at least once, and use is steadily increasing!
430,000 page views to date

Scope of Practice – Interactive Map
The map below from AAD Practice Management Center indicates what services an NPC is allowed to perform in each state.
As of the annual meeting, >2500 members have downloaded >21,000 letters

Prior Authorization Appeal Letter Template

MOST popular feature of Practice Mgt Center

As of the annual meeting, >2500 members have downloaded >21,000 letters

MACRA Resource Center

Visit the MACRA tools to help you determine how to avoid a penalty and earn an incentive...

Acting promptly

DataDerm

DataDerm™: A Robust Clinical Data Registry

Created By Dermatologists, For Dermatologists

• Improves outcomes from registry feedback
• Informs advocacy efforts
• Provides opportunity for quality measures assessment
• Helps dermatologists with quality reporting requirements
• Validates guidelines

Dermatology Data: Interoperability is Critical for All Clinical Registries

Connects data on millions of patients from thousands of dermatologists

• Our dermatologists utilize over 60 EHR vendors, with varying levels of automatic integration with DataDerm
  – DataDerm specialists work directly with each practice individually for EHR integration
• Data is mapped and practices approve that the reports reflect their records
• There is a manual (web portal) entry option for those on paper records

DataDerm by the Numbers

965 active practices
2,700 providers submitted data in the last 12 months
5 million unique patients
11.7 million patient visits

“He who has the data, has the power!”
Do you support a process that evaluates ongoing professional competence to maintain your dermatology board certification?

72% of those who have to participate in MOC support a process that evaluates professional competence to maintain board certification.

Q2. Do you support a process that evaluates ongoing professional competence to maintain your derm board certification?

<table>
<thead>
<tr>
<th>All Respondents</th>
<th>MOC required</th>
<th>MOC not required and actively pursuing</th>
<th>MOC not required and not pursuing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>1684 (63.3%)</td>
<td>1037 (72.0%)</td>
<td>231 (37.7%)</td>
</tr>
<tr>
<td>No</td>
<td>957 (36.0%)</td>
<td>501 (27.6%)</td>
<td>367 (60.0%)</td>
</tr>
<tr>
<td>NR</td>
<td>21 (0.8%)</td>
<td>6 (0.3%)</td>
<td>14 (2.3%)</td>
</tr>
</tbody>
</table>

Total: 2662 (100.0%) 1812 (100.0%) 612 (100.0%)

The ABD/ABMS board certification should be...

- Half of respondents who are required to participate in MOC said certification should be time-limited with CME only. Those not required to participate in MOC favored once in a lifetime milestone, followed by CME.

MOC Poll – Released Oct 2017

- Results have been shared with ABD and American Board of Medical Specialties (ABMS)
- ABMS has recently announced the formation of a Commission to critically examine the re-certification process (Step in right direction)
- AAD leaders met with ABD and vigorously advocated for new MOC processes which correspond to members’ desires
When an Evaluation and Management (E&M) code with modifier 25 are billed by the same provider for the same date of service, plan will compensate the E&M service at a reduction of the otherwise allowed amount.

25% Reduction
50% Reduction

Anthem*  *change in originally announced policy

Blue Cross Blue Shield
Rhode Island
Harvard Pilgrim Healthcare
Independence Blue Cross
Tufts Health Plan

Modifier 25 Advocacy
• Led coordination and development of coalition bringing together state dermatology, state medical, and national medical societies impacted by a reduction
• Introduced and developed broad support for resolution at AMA House of Delegates urging action
– AMA-AADA coordinated efforts led to amendment of Anthem reduction to E/M from 50% to 25%
• Advocacy continued and Anthem rescinded entire proposed Modifier 25 reduction.

Federal Legislative Advocacy Wins
• IPAB repeal
• Stopped extension of misvalued “codes” policy
• MACRA Relief
– MIPS adjustment
– EHR standards
• Access to Care
– Telehealth
– Community Health Centers funding

Alex Azar, HHS Secretary  2-15-2018
• “What we’re doing is taking a whole host of physicians who not only will have reduced reporting burdens but maybe none under the MIPS part of that program.”
Regulatory Relief Advocacy Successes

- Months of advocacy with the HHS Secretary, CMS Administrator and House Ways and Means Committee
- Fewer penalties and less Medicare paperwork in 2018
- 2,800 pages of jargon = we cut the red tape!

Advocating for Access to Compounding

Office-use compounded medications. The FDA has prohibited Section 503A traditional compounding pharmacies from distributing office-use compounded medications to physician practices without a patient-specific prescription.

In-office compounding. Dermatologists are under threat of being held to strict FDA guidelines for buffering lidocaine and reconstituting botox in the office.

Access to Compounding: Progress

- Urging passage of HR 2871, the Preserving Patient Access to Compounded Medicines Act
- Educating high level FDA officials on low risk compounding in dermatology
- Successfully placing dermatologist on FDA Pharmacy Compounding Advisory Committee
- Successfully placing dermatologist on USP Expert Committee on Compounding

Seemal Desai, MD
Allison Vidimos, MD

How to Get in Touch with the AAD

www.AAD.org
Member resource center: 866-503-7546
mrc@aad.org

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Noninvasive Modalities in Lipolysis

Michelle W. Foley, DO, FAOCD, FAAD

Disclosure

Relevant Financial Relationship:
- Compensated speaker for Syneron Candela

Lecture Objectives

- Introduction and overview of the noninvasive options for in-office treatment of unwanted fat
- Discuss use of these modalities in your office
- Review treatment hazards and possible complications
- Live demonstration of a few available modalities

Why this Market Matters

According to the American Society for Aesthetic Plastic Surgery:
- Americans spent 13.5 billion in 2015 on both surgical and non-surgical aesthetic procedures, 42% of that was non-surgical
- Due to the risks associated with invasive body contouring, non-invasive procedures have increased 521% from 1997 to 2013
- Non-surgical fat reduction saw an increase from 2015-2016 of 18.7%

Reasons for this growth

- Aging population
- More men seeking treatments
- Avoidance of surgical procedures: cost, downtime, fear
- Technological breakthroughs and scientific advancements
- Media, social media, celebrity culture
The Technology: FDA Cleared

- Cryolipolysis
- Radiofrequency
- Low-Level Laser Therapy
- High-Intensity Focused Ultrasound

Cryolipolysis

- Exploits temperature sensitivity of adipocytes as compared to other water-rich cells
- Between 1940-1970, case reports showed gradual fat reduction in the lower cheeks of children that sucked on popsicles - “Popsicle panniculitis”
- Cold necrosis
- Gradual reduction over 3 months
- Erythema, numbness and bruising most common
- No change in lipids, LFTs

Radiofrequency

- Electromagnetic wave that can increase deeper skin temperature and lead to adipocyte damage
- Effective in skin tightening and fat reduction in repetition
- Exact protocols unknown and vary with each device
- Smoothing cellulite noted
Low-Level Laser Therapy

- 635nm and 532nm
- Cold red laser
- Reduces size of adipocyte via photoexcitation and release of FFAs, not cell necrosis
- SculpSure* - 1060nm - hyperthermic apoptosis of fat cells
Before and After

High Intensity Focused Ultrasound

- Used since the 1940s to treat organ tumors, uterine fibroids, kindness stones
- External transducer
- Ultrasonic energy to focal areas making molecular vibrations that increase temperature causing coagulative necrosis
- No change in LFTs or lipids

Before and After

Before and After

Minimally Invasive

- Kybella/deoxycholic acid
- FDA approved for the treatment of submental fat
- Series of injections done 4-6 weeks apart
- MANY off-label applications:
  - Back fat, bra fat, knee fat, banana rolls, abdomen, jowls, lateral neck
Before and After

In Practice

- These modalities can operate as stand alone or adjunct therapies for treating unwanted fat, loose skin, and cellulite of both face and body
- Many of the non-invasive modalities discussed do NOT have to be operated by a physician on PAIN
- Most treatments take at least 30 min of face to face time and this builds relationships, rapport, and brand loyalty
- You can offer the patient something they have already read about or watched on Dr Oz, The View, The Doctors
- Part of a multi-modality, all encompassing aesthetics practice

Possible Pitfalls and Perils

- Knowing your contraindications
- Harass, Metal, Old Scars, Pacemakers, Cold-Sensitive Disorders
- Be aware of side effects
- Pain, Swelling, Bruising, Numbness
- Nerve damage (Marginal Mandibular etc)
- Patient selection must be appropriate
- Patient expectations must be reasonable
- Remaining fat can ALWAYS hypertrophy

References

Welcome to Derm Clinic in the Bronx

Charles Gropper, MD
Chair of Dermatology
Saint Barnabas Hospital
Bronx, NY

Disclosures

• No Conflicts of Interest

Day 2 of Admission

Day 4 of Admission
History of Present Illness

• CC: 41 year old Hispanic male presented with left-sided abdominal, chest, and arm pain. Onset approximately 1 month ago after being assaulted with a lead pipe to his left flank. He received medical attention at the time, but has been increasingly sedentary and with poor appetite. Patient reports falling 2 days ago after which pain worsened. He noted purplish discoloration and swelling to his left chest. He also reports lying down on his left side for 16 hours. Also admits he has been using cocaine and heroin in the morning.

• ROS: + left chest pain, +left flank pain, +left arm pain

Past Medical History

• multiple hospital admissions for substance abuse detoxification

Past Surgical History

• none

Allergies

• NKDA

Medications

• none

Social History

– polysubstance abuse (3-5 bags daily of inhaled heroine, crack cocaine, marijuana, 4 pints EtOH daily, cigarette smoker) with multiple prior detox.
– Lives at home alone.
– Has a girlfriend.

HPI

Vitals: T 98.3, HR 88, RR 20, BP 129/89, Sat 96% on RA

Physical per ED:
– General: alert and oriented x3, acutely distressed, appears ill, restless, writhing around in pain
– Skin: “mottled skin on chest, abdomen, and extremities”

Urine Drug Screen

– Barbiturate – negative
– THC – positive
– Cocaine – positive
– Benzodiazepine – negative
– Opiates – positive

Blood alcohol – none detected

Coagulation Studies

– PT 13.8 (H)
– PTT 32.1 (H)
– INR 1.3
– Fibrinogen 552 (H)
HPI

- Imaging Studies:
  - CXR: wnl
  - CT Abdomen/Pelvis without contrast: wnl
  - CTA Chest/Abdomen/Pelvis with contrast:
    - "heart is at upper limits of normal... No acute pulmonary process seen..."
    - liver shows heterogeneous enhancement with multiple wedge-shaped perfusion defects suspicious of multifocal infarction...
    - kidney shows multiple cortical filling defects...
    - main superior mesenteric artery shows enhancement and there is decreased enhancement in the distal vessels...

Events...

- Severe sepsis:
  - Empiric antibiotics (vancomycin and zosyn)
  - IVF with NS → hypothermic at 92.7 → warming protocol
  - Hypotensive → required vasopressors
- Embolic infarct to liver, kidneys
  - Suspect endocarditis → plan for ECHO
- Acute abdomen and ischemic bowel disease
  - General surgery consulted
  - NPO
  - No surgical intervention
- Acute Kidney Injury
  - Nephrology consulted
  - Suspect dehydration and 2/2 rhabdomyolysis

Events...

- Tachypnea → sustained respiratory distressed → intubation
- Over the course of a few hours... skin eruption becomes much more prominent with reports of "bullae"... Dermatology was consulted

Differential Diagnosis

- Necrotizing Fasciitis
- Levamisole-Induced Vasculitis
- Purpura Fulminans
- Staphylococcal Scalded Skin Syndrome
- Streptococcal Toxic Shock Syndrome
- Toxic Epidermolytic Necrosis
- Pemphigus Disorder
- Calciphylaxis

Patient RL

- A. Skin, Left Upper Thigh, Punch Biopsy
- B. Left Hip
Diagnosis:

A. Skin, Left Upper Thigh, Punch Biopsy
- Epidermis without stratum corneum.
- No thrombi or vasculitis seen in the submitted sections.

B. Left Hip
- Epidermis without stratum corneum and with mixed dermal infiltrate.
- No thrombi or vasculitis seen in the submitted sections.

Comment: The histologic findings are suggestive of adult type staphylococcal scalded skin syndrome (SSSS). Clinical images were reviewed. Correlation with clinical findings is recommended.

Outline

• Leading Differential Diagnosis
  — Staphylococcal Scalded Skin Syndrome
  — Necrotizing Fasciitis
  — Purpura Fulminans
  — Levamisole-induced Vasculitis

• Patient Outcome
**Differential Diagnosis**

- **Staphylococcal Scalded Skin Syndrome**
- Purpura Fulminans
- Necrotizing Fasciitis
- Levamisole-Induced Vasculitis

**Staphylococcal Scalded Skin Syndrome**

**Background**
- Superficial blistering disorder caused by *Staph aureus*.
  - Mostly in children and neonates; rarer in adults
- **Exfoliative toxins (ETA and ETB)** by *Staph aureus*
  - Protease that target desmoglein-1
  - Separation of epidermis beneath granular cell layer
  - Spreads hematogenously

**Presentation**
- Diffuse erythematous rash
  - Begins centrally
  - Sandpaper-like \(\rightarrow\) wrinkled appearance
  - Eventual exfoliation (patchy or sheet-like)

What is the pattern of desquamation in Staphylococcal Scalded Skin Syndrome?

- A. Starts on the palms and soles
- B. Starts cephalad and goes caudal
- C. Accentuated in the skin folds
- D. Starts caudal and goes cephalad

**Workup**
- **WBC** – sometimes elevated, often normal
- **ESR**
- Bullae tissue culture - negative
- **Blood culture**
  - Children: usually negative
  - Adults: usually positive
- **PCR for toxin**

**Histology**
- Subcorneal splitting
- Sparse neutrophils
- Immunofluorescence is negative
Staphylococcal Scalded Skin Syndrome

**SUPPORTING**
- H&E with intraepidermal acantholysis

**CONTRADICTING**
- Erythema did not progress to exfoliative dermatosis
- Purpuric dermatosis
- Blood culture negative for staph aureus (+ strep)

Treatment
- **Supportive Care**
  - Fluid rehydration
  - Topical wound care
- **Antibiotics to cover Staph Aureus**

Differential Diagnosis

- Staphylococcal Scalded Skin Syndrome
- **Necrotizing Fasciitis**
- Purpura Fulminans
- Levamisole-Induced Vasculitis

Necrotizing Fasciitis

**Background**
- Rapidly progressive inflammatory infection of the fascia with secondary necrosis of the subcutaneous tissues
- Most frequently develops after trauma
- Frequency is increased in immunocompromised patients
- Mortality rate: 20-40%
- Higher rate with:
  - Female sex
  - Older age
  - Greater extent of infection
  - Delay to first debridement
  - Elevated Creatinine
  - Elevated Lactic Acid
  - 2/2 Group A streptococci
  - Organ Dysfunction

**Pathophysiology**
- Causative bacteria - aerobic, anaerobic, or mixed flora.
  - polymicrobial > monomicrobial
- Three most common:
  - Type I – polymicrobial (most common in adults)
  - Type II – group A streptococcal (most common in children)
  - Type III – gas gangrene or clostridial myonecrosis

**Presentation**
- Intense pain and tenderness
- Erythematous patch that spreads over course of hours to days
- Skin develops dusky or purplish discoloration
- Patches expand and produce large gangrenous skin
Necrotizing Fasciitis

Workup

- CBC
  - WBC >14,000
- CMP
  - Na <135
  - BUN >15
- Imaging
  - X-ray
  - Ultrasound
  - CT with contrast
  - MRI

- Finger Test
- Fascial tissue biopsy
- Blood - Group A Beta-hemolytic Streptococcus
- Tissue cultures - Group A Beta-hemolytic Streptococcus

Necrotizing Fasciitis

Supporting

- History of trauma (assault + fall)
- Intense pain
- Presentation of erythema progressing to purpura and necrosis

Contradicting

- Finger test negative
- CT/US – no gaseous process
- MRI – not done
- Fascial tissue biopsy – not done

Necrotizing Fasciitis

Treatment

- Surgical Emergency
  - SICU, burn center, or trauma center
  - Surgical debridement
- Empiric Broad Spectrum Antibiotic
- Supportive Care
  - Fluids
  - Nutritional support
  - IVIG
- Hyperbaric Oxygen

Differential Diagnosis

- Staphylococcal Scalded Skin Syndrome
- Necrotizing Fasciitis
- Purpura Fulminans
- Levamisole-Induced Vasculitis

Purpura Fulminans

Background

- Rare syndrome of rapidly progressive intravascular thrombosis or hemorrhagic infarction of skin
- Majority arise in infancy and early childhood – rare in adults
- Three forms:
  1. Neonatal - Hereditary deficiency of protein C or S
  2. Idiopathic (post-infectious) - Febrile illness (bacterial or viral) → acquired protein C deficiency
  3. Acute Infectious - Associated with infection and DIC → Most common causes: Neisseria, Group B Streptococcus, VZV

Presentation

<table>
<thead>
<tr>
<th>Neonatal</th>
<th>Acute Infectious</th>
<th>Idiopathic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Within 72 hours after birth</td>
<td>90% of cases Occurs at any age</td>
<td>7-10 days after infection</td>
</tr>
<tr>
<td>Purpuric lesions over many sites</td>
<td>Large purpuric skin lesions</td>
<td>Sudden and progressive erythema → purpura</td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>Fever</td>
<td>Abnormal coagulation factors</td>
</tr>
<tr>
<td>Possible signs of UTI</td>
<td>Hypotension</td>
<td>Major organ dysfunction</td>
</tr>
<tr>
<td>DIC</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Purpura Fulminans

Workup

- Blood cx → group A beta hemolytic streptococcus
- Diagnosis of DIC
  - Thrombocytopenia (171k → 71k)
  - ↑ PTT and ↓ PT
  - Protein C
  - Protein S
  - Antithrombin
  - Fibrinogen
  - D-Dimer

Histology

Hemorrhage
- Subcorneal splitting
- Thrombi in small vessels and mild perivascular infiltrate

Three days later
- Subepidermal bullae
- Epidermal necrosis

Supporting Contradicting

<table>
<thead>
<tr>
<th>SUPPORTING</th>
<th>CONTRADICTING</th>
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<tr>
<td>Sudden and progressive erythema → purpura</td>
<td>Sepsis with Group A Streptococcus</td>
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<tr>
<td>Abnormal coagulation factors</td>
<td>H&amp;E without thrombi or vasculitis</td>
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<tr>
<td>Major organ dysfunction (thromboembolic events to liver and kidneys)</td>
<td></td>
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</tbody>
</table>

Treatment

Neonatal Purpura Fulminans
- Platelet concentrate
- Fresh frozen plasma → low-molecular weight heparin (LMWH) → warfarin
- Debridement of necrotic tissue

Idiopathic Purpura Fulminans
- Antibiotic therapy
- Excision of gangrenous areas
- APC

Acute Infectious Purpura Fulminans
- Antibiotic therapy
- Early administration of APC
- IVIG
- Consider tPA

The Lancet

Purpura fulminans 10 years after contaminated cocaine use

43 year old male with 10 year cocaine use
- With dissemination violaceous plaques with some flaccid bullae
- Abnormal coagulation factors
- Tissue culture: streptococcus pneumoniae
- Diagnosed as purpura fulminans and vasculopathy associated with levamisole-associated cocaine
**Differential Diagnosis**

- Staphylococcal Scalded Skin Syndrome
- Purpura Fulminans
- Necrotizing Fasciitis
- **Levamisole-Induced Vasculitis**

**Levamisole**

- Synthetic antihelminthic agent used in veterinary medicine
- Limited use in humans
  - Previous treatment of autoimmune diseases, pediatric kidney diseases, infections, and cancers
  - Currently FDA approved as adjuvant chemotherapy for colon cancer

**Levamisole Toxicity**

- **Cutaneous reactions:**
  - Lichenoid drug eruptions
  - Fixed drug eruptions
  - Lichen planus
  - Vasculitis
  - Vascular occlusive disease
  - Ulcerations
  - Nodules
  - Erythema Nodosum

- **Most severe reactions:**
  - Agranulocytosis
  - Vascular Occlusive Disease
  - Thrombotic Vasculopathy (with or without vasculitis)

**Levamisole-Induced Vasculopathy**

- **Overview**
  - 1978 - First reported case of levamisole-induced vasculitis in a breast cancer patient who developed a severe cutaneous necrotizing vasculitis.
  - Over 30 cases of levamisole-induced vasculopathy with cocaine use

- **Presentation**
  - Tender purpuric plaques → bullae, necrosis, eschar, ulcers
  - Ears, cheeks, nose, and digits
  - Trunk or extremities with retiform or stellate purpura
  - Asymptomatic ↔ fever with systemic infection
Levamisole-Induced Vasculopathy

Histology

Medium power: showing focally confluent epidermal necrosis with underlying abundant extravasated erythrocytes.

High power: occlusive thrombi and leukocytoclasia.

Levamisole-Induced Vasculopathy

Workup

Neutropenia (initial WBC 1.8k)

↑ ANA
↑ Anticardiolipin antibody
↑ Lupus anticoagulant
↑ p-ANCA

Levamisole Level

• Levamisole urine or blood
  • Must be perform within 48 hours of cocaine use
  • Levamisole half life is 5-6 hours

Levamisole-Induced Vasculopathy

When should Levamisole levels be ordered?

A. < 24 hours
B. <48 hours
C. <72 hours
D. <1 week

Levamisole-Induced Vasculopathy

SUPPORTING

History of chronic polysubstance abuse (including cocaine and crack)
Tox screen +coca ine +opiates
Initial neutropenia
Purpura → necrosis
Generation of autoantibodies (↑ lupus anticoagulant)

CONTRADICTING

Urine levamisole (neg)
ANA, Anticardiolipin - wnl
ANCA studies – not done
H&E without vasculitis

Levamisole-Induced Vasculopathy

Treatment and Prognosis

• Discontinue levamisole
  – Complete clinical resolution after 2-3 weeks
  – Serologies normalize within 2 to 14 months.
• Antibiotics to treat concomitant infection
Back to our patient

- CBC
  - WBC 1.3 → 22.8 → 33.4 → 19.0
  - Pt 171 → 72
- CMP
  - Na (123)
  - Creatinine
  - BUN
- Tox Screen
  - +coca
  - +THC
  - +opiate
- Coagulation Studies
  - PT
  - PTT
  - Fibrinogen (552)
- Protein C
- Protein S
- Anti-cardiolipin Ab – negative
- Beta2-glycoprotein Ab – negative
- ANCA – not done
- Levamisole – none detected

Back to our patient...

- XR wnl
- CT w/o contrast wnl
- CTA with multi-infarcts to liver and kidney
- ECHO and TEE wnl
- Blood cx – Group A beta hemolytic Streptococcus
- Tissue cx – strep pyogenes
- Urine cx/Fungal Cx – negative
- Frozen section
- H&E – acantholytic process

Back to our patient...

- IVIG x 3 days
- Broad spectrum antibiotic/antifungal
  - Vanc and zosyn → meropenem, vancomycin, micafungin, clindamycin, gentamicin (x 1 day)
- Consultants
  - General Surgery
  - Infectious Disease
  - Dermatology
  - Heme/Onc
  - Nephrology
  - Wound Care

Day 6 of Admission

Day 6 of Admission

- Patient continued to have progression of skin involvement with full-thickness skin necrosis
  - Transferred to burn unit
- Per next of kin:
  - 3 surgical operations
  - Hyperbaric oxygen
  - Told it was secondary to “gator” (levamisole)
  - Extubated and eating PO
History of Present Illness

- **CC:** 79 year old female presented with a persistent inguinal rash. Son was present and provided history. Onset was in the spring. Patient denied any pruritus, burning, or pain. She has previously treated the site with clotrimazole cream with no improvement.
  - **ROS:** fevers, weight loss, headache, cough, sore throat, shortness of breath, diarrhea, palpitations, numbness, weakness, dizziness, urinary complaints

HPI

- **Past Medical History:** dementia, HTN, osteopenia, stress incontinence, hyperparathyroidism, BCC on nose
- **Past Surgical History:** Mohs surgery
- **Allergies:** NKDA
- **Medications:** amlodipine, calcium, vitamin D, clotrimazole
- **Social History:** Lives at home with son and has caretaker. No cigarette smoking, alcohol consumption, or illicit drugs.

Differential Diagnosis

- Intertrigo
- Contact Dermatitis
- Inverse Psoriasis
- Lichen Sclerosis et Atrophicans
- Extra-mammary Paget’s Disease
- Hailey-Hailey

Patient MQ

- **A. Skin, Left Inguinal Fold, Punch Biopsy**
St. Barnabas Pathology Report

- Diagnosis:
  - Atypical intraepithelial proliferation, most compatible with *extra-mammary Paget's disease*.
  - Diagnosis supported by positive straining with PAS special stains, CK7, EMA, and CEA. Negative for CK20.
Extra-mammary Paget’s Disease

• Background
• Clinical Presentation
• Histopathology
• Pathophysiology
• Treatment
• Back to our patient

Extra-mammary Paget’s Disease (EMPD)

Background

• In 1874, Sir James Paget reported mammary Paget’s disease (PD)
• In 1889, Crocker recognized and reported EMPD as a distinct clinical entity
• Rare form of intraepithelial adenocarcinoma
• Morphologically and histologically identical to PD
• of nipple with the primary difference being the anatomic location.

Extra-mammary Paget’s Disease

Clinical Presentation

• Typically occurs in 50-60 year olds
• Most often in Caucasians; rare in African Americans
• 4.5 Females : 1 Male
• Most cases involve apocrine-rich areas:
  – Most common site is the vulva
  – Others: perineal, scrotal, perianal, and penile skin
• Average time from symptom onset to accurate diagnosis is ~4 years

“Strawberry and Cream”
Extra-mammary Paget’s Disease (EMPD)

Histopathology
• + Mucicarmine, alcian blue, colloidal iron, PAS
• + Epithelial membrane antigen (EMA)
• + Carcinoembrionic antigen (CEA)
• + CAM 5.2
• + CK7
  – Primary EMPD = CK7+/CK20-
  – Secondary EMPD = CK+7/CK20+

Pathophysiology
• Most cases of EMPD arises as a primary cutaneous adenocarcinoma
  – Derived from pluripotential cells in the epidermis
  – Underlying in-situ adnexal carcinoma which sampling had not discovered
• 25% of EMPD are associated with an underlying in situ or invasive neoplasm (most likely adnexal apocrine carcinoma)
  – Others: carcinomas of the Bartholin’s glands, urethra, bladder, vagina, cervix, endometrium, and prostate
• 10-15% have an internal carcinoma involving rectum, prostate, bladder, cervix, or urethra

What is the significance of EMPD anatomic location?
A. Associated carcinoma
B. Survival rate
C. Depth of invasion
D. Staging

Prognosis
• Anatomic location of EMPD plays a role in predicting risk of associated carcinoma
  – Genital disease is associated with carcinoma in 4-7% of cases
  – Perianal disease is associated with underlying colorectal carcinoma in 25-35% of cases
• Dermal invasion occurs in about 20% of cases
  – Associated with decreased overall survival rate
  – One of the most important prognostic factors
Human epidermal growth factor receptor 2 protein overexpression and gene amplification in extramammary Paget disease

- Subset of EMPD cases show overexpression of HER2 (protein) and amplification of ERBB2 (gene)
- Indicator of biological aggressiveness of EMPD
  - Depth of invasion
  - Lymph-node metastases

Extra-mammary Paget’s Disease

Treatment
- Multi-disciplinary approach:
  - Dermatology, gynecology, urology, gastroenterology, surgery
- Surgery
  - Wide local excision
  - Mohs micrographic surgery
- Topical Imiquimod
- Radiation therapy
- Chemotherapy

Extra-mammary Paget’s Disease

Chemokine Receptors CXCR4 and CXCR7 are Associated with Tumor Aggressiveness and Prognosis in Extramammary Paget Disease

- Expression of CXCR4 and CXCR7 were evaluated by 92 EMPD specimen
- High expression of CXCR7 correlated with:
  - Depth of invasion
- High expression of CXCR4 and CXCR7 correlated with:
  - Regional lymph node metastasis
  - Presence of lymphovascular invasion

Extra-mammary Paget’s Disease

- EMPD frequently extends beyond clinically visible borders
- No standardized resection margin
  - Typically 1-3 cm but recurrence is common (15-43%)
  - Some advise 5 cm margins
- Mohs micrographic surgery is the best option
  - Time-consuming
  - Labor-intensive
  - Limited access
- AIM of study: Correlate conventional fluorescence diagnosis (FD) determinations with histopathologic findings

Extra-mammary Paget’s Disease

A matter of margins: Surgical and pathologic risk factors for recurrence in extramammary Paget's disease

- Medical records of 154 patients (75 F, 65 M)
- Evaluated 5-year follow-up after primary surgery
- Evaluated for risk factors associated with recurrence and margins

Compared to MMS, WLE had
- Significantly higher risk of positive margins
- Greater risk of recurrence among patients with negative margins

Conclusion
- MMS should be considered to improve outcomes for EMPD patients

A matter of margins: Surgical and pathologic risk factors for recurrence in extramammary Paget's disease

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Border of FD = 72.6% samples were +Paget cells
MSB (2 mm past the edge) = 10.3% were +Paget
Average number of stages = 1.78 (max = 3)
Maximum distance beyond FD was 12 mm
Fairly strong correlation borders of FD and histopathology
  - FD combined with MSB was more accurate than FD alone
  - May reduce recurrence rates
  - May reduce number of stages and operation time

Imiquimod
- Targets TLR 7 as a receptor agonist
- Direct antitumor activity
- Systemic review of Imiquimod as adjuvant to surgical excision
  - 71% of cases achieved complete remission
  - 16% achieved partial remission
  - Generally well tolerated with mild-to-moderate local
  - Propose that topical Imiquimod may be used to avoid repeated and mutilating surgeries.

Radiotherapy can be used
- Alternative therapeutic approach for patients with extensive inoperable disease or medical contraindications
- Adjuvant radiotherapy may be considered in presence of risk factors associated with local recurrence

Considerations:
- Prognostic factors: inguinal fold, CK7+/20-
- Son (POA) prefers non-surgical options

Referral to:
- Gyn/Oncology
- Gastroenterology
- Mohs surgeon

Back to our patient...

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- Prognostic factors: inguinal fold, CK7+/20-
- Son (POA) prefers non-surgical options

Referral to:
- Gyn/Oncology
- Gastroenterology
- Mohs surgeon
• CC: Rash on extremities
• HPI: Patient is a 17 year old female presenting with over 5 years of asymptomatic rash on arms and legs. Rash worsened by cold.
• ROS: Denies fever, chills, weight loss, hematuria, hematochezia
• PMH: N/A
• Meds: None
• Allergy: NKDA
Differential Diagnosis

- Livedo Reticularis
- Erythema Ab Igne
- Retiform Purpura
- Reticulated Erythematous Mucinosis
- Viral Exanthem (parvovirus B19)
- Dermatomyositis
- Mycosis Fungoides
Livedo Reticularis

Diagnosis:
- THROMBOTIC VASCULOPATHY, SEE NOTE

Note: The vessels are telangiectatic and contain prominent fibrin deposition. No vasculitis is identified. The differential diagnosis includes coagulation disorders, platelet disorders and cryoglobulinemia. Clinical-pathologic correlation is necessary. This case was studied and reviewed at consensus conference.

PAS stain is negative for fungi and significant basement membrane thickening, and Alcian blue stain demonstrates no significant increase in dermal mucin.

Outline

- Clinical Characteristics
- Disease Associations
- Pathogenesis
- Treatments

Livedo Reticularis (LR)

- Violaceous, red or blue, reticular or mottled pattern of the skin consisting of regular unbroken circles. Appearing “netlike”
- Livedo Racemosa- irregular broken circles. Appearing “branchlike”

Recognize

Retiform Purpura  Livedo Racemosa
Pathogenesis
• Results from alterations in blood flow though the cutaneous microvasculature
• Decreased blood flow to perpendicularly oriented arterioles and stasis of blood in venous plexus leads to the clinical findings of LR

Each arteriole supplies a hexagonal distribution of skin

Causes

Congenital
- Cutis Marmorata Congenita

Acquired w/o Systemic Disease
- Physiologic- temperature dependent
- Primary/idiopathic – some fluctuation w temp

Causes Continued

Secondary to Systemic Disease
- Vasospasm- often have connective tissue d/o (CTD)
- Vessel Wall pathology- medium sized arterioles affected
- Intraluminal pathology- hypercoaguable states

Other
Drugs (amantadine, interferon, norepinephrine), infection (Hep C), malignancy, and neurologic (reflex sympathetic dystrophy)

Causes

Livedo Racemosa
Classically associated with:
• Antiphospholipid syndrome- abortions, raynaud's, vasculitic lesions, livedo
• Sneddon's Syndrome- neurologic, labile HTN, livedo
• Rash may precede the systemic findings by years

Less common:
• PAN
• Livedoid vasculopathy
• SLE
• Polycythemia Vera
• Essential thrombocytemia
Evaluation

Serious systemic causes must be ruled out
Comprehensive history and physical needed

Ask About
- Location of lesions
- Exacerbating or remitting factors
- Duration of attacks
- Symptoms
- Symptoms of CTD
- Hx of hypercoaguability
- Infections
- Medications

Serious systemic causes must be ruled out
Comprehensive history and physical needed

Evaluation

Workup

- Biopsy - ideally a wedge or large punch in a central area of blanching
- Lab work according to history and physical
- No consensus on the best labs to order
- Lupus anticoagulant is the exception

Labs to consider include:
- CBC, CMP, PT/PTT/INR, ANA, ANCA, antiphospholipid, Factor V leiden, cryoglobulins, protein C & S, hepatitis panel, antithrombin III, serum electrophoresis

Treatment

- Treat the underlying systemic condition
- Smoking Cessation
- May try cold avoidance, leg elevation and compression stockings
- Medications have unclear efficacy in treating livedo
- Reserved for symptomatic patient with underlying systemic disease

- Anti-platelet or anti-coagulants like aspirin, clopidogrel, coumadin
- Vasodilators Ca+ channel blockers or ACE inhibitors

http://www.xdiagnosis.net/causes

Case Report of 50y/o F with ulcerations 2/2 Sneddon’s Syndrome

- Treatment with intravenous alprostadil (prostaglandin E1 [PGE-1])
- Doses of 60 μg every 24 hours for 5 days and then a dose of 60 μg every 24 hours monthly as maintenance.
- Rapid amelioration of cutaneous pain
- Within 3 months total resolution of skin lesions

- 98 individuals with history of asymptomatic antiphospholipid antibody were randomized to receive aspirin or placebo
- 48 received aspirin and 50 received placebo
- After ~1 year no significant difference in acute thrombosis incidence
- Suggests no benefit for prophylactic aspirin use in individuals with asymptomatic antiphospholipid antibody positivity
A retrospective analysis of medical records of 4 patients with a clinical and histopathologic diagnosis of LV, with high levels of lipoprotein a [LP(a)] received danazol.

LP(a) has a triple effect: pro-atherogenic, prothrombotic and antifibrinolytic.

Study showing improvement of skin lesions as well as decrease in LP(a) levels by a mean of 70% with low dose therapy, 200mg/day.

Case reports on 2 patients with Livedoid Vasculopathy treated with rivaroxaban having failed prior treatment with warfarin and non-fractionated heparin.

Both patients received 20mg PO daily and within weeks had resolution of pain and LE ulcerations.

Authors suggest rivaroxaban may be an alternative to standard anticoagulant treatments as it does not require laboratory monitoring and is easy to administer.

Strokes in Sneddon Syndrome without Antiphospholipid Antibodies

53 patients with diagnosed Sneddon’s Syndrome, negative for antiphospholipid antibody.

All had prior history of CVA or TIA.

Treated with either anti-platelet or anticoagulant for ~ 6yrs.

No significant difference in stroke recurrence between the two.

Recommend using anti-platelet therapy over anticoagulation.

Relatively new entity called DADA2 first published on in 2014.

~50 individuals now reported who developed early-onset stroke, intermittent fevers, and systemic vasculopathy.

Identified recessively inherited mutations in the CECR1 gene which encodes adenosine deaminase 2 (ADA2) which is implicated in endothelial cell and leukocyte development.

Most common manifestation is livedo racemosa.

May also have CVA, immunosuppression, portal HTN, PAN.

Treatment with TNF-a drugs may reduce CVA.

Significant overlap with Sneddon’s syndrome.
Our Patient

- Asymptomatic livedoid rash for 5 years
- Completely negative lab workup
- Biopsy showing thrombotic vasculopathy
- Normal blood pressure and ROS negative except for occasional fatigue and cold hands
- Family history negative for CTD
- Has been evaluated by both rheum and heme-onc
- Meds- Recently started an OCP
- Previously failed aspirin and plaquenil
- Considering starting her on immunosuppressant or Ca+ channel blocker

Question

- What is the ideal biopsy technique of suspected livedo reticularis?

  A. Punch of peripheral red margins
  B. Normal appearing skin outside net pattern
  C. Punch biopsy of normal appearing skin in center of net pattern
  D. Excisional biopsy of normal appearing skin in center of net pattern

HPI

- Patient is a 87yo F with pmh of CHF, HTN, DM, afib, asthma, and dementia presenting to dermatology consult service with new onset (~2 months) of painful lesion on her R clavicle. Admitted to hospital for acute respiratory failure with sepsis and pneumonia.
- ROS- Denies fever, chills, nausea, vomiting. +weight loss, decreased appetite, fatigue
- Medications- meropenem, eloquis, lantus
- Allergy- None
- Soc hx- Denies ETOH or smoking
- Fam hx- No hx of malignancy
Differential?

- Breast cancer
- Angiosarcoma
- SCC
- BCC
- Lymphoma
- Melanoma
- Other visceral metastasis
- Deep fungal
- Atypical mycobacterial
FINAL DIAGNOSIS

• Metastatic Invasive Ductal Carcinoma, Gr II (tubule formation 3/3, nuclear pleomorphism 3/3, mitotic activity 1/3, combined score 7/9 )
  – CK7 and E-Cadherin positive
• ER, PR, CK20 negative
• HER2 positive (3+ on IHC)

Dr. Richard Hwang Reviewed and approved the report
Saint Barnabas Hospital Pathology Dept.

Cutaneous Metastases

• Occur in 0.6%–10.4% of all patients with cancer
• Represent 2% of all skin tumors
• Can be challenging due to variable clinical presentation
• Leads to delayed diagnosis and poorer outcomes
• Discovery may follow or precede diagnosis of underlying visceral malignancy

Internal Malignancies (frequency)

<table>
<thead>
<tr>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lung</td>
<td>Breast</td>
</tr>
<tr>
<td>Large intestine</td>
<td>Ovary</td>
</tr>
<tr>
<td>Oral cavity</td>
<td>Oral cavity</td>
</tr>
<tr>
<td>Kidney</td>
<td>Lung</td>
</tr>
<tr>
<td>Breast</td>
<td>Large intestine</td>
</tr>
<tr>
<td>Esophagus, pancreas</td>
<td></td>
</tr>
</tbody>
</table>

Cutaneous Metastasis

• Cutaneous metastases herald a poor prognosis
• Average survival time is a few months
• May be the first sign of clinically silent visceral cancer (37% in men and 6% in women)
• Can indicate recurrence of known disease

Clinical Variation
**Pathogenesis**

- Hematogenous, lymphatic spread, direct contiguous tissue invasion, and iatrogenic implantation

Necessary steps include:

- Vessel formation (angiogenesis)
- Cell attachment
- Invasion (matrix degradation and cell motility)
- Cell proliferation

**Regional Distribution of Skin Metastases**

**Histopathologic Patterns and Features**

- Nodular, infiltrative, diffuse, or intravascular

Characteristic of the underlying causative primary tumor

- Dermal deposit of pleomorphic cells
- Mitotic figures

If poorly differentiated may require IHC staining

**Cutaneous Breast Metastasis**

- Most common metastasis to skin in women
- 3.5% presenting sign
- 80% non tender, flesh colored, rubbery, firm, nodules, ant chest wall
- Other presentation: telangiectatic carcinoma, carcinoma erysipeloid, carcinoma-encuirasse, and alopecia neoplastica
Management

- Excision: decrease in total tumor burden, improve quality of life or increased functionality (No evidence-based studies on margins)
- Treatment of primary lesion may improve cut mets
- Electrochemotherapy
- PDT
- Radiotherapy
- Intravascular
- Topical

Entailed 47 studies of 4,313 cutaneous metastases

- Response to SDT is high
- Well tolerated with low recurrence, 9.2%
- Improved QOL
- Complete response rate 35.5%
- Objective response rate 60.2%

Table 5. Multiple regression analysis of factors associated with the overall QOL score of patients with advanced or recurrent breast cancer

- Retrospective study of 24 patients
- Failed numerous other modalities
- Best results with smaller lesions <4cm²
Marked improvement in symptoms of cutaneous metastases: ulceration, pain, bleeding, infiltration, smell
• Improvements in skin often (70%) paralleled systemic
• Symptoms of disease important QOL

Our Patient
Metastatic invasive mammary duct carcinoma HER2+
Heme-onc
• Percocet for pain, Gabapentin for itch
• Mammogram
• US breast/CT abd, chest, pelvis
• PET scan
• Currently on herceptin as palliative, family refuses taxol

Question
• Which malignancy is most likely to be found as a metastasis on the scalp that can potentially lead to alopecia?
  • A. Colon CA
  • B. Pancreatic CA
  • C. Renal CA
  • D. Melanoma
  • E. Ovarian CA

Summary
• Cutaneous metastases are an infrequent, but significant finding in metastatic visceral malignancies
• Denote a poor prognosis and significant morbidity
• Various clinical presentations
• Need improved guidelines for cutaneous metastases
• Treatment can lead to improved quality of life and possibly extend survival
History of Present Illness

- A 17yo M with no significant pmh presents with itchy rash all over trunk and extremities for over a year. Rash relapses and remits. Previous treatment with topical steroids, UV and doxycycline.

- ROS: Denies any fever, chills, nausea, vomiting, blood in stools, diarrhea, arthralgia or shortness of breath
- Medications: None
- Allergy- NKDA
- Soc hx- Denies smoking or ETOH
- Fam hx- non-contributory

Differential

- Lymphomatoid papulosis
- Pityriasis lichenoides
- Anaplastic large cell lymphoma (ALCL)
- Arthropod Bites
- Dermatitis herpetiformis
- Folliculitis
- Mycosis fungoides
Diagnosis:

A. LEFT INFERIOR MEDIAL MID BACK; BIOPSY:
- Lymphomatoid papulosis, favor type A.

B. RIGHT RADIAL DORSAL HAND; BIOPSY
- Lymphomatoid papulosis, favor type A.

Lymphomatoid papulosis (LyP)

- Rare CD30+ lymphoproliferative disorder
- Chronic
- Waxes and wanes
- Self healing, possible PIH or scarring
- Appears as erythematous papules and nodules sometimes with necrotic, crusted or hemorrhagic centers, usually located on proximal legs>arms, trunk

Pathogenesis and Diagnosis

- Unknown pathogenesis
- T cell proliferation disorder
- Usually a CD4 predominant d/o, but CD8 types occur

- Diagnosis by biopsy of suspected lesions and immunohistochemical staining for CD30+ cells

Histologic Subtypes of LyP

- Type A: dermal infiltrate of large pleomorphic lymphocytes in a background of mixed inflammatory cells
- Type B: epidermotropic population of small lymphocytes mimicking mycosis fungoides
- Type C: sheets of large atypical lymphocytes in the dermis mimicking anaplastic large cell lymphoma
- Type D: epidermal hyperplasia with marked epidermotropism of atypical, variably sized CD8 positive lymphocytes and a wedge-shaped, predominantly perivascular dermal infiltrate of monomorphous cells
- Type E: dermal angiocentric, angiodestructive infiltrates of variably sized pleomorphic lymphocytes with positivity for CD4 or CD8, usually with necrosis of adjacent and overlying tissue

Pediatric LyP

- Generally similar to adults, though more rare
- 10% of LyP patients are under the age of 20 (median 7.5yrs)
- Lower risk of secondary lymphomas 5.6-10%
- Pediatric Variants:
  1. Lesions gradually decrease in size and number per outbreak until d/o ceases completely
  2. Chronic localized lesion with slow progression to generalization
  3. Presentation with hundreds of lesions
Treatment

- Some recommend no treatment due to eventual self-resolution
- Treat if severe verruca develops or symptomatic
- Secondary lymphomas cannot be prevented by pharmacologic intervention
- Lifelong follow-up every 6-12 months to monitor for malignancy

Medications:
- High potency topical steroids, ICI
- Oral/topical antibiotics
- Acyclovir/valacyclovir
- UV therapy
- Topical nitrogen mustard
- Methotrexate

- 7/25 patients had concomitant atopic dermatitis
  Suggested AD as a predisposing factor
- A different 7/25 also had preceding infection
  → Activation of CD30

H&E showed large numbers of eosinophils 40% of cases

Lymphomatoid papulosis in children: a retrospective cohort study of 15 cases

- These findings support LyP as reactional rather than neoplastic

Evaluated various Treatment options in multiple articles:
- Topical steroids utilized most often
- Antibiotics second most common—generally poor efficacy
- UV light 3rd
  - 1 treatment: 53.7%
  - 2 treatments: 26%
  - 3 treatments: 16.7%
  - 4 treatments: 1.9%

- 300-380nm UVB treatment twice weekly for 6 wks
- Full resolution of lesions with recurrence 9 months later, successfully retreated with UVB and no relapse after 12 months

- 2/5 children given methotrexate at various doses 2.5-15mg/week
- Both saw drastic improvement/total resolution of lesions
- Significant relapse upon discontinuation
- No complications

20 LyP patients included
- Administered intravenously at 1.8 mg/kg every 21 days for a maximum of eight doses
- 73% overall response rate and 35% complete response rate
- SE-Neuropathy occurred in 67%—grade 1 in 30/31 cases
  If progression to grade 2, decreased dosage
  Fully resolved for 14/31 patients in median of 41.5 wks
Our patient

- Failed PUVA, topicort spray, doxycycline
- Excimer laser showed most improvement
- Regular follow up with PCP

Question

- What is the most commonly associated malignancy in patients with LyP?
  - A. B cell lymphoma
  - B. Mycosis fungoides
  - C. ALCL
  - D. Hodgkin’s lymphoma
HPI

- 9y11m F with PMH asthma consulted for diffuse rash
- Onset: 2 weeks
- Mother reports bumps on face and legs after visiting Coney Island (7/3), believed to be mosquito bites
- Treated in the ED 7/10 with Benadryl and Calamine lotion
- Worsening rash with drainage and crusting; pruritic per patient
- No recent travel history
- No other family members with similar rash
- Admitted and treated for presumed Cellulitis with IV Clindamycin and Benadryl

Differential Diagnosis?

- Which of the following are the most common cause of eosinophilic infiltrates on histopathology?
  - Arthropod reaction
  - Drug reaction
  - Allergic contact dermatitis
  - All of the above

- Which of the following are the most common cause of eosinophilic infiltrates on histopathology?
  - Arthropod reaction
  - Drug reaction
  - Allergic contact dermatitis
  - All of the above
The most common causes of eosinophilic infiltrates include arthropod bites, drug eruptions, allergic contact dermatitis and atopic dermatitis.

Insect bites produce a wide spectrum of clinical lesions and can result in a variety of reactions. Characteristic insect bite reactions are grouped or disseminated, erythematous urticarial papules that are markedly pruritic and often excoriated. Typically, these reactions resolve over 5–10 days but may persist for weeks, sometimes reactivating when new bites occur in different locations. In addition to lesions restricted to the sites of bites, papular urticaria may develop as a generalized phenomenon following insect bites.

Most clinical manifestations relate to the individual's immune response to the bite. The immune response can include a delayed-type hypersensitivity reaction, which is mediated by T cells, and an immediate-type hypersensitivity reaction, which is mediated by IgE antibodies. The delayed-type reaction is more common and typically resolves within a few days, whereas the immediate-type reaction can cause more severe symptoms, such as hives and anaphylaxis.

Insect bites can also lead to cutaneous late-phase reactions mediated by eosinophils. These reactions are often associated with local hypersensitivity and can involve the skin, salivary gland surface proteins, and other target tissues.
**Cellulitis**

- Lower dermis and subcutaneous fat infection characterized by poorly demarcated erythema, swelling, warmth and tenderness
- The tetrad of key physical findings: rubor, dolor, calor, and tumor that are taught in medical school are in actuality non-specific markers of inflammation. As a result, there are many diseases that clinically mimic cellulitis, known as pseudocellulitides.
- In children, cellulitis most often affects the head and neck and is usually caused by S. aureus > GAS
- Cellulitis is often preceded by systemic symptoms such as fever, chills and malaise.
- The borders are usually ill-defined and non-palpable.
- Clinical diagnosis
- In the absence of trauma to both legs, bilateral cellulitis is exceedingly rare.

**Impetigo**

- Most common bacterial skin infection in children
- S. aureus
- Peak incidence in summer
- Extremely contagious
- Occurs at sites of disrupted skin barrier
- Bullous impetigo is more likely to develop on clinically intact skin
- Histology: intense neutrophilic and lymphocytic infiltrate with presence of Gram + cocci

A patient presents with recurrent itchy erythematous indurated plaques on the extremities resembling cellulitis. Which of the following statements are true?

- Peripheral eosinophilia is common
- Flame figures may be present on histology
- Systemic steroids are the treatment of choice
- All of the above
A patient presents with recurrent itchy erythematous indurated plaques on the extremities resembling cellulitis. Which of the following statements are true?
— Peripheral eosinophilia is common
— Flame figures may be present on histology
— Systemic steroids are the treatment of choice
— All of the above

Take Home Points
— History!
— Patients with atopy may often present with more robust response to insect bites
— Bilateral cellulitis is almost always impossible and very unusual
— Impetigo is a superficial self-limited infection with crusting
— Cellulitis presenting with atypical features, consider eosinophilic cellulitis

History of Present Illness
54 year old female from Peru presented with thick, tight skin.
* Onset at 16-17 years old
* Skin on her back became red then purple tight “scar-like” lesions. It progressed to involve her more of her trunk, head, left arm, and left leg.
* Moved from Peru to US with her family 4 years ago.
* ROS: difficulty ambulating, restriction of movement of knee joint, purplish discoloration of bilateral fingers in cold

A Case of Dark Spots
History of Present Illness

• CC: “dark spots”

• HPI: 66 year old Latin American male referred by his primary care physician for evaluation of asymptomatic dark spots on his trunk and extremities present for about 12 months.
  • Non-pruritic
  • No prior treatments
  • ROS: negative

History of Present Illness

• PMH: HTN, IDDM
• Meds: Lisinopril
• Allergies: NKDA
• Surgery: none
• FH: non-contributory
• SM: no alcohol, tobacco, or illicit drug use. Retired.

Differential Diagnosis?
Differential Diagnosis

- Hypertrophic Lichen Planus
- Granuloma Annulare
- Lichen amyloidosis
- Confluent and reticulated papillomatosis
- Sarcoidosis
- Deep fungal
- LSC
- Acanthosis Nigricans
- Epidermal Nevi

Histology:

Biopsy date 10/23/2017
Which plasma cell dyscrasia is most commonly associated with Scleromyxedema?
- IgG Lambda
- IgG Kappa
- IgM Lambda
- IgM Kappa

Lichen Myxedematous (Papular Mucinosis)
- Localized variant of Scleromyxedema
- Patients develop small, firm, waxy papules (or nodules and plaques produced by the confluence of papules) that are limited to only a few sites – usually the upper and lower limbs and/or trunk.
- The skin is the only site of involvement and these variants, in contrast to scleromyxedema, are not associated with sclerosis, paraproteinemia or systemic involvement, nor are they associated with thyroid disease.

Localized Variants of Lichen Myxedematous
- 4 Subtypes:
  - Discrete papular form
  - Acral persistent papular mucinosis
  - Cutaneous mucinosis of infancy
  - A pure nodular form
    - Nodular lichen myxedematous is characterized by multiple nodules on the limbs and trunk, with a mild or absent papular component.
- May be observed in association with HIV or Hepatitis C
- Incidence and prevalence unknown

Atypical Lichen Myxedematous
- Occasional patients with lichen myxedematous have atypical features or features intermediate between scleromyxedema and localized lichen myxedematous.
- Atypical classification:
  - Patients with scleromyxedema who lack a monoclonal gammapathy
  - Individuals with localized forms of lichen myxedematous who also have a monoclonal gammapathy and/or systemic symptoms
  - Localized forms with mixed features of the subtypes
  - Other not well-specified cases
Discrete Papular Type

Acral Persistent Papular Mucinosis

Cutaneous Mucinosis of Infancy

Scleromyxedema
- Generalized
- Multiple firm papules, often linear
- Face/neck
- Sclerodermoid changes
- Evidence of plasma cell dyscrasia

Lichen myxedematosus
- Localized
- Multiple papules
- Symmetric
- Trunk and Extremities
- Facial sparing
- No laboratory abnormalities

Scleromyxedema

Treatment Options
- Localized lichen myxedematosus does not require therapy and there is no definitive treatment.
- Topical application of corticosteroids, pimecrolimus or tacrolimus may be of some benefit.
- Spontaneous resolution may occur, even in the setting of HIV infection.
Clinical Course

- **09/12/2017 – INITIAL VISIT**
  - Dermatitis, unspecified
  - DDx included LSC and Prurigo nodularis
  - Treated with Triamcinolone acetonide 0.1% ointment BID
- **10/23/2017 – Follow-up**
  - No improvement with TAC
  - Punch biopsy performed of both nodular and papular/plaque component

Clinical Course

- **11/23/2017 – Follow-up**
  - Punch Biopsy: Lichen myxedematosus
  - SPEP/UPEP ordered
  - Rx Halobetasol propionate 0.05% ointment BID
- **12/18/2017 – Follow-up**
  - Improved texture and decreased pigmentation with Halobetasol per patient
  - Rx for Tacrolimus ointment
Morphea (Localized Scleroderma)
- Fibrosing inflammatory condition limited to the skin, subcutaneous tissue, bone, and (rarely) the underlying central nervous system
- Incidence of 0.5-2.7 per 100,000 people
- Female: 1 Male
- Prevalence is equal in adults and children
- In adults, peak incidence is in 3rd and 4th decade
- Prognosis and morbidity varies according to variant

Morphea – Clinical Presentation
- Early lesion: erythematous to dusky violaceous patches and plaques
- Later lesion: sclerotic, hairless, anhidrotic plaques with varying amounts of post-inflammatory hyperpigmentation

Morphea - Subgroups and Variants

Parry-Romberg Syndrome
- Hemifacial atrophy with progressive loss of subcutaneous fat, little or no sclerosis
- Distribution of trigeminal nerve (may have neuropathy)
- 20% of patients will have intracranial manifestations
- e.g. cerebral atrophy, seizures, ophthalmic changes
- Onset usually 1st and 2nd decade of life → Progressive for 2-20 years → quiescent
- Diagnosis usually done via clinical and exclusion of other diseases with cutaneous involvement

Differential
- Scleroderma
- Morphea
- Nephrogenic Systemic Fibrosis
- Scleredema
- Scleromyxedema
Thank you!

Charles Gropper, MD
Chair of Dermatology
Saint Barnabas Hospital
Bronx, NY
Sunday, March 25, 2018

6:30 a.m. - 7:30 a.m.  Breakfast with Exhibitors

7:30 a.m. - 8:30 a.m.  Prescribing Laws and Rules for Florida Licensed Healthcare Professionals
                       Edwin A. Bayo, JD

8:30 a.m. - 9:30 a.m.  Florida Laws and Rules Osteopathic Medicine
                       Jason Winn, PA, Attorney at Law

9:30 a.m. - 10:00 a.m.  Break

10:00 a.m. - 11:00 a.m.  Professional Medical Ethics
                         Ray Moseley, Ph.D.

11:00 a.m. - 1:00 p.m.  Prevention of Medical Errors
                         Arnold Mackles, MD, MBA, LHRM
Resident Poster Presentations

Triple-Hit Primary Cutaneous Diffuse Large B-Cell Lymphoma, Leg-Type: A Case Report and Review of Literature
Dana Baigrie, D.O.
OMNEE/Sampson Regional Medical Center

Necrobiotic Xanthogranuloma in Female with Monoclonal Gammopathy of Undetermined Significance
Carl Barrick, D.O.
PCOM/Lehigh Valley Health Network

Lichen Planus Pigmentosus Inversus: The Fraternal Twin of Lichen Planus Pigmentosus
Trevor Batty, D.O.
NSUCOM/Broward Health Medical Center

Isotretinoin Induced Periungal Pyogenic Granuloma Resolution with Combination Therapy
Jonathan Bellew, D.O.
MWU/OPTI/Advanced Desert Dermatology

Oral Hemorrhagic Bullae in a Case of Immune Thrombocytopenic Purpura
Conrad Benedetto, D.O.
OPTI-West/Chino Valley Medical Center

An Old World Disease in Oregon
Collin Blattner, D.O.
OPTI-West/Silver Falls Dermatology

Case Report: Aneurysmal Fibrous Histiocytoma on the Back
Devin Burr, D.O.
OPTI-West/Aspen Dermatology

Cutaneous Manifestations of Amyloidosis: Case of Biphasic Variant
Vladyslava Doktor, D.O.
LECOMT/St. John's Episcopal Hospital, South Shore

Epidermolytic Acanthomas of the Scrotum
Claire Dorfman, D.O.
PCOM/Lehigh Valley Health Network

A Rare Case of Ectopic Extramammary Paget's Disease
Charles Elias, D.O.
NYCOMECE/Palisades Medical Center
Case Report: Persistent Lofgren's Syndrome in an African-American Patient  
Maheera Farsi, D.O.  
NSUCOM/Largo Medical Center

Dermatophilus Congolensis Infection in a Hunter  
Elizabeth Foley, D.O.  
NSUCOM/Larkin Community Hospital

Acute Tissue Injury Following Tattooing: A Confocal Microscopy Analysis  
Andrei Gherghina, D.O.  
NSUCOM/Larkin Community Hospital

Histiocytoid Bullous Sweet Syndrome: A Case Presentation and Discussion  
Derek Hirschman, D.O.  
SCS/MSUCOM/Botsford Hospital

Clear Cell Acanthoma: A Clinical, Dermatoscopic, and Histological Review  
John Howard, D.O.  
NSUCOM/Larkin Community Hospital

A Rare Histologic Variant of a 'Not So Sweet' Rash  
J. Ryan Jackson, D.O.  
Still OPTI/Northeast Regional Medical Center

Atypical ANCA Associated Vasculitis with Rheumatoid Arthritis Overlap and Literature Review  
Andrew Jensen, D.O.  
NSUCOM/Larkin Community Hospital

The Clinical Importance of Cutaneous Angiomyxomas  
Karsten Johnson, D.O.  
OPTI-West/Silver Falls Dermatology

Dual CD4 & CD8 Positive Mycosis Fungoides: A Report of A Rare Cytological Phenomenon  
Ryan Jones, D.O.  
SCS/MSUCOM/Lakeland Regional Medical Center

Isolated Cutaneous Langerhans Cell Histiocytosis in a Three Months Old Infant- A Case Report  
Adeline Kikam, D.O.  
Texas OPTI/Bay Area Corpus Christi Medical Center

Granular Parakeratosis: A Case Report  
Jessica Kim, D.O.  
CEME/Palm Beach Consortium for GME

Keloid Excision with Superficial Radiation Therapy (SRT)  
Michael Lipp, D.O.  
LECOMT/Larkin Community Hospital - Palm Springs Campus

A Rare Case of Chronic Smoldering Adult T-cell Lymphoma  
Gabriela Maloney, D.O.  
CORE/O'Bleness Memorial Hospital

A Rare Conjunctival Melanoma  
Leslie Marshall, D.O.  
Still OPTI/Northeast Regional Medical Center

Cutaneous Blastomycosis: A Diagnostic Challenge  
Alyssa Miceli, D.O.  
OMNEE/Park Avenue Dermatology
Rubenstein-Taybi Syndrome
Nathan Miller, D.O.
OMNEE/LewisGale Hospital - Montgomery

Antiphospholipid Syndrome: A Case Report
Leslie Mills, D.O.
CEME/Palm Beach Consortium for GME

A Case of Perforating Granuloma Annulare
Dustin Mullens, D.O.
MWU/OPTI/affiliated Dermatology

An Atypical Presentation of Bullous Systemic Lupus Erythematosus
Robert Murgia, D.O.
OMNEE/LewisGale Hospital - Montgomery

Loose Anagen Syndrome in a 2-year-old Female: A Case Report and Review of the Literature
Anne Nguyen, D.O.
OPTI-West/Aspen Dermatology

Sebaceous Adenoma in an Immunosuppressed Male Suggestive of Muir-Torre Syndrome: A Case Report
Caitlin Porubsky, D.O.
PCOM/North Fulton Hospital Medical Campus

An Uncommon Case of Mucormycosis
Joseph Prohaska, D.O.
OMNEE/Sampson Regional Medical Center

A Case of Nevus Lipomatosus Superficialis with Features of a Spindle Cell Lipoma
Roxanne Rajaii, D.O.
SCS/MSUCOM/Botsford Hospital

Novel Use of Combination Therapeutic Plasma Exchange and Rituximab in the Treatment of Nivolumab-Induced Bullous Pemphigoid
Alyson Ridpath, D.O.
CORE/O’Bleness Memorial Hospital

A Rare Etiology of Flagellate Erythema: A Case Report & Review
Ryan Schuering, D.O.
NSUCOM/Larkin Community Hospital

Basaloid Follicular Hamartoma: Case Report and Novel Cosmetic Treatment
Kelley Segars, D.O.
NSUCOM/Largo Medical Center

Worsening Indurated Pink Translucent Nodules and Severe Hyperkeratosis of the Lower Extremities: A Case of Elephantiasic Pretibial Myxedema
Jason Solway, D.O.
NSUCOM/Largo Medical Center

DRESS Syndrome with AGEP Features
Adrian Tinajero, D.O.
LECOMT/St. John's Episcopal Hospital, South Shore

Persistent Scabs: A Case of Eruptive Pustular Dermatosis
Matthew Uhde, D.O.
CEME/Palm Beach Consortium for GME

Paronychia with Excessive Granulation Tissue as a Side Effect of Isotretinoin Treatment
Shannon Wiedersum, D.O.
NYCOMEC/Palisades Medical Center
Primary cutaneous diffuse large B-cell lymphoma, leg-type is an aggressive form of cutaneous lymphoma with an overall poor prognosis. The mean age of patients with this type of cutaneous lymphoma is 78 years old. Most of the patients affected are elderly females.¹

Patients present with rapidly growing bluish-red tumors on the lower legs. Approximately 10-20% of PCDLBCL-LT are located at sites other than the legs, and extracutaneous spread of this cutaneous lymphoma subtype is not uncommon, most commonly to the lymph nodes, bone marrow, and CNS.¹

B-cell lymphoma with two gene rearrangements, double-hit lymphoma (DHL), is uncommon, and the triple-hit type of lymphoma (THL) is even rarer. There are only case reports and a few small case series published of THL with BCL2, BCL6, and MYC rearrangements.²⁻³ They tend to have an aggressive clinical course and spread to distant sites including the bone marrow and CNS.⁴ The average survival rate for these lymphomas is reported to be approximately 4 months.⁵ THL may also show a high resistance to chemotherapy and greater likelihood of recurrence.⁶

**CASE PRESENTATION**

A 70-year-old Caucasian male presented with an enlarging skin lesion of 3-month duration on his right posterior lower thigh. Physical exam revealed a slightly tender, deeply erythematous and focally indurated plaque with irregular borders on his posterior right lower thigh. The patient denied constitutional symptoms.

PET scan revealed hypermetabolic cutaneous lesions in the left thigh and right lower thigh. Hypermetabolic activity in the left testicle was later visualized by ultrasound to represent a lymphomatous infiltration. The patient was therefore diagnosed with Stage IVA B-cell lymphoma/leukemia according to the new WHO-EORTC classification for cutaneous lymphomas: comparison with previous classifications and identification of prognostic markers. J Clin Oncol. 2008;26:4470-4478.

Histopathology revealed a dense, diffuse proliferation of atypical lymphoid cells in the dermis with hyperchromatic enlarged nuclei and scattered tingible body macrophages extending deeply into the subcuticular fat. Immunohistochemistry revealed strong positivity for CD-20 and CD-45, confirming a B-cell process of lymphoid origin. The cells were also positive for BCL2, BCL6, and negative for CD-10. C-MYC immunohistochemical studies showed strong positivity of 75%.

**CONCLUSION**

Triple-hit primary cutaneous diffuse large B-cell lymphoma, leg-type is a rare and aggressive form of cutaneous lymphoma. Similar to DHL cases, THL patients usually have an aggressive clinical course and poor prognosis. Since reports of triple-hit lymphomas are sparse in the literature, it is important to bring attention to this entity, as prognostic and therapeutic implications make solidifying a correct and early diagnosis crucial to best patient outcome.

Our patient was started on R-mini-CHOP—a decreased dose of CHOP (doxorubicin, cyclophosphamide, vincristine, and prednisone) chemotherapy with the conventional rituximab dose due to his age and comorbidities.

**References**


The authors have no conflicts or disclosures to report.
Necrobiotic Xanthogranuloma in Female with Monoclonal Gammopathy of Undetermined Significance

Carl Barrick, DO and Tanya Ermolovich, DO
Lehigh Valley Health Network, Allentown, PA

Case Presentation


History of Present Illness: Patient presents with yellow to pink dermal nodule on midline superior pubis, overlying pre-existing cesarean-section scar. A biopsy was performed and the histopathology listed a vast differential diagnosis including ruptured cyst. A few months later a similar lesion developed on the left abdomen and then left anterior neck. With further questioning a similar lesion was removed on her left anterior neck by a plastic surgeon a few years ago.

Medical/Surgical History: Monoclonal gammopathy of undetermined significance, hip replacement, cholecystectomy, breast lumpectomy, cesarean-section, hysterectomy, bladder sling, liposuction, back, shoulder surgery

Family History: Hypertension, lung cancer, non-melanoma skin cancer

Medications:

- Desoximetasone 0.25% cream, mometasone inhaler, levothryoxine sodium, vitamin D-3, calcium, curcumin, potassium, vitamin C

Current Treatments:

- Intralesional triamcinolone 0.5cc of 20mg/cc, desoximetasone 0.25% cream, ongoing hematologic monitoring under direction of hematology oncology

Physical Examination: Yellow-to-pink firm 1.3cm x 0.7cm dermal/subcutaneous nodule on left anterior neck overlying a thin scar. Additionally, patient has a 3cm x 1.4cm yellow plaque with red-brown rim on midline superior pubis and left abdomen.

Laboratory Data:

- CBC: WBC 1.0thou/cc (1.6-7.0); platelet 299,000 (150-400); hemoglobin 10.0g/dL (12.0-16.0); hematocrit 30.0% (40.0-50.0); MCV 82fl (80-100); MCH 27pg (27-32); MCHC 32g/dL (32.5-36.5); RBC 3.8 million/cumm (4.5-5.5); Differential: lymphocytes 27% (25-40); monocytes 4% (5-13); eosinophils 0% (0-5); Basophils 0% (0-1); neutrophils 69% (60-70)
- Liver function tests: AST 48 U/L (0-52); ALT 46 U/L (0-40); ALP 102 U/L (65-252); GGT 12 U/L (0-50); total bilirubin 1.0mg/dL (0.2-1.2); albumin 3.1g/dL (3.5-4.8); ESR 110 (0-30)
- Renal function: creatinine 1.1mg/dL (0.7-1.4); BUN 32mg/dL (8-22); sodium 139mEq/L (137-145); potassium 4.8mEq/L (3.5-5.1; calcium 10.8mg/dL (9.5-10.4); magnesium 2.6mg/dL (1.7-2.8)
- Thyroid function tests: TSH 0.56mIU/L (0.45-5.6); T3 1.6ng/dL (1.3-2.7); T4 6.7mcg/dL (4.9-15.6); T4U 1.3mcg/dL (0.7-2.0), reverse T3 1.1mcg/dL (0.4-2.0)
- Vitamin B12 390pg/mL (200-1200), folic acid 18ng/mL (7-20)
- Thyroglobulin 0.7ng/mL (0-16), anti-thyroglobulin antibody 0.05 (0-2.0), anti-thyroid peroxidase antibody 0.22 (0-2.0)
- Platelet function tests: P2Y, GPIIb-IIIa, GPIb-IX, platelet aggregation: ADP, collagen, ristocetin.
- Thrombotic risk acquired panel: INR 1.0 (0.8-1.2), ESR 110, CRP <0.5, D-dimer <0.5, ECG, echocardiogram, mammogram, DDimer (
- CXR, EKG, echocardiogram, mammogram all WNL

Studies: CBC, EKG, echocardiogram, mammogram all WNL

Biopsy: Advanced Dermatology Associates, LTD (AD16-12308, 10/19/2016) Left anterior neck: “Effacing the dermis is a large nodule characterized by dense collections of epithelioid cells, numerous multinucleate giant cells (many of which are Touton type) and broad ribbon-like, intersecting zones of degenerating collagen that sometimes contain prominent collections of cholesterol clefts.”

Reason for Presentation: interest

Discussion

Necrobiotic xanthogranuloma (NXG) is a rare multisystem disease with cutaneous findings that were first described by Kossard and Winkelmann in 1980. NXG is characterized by red-brown, violaceous, or yellowish cutaneous papules and nodules that progress to form infiltrated plaques. The cutaneous lesions are most commonly located in the peri-orbital region in more than 80% of patients. The trunk and proximal extremities can also present with large plaques that may present within scars. These skin lesions can grow to 25cm in diameter. The plaques may ulcerate centrally and heal with atrophic scars.

Extracutaneous involvement most commonly affects the eyes, but can involve the lungs, heart, larynx, and kidneys. Eye involvement can present as burning, itching, or eye pain. Conjunctivitis, uveitis, iritis, scleritis, keratitis, ectropion, and proptosis have been reported with NXG. In approximately 80% of NXG cases, a plasma cell dyscrasia is reported including monoclonal gammopathy of undetermined significance (MGUS), smoldering multiple myeloma, and multiple myeloma. IgG monoclonal gammopathy is the most common associated paraproteinemia. Other hematologic malignancies including non-Hodgkin lymphoma, chronic lymphocytic leukemia, Hodgkin lymphoma, and lymphoplasmacytic lymphoma have been reported in association with NXG.

Histopathology reveals areas of disrupted and degenerated collagen (necrosis) surrounded by palisading foamy macrophages. The alternating necrosis and granulomas give a layered appearance to the overall architecture of the specimen. Abnormal Touton giant cells are found throughout the affected tissue. Cholesterol clefts, lipid deposits, lymphoid follicles, and plasma cells are often seen. The histopathological differential includes necrobiosis lipoidica, which has less atypical giant cells, lymphoid nodules, and cholesterol clefts.

The treatment of NXG is directed at the underlying paraproteinemia or malignancy. Treatment of the underlying disease can lead to resolution of cutaneous manifestations. If the lesions remain persistent there are several cutaneous directed therapies including, systemic corticosteroids, interferon alpha, alkylating agents, and plasmapheresis. Patients with NXG have an increased risk of hematologic malignancies and therefore should undergo appropriate screening with hematologic oncology.

References:

Lichen Planus Pigmentosus Inversus: The Fraternal Twin of Lichen Planus Pigmentosus
Trevor Battty, DO (PGY-3), Brandon Basehore, DO (PGY-2), Asfa Akhtkar, DO, FAOCDF, FAAD
1Broward Health Medical Center Dermatology Residency, Fort Lauderdale, FL; 2Department of Dermatology, Cleveland Clinic Florida

INTRODUCTION
In 2001 Pock et al proposed the term lichen planus pigmentosus inversus (LPPI) after reporting seven Caucasian patients who presented with lichen planus pigmentosus (LPP) limited to intertriginous and flexural areas.1 There has been debate in the literature whether LPPI represents a unique disease entity or if it is a variant of LPP. LPPI may present in any skin type, and lesions typically appear on non-sun-exposed areas as non-pruritic macules that may coalesce into patches. Diagnosis is made with a correlation of clinical history, presentation, and biopsy results. We present a rare case report of LPPI with lesions similar in clinical appearance to LPP but with inverse distribution in non-sun-exposed locations. Recognition and diagnosis of this rare disease requires direct clinical-pathologic correlation in order to prevent confusion of this disease with other similar entities.

CASE PRESENTATION
A 66 year old female with no significant medical history presented to the dermatology clinic with a non-pruritic dermatitis of 3 months duration located on bilateral medial thighs and superior gluteal crease. Examination of the patient revealed well-circumscribed violaceous macules on the bilateral medial thighs and superior gluteal crease. No involvement of the gingival or oral mucosa and no perioral edema or erythema was noted. Nails were normal. Punch biopsies of lesions on the left inner thigh and popliteal fossa, inguinal, and anterior neck fold areas. Additional lesions with histopathological findings consistent with lichen planus (Wickham's striae) or lichen planus pigmentosus have been reported outside the flexural areas in approximately 10% of cases, but when present account for less than 10% of the entire area of involvement.1,2,3 The subtle differences in distribution of these lesions allows physicians to differentiate LPPI from other similar entities.

DISCUSSION
Lichen planus pigmentosus (LPP), a variant of lichen planus, was initially reported in 1974 by Bhutani et al. This case study reported dark-brown, violaceous macules located mainly on the neck, upper arms, and upper anterior chest. Other reported lesions include the axillae, intertriginous, and anogenital areas.2 There have been reports of other diseases associated with LPP, including frontal fibrosing alopecia, Hepatitis C infection, and lichen planus inversus.2,4

Lichen planus pigmentosus inversus (LPPI) is a chronic inflammatory disorder with considerable clinical overlap to other similar entities such as lichen planus pigmentosus and erythema dyschromicum perstans (ashy dermatosis). The characteristic clinical presentation of LPPI is that of non-pruritic hyperchromic dark-brown to violaceous macules and patches. These lesions are typically located in intertriginous or flexural areas and skin folds, unlike lichen planus pigmentosus which develops in photosensitized areas.4,5 Asy dermatosis is another mimic of LPPI that has a predilection for torso and limbs and demonstrates erythematous active borders.6 Larger lesions of LPPI can develop with the long axis of the lesion following cleavage lines in a linear or angular configuration. While axillary involvement is reported in greater than 90% of cases, other reported areas include the submammary, popliteal fossa, inguinal, and anterior neck fold areas. Additional lesions with findings consistent with lichen planus (Wickham’s striae) or lichen planus pigmentosus have been reported outside the flexural areas in approximately 10% of cases, but when present account for less than 10% of the entire area of involvement.2,3,4

Histopathology of previous cases have demonstrated irregular vacuolar degeneration of basal keratinocytes, orthokeratosis, marked pigmentary incontinence, and epidermal atrophy. It is theorized that the rapid onset of the intense lichenoid reaction is the cause of the epidermal atrophy and pigment incontinence without subsequent epidermal hyperplasia (as seen in lichen planus). Our patient demonstrated the characteristic hydropic degeneration of basal keratinocytes without the compensatory epidermal hyperplasia that would be expected in lichen planus. The level within the dermis of the pigment incontinence is another characteristic that helps to differentiate from the similar disease entity of ashy dermatosis. In LPPI the pigmentary incontinence is present in the superficial dermis (the location of melanin and melanophages) and is present in the deeper dermis.2,3,4

Due to the lack of published cases of LPPI and the varied results of numerous treatments, standardized treatment recommendations have yet to be determined. There are various treatment responses recorded to a number of different systemic and topical therapies. While some authors have reported a response of the condition to topical medium or high-potency steroids, there are reports of resistance to LPPI with a variety of treatments including topical steroids, oral steroids, and topical calcineurin inhibitors.4,6 In our patient we elected to initiate treatment with Fluticasone propionate 0.05% cream BID and will assess her response to treatment on subsequent follow-up visits.

CONCLUSION
Lichen planus pigmentosus-inversus is a rare variant of lichen planus pigmentosus with unique presentation, histopathological findings, and varied response to treatment regimens. The non-pruriginous nature of the lesions, clinical presentation, and histopathological findings seen in our patient are consistent with previously reported cases of LPPI. Due to the clinical overlap with other similar entities and the reported resistance to certain medications, it is imperative to correlate clinical-pathologic findings in order to recognize the disease entity and determine the appropriate treatment regimen.

REFERENCES
Isotretinoin Induced Periungal Pyogenic Granuloma Resolution with Combination Therapy

Jonathan G. Bellew, DO, PGY3; Chad Taylor, DO; Jaldeep Daulat, DO; Vernon T. Mackey, DO
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Abstract
Pyogenic granulomas are vascular hyperplasias presenting as red papules, polyps, or nodules on the gingiva, fingers, lips, face and tongue of children and young adults. They are commonly associated with trauma, but systemic retinoids have rarely been implicated as a causative factor in their occurrence. We report a case of eruptive pyogenic granulomas of the periungual region caused by isotretinoin, highlighting this important but rarely reported adverse effect of systemic isotretinoin therapy. These periungual granulomas did not resolve spontaneously with discontinuation of isotretinoin, or first line therapeutic modalities. Their resolution did occur with administration of intralesional steroids and ablation with topical silver nitrate.

Introduction
Pyogenic granulomas represent vascular hyperplasias of unknown etiology. They are characterized by rapid growth with tenderness and associated pain. Morphologically they present as a solitary red papule, polyp, or nodule that usually ulcerates and bleeds excessively with minor trauma. They may develop at any age but are more common in children and adolescents. Although idiopathic, approximately one-third develop after trauma. Most common sites include the gingiva, fingers, lips, face and tongue. Pyogenic granulomas have been reported in association with systemic retinoids, indinavir, and epidermal growth factor receptor inhibitors.

History of Present Illness
A 15-year-old male presented to our dermatology clinic with multiple painful bright red papules/papulonodules located at the dorsal surface of the distal portion of the periungual third and fourth fingers extending from the hyponychium distally down through the nail grooves with extension proximally to the nail folds of the fingernails (Figure 1). The lesions appeared abruptly and were enlarging over several weeks. Associated pain with easy bleeding on minor trauma was reported in the lesions. The patient denied significant trauma or prior contact with chemicals or allergens before the outbreak. His primary care provider initiated treatment with trimethoprim-sulfamethoxazole twice daily for two weeks. After the patient experienced no significant response to therapy, he was referred to our dermatology office for evaluation.

Management & Clinical Course
At the time of the periungal eruption on the distal fingernails, the patient was undergoing isotretinoin therapy for severe nodulocystic acne with significant scarring. He was in his fifth month of isotretinoin therapy with a cumulative dose of 140 mg/kg. He began isotretinoin therapy at a dose of 40 mg daily (0.52 mg/kg/day) for the first month and his dose later increased to 80 mg daily (1.04 mg/kg/day). Prior to undergoing isotretinoin therapy the patient was treated for three months with topical benzoyl peroxide, tretinoin, clindamycin, and oral doxycycline without clinically significant improvement. Monthly laboratory evaluations during isotretinoin therapy were within normal range with no abnormalities in the hematopoietic, renal, or hepatic systems.

The patient’s nodulocystic acne was much improved after five months of isotretinoin therapy having reached the targeted cumulative isotretinoin dose between 130 to 150 mg/kg, thus we elected to discontinue this medication in light of the patient’s painful eruption on the distal periungual nails. Local treatment to the fingernails was initiated with topical mupirocin 2% ointment in the morning and ketoconazole 2% cream at night to prevent secondary infection. Two weeks later at follow-up, the patient exhibited significant improvement. Monthly laboratory evaluations throughout three months with topical benzoyl peroxide, tretinoin, and silver nitrate over a period of six weeks led to complete resolution of these irritating lesions.

In 1983, Campbell et al. first reported the association between systemic retinoid therapy and excess granulation tissue responses. According to the available literature, a course of topical therapy with topical steroids and antibiotics under occlusion for two to three weeks is the first line treatment for periungal pyogenic granulomas.3 In our patient’s case, this local treatment along with discontinuation of oral isotretinoin was ineffective in resolving the painful nailfold pyogenic granulomas. Intralesional triamcinolone and silver nitrate over a period of six weeks led to complete resolution of these irritating lesions. Ultimately a combination of intralesional corticosteroids with silver nitrate therapy resulted in complete resolution of periungal pyogenic granulomas in our patient. We hope that this case report will assist others in the future recognition and management of this rare but painful adverse effect of oral retinoid therapy for severe nodulocystic acne.

Discussion
Excess granulation tissue and pyogenic granulomas have been described in both previous acne scars and periungal locations. Literature review illustrates rare reports of this adverse event. In addition, the mechanism by which retinoids cause excess granulation tissue of the skin is not well known. According to the available literature, a course of occlusive therapy with topical steroids and antibiotics under occlusion for two to three weeks is the first line treatment for periungal pyogenic granulomas. Literature supported first line medical treatment for pyogenic granulomas includes shave excision with electrodesication and curettage, pulsed dye laser, and sclerotherapy utilizing monooestanolamine oleate.

Conclusion
It has been reported that the resolution of excess granulation tissue secondary to systemic retinoid therapy occurs on withdrawal of isotretinoin. Unfortunately for our patient, discontinuation of isotretinoin and prevention of secondary infection in areas of excess granulation tissue was insufficient in resolving these lesions. To date, there is no consensus evidence based approach to the treatment of isotretinoin induced pyogenic granulomas. Literature supported first line medical treatment for pyogenic granulomas includes shave excision with electrodesication and curettage, pulsed dye laser, and sclerotherapy utilizing monooestanolamine oleate.

References
Oral Hemorrhagic Bullae in a Case of Immune Thrombocytopenic Purpura

Conrad Benedetto, DO; Leila Ettefagh, MD; Matthew Koebler, DO
Western University of Health Sciences/Chino Valley Medical Center

CASE PRESENTATION

Chief Complaint: Black spots on the tongue

History of Present Illness: Patient is a seventy-nine-year-old man who presented to clinic with a several day history of sudden onset of subepithelial, painless, blood filled vesicles or bullae, most commonly located on the soft palate, buccal mucosa, tongue and lips [1]. The exact etiology of ABH remains a question. Precipitating factors that have been identified include arteritis hemorrhagic, diabetes mellitus, inhaled steroid use, and oral trauma [2]. Although not commonly associated with an underlying systemic disorder, rare reports of ABH-like lesions associated with drug-induced thrombocytopenia and Dengue Fever exist [3, 4].

The differential diagnosis for ABH is expansive and includes autoimmune vesiculobullous disorders including mucous membrane pemphigoid, pemphigus vulgaris, linear IgA dermatosis, bullous lichen planus, erythema multiforme and epidermolysis bullosa acquisita; however, histopathology of ABH commonly reveals hemophilia subepithelial bullae, with nonspecific ulceration and a chronic inflammatory cell infiltrate in the lamina propria. Direct immunofluorescence staining for IgA, IgG, IgM, C3 and fibrin in it is usually negative [5]. ABH-like lesions can be mistaken for oral mucosal melanomas as the lesions appear black in color, are asymptomatic, occur in a similar age group (50 to 70) and go unnoticed until there is a breakdown of the overlying epithelium or hemorrhage [6]. The spontaneous, rapid onset of lesions make the latter diagnosis less likely.

Dermatohistopathology: A biopsy was performed on the mucosal lip for H&E that demonstrated squamous mucosa with extensive necrosis and hemorrhage; no other abnormalities were noted.

Patient Course/Treatment: The patient was sent to the ER for laboratory testing after a biopsy was performed. In the ER his platelet count was found to be 5,000 cells/µL and was positive for Influenza A. Despite initially denying any history of hematologic disorders at our clinic, he was known to the hematology/oncology service for a history of ITP. He was treated with multiple rounds of intravenous platelet transfusions and oral prednisone. His platelet count eventually rose to 10,000 cells/µL. He noted to have petechiae of the bilateral lower extremities. He reported flu-like symptoms one week prior to the onset of the oral lesions. He denied any trauma to the mouth.

Past Medical History: Hypertension, Hyperlipidemia, Immune Thrombocytopenic Purpura (ITP)

Medications: Lisinopril, Rosuvastatin

Allergies: NKA

Physical Exam: Patient is a well-nourished, well-appearing male who presented in no acute distress. Mucocutaneous exam revealed a well-delineated, soft, black nodule on the lower mucosal lip and plaque on the right lateral tongue. Skin exam revealed numerous scattered, non-blanchable, red to violaceous mucules on his bilateral lower extremities.

Laboratory Tests: Platelet count 5,000 cells/µL, nasopharyngeal swab Influenza A positive.

REFERENCES


DISCUSSION

Hemorrhagic bullae of the oral mucosa is a rare presentation of thrombocytopenia. The usual presentation of thrombocytopenia includes petechiae, prolonged bleeding from minor cuts, epistaxis, gingival bleeding, abnormal vaginal bleeding, prolonged bleeding after surgery or dental work, and hematuria or hematochezia. The most worrisome complication of thrombocytopenia is intracranial hemorrhage. There have only been rare reports of thrombocytopenia presenting with angioid bullous hemorrhagica-like lesions.

Angina bullosa hemorrhagica (ABH), first described by Badham in 1967 as “Blood Blisters in the mouth,” is a condition characterized by sudden onset of subepithelial, painless, blood filled vesicles or bullae, most commonly located on the soft palate, buccal mucosa, tongue and lips [1]. The exact etiology of ABH remains a question. Precipitating factors that have been identified include arteritis hemorrhagic, diabetes mellitus, inhaled steroid use, and oral trauma [2]. Although not commonly associated with an underlying systemic disorder, rare reports of ABH-like lesions associated with drug-induced thrombocytopenia and Dengue Fever exist [3, 4].

Oral hemorrhagic bullae in a case of immune thrombocytopenic purpura. The differential diagnosis for ABH is expansive and includes autoimmune vesiculobullous disorders including mucous membrane pemphigoid, pemphigus vulgaris, linear IgA dermatosis, bullous lichen planus, erythema multiforme and epidermolysis bullosa acquisita; however, histopathology of ABH commonly reveals hemophilia subepithelial bullae, with nonspecific ulceration and a chronic inflammatory cell infiltrate in the lamina propria. Direct immunofluorescence staining for IgA, IgG, IgM, C3 and fibrin in it is usually negative [5]. ABH-like lesions can be mistaken for oral mucosal melanomas as the lesions appear black in color, are asymptomatic, occur in a similar age group (50 to 70) and go unnoticed until there is a breakdown of the overlying epithelium or hemorrhage [6]. The spontaneous, rapid onset of lesions make the latter diagnosis less likely.

PETECHIAE, the most common presentation of thrombocytopenia, is seen with hemostatically relevant thrombocytopenias (platelets <50,000 cells/µL) including immune and thrombotic thrombocytopenic purpura, thrombocytopenia secondary to disseminated intravascular coagulation, peripheral-arterial destruction (quinine, quinidine), decreased production (chemotherapy), bone marrow infiltration or failure, and certain infections including viral illnesses [7]. Spontaneous bleeding, including intracranial hemorrhage, can occur with a platelet count less than 20,000 cells/µL [8].

Treatment for ITP includes systemic corticosteroids with or without IVIG as first-line therapy. Additional treatments include rituximab, splenectomy, immunosuppressive medications and thrombopoietin receptor agonists [9]. Treatment for an underlying cause, if one exists, should also be performed.

Despite ABH lesions presenting in patients without underlying systemic disorders in the majority of cases, it is prudent to do regular laboratory evaluations, most importantly a CBC, to rule out hemorrhagic bullae associated with thrombocytopenia and its various causes. Other authors have also urged for a keen awareness for hemorrhagic bullae of mucous membranes as they may be the only signs of severe thrombocytopenia [10, 11]. Although rarely reported in dermatologic literature, ABH is a dermatologic entity that should be recognized and an effort made to rule out underlying diseases, including thrombocytopenia, which can present similarly.
ABSTRACT

- Leprosy is a chronic and progressive granulomatous disease affecting the skin and nerves that is caused by Mycobacterium leprae[1]. Transmission is typically through nasal and oral droplets from close contact with the bacilliferous patient and less often from eroded skin [2].

BACKGROUND

Leprosy is an ancient and biblical disease that is rare in the United States. Leprosy can be classified into two major forms, tuberculoid and lepromatous leprosy, based on clinical, histological and immunological features [3]. Tuberculoid Leprosy is characterized as a Th1 reaction. Paucibacillary skin lesions are often isolated to one erythematous patch or a few asymmetric hypopigmented macules or patches and significant nerve damage with anesthesia or dysesthesia can ensue. The Lepromatous form presents with more acute onset and is characterized by increased disease burden and a Th2 response. There are hybrid borderline forms, borderline tuberculoid (BT), borderline leprosy (BB) and borderline lepromatous leprosy (BL). Standard treatment of Leprosy consists of multidrug therapy with rifampicin and dapsone, with or without clofazimine. For paucibacillary cases, the recommended treatment duration is 6 months whereas for multibacillary cases, it is 12 months [4]. If only a single lesion is present then a one time dose of rifampicin, oxofloxacin and minocycline may be given.

CASE HISTORY

- A 23-year-old female who recently immigrated to the United States from Micronesia presented with a 1-week history of painful nodules on left lower extremity. She also complained of decreased sensation throughout her upper and lower extremities. Dermatologic examination revealed bilateral leg edema and erythema. Painful subcutaneous nodules varied in size from 0.5 to 2.0 cm. Further exam demonstrated hypopigmented macules on the bilateral upper arm, legs, and trunk.

PATHOLOGY

- Histological examination demonstrated non-caseating granulomatous inflammation with numerous bacilli in the granulomas that were highlighted by the Fite stain above. Acid-Fast Bacilli smear further confirmed the diagnosis of Leprosy.

REFERENCES

Case Report: Aneurysmal Fibrous Histiocytoma on the Back

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The above physicians are part of Aspen Dermatology Residency Program in Spanish Fork, Utah.

INTRODUCTION

Dermatofibroma is one of the most common subcutaneous dermatologic tumors. In its classic variant, a dermatofibroma is easily recognized by dermatologists. However, numerous variants of the dermatofibroma have been identified which oftentimes do not present with a classic clinical picture. Aneurysmal fibrous histiocytoma, one of these variants, is not easily recognized given its bizarre growth and potentially malignant appearance. Microscopically, aneurysmal fibrous histiocytoma can be difficult to identify, as the lesion will display some similarities to a classic dermatofibroma along with distinguishing characteristics like large blood-filled cavernous spaces. Aneurysmal fibrous histiocytoma is a benign lesion with a low risk for recurrence if adequately excised. On this poster, we will present a case of aneurysmal fibrous histiocytoma and review the current literature on this rare dermatofibroma variant.

CASE REPORT

A 28-year-old healthy male presented to the clinic for evaluation of an enlarging nodule on his right scapula. He reported that it had been present for about one year and initially appeared as a 1-2 mm purple papule. It slowly grew for the first 6 months and then rapidly enlarged in size over the last half year. The patient regularly lifted weights and stated that often the squat bar rubbed against the nodule, but it had never ruptured or bled. He reported no pain with the lesion unless firmly palpated. He had no family history of cutaneous malignancy. On physical examination, this patient presented with a 3.2 x 3.2 cm exophytic, slightly scaly, well-circumscribed, spherical nodule with minimal surrounding erythema that was slightly compressible and some blanching with palpation. (SEE FIGURES 1 AND 2) The rest of his examination was unremarkable, including no lymphadenopathy. On the differential diagnosis, we considered malignant melanoma, nodular Kaposi’s sarcoma, leiomyosarcoma, pyogenic granuloma, and dermatofibrosarcoma protubersans. An excisional biopsy was performed that day. Interestingly, upon histological examination, there was a lesion which displayed some features of a classic dermatofibroma along with distinguishing characteristics like large blood-filled cavernous spaces. Histologically, on hematoxylin and eosin staining, there was a solid area that looked like the common fibrous histiocytoma given the stroma that have the potential to hemorrhage and deposit hemosiderin. Mitoses can be visualized, but atypical mitotic figures are not expected. An immunohistochemical stain for CD10, and CD68 confirmed this as a fibrous histiocytoma given its large size and not necessarily because of a biological component. One can visualize numerous small capillaries in the stroma that have the potential to hemorrhage and deposit hemosiderin. Aneurysmal fibrous histiocytomas can present histologically in a variety of ways, one must distinguish it from other vascular or fibrous tumors such as dermatofibrosarcoma protubersans, Kaposi’s sarcoma, and angiomatoid malignant fibrous histiocytoma.

Aneurysmal fibrous histiocytoma was originally described by Santa Cruz and Kyratsis in 1981. When compared to a classical dermatofibroma, the aneurysmal fibrous histiocytoma is typically larger in diameter, more elevated, and has an accelerated growth phase. The rapid growth is thought to be due to vast hemorrhage within the lesion. Patients usually do not complain of pain or tenderness. While aneurysmal fibrous histiocytoma tumors can present in various locations such as the head, neck, and trunk, they are more commonly seen on the extremities. Aneurysmal fibrous histiocytoma portrays less than 2% fibrous histiocytoma because of the large blood-filled spaces occupying up to one-half of the tumor. These spaces appear from thin clefts to broad gaping cysts lacking an endothelial lining and can be either focal or involve most of the lesion. Aneurysmal fibrous histiocytoma usually contains some solid areas that look like the common fibrous histiocytoma given the hypercellularity. One can visualize numerous small capillaries in the stroma that have the potential to hemorrhage and deposit hemosiderin. Aneurysmal fibrous histiocytomas can present histologically in a variety of ways, one must distinguish it from other vascular or fibrous tumors such as dermatofibrosarcoma protubersans, Kaposi’s sarcoma, and angiomatoid malignant fibrous histiocytoma.

Aneurysmal fibrous histiocytoma does have a good prognosis, but there is a high potential for recurrence, up to 10%. This recurrence rate is significantly higher than common fibrous histiocytoma, which occurs in less than 2% of cases. The most likely this is due to an incomplete removal of the tumor given its large size and not necessarily because of a biological component. Regular reevaluations are thus recommended to ensure that the aneurysmal fibrous histiocytoma does not recur.

CONCLUSION

Aneurysmal fibrous histiocytoma is a rare variant of the fibrous histiocytoma. While it is benign, the lesion can appear malignant and one should consider an excisional biopsy to rule out other malignant conditions, such as malignant melanoma, nodular Kaposi’s sarcoma, and angiosarcoma. Histologically, aneurysmal fibrous histiocytomas will present with large cavernous blood-filled spaces along with hypercellularity that is seen in the classic fibrous histiocytoma variant. Our patient had regular repeated trauma to the lesion from frequent pressure from a squat bar. We believe that contributed to the rapid growth of the lesion because of the increased hemorrhage. While not all cases of aneurysmal fibrous histiocytomas will increase in size from repeated trauma, we consider this as a potential cause for rapid growth in some. Given its propensity to recur if not adequately excised, we recommend regular, long-term reevaluations to ensure it does not redevelop.
The deposition of amyloid in the skin can occur as a skin-limited disorder or as a manifestation of systemic amyloidosis. In primary cutaneous amyloidosis, the deposits derive from keratin intermediate filament proteins. The various forms include macular, lichen, nodular, bicipial, and dyschomatic amyloidosis. The causative range from friction-induced, type 2 diabetes, medullary carcinoma of the thyroid, insulinoma to multiple endemic neoplasia type 2A. Systemic causes of cutaneous amyloidosis consists of plasma cell dyscrasia, chronic inflammatory disorder (e.g. rheumatoid arthritis), autoinflammatory disorders (e.g. familial Mediterranean fever) and chronic infections.

Our case describes a 33 year old female with no past medical history who presented to our clinic with pruritic mixture of reticulated and papular thin plaques on upper back and lower extremities.

The rarity of biphasic amyloidosis and its association with diabetes mellitus makes this case of interest. Our case emphasizes the importance of prompt diagnosis of cutaneous amyloidosis. This will lead to timely treatment of any possible associated systemic disease.

**Case Presentation**

A 33 year old female with no past medical history presented to clinic with pruritic mixture of reticulated and papular thin plaques to upper back (Fig. A) scapular region) and bilateral shins (Fig. B) of three years duration. Patient denied any inciting events prior to rash appearance. However, patient did complain of mild pruritus on lower extremities. After a through history, physical exam and biopsy of the upper back (Fig. D), the patient was diagnosed with biphasic amyloidosis. This is consistent with biopsy proven macular amyloidosis on the back and clinically diagnosed lichen amyloidosis on the legs. The patient was subsequently tested to rule out possible systemic causes blood work including CBC, CMP, thyroid and renal function tests, HgA1C, ESR and hepatitis panel. All but one test were within normal limits (HgA1C was 6.7).

On further questioning, patient admitted to familial history of diabetes mellitus (i.e. both parents). Patient was treated with topical corticosteroid therapy to ameliorate pruritus for 3 weeks. Subsequently, patient reported improvement not only in pruritic symptoms but also in texture and color of lower extremities (Fig. C). Patient was also advised to follow up with primary care physician for treatment of her newly diagnosis diabetes mellitus.

**Differential Diagnoses**

**Table 1: Macular Amyloidosis DDx:**
- Notalgia Paresthetica
- Tinea Versicolor
- Confluent and reticulated papillomatosis
- Drug-induced hyper pigmentation
- Exymma Dyschromicum Persians
- Actinic lichen planus

**Table 2: Lichen Amyloidosis DDx:**
- Lichen Simplex chronics
- Prurigo Nodularis
- Hypertrophic Lichen Planus
- Localized lichen myxedematous
- Pruribil myxema
- Elephantis nostri verrucosa

**Table 2: Clinical classification of amyloidosis**

- Cutaneous
  - Primary: macular, lichen, bicipial, dyschomatic, nodular
  - Secondary: incidental finding within various skin tumors (e.g. dermatofibroma, intradermal melanocytic nevi, seborrhoea keratoses, adenoid cystic carcinoma, Bowen disease, porokeratosis); following PUVA therapy
- Endocrine
  - Medullary carcinoma of the thyroid, insulinoma, type 2 diabetes
- Cerebral

Amyloidosis is considered an abnormal deposition of amyloid protein, which is made up of insoluble fibrils of beta-pleated sheets, in extracellular tissues. When it is directly deposited in the skin it is known as a primary localized cutaneous amyloidosis. Table 2 lists different types of localized amyloidosis. Biphasic amyloidosis, a rare variant, which signifies presence of concurrent lesions on macular and lichen amyloidosis. Literature describes several reports of biphasic amyloidosis in Asia and South America. However, no clear cause has been established to this date. It has been suggested that chronic friction, scratching and rubbing, for instance with towels, can cause several types of cutaneous amyloidosis. Amyloid deposition in macular amyloidosis and lichen amyloidosis is primarily caused by epidermal keratinocyte degeneration. Cytokeratin released from apoptotic basal keratinocytes is co-ordinated with autoantibodies, phagocytosed by macrophages, and enzymatically degraded to form amyloid. Patients with diabetes mellitus who inject insulin at the same site can also develop localized insulin-derived amyloidosis at the injection site.

Our patient, however, lacked history of physical rubbing or any systemic illnesses at initial presentation. As it was discovered after thorough testing, patient had diabetes mellitus. Recent studies have found that degeneration of pancreatic islet cells that is found in diabetes mellitus is also associated with biphase amyloidosis. Perhaps this plays importance in our patient and offers a plausible explanation on the origins of cutaneous presentation. Endocrine connection has also been established between localized amyloidosis and Multiple Endocrine Neoplasia Type 2, a group of endocrine disorders. More specifically, medullary thyroid carcinoma is associated with amyloid production.

**Treatment**

There are some anecdotal cases of various treatment options. One case of 73-year-old man with a 15-year history of biphase amyloidosis was treated with acitretin 35 mg once daily dose (0.5 mg/kg/day) for 8 months with success. Another report case of 26-year-old woman with a 7-year history of biphase amyloidosis was treated with topical tacrolimus ointment 0.1% and narrow band ultraviolet B treatment simultaneously for 8 weeks. At that point significant improvement was noticed, however, the patient had moved away from the treatment center and could not continue with the treatment any longer. More common therapeutic options include antihistamines, potent topical corticosteroids, keratolytic agents, intralesional corticosteroids, and capsain. It is certainly important to hold a discussion on avoidance of aggravating factors such as itching or rubbing the affected skin areas.

**References**


**Epidermolytic Acanthomas of the Scrotum**

Claire O. Dorfman, DO and Tanya Ermolovich, DO

Lehigh Valley Health Network, Allentown, PA

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**Case Presentation**

**Patient:** 64 year-old Caucasian male.

**History of Present Illness:** The patient presented in August 2016 with multiple, new, asymptomatic growths located on the scrotum. He denied itching, pain or burning. The patient was treated with Imiquimod 5% cream 3 times per week. After 4 weeks of treatment, he developed erythema and irritation primarily on the bilateral inguinal creases and penile shaft, without significant erythema on the scrotum. Imiquimod was continued for a total of 10 weeks. There was little improvement of the scrotal lesions. Interestingly, the patient was treated for pruritus scroti from 2009 to 2011.

**Medical/Surgical History:** Hypertension, asthma, benign prostatic hyperplasia, rosacea, seborrheic dermatitis, folliculitis, basal cell carcinoma, tonsillectomy, appendectomy, wisdom tooth extraction

**Family History:** Hypertension, heart disease, asthma

**Medications:** Lisinopril-hydrochlorothiazide, aspirin, tamsulosin, rosuvastatin, ciclesonide inhaled, albuterol inhaled, pseudoephedrine, azelastine/fluticasone propionate nasal, doxycycline 20mg BID, ketoconazole 2% shampoo, ketoconazole 2% cream, metronidazole 0.75% gel, clindamycin 1% gel

**Previous Treatment:** Imiquimod 5% cream 3 times per week for 10 weeks total, hydrocortisone 2.5% cream, triple paste

**Allergies:** Sulfonamides

**Physical Examination:** Multiple tan to light pink flat-top 2-3mm keratotic papules located on the scrotum.

**Biopsy:** Advanced Dermatology Associates, LTD. (AD16-09835, 8/29/2016) Left inferior scrotal sack: "Epidermolytic acanthoma is rendered." The lesion was biopsied and histopathologic analysis revealed compact hyperkeratosis, and epidermolytic hyperkeratosis.

**Diagnosis:** Epidermolytic acanthoma is a rare benign tumor that can appear in two varieties; an isolated form which was initially described in 1970, as well as a disseminated form described in 1973. Individual lesions appear as an asymptomatic tumor less than 1cm in diameter with a verrucous surface. Epidermolytic acanthomas may occur anywhere on the body with a predilection for the trunk, although involvement of the genitalia has been increasingly reported. They present during adulthood, with no particular predilection for a particular race or gender.

**Discussion**

Epidermolytic acanthoma is a rare benign tumor that can appear in two varieties; an isolated form which was initially described in 1970, as well as a disseminated form described in 1973. Individual lesions appear as an asymptomatic tumor less than 1cm in diameter with a verrucous surface. Epidermolytic acanthomas may occur anywhere on the body with a predilection for the trunk, although involvement of the genitalia has been increasingly reported. They present during adulthood, with no particular predilection for a particular race or gender.

Epidermolytic hyperkeratosis is a characteristic histopathologic feature consisting of compact hyperkeratosis and vacuolar degeneration of keratinocytes in the spinous and granular layers. These findings are also seen in bullous congenital ichthyosiform erythroderma, which is also known as epidermolytic hyperkeratosis (EHK). A genodermatosis caused by mutations in keratin 1 and keratin 10 genes. Therefore, it has been hypothesized that epidermolytic acanthomas may represent a localized variant of generalized EHK. However, recent studies have failed to find gene mutations associated with the isolated form.

The pathogenesis of epidermolytic acanthomas remains unclear. Both clinically and histopathologically, these lesions are often mistaken for verrucae and condyloma acuminata. Therefore, a viral etiology has also been considered. There has been little evidence of human papillomavirus DNA found in these lesions, although Jung et al. recently reported a case of multiple epidermolytic acanthomas on the scrotum associated with human papillomavirus 16. Other exogenous factors may play a role such as other viral infections, ultraviolet radiation, sunburn, immunosuppression, and trauma.

Treatment is not necessary unless the appearance or associated symptoms are particularly bothersome to the patient. Some treatment modalities include surgical excision or other destructive options such as cryotherapy. Jang et al. also reported a case of multiple epidermolytic acanthomas successfully treated with topical imiquimod, and Tan et al. reported effective treatment of associated pruritus with 0.1% tacrolimus ointment.

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**References:**


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A Rare Case of Ectopic Extramammary Paget’s Disease

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Hackensack Meridian Health Palisades Medical Center Dermatology Residency

Abstract
Extramammary Paget’s disease is a rare cutaneous neoplasm, sometimes associated with internal visceral malignancies. It is very rarely encountered in “ectopic” areas that are not associated with the presence of apocrine glands. Treatment with wide surgical excision can be curative although recurrences are not uncommon. We present a case of ectopic extramammary Paget’s disease found on the forearm of a male.

Case
A 67 year old male presented with an asymptomatic lesion on his left dorsal forearm that had been present for about 5 months. Surgical history was significant for a normal colonoscopy 4 years prior to presentation. His only medication was daily aspirin. On examination, the patient was found to have an erythematous, eczematous-appearing patch on his left dorsal forearm, measuring about 3x1 centimeters. A shave biopsy was obtained which showed intraepidermal proliferation of large, atypical, epitheloid cells arranged both as small nests and single cells. The tumor cells were described as having ample cytoplasm, prominent nuclei, and conspicuous nucleoli. These findings were interpreted as being consistent with extramammary Paget’s disease. The patient returned to the office for wide local excision. Pathology showed residual extramammary Paget’s disease with clear margins. The patient was referred to oncology to rule out occult malignancy. Work-up, including blood tests, radiological scans, and colonoscopy were negative.

Discussion
Extra-mammary Paget’s disease (EMPD) is a cutaneous, intraepithelial adenocarcinoma found outside of the breast. There are rare cases in which EMPD has been found in areas of the body that do not typically contain apocrine glands. These cases have been referred to as “ectopic” EMPD. 3 Ectopic EMPD cannot be distinguished from EMPD other than by its location in a non-apocrine bearing area. While it remains unclear how Paget’s disease arises in non-apocrine bearing skin, it is hypothesized that an epidermal multipotent stem cell may eventually gain an apocrine phenotype and result in EMPD occurring in non-apocrine gland bearing areas. 4 Clinically, EMPD presents as red to brown plaques with secondary changes of scale or ulceration. 4,3 The location of the lesion on apocrine bearing skin may provide a clue in the diagnostic process. For this reason, ectopic lesions require a high level of clinical suspicion and necessitate a biopsy.

Treatment of EMPD is with wide local excision, although there are high rates of recurrence. Moh’s micrographic surgery and 5-fluorouracil have also been used successfully.

References
Case Report: Persistent Lofgren’s Syndrome in an African-American Patient

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INTRODUCTION

HPI: 31-year old African-American male presented for evaluation of a mildly tender lesion on the nose for 3 months duration. Prior evaluation by pulmonology for a chronic dry cough revealed bilateral hilar lymphadenopathy, biopsy on mediastinoscopy was inconclusive. The patient was placed on a prednisone taper, and 10mg daily of prednisone thereafter without improvement of symptoms.

PMHx, SoFx, FamFx – Non-contributory.

ROS: (+) migratory arthralgias, fatigue, fevers, dry cough, visual changes.

PE: 3 mm skin-colored papule with yellowish-green hue on nasal tip (Fig. 1). Scattered 0.5-1.5 cm firm, slightly tender subcutaneous nodules on the left upper arm (Fig. 2, Fig. 3), and right lower back (Fig. 4).

LABORATORY DATA

Table 1

<table>
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<tr>
<th>Test</th>
<th>Result</th>
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</thead>
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<tr>
<td>Acid Fast Stain</td>
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<tr>
<td>GMS Stain</td>
<td>Negative</td>
</tr>
<tr>
<td>Tissue Cultures</td>
<td>Negative*</td>
</tr>
<tr>
<td>Quantiferon Gold</td>
<td>Negative</td>
</tr>
<tr>
<td>Angiotensin Converting Enzyme</td>
<td>172 U/L (Elevated)</td>
</tr>
</tbody>
</table>

DISCUSSION

Lofgren’s syndrome is an acute form of sarcoidosis characterized by the triad of hilar lymphadenopathy, polyarthralgias, and erythema nodosum.

There is a bi-modal age distribution with peaks at ages 25-35 and 45-65 and it is more common in females and patients of Scandinavian and Irish descent; rare in African-Americans.

Patients may exhibit symmetric red-brown or yellow-brown to erythematous-violaceous papules and plaques. Most cases resolve spontaneously within 1 year. After onset of symptoms, 8% of patients have active disease at 2 years; 6% have recurrent episodes up to 20 years after diagnosis.

Elevated ACE level at diagnosis predicts persistent or recurrent disease. This patient was symptomatic with elevated ACE level despite being on daily prednisone.

The duration of symptoms in our patient along with his ethnic background make this case a unique and interesting presentation of Lofgren’s syndrome.

REFERENCES

Dermatophilosis congolensis infection in a Hunter

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Abstract:
Dermatophilosis congolensis (D. congolensis) infection is a bacterial infection that primarily affects animals and is rather uncommon in humans. Clinically, it presents with cutaneous manifestations such as crusted, pustular, or nodular lesions, and aerobic colonization may be demonstrated through the Gram stain. In the present case report, a 76-year-old male hunter who gutted and cleaned the animal with his bare hands subsequently developed lesions on his hands and flank. The lesions were characterized with respect to dermatopathology, which revealed Gram-positive bacilli and thick, sebaceous, branching filaments found only in upper epidermal layers. No organisms were found in the dermis.

Background:
Dermatophilosis congolensis is an aerobic and facultative anaerobic Gram-positive bacillus that primarily affects animal species causing dermatophilosis. It most commonly affects cattle, sheep, horses, and goats. A review of the literature reveals that this organism rarely infects humans. Dermatophilosis is most commonly associated with cattle, sheep, goats, and horses but has also been reported in humans. D. congolensis is an aerobic and facultative anaerobic Gram-positive bacilli. It is catalase and urease positive, and it does not grow on Sabouraud dextrose agar, MacConkey agar, or Lowenstein Jensen medium. This organism does not grow on human skin tissue culture, which is consistent with the observation that human infections are self-limiting and may resolve without treatment, but can recur if the skin remains moist.

Microscopic Findings:
Figure 1. Scaly, erythematous papule with crusted lesion on dorsal aspect of right wrist. Figure 2. Clear view of lesion or denudation of skin aspect of right wrist. Figure 3. Thick yellowish-crusted nodule with scale and surrounding erythema on right flank. Figure 4. H&E. Faint patchy hyperplasia lesions on right dorsal wrist revealed subepithelial parasites with filaments. Figure 5. GMS revealed Gram-positive bacilli thick, sebaceous branching filaments found only in upper epidermal layers. No organisms were found in the dermis.

Discussion:
Dermatophilus congolensis infection is very uncommon in humans. For its case presented, the differential diagnosis included dermatozoonosis and Franciscella tick-borne encephalitis but both were ruled out and the organism and clinical picture of the patient are consistent with the case of D. congolensis described in 1915 by van Saceghem. The patient was successfully treated with oral minocycline at 100 mg twice a day and topical mupirocin ointment twice a day for 30 days. He was seen three weeks after initial presentation and reported resolution of his lesions. No evidence of recurrence was noted on 2 months follow-up examination.

References:

Conclusion:
The case presented in this report illustrates a rare example of Dermatophilus congolensis infection in an individual who handled and cleaned the carcass of a frozen raccoon that was frozen for more than a year. This finding indicates that bacteria can remain viable in the frozen tissue and reach the rewarmed tissue. Dermatophilus congolensis has a "watch and wait approach" would have been an acceptable alternative to the patient. It has been suggested that D. congolensis infections are self-limiting and may resolve without treatment, but can recur if the skin remains moist. Although this transmission mechanism is not entirely known, transmission of infection in the environment is often associated with contact with infected animals or their carcasses. The lesions do not appear to be contagious, and there is no evidence of spread to others. Despite the rarity of this condition, it is important to keep this entity in the differential diagnosis when working up cutaneous lesions in humans who are exposed to wild or domesticated animals.

DISCUSSION (cont’d)
Although the transmission mechanism is not entirely known, transmission of infection in the environment is often associated with contact with infected animals or their carcasses. The lesions do not appear to be contagious, and there is no evidence of spread to others. Despite the rarity of this condition, it is important to keep this entity in the differential diagnosis when working up cutaneous lesions in humans who are exposed to wild or domesticated animals.

Dermatophilus congolensis
D. congolensis is an aerobic and facultative anaerobic Gram-positive bacilli. It is solitary and sparse, and generally grows in thin agar at 37°C in 5% CO2. Colonies are browntan and grow to size 0.5 to 1 mm after 24 hours. This organism does not grow on Sabouraud dextrose agar, MacConkey agar, or Luria-Bertani broth media.
Acute Tissue Injury Following Tattooing: A Confocal Microscopy Analysis

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Abstract

Purpose
To define progressive cytarchitectural changes after acute tissue injury days to weeks after acquiring a tattoo, using in vivo reflectance confocal microscopy (RCM).

Method and Design
We report the use of in vivo RCM in the investigation of acute tissue injury days to weeks after acquiring a tattoo, and examine changes in the stratum corneum, the spino-granular (SG) layer, and at the dermal-epidermal junction (DEJ). Patients were examined in a clinic setting using the Calleo ID Vitalscope 1500 multi-laser reflectance confocal microscope. Encounters were carried out as follows:

<table>
<thead>
<tr>
<th>No</th>
<th>Present</th>
<th>Procedure</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>Post-patellar</td>
<td>Post-tattoo day 10</td>
</tr>
<tr>
<td>2</td>
<td>L-shape</td>
<td>Baseline, &amp; post-tattoo day 3, 10, 17, 24</td>
</tr>
<tr>
<td>3</td>
<td>L-shape</td>
<td>Baseline, &amp; post-tattoo day 3, 7, 10, 17, 24</td>
</tr>
</tbody>
</table>

Discussion

Acute tattoo-related complications of the skin

Needle trauma

Ink foreign reaction

Reaction to ink-diluent

Reaction to alcohol and post-tattoo creams

Material-related trauma

Infectious

Material causally normal skin flora

Contaminated water used to dilute ink

Viral, fungal, allergy, parasitic, chronic plaque, tumors

Non-Reflected

Feasibility of tattoo colors

Pit: most common color causing DTR (pinprick, x-ray)

Black: most common color used, second most common cause of DTR

Green: chronic tattoo often reactive on patch testing

Other colors: scar-like granulomatous reactions, keloidal reactions

Histopathology of acute tissue injury

In the first 24 hours, neutrophils phagocytose the pigment, later followed by evidence of pigment aggregates in keratinocytes, macrophages, mast cells, and fibroblasts. Eventually a lymphocytic response may emerge as Langerhans cells. The disruption of the basement membrane allows pigment to travel back to the epidermis from the dermis. This is termed “transdermal elimination” and can be seen one month later with pigment in macrophages, and fibroblasts. Transdermal elimination ceases once the basement membrane renews. Pigment however may still be evident in corresponding lymph nodes.

Older tattoos show pigment in mononuclear cells, fibroblasts, and extracellular tissues. Larger particles remain in the dermis, too large to be transported. Later these particles are found within dermal fibroblasts as a nonspecific response of foreign nontoxic substances. As tattoo continues to age, the pigment absorbs further into the dermis, eventually draining to lymph nodes.

Introduction

Tattooing has increased exponentially in recent years, being embraced, especially in the younger generation, where approximately 50% of current Americans aged 18-25 and 40% of Americans aged 26-40 are presently tattooed. 1,2 Interestingly, in a large-scale trial of increased scrutiny and regulation, tattooing is largely unregulated and is regarded by some as an ongoing human experiment on the injection of chemicals into the human skin, where adverse reactions are not uncommon. 3,4

Tattoos have the potential for a variety of complications including bacterial, viral, fungal infections, and allergic reactions, and even localized skin disease such as poikiloderma and keloid plaques, and tumors have been documented. A majority of these complications are probably due to the implantation of unsterile pigments in unsterile settings. 5,6,7 The extent of such complications, often occurring days, months, or even years later. 8 These adverse events can be seen in medical uses of tattooing as well, which have recently increased in the cosmetic and reconstructive medical arena.

Results

Patient 1 was imaged on only one occasion, post-tattoo day 10. On this day they had similar results to patients 2 & 3 on their corresponding post-tattoo day 17 & 24. Patients 2 & 3 had similar results on every examination from their baseline pre-treatment to the last imaging. Patients 2 & 3 were tattooed by the same person using similar equipment, however Patient 1 was tattooed elsewhere, likely with different pigment and technique.

Patient 1

- Disrupted architecture in the stratum corneum in locations of needle entry, shown as hyperreflectivity areas.
- Hyporeflective areas with an inflammatory cell infiltrate.
- SG layer has multiple spindle-shaped cells consistent with Langerhans cells. These dendritic cells appear grouped together.

Patient 2 & 3

- Disrupted architecture in the stratum corneum in locations of needle entry, shown as hyperreflectivity areas.
- Hyporeflective areas with an inflammatory cell infiltrate.
- SG layer has multiple spindle-shaped cells consistent with Langerhans cells. These dendritic cells appear grouped together.

Above: Photos are of black rose tattoo on patient 1 at post-tattoo day 10. (A) Dermascopic photo. (B) RCM image of the stratum corneum. The tattoo design can be noted by transeuclamerasted areas of needle entry noted here as hyporeflective areas. (C) Close-up of cornal layer showing traumacised areas surrounded by a typical honeycomb pattern and several hair follicles. (D) RCM image of SG layer. (E) Close-up of SG layer showing hyperreflective spindle-shaped Langerhans cells present in increased density in areas of trauma. (F) Confocal features of tattoos. (F) Patient 2 & 3 on post-tattoo day 3, 7, 10, 13, (G) Patient 1 on post-tattoo day 10 as well as patient 2 & 3 on post-tattoo day 17, 24. Note: this correlates with the above confocal images of patient 1 at post-tattoo day 10.

Conclusion

We are the first to describe progressive cytarchitectural changes using RCM in patients with pre-procedure baseline confocal images. Although a lymphocytic response to tattooing has been described, confocal images of these changes in the form of a lymphohistiocytic infiltrate including Langerhans cells has not yet been shown to be the best of our knowledge. This work has the potential to enhance understanding of how tattooing leads to inflammatory cell recruitment and ultimately the dynamics of what drives the degradation of permanent tattoos. This work is especially significant in an era where tattoos have gained popularity and acceptance both as a form of art and as a reconstructive modality, and are seen in a growing population of adults.

References

Safety

No adverse events were observed or reported during the study.

No adverse events were observed or reported during the study.

No adverse events were observed or reported during the study.

No adverse events were observed or reported during the study.
INTRODUCTION

Histiocytoid Sweet Syndrome (HSS) is a rare inflammatory cutaneous disease classified as a histological variant of Sweet's Syndrome (SS) also known as acute febrile neutrophilic dermatosis.  Described in 1964 by Robert Douglas Sweet, SS often presents as a sudden onset of painful erythematous plaques and nodules associated with fever, leukocytosis, and neutrophilia.  Microscopically, lesions have dense diffuse infiltration of mature neutrophils with papillary dermal edema.  In 2005, Requena, with colleagues, classified HSS as a variation of SS.  Although the two are undistinguishable clinically, microscopically the diseases show infiltrate of different cell lineage.

CASE REPORT

Patient is a 62-year-old Jordanian female who presented with an acute onset of painful, pink-to-flesh colored, edematous annular plaques with associated hemorrhagic bullae affecting mainly the bilateral ventral forearms and bilateral hands.  Clinical presentation is illustrated in Figures 1-3.

No further workup was deemed necessary by the hematology/oncology department at this time.

DISCUSSION

The pathogenesis of SS continues to be unknown, but SS often presents in three clinical settings: classical (or idiopathic), drug induced, or malignancy associated (especially myeloid leukemias and myelodysplastic syndromes).  The classic form often most commonly occurs in healthy, middle-aged (30-60) females and may be associated with preceding infection (URI, strep., etc.), connective tissue disease, inflammatory bowel disease, or even pregnancy.  Initial published data suggested that HSS typically behaved in a benign fashion and lacked an association with possible underlying malignancy.  However, more recent case series suggest that between 36%-53% of HSS cases are in fact associated with some hematologic disorder, again, most commonly myelodysplastic syndrome and myelogenous leukemias.  Both HSS and SS prognosis seem to be predominantly dictated by the medical associated conditions, instead of the skin disease itself.  Sweet's syndrome may recur following either spontaneous remission or secondary to treatment induced clinical resolution and the duration of remission is variable depending on the underlying cause.  Typically classical cases show less risk of recurrence whereas, in cancer patients, Sweet's syndrome recurrences are more common.  Systemic corticosteroids are the first-line therapies for HSS.  Corticosteroids remain the mainstay of treatment for HSS.  In the setting of active disease and no remission with corticosteroids, azathioprine or dapsone may be considered.  Treatment of aggressive disease or corticosteroid unresponsive disease includes cytarabine, cladribine, or all-trans retinoic acid.  For HSS patients who are refractory to dapsone, azathioprine monotherapy has been reported to be successful.

Figure 1. Clinical photograph of right ventral forearm just after biopsy.

Figure 2 and 3.  Clinical photographs of right and left hands.

CONCLUSION

Presented is a case report of classic histiocytoid sweet syndrome (clinically bullous variant) with an unknown etiology that showed rapid clearance with systemic corticosteroids and sustained clearance with dapsone.  Despite the negative workup in this patient, due to the possible association with hematologic malignancy, it is important to screen all patients diagnosed with SS or HSS.

REFERENCES


**Clear Cell Acanthoma: A Clinical, Dermatoscopic, and Histological Review.**

**Abstract**

Clear cell acanthoma (CCA) is an uncommon benign epidermal tumor, presenting as an erythematous solitary papule with a peripheral scale, usually on the lower extremity. Although biopsy is commonly performed for diagnosis, dermatoscopically, clinicians may suspect a CCA with the use of clinical and dermatoscopic findings. We present our case of a suspected clear cell acanthoma confirmed by biopsy along with a clinical, dermatoscopic and histological review.

**Clinical Findings**

CCAs are generally solitary, asymptomatic, red or brown dome shaped papules or nodules. They may be covered by scaled edges or have a moist appearance. The size of the lesion can range from approximately 3-20mm and can slowly grow for up to 10 years. When closely examining the surface of the lesion, vascular puncta are present, which easily bleed following minor trauma. These lesions are usually found on the lower extremities in middle aged to elderly adults, males and females alike [5-6]. Although this description is the most common presentation, there are a variety of clear cell acanthoma types creating a large list of differential diagnosis. These types include: giant, polypoid, pigmented, erosive, atypical and cystic [6].

**Histopathology**

Typically, CCAs are characterized by well demarcated epidermal hyperplasia made up of large keratinocytes and basal cells full of a glycol-synthetic cytoplasm with abundant keratinized cells that stain bright pink. An abundance of densely packed dilated capillaries are seen in a well-demarcated distribution, which correlate with the dermatoscopic vascular features or red dots and globules outlined above. Parakeratosis, neutrophilic exocytosis and mild spongiosis are also present.

**Management and Therapy**

Management of CCA is excisional removal of a solitary lesion. This can be done through a variety of methods including, but not limited to, standard surgical excision, Mohs micrographic surgery, cryotherapy, electrofulguration, curettage or carbon dioxide laser. For cases of multiple or larger size lesions, cryotherapy and carbon dioxide laser have been successfully used [6]. In addition, with the theorized inflammatory reactive cause, a case report showed the regression of CCA after a two month trial of calcipotriol [4]. In the case of our patient, shave excision combined with electrofulguration was used for diagnosis and treatment.

**Conclusion**

CCAs have a large differential including many lesions that are less benign and which occur with much higher frequencies in the population. Under these conditions, the diagnosis of a CCA is usually one that is made histologically, after a biopsy has been performed. Since the features of this lesion are dermatoscopically distinct, this may afford the clinician more diagnostic confidence. The use of routine dermatoscope may therefore lessen biopsies of this benign dermatologic entity.

**BIBLIOGRAPHY**

A Rare Histologic Variant of a ‘Not So Sweet’ Rash

J Ryan Jackson, D.O., Nathan Cleaver, D.O., David Cleaver, D.O.

Introduction

The neutrophilic dermatoses are a clinically diverse group of disorders with characteristic histology of intense epidermal and/or dermal inflammatory infiltrates composed primarily of neutrophils without evidence of infection.1

The neutrophilic dermatoses include Behçet’s disease, bowel (intestinal) bypass syndrome, erythema elevatum diutinum, neutrophilic dermatosis of the dorsal hand, neutrophilic eccrine hidradenitis, pyoderma gangrenosum, and Sweet’s syndrome (SS).1

The pathogenesis of neutrophilic dermatoses is unknown, however these disorders may represent a state of immunologic reactivity.2

SS (Acute febrile neutrophilic dermatosis) is an uncommon benign disease, with a worldwide distribution and female predominance except in the internal malignancy subgroup. SS is a reactive process that is characterized by fever, peripheral blood neutrophilia, and painful erythematous plaques that are occasionally bullous and favor the face, and upper extremities and contain dense neutrophilic dermal infiltrates histologically.3

SS can be subdivided into subacute SS and histiocytoid SS. Histiocytoid SS is a rare variant of SS with a strong association with hematologic malignancies but rarely renal cell carcinoma.4 We present a case of a 65-year-old Caucasian male with an 8-year history of a diffuse unspecified rash previously managed by an outside provider. Subsequent punch biopsy of the left inferior postauricular skin confirmed the diagnosis of histiocytoid Sweet’s syndrome.

Case Presentation

A previously healthy, 65-year-old Caucasian male presented to the dermatology clinic with an 8-year history of a diffuse unspecified rash previously managed by an outside provider. Upon examination, multiple edematous red plaques were located on the neck, trunk and upper and lower extremities (Figure 1).

A 4 mm punch biopsy was obtained from the left inferior postauricular skin, which demonstrated mixed dermal infiltrate with karyorrhexis and a prominent histiocytoid cellular component (Figure 2). The biopsy was consistent with histiocytoid Sweet’s syndrome.

Our patient was initially treated with prednisone 20 mg daily and recommended to obtain a full malignancy workup. Upon completion of the malignancy workup, the patient was diagnosed with renal cell carcinoma of the right kidney and subsequently underwent nephrectomy.

Throughout the patient’s dermatologic management, the patient’s cutaneous findings were reluctant to prednisone alone; thus various doses of prednisone were tried along with indomethacin 150 mg daily in combination with multiple class I topical steroids which produced variable success. While on systemic steroids, daily calcium and vitamin D supplements were recommended. Steroid sparing agents such as dapsone were considered, but patient’s anemic state prevented safe and successful use. A trial of dapsone and nicotinamide was attempted with limited benefit.

Along with the recalcitrant cutaneous findings, the patient also experienced intermittent episodes of vision loss, palpitations, fatigue, anemia, dyspnea, atrial fibrillation, diffuse cutaneous fungal infections, and ultimately, metastatic renal cell carcinoma to the contralateral kidney and lung. Currently, the prednisone dose is being tapered in order to limit the systemic and immunomodulatory effect while the patient is being managed by oncology.

Neutrophilic Dermatoses

<table>
<thead>
<tr>
<th>Disease</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Behçet’s disease</td>
<td>Aphthous stomatitis, recurrent genital ulceration, ocular lesions Pathergy (+), HLA-BS1 allele association</td>
</tr>
<tr>
<td>Bowel bypass syndrome</td>
<td>1-6 years post GI surgery, serum sickness like symptoms precede rash, tender SES nodules on EN-like lesions</td>
</tr>
<tr>
<td>Erythema elevatum diutinum</td>
<td>Red-brown violaceous papules on extensor surfaces near joints, HIV and IgA paraproteinemia, arthritis, skin ulcers</td>
</tr>
<tr>
<td>Neutrophilic dermatosis of the dorsal hand</td>
<td>Ulcerative red-violaceous plaques on dorsal hands</td>
</tr>
<tr>
<td>Pyoderma gangrenosum</td>
<td>NAP, patients undergoing chemotherapy, erythematous plaques on extremities, trunk and face, neutrophilic infiltrate around eccrine glands and sebaceous coils</td>
</tr>
</tbody>
</table>

Discussion

SS was first described in 1964 by Dr. Robert Douglas Sweet as “acute febrile neutrophilic dermatosis.”1 In 1968 Whittle et al. reported a similar case and named it “Sweet’s syndrome.”2 SS is characterized by painful, erythematous, cutaneous plaques and nodules of rapid onset accompanied by fever, leukocytosis, and neutrophilia.3 The face, neck, and upper extremities are frequently involved.4 The characteristic histologic presentation consists of a diffuse dermal neutrophilic infiltrate with karyorrhexis and massive papillary dermal edema, which is responsible for the pseudoexudative clinical morphology.5 SS generally lacks leukocytoclastic vasculitis.6

SS can be further subdivided histologically into subacute Sweet’s syndrome and histiocytoid Sweet’s syndrome. Histiocytoid Sweet’s syndrome is a rare variant of SS. Histologically it is characterized by dermal and/or subcutaneous infiltrate of neutrophils and “histiocytoid” cells, which are immature myeloid cells that stain positively for myeloperoxidase.6 Recent studies suggest this variant may have a stronger association with hematologic malignancies but rarely with renal cell carcinoma.7 To the best of our knowledge this is the only reported case of renal cell carcinoma causing histiocytoid SS in the literature.

SS is benign and lesions typically involve in weeks to months without treatment, however recurrences occur in approximately 30% of cases.8 The most effective therapy for SS is oral prednisone (0.5–1.0 mg/kg/day) for 4–6 weeks. Other major alternative treatments for SS are potassium iodide (900 mg/day), dapsone (100–200 mg/day), and colchicine (1.5 mg/day).9 Nonsteroidal anti-inflammatory drugs, ciclosporine, cyclosporine, thalidomide and interferon-a have also been reported to lead to improvement of Sweet’s syndrome.10

References

Our understanding of the real-world value of vaccines has dramatically improved over the past several decades. However, despite the tremendous progress, anti-neutrophil cytoplasmic autoantibody (ANCA)-associated vasculitides (AAV) remain among the most common autoimmune disorders associated with vasculitis. AAV has been associated with a variety of clinical manifestations, including renal disease, pulmonary hemorrhage, and peripheral vasculitis. Despite advances in treatment, AAV remains a challenging condition to manage, with significant morbidity and mortality.

**Background**

Antineutrophil cytoplasmic antibodies (ANCA) are antibodies that target proteins in the cytoplasm of neutrophils. They are classified as either perinuclear anti-neutrophil cytoplasmic antibodies (c-ANCA) or diffuse cytoplasmic anti-neutrophil cytoplasmic antibodies (p-ANCA). ANCA are associated with a variety of autoimmune disorders, including AAV.

**Patient Presentation**

A 76-year-old female presented to our dermatology office complaining of a rash on her lower extremities for several months. She stated the rash was mildly itchy with persistent color changes of the extremities. She had previously been treated with prednisone 40mg daily with vitamin D and calcium supplementation. She was also given colchicine for treatment of recurrent gout flares. She had a history of hypertension, hypercholesterolemia, and diabetes mellitus. A complete blood count revealed a leukocytosis with a left shift. Urinalysis was unremarkable. A skin biopsy revealed a perivascular infiltrate with neutrophils and eosinophils. Autoantibody testing was performed and revealed positive c-ANCA and negative p-ANCA.

**Discussion**

AAV is a subset of ANCA-associated vasculitides, which are a group of autoimmune diseases characterized by inflammation of the blood vessels. AAV is typically associated with ANCA positivity, but not all patients with ANCA positivity will develop AAV. It is important to note that AAV can present with a variety of clinical manifestations, including renal disease, pulmonary hemorrhage, and peripheral vasculitis. The diagnosis of AAV is typically made using a combination of autoantibody testing and clinical findings. Autoantibody testing in AAV can be challenging, as ANCA positivity can be present in a variety of conditions. It is important to consider the clinical presentation and other laboratory findings when interpreting autoantibody testing results.

**Conclusion**

AAV is a complex autoimmune disease that requires a multidisciplinary approach to management. Early recognition and treatment of AAV can improve outcomes and prevent potential complications. Further research is needed to better understand the pathogenesis of AAV and improve treatment options for patients.
Clinical Significance of Cutaneous Angiomyxoma

ABSTRACT

• Cutaneous angiomyxomas are rare benign tumors of the skin that do not metastasize. Physician awareness is essential since this tumor can be the harbinger of the more deadly Carney complex. Carney complex may involve the integumentary, cardiac, and endocrine systems. This case reminds the reader to be familiar with the clinical manifestations of Carney complex when confronted with cutaneous angiomyxoma.

BACKGROUND

• Cutaneous angiomyxomas (CA) are uncommon benign (multilobulated) tumors of the dermis and subcutis with a high incidence of recurrence following excision. Superficial angiomyxoma is a synonymous term used to differentiate these tumors from “aggressive angiomyxomas.”1 Diagnosis of CA is based on distinctive histological features.2 The tumor was first described as part of Carney complex in 1985.3 CA can be the initial presenting lesion of Carney complex which is associated with significant morbidity and mortality. Dermatologists should be aware of the clinical and pathologic features of CA, as well as the manifestations of Carney complex. We present a case of a CA in a 49-year-old male with an asymptomatic raised lesion of the nose.

CASE HISTORY

• A 49-year-old male presented complaining of a light brown, raised lesion on the right nasal sidewall. The lesion had been present for many years and had gradually increased in size. The lesion was not pruritic, painful or bleeding. He denied previous treatment for the lesion. Personal and family history was negative for cancer. Physical examination revealed an 8x8 mm flesh colored papule on the right nasal sidewall. Shave biopsy was performed. Histopathology revealed a proliferation of hypocellular myxoid tissue forming a nodule in the dermis with no cytologic atypia. No increase in staining was seen with MelanA or S100. Dermatopathology confirmed the diagnosis of CA.

PATHOLOGY

• Histopathology revealed a proliferation of hypocellular myxoid tissue forming a nodule in the dermis with no cytologic atypia. No increase in staining was seen with MelanA or S100. Dermatopathology confirmed the diagnosis of CA.

REFERENCES


We present the case of a 63 year old male who sought care in our office for a rash located on his right anterior thigh. The patient claimed to have had the rash for over 30 years and started shortly after he was involved in a motor vehicle accident. The rash is red in color and currently does not have any associated symptoms outside of a minor itch that is not persistent. Over the past 30 years he denied any change in size, shape, or color. He has been to many (non-dermatology) physician offices over the years where he was told the etiology of the rash was unknown. Previously the patient has tried applying hydrocortisone and a moisturizer cream to the rash which did not result in any change. His only other complaint is a new onset rash located on the posterior-medial side of his right arm proximal to the elbow that has been present for around 1 year. He denied any other complaints or symptoms.

Past medical history is positive for arthritis, cardiomyopathy, bladder cancer (urothelial carcinoma) cluster headaches, dyspnea, hyperlipidemia, hypertension, neuropathy, obesity, peripheral vascular disease, scoliosis, sleep apnea, and cerebrovascular accident secondary to aortic aneurysm rupture. His surgical history includes aortic aneurysm repair, bladder cancer surgical resection, cardiac stenting, coronary artery bypass grafting, left hip arthroplasty. His family history includes his mother passing from breast cancer at age 76, but is otherwise largely unknown to him.

Social history includes alcohol consumption of 1-2 drinks per day, former smoker (quit 15 years prior), denies illicit drug use, employed at an auto parts store, and is married. Current medications include aspirin (81mg), Benadryl, Biotin, Coreg, Enalapril, Flomax, Hydrochlorothiazide, Krill Oil, Lovastatin, Tramadol, Vitamin C, Zinc. His only known allergies are Penicillin and Codeine.

Upon physical examination the patient was noted to have a well-demarcated, erythematous patch negative to diascopy with fine wrinkling and mild scale. The patch was non-tender and did not have any further characteristic change including no ulceration (Figure 1). The patient was originally given a class I topical steroid to apply twice daily for 2 weeks and was instructed to return for a follow-up appointment in 4 weeks. Upon return for the follow up visit the patient’s lesions were unchanged and the decision to perform punch biopsies was made. Two 4 millimeter punch biopsies were obtained and sent to a Dermatopathology lab for H&E review.

Histopathologic examination of the biopsy specimens revealed a superficial and deep perivascular infiltrate of atypical lymphocytes. In addition there was a lichenoid infiltrate of atypical lymphocytes with associated epidermotropism (Figures 2 and 3). Immunohistochemistry showed positive staining for CD2, CD3, CD4, CD8 and CD43 along with negative CD7 staining. Interestingly the staining pattern showed a majority of cells with dual CD4 and CD8 positive markers (Figures 4 & 5). This yielded a diagnosis of Patch Stage Cutaneous T-Cell Lymphoma, Mycosis Fungoides with aberrant co-expression of CD4 and CD8. The patient was referred to a Hematology/Oncology specialist to obtain a decision to perform phototherapy alone combined with topical steroids, which he is currently completing.

Discussion

Cutaneous T Cell Lymphoma represents a group of variable, heterogeneous tumors of the skin. Mycosis fungoides (MF) is considered an indolent subtype of CTCL with a good prognosis when discovered in early stages. MF represents less than 1% of all Non Hodgkins Lymphomas but has the highest skin infiltration incidence among all lymphomas. The incidence has been reported to be around 0.36 per 100,000 people in the United States. Patch Stage MF is commonly overlooked and mistaken for more common conditions like eczema, psoriasis, parapsoriasis, pityriasis, and others (such was the case with our patient having been misdiagnosed for over 30 years). Due to the indolent course early patch stage MF is well known to require several biopsies over months to years before definitive diagnosis1.

The first known report of dual CD4 and CD8 positive mycosis fungoides was reported by Knapp et al in 2012 involving a elderly female patient1. A second case was reported by Tournier et al in 2014 involving a 31 year old female patient2. Yang and Shen reported a third case in 2015, the first in a male patient3. Knapp reported that the patient presented with sclerodermoid lesions on her abdomen and thigh, while Tournier reported infiltrated plaques in a generalized photo protected areas of the skin1. In Yang and Shen’s case the patient presented in the plaque stage and quickly progressed to systemic disease which resulted in death four months after presentation2. To our knowledge our case is the fourth reported case of this phenomenon and the second in a male patient1,2,3. It is not known if this unique immunologic type has an impact on prognosis or treatment. As more of these cases are reported, future reports will likely uncover these questions and help guide more individualized treatment.

References

**Introduction**

- Cutaneous Langerhans Cell Histiocytosis (LCH) is a rare disorder which typically presents in children less than two years of age with a variable clinical course ranging from spontaneous resolution to fatal outcome.
- LCH in infants can be similar in presentation to other pediatric rashes such as atopic dermatitis, seborrheic dermatitis, arthropod assault and diaper dermatitis. Misdiagnoses can result in delayed diagnosis and ineffective treatment due to low awareness and understanding of LCH.
- We present the case of a three months old infant promptly diagnosed with LCH following biopsy of the lesion after unsuccessful treatment for atopic dermatitis and arthropod assault.
- Given the rarity of the condition and variable clinical presentations, there is no consensus treatment guideline of LCH. This case report outlines the initial clinical work-up and treatment of an infant with isolated cutaneous LCH and subsequent response to therapy.

**Case Report**

A three-month-old Hispanic female presented to dermatological consultation with generalized pruritic erythematous papules, pustules and nodules on the scalp, trunk, and bilateral upper and lower extremities of two months’ duration (Figures 1 & 2). She was empirically treated by her pediatrician with scabies therapy followed by different topical steroids and antibiotics. Other than a history of premature birth and a heart murmur, parents denied any other complaints. The mother did not feel the baby was drinking or urinating more often than usual.

**Intervention:** Treatment was initiated with oral low dose methotrexate (20mg/m2) for a planned duration of six months with disease evaluation every two weeks for routine labs and disease monitoring. Chemotherapy regimen consisting of vinblastine and an oral steroid was decided as the alternative treatment should disease progress to multisystem LCH.

**Response to Treatment:** After two months of methotrexate therapy, the patient significantly improved with a decrease in the overall number of lesions. No adverse effect to treatment has been noted and baby is still undergoing therapy.

**Discussion**

- LCH is a rare disease of idiopathic monoclonal proliferation of abnormal Langerhans cells and cytokine overproduction resulting in inflammation, infiltration and destruction of many tissues in the body.
- There is poor understanding of the true incidence and burden of LCH due to the rare nature of the disease and its diverse clinical presentations.
- Clinical features of LCH can range from localized, single-organ lesions to multifocal, multi-organ involvement.
- LCH confined to the skin as presented in this report is rare and accounts for only about 5% of LCH cases.
- LCH can result in spontaneous regression, occurrence or progress to rapid deterioration, and even death.3,4,5 Isolated cutaneous LCH tends to regress spontaneously, but progression to MS-LCH is also common.4
- There is no consensus management guideline for LCH and the rarity of the condition complicates proper research on the most effective treatment.
- For LCH recalcitrant to topical steroid therapy where there is involvement of an extensive area, systemic therapy with oral steroids with or without vinblastine or oral low dose methotrexate can be used. It should however be noted that the level of evidence with this therapy is low.5

**References**

Granular Parakeratosis: A Case Report

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*PBCGME/JFK Medical Center North Campus, West Palm Beach, FL
**VA Medical Center, West Palm Beach, FL

Introduction

• Granular parakeratosis (GP) is a rare, benign skin condition
• Affects males and females of all ages equally with no racial predilection
• Self limited cutaneous condition
• First case of GP, in 1991, was present in the axilla, so the condition was initially termed axillary granular parakeratosis. Since then studies have shown GP in other skin folds so the definition has expanded and is now just called granular parakeratosis.
• Two major theories exist as to the origin of GP:
  - Affects males and females of all ages equally with no racial predilection
  - Affects males and females of all ages equally with no racial predilection

Case

• 59 year old male presents to clinic with chief complaint of a progressive rash over the course of a year (Figure 1)
• Has tried changing deodorants and using talcum powder without improvement
• Rash is asymptomatic without any blisters or weeping, only located in axilla
• Shave biopsy of right axilla consistent with findings of GP (Figure 2)
• Instructed to stop topical applications and clean daily with mild soap
• Rash is asymptomatic without any blisters or weeping, only located in axilla
• Instructed to stop topical applications and clean daily with mild soap
• Five days later: Rash had cleared
• Patient is instructed to stop topical applications and clean daily with mild soap
• Rash is asymptomatic without any blisters or weeping, only located in axilla

Discussion

• Etiology of GP initially thought to be secondary to an external irritant or contact allergy, (specifically deodorant), a possible defense mechanism
• But many cases show presence of GP without use of an irritant, in non-intertriginous areas, and unilaterally.
• The other line of thought is a disorder of cornification due to improvement with isotretinoin
• General consensus is that there is a disorder of cornification from stratum granulosum layer to stratum corneum layer.
• Speculation that this is due to inability to process profilagrin to fillagrin extrapolated from electron microscopy studies.
• With failure to degrade keratohylain granules and aggregate keratin filaments
• Main differentials to rule out are allergic contact and irritant contact dermatitis, Hailey Hailey disease, Darier’s disease, intertrigo, and acanthosis nigricans
• Due to unknown origins and spontaneous remission, there is no exact effective treatment method.
• Prior treatments have included topical and systemic steroids, retinoids, calcipotriene, antibiotics, antifungals
• Simple discontinuation of topical applications have proven effective

Conclusion

• This case supports the theory of external irritant as etiology of GP; the axillary lesions quickly resolved after discontinuation of deodorant and talcum powder
• It is possible that this patient has a predisposition to GP which was triggered by topical irritant
• More information is needed to determine the etiology of GP and determine a possible genetic predisposition

References

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KELOID EXCISION WITH SUPERFICIAL RADIATION THERAPY (SRT)

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INTRODUCTION

Derived from the greek word “chelē,” meaning crab’s claw, to describe the lateral growth of scar onto unaffected skin, keloids are among one of the challenging conditions to treat that face dermatologists. Keloids have been documented in all racial groups, but most commonly seen in individuals of Black, Asian, Latino ancestry. Darker skinned individuals have been reported to develop keloids as high as 20 times more likely than lighter-skinned individuals (1). Keloids can occur at any age, but most commonly occur in the 2nd through 3rd decades of life (2).

Keloids are benign dermal fibro-proliferative tumors with no malignant potential. Histologically, keloid tissue shows disorganized type I and III collagen bundles that extend beyond the margins of the original wound and spread by invasion of the neighboring skin rather than extend beyond the margins of the original wound and spread by invasion of the neighboring skin rather than expansion. Although the exact etiology for keloids is unknown, the persistence or failure to downregulate wound healing signals is the current pathological theory. Gene profiling of keloid fibroblasts has shown altered expression in multiple fibrosis-associated pathways, including TGF-binding and TGF-binding-related proteins, decreased expression of a subset of Wnt pathway inhibitors and multiple IL-1 inducible genes (3). Certain growth factors such as the TGF-beta family and their downstream target (SMAAD signal transduction pathway) have been identified in normal wound healing with dysregulation leading to keloid formation (4-7).

OBJECTIVE

The surgical removal of keloids is usually not difficult on its own. However, preventing recurrence can be unique clinical challenge. Intraluminal corticosteroids, cryotherapy, intraluminal 5-fluorouracil (5-FU), intraluminal bleomycin, topical imiquimod, pulse dye laser, fractionated CO2 laser and radiotherapy amongst others are several treatment options used on keloids. Surgical revision alone will not put the healing process back into a balance and other forms of treatment are needed to correct this matter. Past methods using surgical revision alone lead to failure due to the continuation of collagen production with recurrence rates ranging from 45-100% (8). Surgery combined with radiotherapy results in recurrence rates between 0-20% (9). Herein, we present our experience and success using surgery followed by superficial radiation therapy (SRT) for the treatment of keloids.

MATERIALS AND METHODS

Excisions were performed removing all collagen bundles and closed under no tension. Twenty-four hours post-surgery, SRT consisting of 3 fractions of 600 Gy at energies of either 70kV or 100kV with 5mm margins around the entirety of the closures were performed (BED 30 Protocol)(10).

RESULTS

A total of 17 patients with 24 keloids were treated between June 2015 to February 2017. Keloids from 5 areas of the body were treated in the study, which included head/neck, scalp, ears, back and chest (Figure 1). No recurrences have been reported at present time. The main side effect has been hyperpigmentation.

CONCLUSION

Keloids represent a challenging condition to treat. The efficacy of many treatment modalities (i.e. intraluminal corticosteroids, 5-FU, etc.) by their attenuation of the inflammatory response is a reasonable approach. However, achieving short-term and long-term efficacy by complete removal of keloids and factors that mitigate its future growth are paramount (11). Complete surgical removal of keloids with SRT has promising results and should be considered a powerful tool in treating keloids.

REFERENCES

A rare case of Chronic Smoldering Adult T-cell Lymphoma

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Introduction

**Adult T-cell lymphoma** is a rare and often aggressive T-cell lymphoma resulting from **Human T-cell Lymphotropic virus type I (HTLV-1)**. Endemic areas of HTLV-1 include South America, the Caribbean basin, southern Japan, and Iran. More than 90% of carriers remain asymptomatic1, and less than five percent of individuals with HTLV-1 will develop ATLL. We present a case of smoldering adult T-cell lymphoma that was originally thought to be mycosis fungoides until the patient was found to have HTLV-1 positivity.

**Background**

A 67 year old male developed an erythematous and pruritic lesion on his right extensor arm after a boating trip. He thought it was an insect bite due to edema and pruritus overlying the area; however, the lesion remained fixed for several months. Patient started developing extremely pruritic eczematous patches in the intertriginous folds and sought dermatologic care. He was diagnosed with atopic dermatitis at that time and treated with triamcinolone and fluocinonide without improvement. His past medical history included a pituitary tumor, HTLV-1 infection diagnosed 4 years, and Sarcoidosis. He retired from GM, which took him to Brazil on an annual basis for several years. The patches continued to spread to involve his extremities and buttocks, with some developing a plaque-like quality. The patient then underwent a biopsy with a differential including granuloma annulare and sarcoidosis. Since that visit, he rapidly developed further edematous plaques along the right arm. His initial biopsy was read as peripheral T-cell lymphoma with large cells, and he was referred to the Cutaneous Lymphoma Clinic at The James. At that time he denied weight loss, fever, night sweats, chills, or lymphadenopathy. Patient underwent a repeat biopsy, which was read as mycosis fungoides with large cell (CD30) transformation.

**Test Results**

**Initial skin biopsy**: Peripheral T cell lymphoma with large cells. The differential included mycosis fungoides with large cell transformation and adult T-cell lymphoma. The CD4:CD8 ratio was more than 10:1. CD7, CD5 with some positivity. Ki-67 proliferation index of 50-70%. CD30 positive in less than 10% of cells. Malignant cells were positive for CD45, CD2, and strongly for CD25.

**Repeat skin biopsy**: Mycosis fungoides with large cell (CD30+) transformation. The CD4:CD8 ratio was normal, and a discrete subset of lymphocytes were CD3+, CD7- (41%) and CD26- (77%). T-cell receptor Vb analysis showed a pattern consistent with polyclonal population of T lymphocytes. CD30 positivity was present in approximately 75% of cells.

**PET scan**: Hypermetabolic lymph nodes within the chest and inguinal region. Focal uptake noted along the skin of bilateral arms, and nonspecific bilateral tibial uptake.

**Bone marrow biopsy**: Mildly hypercellular bone marrow (50%) with tetrilineage hematopoiesis with no evidence of a T-cell lymphoproliferative neoplasm. An atypical T cell population was detected by flow cytometry.

Discussion

**Adult T-cell lymphoma (ATLL)** is a rare and aggressive T-cell lymphoma. It involves the blood, lymph nodes, and skin, and may affect other areas of the body.

There are four subtypes of ATLL: acute, lymphomatous, chronic, and smoldering. Acute and lymphomatous subtypes are fast-growing forms of ATLL, whereas chronic and smoldering are less aggressive. The smoldering ATLL is associated with very mild symptoms, such as a few skin lesions. Observation without treatment may be appropriate for patients who have the slower-growing subtypes of ATLL with mild or no symptoms, but follow-up monitoring is required. For ATLL affecting the skin, skin-directed therapies including topical steroids or local radiation may be needed.

Conclusion

Due to the patient’s HTLV positivity, a biopsy supporting the diagnosis of ATLL with strong CD25+ expression on IHC, and a bone marrow biopsy with no evidence of involvement, the patient was diagnosed with **chronic smoldering adult T-cell lymphoma**.

He was started on Bexarotene 150 mg PO, NBV/B phototherapy, Clobetasol cream BID PRN pruritus, and transitioned to IFN 7 MIU 5x.week and Zidovudine 300mg BID as antiretrovirals. At the current time we are considering bone marrow transplant as even the smoldering type can have an aggressive behavior pattern.

It is important for dermatologists to be aware of this entity as the morphology of mycosis fungoides and ATLL are practically indistinguishable, with a strong CD25 expression being one of the biggest differentiating factors. With international travel to endemic areas of HTLV-1 being so common, it is important to screen patients for HTLV-1, especially if the pathology looks like transformed MF.

References

Primary conjunctival melanomas (CM) are a rare type of melanoma. They account for approximately 5% of all cases of oculc melanomas, with ocular melanomas representing only 3.7% of all melanoma cases.1,2,14 The majority of primary ocular melanomas occur in the uvea, but interestingly, CM and uveal melanomas bear little genetic similarity to one another. CM has far greater mutation commonality with mucosal and cutaneous melanoma.2,4,14,16,17

In this report, we describe a patient with a rare epitheloid cell type conjunctival malignant melanoma who was successfully treated with wide local excision.

A 72 year-old Caucasian male with a history of basal cell carcinoma presented for routine outpatient dermatologic examination. On examination, he is noted to have a 0.4 cm firm, black pedunculated nodule on the right medial canthal conjunctiva (Figure 1). The patient was previously unaware of the lesion. A conjunctival biopsy was performed due to suspicion for malignant melanoma. Of note on the biopsy, the lesion was not fixed to the sclera.

Histopathology revealed an epitheloid cell type conjunctival malignant melanoma with a Breslow thickness of 2.2 mm involving the deep and lateral margins (Figures 2,3). Immunohistochemistry of the tumor revealed positive staining with S100, melan-A, and HMB-45. PHH3 staining showed rare invasive tumor cells consistent with rare mitotic activity. The patient was referred for treatment including wide local excision with cryotherapy to the margins and subsequent sentinel node biopsy. The patient had negative sentinel lymph node biopsy and is over 2 years disease free (Figure 4).

CM is a disease mainly of 55 to 65-year-old Caucasians and lacks notable gender predilection. Rarely it has been reported in pediatric, Asian, and black populations.7,14 Clinical presentation typically consists of an enlarging fixed pigmented nodule in the peri-limbal area with the presence of feeder vessels. Poor prognostic factors include tumor thickness greater than 2 millimeters, de novo origin, ulcerated or nodular tumors, involvement of adjacent tissue structures, older age, nonwhite race, male gender, and local recurrence. Local recurrence is estimated to be 45% and 59% at five and ten years, respectively.1,2,9,13,14

CMs are thought to arise de novo in 16-26% of cases. These lesions portend a poorer prognosis than CM arising from a precursor lesion.13-15 The risk factors for development of CM are largely unknown, but ultraviolet radiation is thought to play a role in pathogenesis, as the conjunctiva is the only mucosal surface with natural exposure to sunlight. Furthermore, NRAS (neuroblastoma RAS viral (v-ras) oncogene homologue) and BRAF (v-RAF murine sarcoma viral oncogene homologue B1) mutations may be found in CMs and are also found in sun exposure related cutaneous melanomas.1,15-17

The most common treatment modality utilized is excision with 3-6 mm of tumor free conjunctival margin with double freeze slow thaw cryotherapy to the margins. Exenteration is no longer commonly recommended, except in extensive cases of orbital invasion.19,20

References

CASE PRESENTATION
A 69-year-old Caucasian male presented with a 4-month history of a slowly expanding, verrucous plaque of the left upper cutaneous lip. The lesion reportedly began as an abscess and had undergone incision and drainage, followed by multiple unsuccessful courses of oral antibiotics prior to presentation to our clinic. The patient reported that the area was occasionally itchy, but otherwise asymptomatic. Review of systems was negative for any systemic symptoms. The patient denied any preceding illnesses, changes in medications, or previous skin cancers. His social history was significant for an extensive international travel history, though he denied any known exotic exposures. His hobbies included gardening and tree planting near his home in the mountains of western North Carolina, where the patient was residing when the lesion started. Physical examination revealed a verrucous, erythematous plaque with papillomatous borders and central ulceration on the left upper cutaneous and vermilion lip extending to the nasolabial fold (Figure 1). A small erythematous plaque with overlying serous crust was noted on the patient’s right upper back, which the patient reported had been present for approximately 20 years.

HISTOPATHOLOGY
Histopathologic examination of multiple punch biopsies showed pseudopodipheliomatosus hyperplasia with intraepidermal pustules containing neutrophils and eosinophils. Stains were Periodic acid-Schiff (PAS), H&E, and VZV-negative. A pancytokeratin stain showed no evidence of squamous cell carcinoma. Direct immunofluorescence was negative. Indirect immunofluorescence for skin autoantibodies was negative. Three separate tissue culture specimens showed no bacterial, fungal, or mycobacterial growth. Leishmania PCR and DNA sequencing was negative. An additional punch biopsy (Figure 7) revealed yeast forms with broad-based budding and refractile walls highlighted with Gomori Methenamine Silver (GMS) stain of the tissue, consistent with cutaneous blastomycosis (Figures 3-5).

CLINICAL COURSE
A chest X-ray demonstrated no pulmonary involvement. In collaboration with an infectious disease specialist, the patient was initiated on therapy with itraconazole 200 mg twice daily for a total of 6 months. Side effects during therapy included occasional headache and nausea. At six-month follow-up, residual scarring and alopecia were noted in parts of the previously affected areas of the beard and nasolabial fold (Figure 2). Complete resolution of both the gastrointestinal upset, blurred vision and a transient increase in blood pressure, face and back lesions was noted at the completion of the treatment course. At six-month follow-up, residual scarring and alopecia were noted in parts of the previously affected areas of the beard and nasolabial fold (Figure 2). At six-month follow-up, residual scarring and alopecia were noted in parts of the previously affected areas of the beard and nasolabial fold (Figure 2). At six-month follow-up, residual scarring and alopecia were noted in parts of the previously affected areas of the beard and nasolabial fold (Figure 2). At six-month follow-up, residual scarring and alopecia were noted in parts of the previously affected areas of the beard and nasolabial fold (Figure 2). At six-month follow-up, residual scarring and alopecia were noted in parts of the previously affected areas of the beard and nasolabial fold (Figure 2).

FIGURES
- **Figure 1**: (December 2016): Verrucous, ulcerative plaque with papillomatous borders and central ulceration on the left upper cutaneous and vermilion lip extending to the nasolabial fold.
- **Figure 2**: (January 2018): Residual scarring and alopecia in parts of the beard area and nasolabial fold 4 months after completing itraconazole therapy.
- **Figure 3**: Pseudopodipheliomatosus hyperplasia with intraepidermal pustules containing neutrophils and eosinophils (H&E, x 40).
- **Figure 4**: Yeast forms with broad-based budding and refractile walls (H&E, x 400).
- **Figure 5**: GMS stain highlighting single nonbudding yeast forms (x 400).
- **Figure 6**: Yeast forms with broad-based budding and refractile walls (H&E, x 400).
- **Figure 7**: Yeast forms with broad-based budding and refractile walls (H&E, x 400).
- **Figure 8**: Yeast forms with broad-based budding and refractile walls (H&E, x 400).
- **Figure 9**: GMS stain highlighting single nonbudding yeast forms (x 400).

DISCUSSION
Blastomycosis is a fungal infection caused by *Blastomyces dermatitidis*, a thermally dimorphic fungus endemic in the soils of the Ohio and Mississippi River Valleys and southeastern United States. It most commonly manifests as a pulmonary infection following inhalation of spores, which may be asymptomatic and therefore undetected. Extrapulmonary disease occurs in ~ 25-30% of patients after hematogenous dissemination from the lungs, with the skin being the most common site of extrapulmonary disease. Primary cutaneous blastomycosis is quite rare and occurs due to direct inoculation after trauma to the skin via an infected animal bite, direct inoculation in laboratory settings, or due to injury during outdoor activities involving contact with soil. Given our patient’s horticultural hobbies, lack of pulmonary symptoms, and negative radiological examination, primary cutaneous blastomycosis infection is a possibility, though it is difficult to definitively ascertain whether our case represents primary or secondary cutaneous blastomycosis.

Clinically, cutaneous blastomycosis starts as papules that evolve into crusts, vegetative plaques often with central clearing or ulceration. It can be mistaken for squamous cell carcinoma, penicillus vegetans, leishmaniasis, bacterial pyoderma, and other deep fungal infections, therefore histopathologic examination and tissue culture are crucial to the diagnosis. On histopathology, pseudopodipheliomatosus hyperplasia with neutrophile abscesses is seen. Organisms can be difficult to identify and are often found within histiocytes or abscesses in the dermis. The yeasts are 8 to 15 µm in diameter with thick, double-contoured walls and display broad-based budding. Despite stains including GMS and PAS, blastomycosis can be a very difficult diagnosis and it is important to note that a negative result does not exclude the possibility of blastomycosis, as demonstrated in this case. Culture is certainly the most sensitive method for detecting and diagnosing blastomycosis. Growth is typically detected in 5 to 10 days, but can take up to 30 days if few organisms are present in the specimen. Though spontaneous remission can occur, it is recommended that all patients with cutaneous blastomycosis be treated to avoid dissemination and recurrence. Itraconazole is currently the treatment of choice. Doses are typically 200 to 400 mg per day for 6-12 months.

REFERENCES
Rubinstein-Taybi Syndrome
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Introduction:
Rubinstein-Taybi syndrome (RTS) is a rare congenital disorder involving a combination of both intellectual impairment and physical abnormalities. It is caused by a microdeletion in the gene encoding for CREB Binding Protein (CREBBP) found on chromosome 16p13.3. Typical features include broad, angulated thumbs and halluces, short stature, beaked nose, small maxilla, talon cusps, and down-slanting palpebral fissures. In addition, many individuals with RTS have a propensity toward keloid formation.

Case Report:
In this case we present a 38-year-old Caucasian male with distinct physical characteristics who presented to our clinic to have his keloids treated. His past medical history is varied but is typical of the syndrome. He was born without tear ducts. He was born with hard palate and jaw deformities, with undescended testicles and without an Odontoid process. Seven years prior the patient was noted to have severe obstructive sleep apnea secondary to a significantly enlarged lingual tonsil. Due to the size of the tonsil, during the surgery, the patient’s epiglottis was damaged and scarred open. A feeding tube had to be placed to prevent aspiration. He is Currently undergoing chemotherapy for stage IV non-Hodgkin’s lymphoma (NHL).

Family history: Cousin diagnosed with Ehlers-Danlos syndrome; all else unremarkable
Social history: graduated from high school; attends church; very sociable and has a great memory
Medications: Rituxan Q2 months for NHL; testosterone crème

Differential Diagnosis:
Conditions with Broad thumbs:
- Rothmund-Thomson syndrome (Poikiloderma Congenital)
- Stapes ankylosis with broad thumb and toes

Conditions with Short Stature:
- Bloom Syndrome
- Trichothiodystrophy (PIBIDS)
- Cockayne Syndrome

Discussion
RTS is an inherited disorder in either a sporadic or autosomal-dominant fashion resulting in a CREBBP defect. RTS is characterized by distinct facial features, broad angulated thumbs and great toes, short stature, intellectual impairment, keloid formation, congenital heart defects, vascular malformations, cryptorchidism, and increased susceptibility to solid tumors. Birth prevalence is uncommon occurring in 1 in 100,000 to 125,000 births.

“CREBBP (CRE binding protein or CBP) encodes for a large ubiquitously expressed protein of the same name that performs multiple roles in transcriptional co-activation, including the acetylation of histone and nonhistone targets… Somatic mutations in CREBBP have recently been noted in more than one third of diffuse large-cell non-Hodgkin lymphoma and follicular lymphoma at diagnosis.”

Conclusion
RTS is an uncommon congenital disorder with no known cure characterized by intellectual impairment and multiple physical deformities. It has a number of dermatologic manifestations including keloid formation. The patient presented in this case had several keloids on his abdomen that were in the process of being treated with intralesional steroid injections to which he appeared to be responding well.

References

Distinct Facial Features:
- Down slanted palpebral fissures
- Low hanging columella
- High palate
- Grimacing smile

Multiple Keloids
Talon Cusps

Broad angulated thumbs and great toes
Antiphospholipid syndrome (APS) is a rare acquired autoimmune disease, characterized by the formation of autoantibodies to cardiolipin, lupus anticoagulant, and beta-2-glycoprotein I. It affects primarily young women. Predisposes to thrombosis and obstetric morbidity. Most commonly associated with an underlying autoimmune disease such as systemic lupus erythematosus (SLE). Less frequently, occurs in the setting of infection or lymphoproliferative malignancy. In addition, several medications are associated with APS. Empiric anticoagulation, anti-platelet, and antimarial agents are the treatment of choice in those with concurrent lupus. Featured is a case of anti-phospholipid syndrome presenting as discoid lupus erythematosus (DLE), perniosis, and cerebral infarctions in a young woman with SLE.

HEADLINE
“Antiphospholipid Syndrome: A Case Report”

INTRODUCTION
- Anti-phospholipid syndrome (APS) is a rare acquired autoimmune disease, characterized by the formation of autoantibodies to cardiolipin, lupus anticoagulant, and beta-2-glycoprotein I.
- Affects primarily young women.
- Predisposes to thrombosis and obstetric morbidity.
- Most commonly associated with an underlying autoimmune disease such as systemic lupus erythematosus (SLE). Less frequently, occurs in the setting of infection or lymphoproliferative malignancy. In addition, several medications are associated with APS.
- Empiric anticoagulation, anti-platelet, and antimarial agents are the treatment of choice in those with concurrent lupus.
- Featured is a case of anti-phospholipid syndrome presenting as discoid lupus erythematosus (DLE), perniosis, and cerebral infarctions in a young woman with SLE.

PRESENTATION
- A 26-year-old African-American female complaining of a two-week history of progressive, throbbing headache and rash involving her nose and distal extremities following a flu-like illness.
- Associated myalgia, arthralgia, light-headedness, blurred vision, and right lower extremity weakness were reported.
- Recent history of upper-respiratory illness with rhinorrhea, nasal congestion, and shortness of breath relieved by over-the-counter decongestants.
- Denied recent sun exposure, chemical exposures, travel, vaccinations, or drug contacts.
- Past medical history included SLE, DLE, arthritis, and chronic lower back pain.
- Home medications included hydroxychloroquine and mycophenolate mofetil.
- Family history, allergies, and social history noncontributory.
- Physical examination revealed scar-like, atrophic, erythematous plaques covered with adherent scales of the nose and malar cheeks (Figure 1). Multiple purpuric, tender erythematosus to violaceous muscles and edematous papules were noted on the palm, hands, and plantar feet (Figure 2).
- Laboratory tests revealed leukocytosis and elevated ESR.
- Renal function tests, coagulation profile, cultures, urine analysis, and chest radiograph were unremarkable.
- Lupus anticoagulant and anti-cardiolipin antibodies positive.
- Lumbar puncture and cerebral spinal fluid (CSF) analysis negative.
- Head computed tomography (CT) showed nonspecific leukoencephalopathy (Figure 3).
- Head magnetic resonance imaging (MRI) showed multifocal areas of T2 hyperintensities throughout the subcortical and deep white matter, multiple areas of restricted diffusion bilaterally suggestive of multiple infarctions, possibly from lupus vasculitis (Figure 4).
- Findings were consistent with APS.
- Resolution of her neurologic status with improvement of her overall skin findings was achieved following initiation of high-dose prednisone, aspirin, and anti-coagulation.

DISCUSSION
- The antiphospholipid syndrome is an autoimmune disease characterized by the presence of circulating antiphospholipid antibodies that result in vascular thrombosis and obstetrical complications.
- Occurs mainly young women.
- Predisposition to thrombosis, the major complication of APS.
- Clinical presentation
  - History of vascular thrombosis, premature birth, recurrent miscarriage, and labile blood pressure.
  - Lupus retinopathy is the most common cutaneous finding.
  - Other cutaneous findings include atrophic blanche, leg ulcers, vasculitis, pseudovasculitis, digital gangrene, cutaneous necrosis, splinter hemorrhages, cyanosis, perniosis, and reformation purpuric suggestive of occlusion.
  - Systemic features include DVT, PE, stroke, renal infarct, myocardiial infarction, arthritis, and seizure.
- Often with autoimmune disorders, such as SLE (most common), rheumatoid arthritis, and ulcerative colitis.
- SLE associated with increased risk of stroke and premature death due to cerebral infarction.
- Stroke has been reported in up to 19% of patients with SLE.
- Precipitants may include surgery, infection (e.g. HIV, hepatitis C), and medications (e.g. hydroxychloroquine, oral contraceptives, ace inhibitors).
- Pathology
  - Occlusion of arterioles and arteries with fibrin thrombi, occlusion of veins or veins; minimal inflammation; typically lacks leukocytoclastic vasculitis.
  - Vasculopathy may cause direct injury and affect the blood-brain barrier, allowing antibodies to enter the nervous system, characterized by a small to moderate perivascular accumulation of mononuclear cells in blood vessels, resulting in small infarcts due to luminal occlusion.
- Laboratory findings
  - Positive antiphospholipid antibodies: anti-cardiolipin antibodies (most sensitive), lupus anticoagulant, anti-β2-glycoprotein I antibody (most specific).
  - Increase the risk of stroke and seizures.
  - Associated with an increased prevalence of abnormal findings on MRI.
  - False-positive lupus serology.
- Sapporo criteria for diagnosis of APS were revised in 2006 - one clinical finding along with one positive laboratory criterion must be present.
- Treatment of APS is typically a combination of anticoagulation (e.g. warfarin), antiplatelet agents (e.g. aspirin), and antimarial agents (e.g. hydroxychloroquine).
  - To date, long-term anticoagulation has been the only treatment shown to reduce vascular complications. However, that regimen does not prevent organ deterioration and death in high-risk patients.
  - Treatment underlying cause such as infection and discontinuation of any responsible drugs.

CONCLUSION
- This case highlights the predisposition to thrombosis and vasculitis noted in patients with APS.
- Clinicians must have a high degree of suspicion for the presence of antiphospholipid antibodies in women with a history of SLE presenting with neurologic symptoms.
- The presence of antiphospholipid antibodies in the appropriate clinical setting is key to establishing diagnosis.
- Prolonged anticoagulant therapy is the mainstay of treatment.
- Low-molecular weight heparin and low-dose aspirin preferred for pregnant patients, and pravastatin may improve pregnancy outcomes when taken at onset of preeclampsia and intrauterine growth restriction.
- Treatment often includes antplatelet agents and antimarial agents as well.

REFERENCES
Granuloma annulare (GA) is a chronic skin condition that presents as discolored plaques in a ring formation. This skin condition is seen in 0.1-0.4% of patients presenting to dermatology offices and is 2.5 times more common in females. The exact pathogenesis is unknown, however, inciting factors such as trauma, insect bites, tuberculosis skin tests, vaccines, sun exposure, and infections have been implicated. The most common histopathological findings include dermal lymphohistiocytic infiltration and degenerated collagen.

Granuloma annulare has been associated with numerous disorders including diabetes mellitus, dyslipidemia, thyroid disorder, malignancy, and HIV infection. The five identified variants of granuloma annulare include localized GA, generalized GA, subcutaneous GA, patch GA, and perforating GA. The most common subtype, localized GA, is a non-scaly, erythematous annular plaque on the distal extremities seen in the first three decades of life. Generalized GA accounts for about 15% of all GA cases, is most common in the 4th through 7th decades, and consists of numerous erythematous papules and plaques found on the trunk and extremities. Subcutaneous GA is the most common type of GA found in children. Perforating GA (PGA) is most often found in children or young adults with an increased prevalence in Hawaii. PGA presents as erythematous papules that can be either localized to the extremities or widespread and may develop into umbilicated papules with clear-to-white discharge. The pathology of PGA consists of trans-epidermal elimination of mucinous degenerated collagen surrounded by palisading lymphohistiocytic granulomas.

Case Report

A 60 year-old Caucasian male with past medical history of diabetes and hypertension treated with lisinopril who presented with a 6-week history of multiple skin lesions on the left and right arms. Patient denied any systemic symptoms such as fever, chills, night sweats or weight change. Patient reported the lesions were tender when palpated. He denied any previous treatment.

Laboratory Tests:
- Glucose 340 mg/dL
- Triglyceride 256 mg/dL
- RPR Screen Reactive
- RPR Titer 1:32
- Treponema pallidum Antibody-PA Reactive

Histopathology:
Two punch biopsies showed a patchy lymphocytic infiltrate in the dermis, accompanied by histiocites and some histiocytic giant cells palisaded around less cellular areas. The latter have thick collagen bundles with diminished numbers of fibroblasts in their centers. There is irregular epidermal hyperplasia with small collections of neutrophils within some of the more jagged foci, and granulation tissue near them.

Discussion

Granuloma annulare (GA) is a granulomatous inflammatory disorder of the skin not uncommonly seen. Perforating granuloma annulare (PGA) is a rare clinical variant occurring in up to 5% of patients with GA, first described in 1971 by Owens and Freeman. PGA has a chronic course with predilection for the extremities or less commonly generalized to involve the trunk and extremities. The primary lesion is a small umbilicated papule, scale-crust or focal ulceration primarily on the dorsal hands and fingers and histologically exhibits transpidermal elimination of degenerating collagen.

Two punch biopsies showed mucin deposition in the centers of the granulomatous foci, although not as much in most cases of granuloma annulare. Some transpidermal elimination evidence was observed, which histologically correlates with the crusted areas seen clinically. Atypical presentation of necrobiosis lipoidica was also considered as a possibility. However, most cases of this variant are now considered to be annular elastolytic giant cell granuloma (AEGCG), also known as actinic granuloma, which is in turn regarded by many as a variant of granuloma annulare. The sparing of elastic fibers in an elastic van Giesen stain is against necrobiosis lipoidica.

Secondary syphilis was treated with doxycycline 100mg by mouth twice daily for 14 days. The patient was treated with topical Clobetasol 0.05% ointment twice daily to the skin lesions until flat and no longer erythematous. Treatment of the localized form of PGA using intralesional corticosteroids or topical high-potency corticosteroids may also be used. Topical imiquimod, topical calcineurin inhibitors pimecrolimus and tacrolimus, cryotherapy, and simple excision have been reported treatments for PGA. The treatment of perforating granuloma annulare is often unsuccessful.

References
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Bullous Systemic Lupus Erythematosus (BSLE) is a particularly uncommon manifestation of nonscarring subepidermal bullous eruption of systemic lupus erythematosus (SLE) with an incidence of 0.26 per million persons per year in adults. BSLE presents with a context of bullae, vesicles, or a combination of vesicles and bullae distributed throughout the body, concentrated in the face, neck, extensor sites, and trunk. The simultaneous occurrence of a primary bullous eruption, including subepidermal bullae, indicates a significant risk of extravasation, and necrosis of blood vessels, may be evident, which are rarely seen in DH. Classic features of primary lupus lesions, such as epidermal atrophy, basal keratinocyte vacuolization, and the presence of a lupus band test, are not found in bullous lesions. Direct immunofluorescence (DIF) studies show deposits of IgG, C3, IgA, and IgM at the BMZ in two types of lesions, lichenoid, and papulosquamous, with an occasional mixed granular-linear configuration. Indirect immunofluorescence with split-skin specimens demonstrates immunoglobulin deposition on the dermal side of the split.

The criteria for the diagnosis of BSLE proposed by Gammon and Briegeman et al. include a diagnosis of SLE based on the criteria of the ACR, vesicles and bullae located on but not limited to sun-exposed skin; histopathologic findings similar to DH; and deposition of IgG and/or IgM and/or IgA at the BMZ by DIF. BSLE has been further classified into two distinct subtypes by Gammon and Briegeman: patients with circulating antibodies to type VII collagen are designated as cases of BSLE-1 while patients designated as cases of BSLE-2 do not have these antibodies. Clinically, it is not possible to distinguish between the two types of BSLE.

Differential diagnosis of BSLE (Table 1) includes EBA, BP, DH, and linear IgA dermatosis, which can be distinguished on the basis of clinical, histopathologic, and immunologic findings. Unlike patients with EBA, most patients with BSLE respond dramatically to dapsone. The bullous lesions of EBA can remain for weeks or months, but will often heal with scar formation. EBA should be considered in the differential diagnosis of patients with BSLE. The activity of the systemic and skin disease is generally unrelated, however. Unfortunately, many patients with BSLE do not respond to dapsone, and the lesions may persist despite treatment. In these patients, alternative therapies such as corticosteroids, ciclosporin, methotrexate, azathioprine, chloroquine, and antimalarials have been reported to be useful.

The prognosis in patients with SLE and bullous lesions is determined largely by the visceral manifestations of the SLE. The activity of the systemic and skin disease is generally unrelated, however. Unfortunately, many patients with BSLE do not respond to dapsone, and the lesions may persist despite treatment. In these patients, alternative therapies such as corticosteroids, ciclosporin, methotrexate, azathioprine, chloroquine, and antimalarials have been reported to be useful.

The clinical presentation of BSLE is generally that of an acutely generalized vesiculobullous eruption in patients who meet the American Rheumatism Association revised criteria for SLE. Lesions may involve flexural or extensor skin and mucosal surfaces of the mouth and pharynx. Blisters may form on erythematous skin and may be preceded by erythematous macules and plaques. Lesions may be large and tense and resemble those of BP or small and grouped and resemble those of DH. BSLE does not exhibit mechanical fragility of the skin with tense bullae that subsequently heal with scars and relia contrasting with EBA. Patients may complain of itching, which may sometimes be severe. However, pruritus is not prominent. Additionally, the primary lesions seen in both SLE and discoid lupus are not commonly associated with BSLE. BSLE is characterized by recurrent bullous eruptions, which are not limited to the scalp or sun-exposed skin but are seen over a wide range of sites. Lesions may be unilateral or bilateral, and may occur on both the upper and lower extremities. Lesions may be large and tense and resemble those of BP, or small and grouped, and may occur on both the upper and lower extremities. Lesions may be large and tense and resemble those of BP, or small and grouped, and may occur on both the upper and lower extremities.

In summary, BSLE presents with subepidermal vesicles and neutrophilic microabscesses in the papillary tips, making it difficult to distinguish from DH. In many cases, neutrophils may not be solely confined to the papillary tips. BSLE presents with a context of bullae, vesicles, or a combination of vesicles and bullae distributed throughout the body, concentrated in the face, neck, extensor sites, and trunk. The simultaneous occurrence of a primary bullous eruption, including subepidermal bullae, indicates a significant risk of extravasation, and necrosis of blood vessels, may be evident, which are rarely seen in DH. Classic features of primary lupus lesions, such as epidermal atrophy, basal keratinocyte vacuolization, and the presence of a lupus band test, are not found in bullous lesions. Direct immunofluorescence (DIF) studies show deposits of IgG, C3, IgA, and IgM at the BMZ in two types of lesions, lichenoid, and papulosquamous, with an occasional mixed granular-linear configuration. Indirect immunofluorescence with split-skin specimens demonstrates immunoglobulin deposition on the dermal side of the split.

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Loose Anagen Syndrome in a 2-year-old Female: A Case Report and Review of the Literature

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INTRODUCTION

Loose anagen syndrome is an uncommon condition characterized by excessive and painless loss of anagen hairs from the scalp. The condition most commonly affects young females with blonde hair, but males and those with darker hair colors can be affected. Patients are known to have short, sparse hair that does not need cutting, and hairs are easily and painlessly plucked from the scalp. No known treatment exists for this rare disorder, but many patients improve with age.

CASE REPORT

We present the case of a 27-month-old female presenting to the clinic with a chief complaint of diffuse hair loss for the past five months. The mother stated that she began finding large clumps of hair throughout the house, most notably in the child’s play area. She stated that the condition had progressed to where the hair was fallen out in clumps, and the child was combing her hair using a large sock. The mother stated that the hair was falling out as opposed to being pulled out. The child’s hair was fine, straight, and no hair will grow past her neckline. The child had no notable medical history and took no medications. The child has no other skin, dental or nail findings. Her eyebrows, body hair, and eye lashes were unaffected. Laboratory evaluation done by her pediatrician, including complete blood count, renal panels, liver panel, and eye lashes were unaffected. The child has no other skin, dental or nail findings. Her eyebrows, body hair, and eye lashes were unaffected.

A hair-pull test or trichogram was done, which revealed the presence of loose anagen hairs that when examined under the microscope display derangements involving the inner root-sheath leading to poor adhesion between the cuticle of the inner root-sheath and that of the hair shaft, causing poor anchoring. Normal anagen hairs are a complex structure requiring orderly development and maturation in order to achieve the proper hair follicle. Deranged anagen follicles of LAS exhibit characteristic features under both light and electron microscopy. The keratinized cell sheath of the inner root-sheath is abnormally thickened and tortuous. The cuticle cells of the inner root-sheath contain irregularly arranged cells. The hair bulb is long, tapered, and twisted along the irregularly arranged cells. The Huxley layer also exhibits premature keratinization with edema. Lastly, the cuticle cells of the hair shaft and Henle layer contain vacuoles with nuclear debris. In addition, there is premature keratinization and dysplasia with pyknotic nuclei, sparse filaments, and intracellular debris. The trichohyalin filaments, in an electron micrograph, are reduced and disorganized. The haematoxylin-eosin stain of the abnormal inner root sheaths is characterized by distorted anagen hair morphology. The clinical picture is that of a young girl with blonde hair that can be easily and painlessly plucked. Even so, cases do occur in males and dark-haired individuals, as well as in individuals with dark hair. Recent reports document cases from Egypt and India 8.3. These phenotypes, types A, B, and C, have been described. In Type A, hair is sparse and does not grow past the shoulders. In Type B, the individual has unruly hair that is either diffused or patchy. In Type C, the hair appears normal but has excessive shedding and loose anagen hairs.10 The eyebrows and eyelashes are unaffected.

A diagnosis relies on the presence of loose anagen hairs that when examined under the microscope display derangements involving the inner root-sheath leading to poor adhesion between the cuticle of the inner root-sheath and that of the hair shaft, causing poor anchoring. Normal anagen hairs are a complex structure requiring orderly development and maturation in order to achieve the proper hair follicle. Deranged anagen follicles of LAS exhibit characteristic features under both light and electron microscopy. The keratinized cell sheath of the inner root-sheath is abnormally thickened and tortuous. The cuticle cells of the inner root-sheath contain irregularly arranged cells. The hair bulb is long, tapered, and twisted along the irregularly arranged cells. The Huxley layer also exhibits premature keratinization with edema. Lastly, the cuticle cells of the hair shaft and Henle layer contain vacuoles with nuclear debris. In addition, there is premature keratinization and dysplasia with pyknotic nuclei, sparse filaments, and intracellular debris. The trichohyalin filaments, in an electron micrograph, are reduced and disorganized. The haematoxylin-eosin stain of the abnormal inner root sheaths is characterized by distorted anagen hair morphology. The clinical picture is that of a young girl with blonde hair that can be easily and painlessly plucked. Even so, cases do occur in males and dark-haired individuals, as well as in individuals with dark hair. Recent reports document cases from Egypt and India 8.3. These phenotypes, types A, B, and C, have been described. In Type A, hair is sparse and does not grow past the shoulders. In Type B, the individual has unruly hair that is either diffused or patchy. In Type C, the hair appears normal but has excessive shedding and loose anagen hairs.10 The eyebrows and eyelashes are unaffected.

DISCUSSION

Loose anagen syndrome is an uncommon condition characterized by loosely attached hairs of the scalp leading to diffuse thinning with poor growth, thus requiring few haircuts. It was first described in 1984 by Zaun.

The anagen phase is essentially a state of cell proliferation. The process is controlled by a growth factor secreted by one of the layers of cells surrounding the growing hair. When the supply of this growth factor is reduced, the cells are no longer able to divide and leave the anagen phase. This leaves the hair in a dormant state. When the growing factor is again supplied, the hair returns to the anagen phase.

The condition is usually diagnosed in the first year of life and is more common in females. The diagnosis is based on clinical findings and, in some cases, histological examination of scalp biopsies. The condition is usually self-limiting, and most cases resolve by the age of 5.

While the exact cause of the condition is unknown, it is thought to be related to a disturbance in the growth factor supply or a decreased sensitivity to the growth factor.

REFERENCES

Sebaceous Adenoma in an Immunosuppressed Male Suggestive of Muir-Torre Syndrome: A Case Report

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INTRODUCTION

Muir-Torre syndrome (MTS) is a rare genetic disorder that causes internal malignancy as well as cutaneous manifestations. The criteria for diagnosis are having one sebaceous neoplasm and one internal malignancy.1 The most common associated malignancy is colorectal carcinoma. An associated internal cancer may appear before, after, and at the same time as the sebaceous tumor.2 Treatment involves excision of the skin lesion, resection of internal cancer, and/or screening for future malignancies. We present a case of microsatellite instability noted in a sebaceous adenoma with implications of possible MTS, in the background of immunosuppression and no current known malignancy.

CASE REPORT

A 64 year old Caucasian male presented to our clinic for a complete skin examination. His past cutaneous medical history included two squamous cell carcinomas in situ and one squamous cell carcinoma. His past medical history was significant for hypertension, hyperlipidemia, gout, and immunosuppression status post right kidney transplant in 1994 for IgA nephropathy. Medications that he takes daily were cyclosporine, azathioprine, prednisone, metoprolol, and atorvastatin. No significant family history reported.

The patient presented with a pale pink to yellow, umbilicated papule on the right nasal sidewall (Figure 1). The differential diagnosis included sebaceous, sebaceous hyperplasia, basal cell carcinoma, and molluscum contagiosum. A shave biopsy was performed on the papule after obtaining verbal and written consent, along with local anesthesia with 1% lidocaine with epinephrine. Histopathological examination revealed this papule to have basaloid cells and mature sebocytes, consistent with a sebaceous adenoma and had noted atypia; margins were still involved (Figure 2). This diagnosis led the dermatopathologist to perform further studies which included staining for microsatellite instability involved in Muir-Torre syndrome. The studies showed loss of nuclear staining for MSH2 (Figure 3) and MSH6, suggestive of MTS. Our patient was 64 years old when his first sebaceous neoplasm was discovered, and at this time, does not have a known visceral malignancy.

MTS is inherited in an autosomal pattern and caused by germline mutations in mismatch repair genes. The mutations cause microsatellite instability, specifically at MSH2, MLH1, MSH6, and PMS2. The most commonly found gene associated is MSH2,6 and only a small subset shows mutations in MSH6.7,8 However, a newly described subtype has been described, MTS II, that does not have microsatellite instability mutations associated with it but a base excision repair gene mutation in MSH6,9,10 and shown to be autosomal recessive.10 Although MTS is autosomal dominant, immunosuppression has been reported to increase the development of tumors. Two cases of patients’ status post organ transplants that had expensive sebaceous neoplasms while on tacrolimus.9,10 Our patient was on cyclosporine, azathioprine, and prednisone for immunosuppression and had a solitary sebaceous adenoma, but one may reason that these medications increased the development of the neoplasm. His tumor did reveal loss of nuclear staining for MSH2 and MSH6.

Clinical findings associated with MTS are a variety of sebaceous neoplasms, including sebaceous adenomas, carcinomas, and epitheliomas, and keratoacanthomas. The individual must also have an internal malignancy.11 Gastrointestinal malignancy, specifically colorectal adenocarcinoma, is most commonly associated.

Figure 1. Pink to yellow, umbilicated papule on the right nasal sidewall.

However, other have been reported, such as, genitourinary, breast, hematological, endometrial, and central nervous system.9,11 The internal malignancy may present before or after the cutaneous manifestations of MTS. In 205 cases of MTS, it is calculated that 12% of cases the cutaneous neoplasm appeared concurrently, 22% appeared prior, and 56% of the time after a finding of internal malignancy.2 At present, our patient does not have an internal malignancy, but will continue to have close monitoring. Diagnosis is a clinical one directed by the finding of a sebaceous adenoma and an internal cancer.1 This can be expanded to having a high index of suspicion with pathology findings and family history for MTS or colon cancer, leading to MMR genetic sequencing or loss of satellite staining on tissue. Histologically, MTS is most notably categorized by sebaceous neoplasms. The sebaceous neoplasms show lobules of sebocytes with varying percentage of basaloid cells, and include sebaceous adenoma, epithelioma, and carcinoma.1,5,11

The treatment for MTS is surgical excision for cutaneous and internal lesions. Sebaceous carcinomas can be excised using Mohs micrographic surgery. Fortunately, many of the sebaceous neoplasms in MTS are often curative with excision. Management after diagnosis is monitoring for internal cancer. Current recommendations include screening in individuals with MTS and their first-degree relatives with annual colonoscopy, pelvic exam, and physical exam for breast, testicular, and prostate.2 It is reasonable to consider renal ultrasounds, upper endoscopy, and thorough neurological exam as precautions for the rarer associated malignancies.1,5 This case report’s patient undergoes yearly examination, especially due to his history of renal transplant, and does not currently have an internal malignancy. The patient will continue to cancer screenings and made aware of his increased risk for colon cancer.

DISCUSSION

Muir-Torre syndrome (MTS) was first described in 1967 by Muir3, and in 1968 by Torre.4 It is a rare genodermatosis that predisposes the individual to sebaceous neoplasm and internal malignancies, most notably colorectal adenocarcinoma. This syndrome is described as a variant of hereditary nonpolyposis colorectal cancer (HNPCC) or Lynch syndrome.1,5 It is known to have a higher incidence in males than in females, stating a ratio of 3:2.2 The mean age for cutaneous manifestations is 53 years, and the internal malignancy may appear prior or after the discovery the sebaceous neoplasm. Our patient was 64 years old when his first sebaceous neoplasm was discovered, and at this time, does not have a known visceral malignancy.

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CONCLUSION

Our patient shows an unusual presentation for this already rare syndrome as he had a nephrectomy and status post kidney transplant due to IgA nephropathy years prior. He currently has no known visceral malignancy. His presentation of sebaceous adenoma with atypia led to the staining for microsatellite instability. The patient has had cancer screenings and will continue to be followed, allowing for early detection. This interesting case urges practitioners to remember MTS when pathology of sebaceous neoplasms arise in an atypical setting and promote further awareness of this genodermatosis.

REFERENCES

An Uncommon Case of Mucormycosis

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Introduction

- Mucormycosis is caused by a family of ubiquitous saprophytic fungi known as mucoraceae. Rhizopus spp is by far the most common pathogen. These are angiotropic pathogens that will grow within and eventually occlude blood vessels ultimately resulting in necrosis of affected tissue.1
- Mucoraceae are opportunistic pathogens that typically infect patients with diabetes mellitus, but it can also infect other immunocompromised hosts, such as transplant or burn patients. Due to an increase in incidence, mucormycosis has been identified as an emergent disease, likely due to a rise in susceptible hosts.2
- There are five distinct clinical presentations of mucormycosis; rhinocerebral, cutaneous, pulmonary, gastrointestinal, and cardiac mucormycosis.
- Rhinocerebral and cutaneous mucormycosis are the presentations more likely to be diagnosed by a dermatologist.
- Rhinocerebral mucormycosis may present initially with banal symptoms such as sinusitis, which progresses to necrotic discharge or even visibly necrotic mucous membranes. Symptoms of the infection will be specific to the structures and extent of involvement. Bilateral involvement is uncommon.1
- Cutaneous Mucormycosis can be seen as a primary infection or represent secondary spread from another site. Primary cutaneous mucormycosis spreads via direct inoculation either by trauma such as an IV site or via a contaminated dressing of an open wound. Cutaneous mucormycosis can present as papules, plaques, or nodules. These lesions will characteristically progress to necrotic lesions. Other presentations can include hemorrhagic bullae or non-healing ulcers.3
- Clinical suspicion and early diagnosis is essential as this is a rapidly progressing infection with a high mortality rate. Treatment is emergent surgical debridement of all necrotic tissue; performed in an attempt to limit the spread of the infection. Liposomal amphotericin B is the antifungal of choice.1,4 It can be combined with other antifungals, such as posaconazole or caspofungin, for salvage therapy in whom surgical treatment has failed.4,5

Case Description

A 55 year-old female with past medical history of uncontrolled diabetes with a below the knee amputation and chronic kidney disease presented with acute onset necrosis of her forehead and scalp. Ten days prior to admission, she was admitted for intractable headache and periorbital edema. Her symptoms had been present for at least 3 days (13 days prior to presentation total). A work up for angioedema and acute kidney failure was negative and the patient improved on steroid therapy. The patient was discharged on a two week steroid taper.

At the time of admission, patient was awake and alert with bilateral periorbital edema. Her symptoms had been present for at least 3 days (13 days prior to presentation total). A work up for angioedema and acute kidney failure was negative and the patient improved on steroid therapy. The patient was discharged on a two week steroid taper.

Surgical debridement progressed to involve the entire forehead and scalp, revealing invasion into cranial blood vessels. The H&E staining of the tissue revealed extensive necrosis with non-septate broad branching hyphae within dermal blood vessels consistent with Mucormycosis. Fungal cultures did not reveal the causative organism. The patient died in hospice three days after admission.

Discussion

- This case also represents an unusual presentation of Mucormycosis due to the subacute timeframe for the initial presentation of her symptoms and for the stepwise fashion which the infection progressed.
- Her initial symptoms, headache and periorbital edema, had been present for 13 days prior to the onset of scalp necrosis. This likely represented the first cutaneous indication of the fungus spreading outside the sinuses.
- These symptoms persisted for nearly two weeks before the supratrochlear and supraorbital arteries were compromised resulting acute onset necrosis.
- This case highlights progression of Mucormycosis from non-descript skin and clinical findings to a more characteristic presentation of the disease.

Conclusions

- Mucormycosis is an opportunistic infection caused by Rhizopus, Mucor, and Absidia.
- Rhinocerebral and cutaneous mucormycosis result in necrotic or ulcerated lesions
- Treatment of choice is urgent surgical debridement and Liposomal Amphotericin B
- Rhinocerebral Mucormycosis can present with bilateral periorbital edema

References

Nevus lipomatosus superficialis (NLS) and spindle cell lipoma (SCL) are both relatively rare benign neoplasms. NLS can further be subdivided into two clinical types: the classical type and the solitary type. The classical type typically presents in the lumbar area at birth or within the first three decades of life as multiple, non-tender papules or nodules, which commonly coalesce to form plaques. The solitary form has no location preference and usually occurs later in life as a nodular lesion. SCL is usually found in the subcutaneous tissues, with rare intradermal cases reported in the literature. This neoplasm most commonly occurs on the neck, shoulders, or back of middle-aged to elderly males as a subcutaneous nodule. In this case report, the authors present a rare and interesting presentation of a NLS with co-existing features of a dermal SCL.

A Case of Nevus Lipomatosus Superficialis with Features of a Spindle Cell Lipoma

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ABSTRACT

Nevus lipomatosus superficialis (NLS) and spindle cell lipoma (SCL) are both relatively rare benign neoplasms. NLS can further be subdivided into two clinical types: the classical type and the solitary type. The classical type typically presents in the lumbar area at birth or within the first three decades of life as multiple, non-tender papules or nodules, which commonly coalesce to form plaques. The solitary form has no location preference and usually occurs later in life as a nodular lesion. SCL is usually found in the subcutaneous tissues, with rare intradermal cases reported in the literature. This neoplasm most commonly occurs on the neck, shoulders, or back of middle-aged to elderly males as a subcutaneous nodule. In this case report, the authors present a rare and interesting presentation of a NLS with co-existing features of a dermal SCL.

CASE REPORT

A sixty-two-year-old male presented with a chief complaint of an asymptomatic, enlarging growth located on the left lower extremity that had been present for approximately three years. He denied associated symptoms including pain, tenderness, pruritus, bleeding, ulceration, and discharge. Furthermore, he denied any previous evaluation and treatment of the lesion. Patient reported no prior personal or family history of skin cancer.

On examination, the patient was a Fitzpatrick skin type II with a solitary 1.5 cm skin-colored to pink pedunculated papule on the left proximal posterior thigh as shown in Figure 1. A shave biopsy of the lesion was performed and a differential diagnosis included neurofibroma, benign intradermal nevus, fibroepithelial polyp, basal cell carcinoma, and amelanotic melanoma.

Histologic sections demonstrated a pedunculated papule with basket-weave stratum corneum and a relatively normal appearance to the epidermis. Within the dermis, relatively normal collagen bundles with increase in fibroblasts within the superficial dermis were observed. Of note, lobular aggregations of adipocytes were found to be replacing much of the dermis with many areas of the adipocytes showing spindle cells and abundant mucin (Figures 2a-c). Based on the histology, a diagnosis of nevus lipomatosus superficialis with features of a spindle cell lipoma was made. Due to the benign nature of this entity, no further treatment was necessary or recommended. Excision was discussed with the patient in case of recurrence of the lesion, if desired.

DISCUSSION

Nevus lipomatosus superficialis (NLS) is a benign hamartomatous condition characterized by ectopic adipocytes in the dermis. The condition is divided into two clinical presentations: the classical Hoffman-Zurhelle subtype and the solitary subtype.1,4 In the classical Hoffman-Zurhelle subtype, clusters of soft skin-colored or yellowish papulonodules or plaques may be appreciated. In the solitary subtype, lesions present later in life as a single dome-shaped or sessile papule. The classical subtype most commonly presents in the pelvic or gluteal region at birth or within the first three to four decades of life. In contrast, there is no site predilection for the solitary subtype.4

NLS may be differentiated from other entities in the differential diagnosis such as nevus sebaceous, neurofibromas, fibrolipomas, hemangiomas, lymphangiomomas by clinical presentation, and definitively by histology. Histopathology of NLS shows a dermal proliferation of mature adipocytes that may be connected to the subcutaneous tissue or separated from the subcutis by collagen.7 The adipocytes may present solitarily between collagen bundles or form aggregates around blood vessels or eccrine glands. Infrquently, spindle cells representing immature fat cells may be present. Cases of co-existing café-au-lait macules, scattered leukoderma, hypertrichosis, and comedo-like lesions within a NLS have been reported.1,4

A spindle cell lipoma (SCL), and its pleomorphic subtype, in contrast to NLS, most commonly presents in the fourth to seventh decade of life as a well-circumscribed mass in the subcutaneous tissue of the upper back, posterior neck, or shoulders.9 Diagnosis of SCL and the pleomorphic subtype requires mature fat cells, spindle cells, and strands of strongly eosinophilic collagen.7 Although SCL typically arises in subcutaneous tissue, rare cases of SCL and the pleomorphic subtype occurring within the dermis have been reported. The dermal SCL and pleomorphic lesions differ from the classic SCL as they are poorly circumscribed and unencapsulated.10 In addition, the dermal variant of a SCL may not have a predilection for any specific site or may have a slight predilection for the thigh-th buttocks-groin area.9

Our case highlights a rare and interesting presentation of a NLS with co-existing features of a dermal SCL, one of only a few reported in the literature. Neither NLS or SCL have concern for systemic involvement or malignancy. Therefore, treatment is not necessary for any reason other than cosmesis, and excision is curative with rare recurrence. Rarely, these lesions may ulcerate with associated foul-smelling discharge. In such instances, surgical excision may be warranted.1,2

REFERENCES

Novel Use of Combination Therapeutic Plasma Exchange and Rituximab in the Treatment of Nivolumab-Induced Bullous Pemphigoid

Alyson V. Ridpath, DO1, Polina V. Raspa, MD2, Sabrina M. Shearer, MD3, Benna K. Pignotti, OMS IV3, Scott R. Scrapes, MD1, Thomas Chenki, DO1, Benjamin H. Kaffenberger, MD1
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Introduction

Immune checkpoint inhibitors are a new class of medication for the treatment of metastatic melanoma. Recently, there have been seven documented cases of bullous pemphigoid (BP) associated with the programmed death-1 (PD-1) and programmed death ligand-1 (PD-L1) inhibitors.

While corticosteroids are effective in Bullous Pemphigoid, we report a case of severe, refractory nivolumab-associated BP successfully treated with plasmapheresis and rituximab, to maintain the enhanced cellular immunity without need for further oral corticosteroids.

Clinical Case

A 67-year-old male with stage IV BRAF- and c-KIT-negative, NRAS-positive melanoma of unknown primary with metastases to the liver, lung and brain was started on nivolumab 3 mg/kg every two weeks. After 16 cycles over 32 weeks, he presented to the emergency department with a new, severe, pruritic, bullous eruption covering approximately 90% body surface area, and altered mental status. (Fig. 1)

Laboratory Studies

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Table 1: Relevant laboratory and pathology results

Therapeutic Challenges:

• Finding an effective treatment that balanced immunosuppression with immune checkpoint inhibitor mechanism of action
• Failure of first line agents
• Therapy with a rapid onset of action
• The need to resume therapy for underlying metastatic melanoma

Initial Treatment:

• Prednisone up to 1 mg/kg daily
• Betamethasone dipropionate 0.05% cream twice daily
• Deferral of nivolumab until completion of steroid taper

Complications:

• Bacteremia
• Worsening of BP
• Inability to taper the patient to low dose steroids

A New Approach

• Initiation of plasma exchange for a total of 5 treatments
• Continuous rituximab therapy consisting of two 1000 mg infusions 15 days apart.

Patient Progress:

• Near clearance of BP and a significant reduction of anti-bullous pemphigoid 180 autoantibodies. (Fig. 2, Table 1)
• He continues to use betamethasone dipropionate 0.05% cream twice daily without systemic corticosteroids.

Initial Treatment of Oral and Topical Steroids:

• Of the reported cases of PD-1/PD-L1 inhibitor-induced BP, the majority were controlled with topical and/or oral steroids alone.1,2

Therapeutic Plasma Exchange:

• Targeted immunosuppressive effects
• Rapid onset of action
• Used successfully to treat BP

Rituximab:

• Previous single agent success in treating and preventing relapse of nivolumab-induced BP
• Anti-CD20 may assist in treatment of melanoma if tumor has a CD20 positive cell population

Discussion

Autoimmune bullous dermatoses are a recently described adverse event to the PD-1/PD-L1 inhibitors.3 The pathogenesis of BP may be due to excessive B-cell co-stimulation and increased autoantibody production with blockade of PD-1/PD-L1 receptors.1,2,5 None of the prior patients mentioned neurologic or cutaneous metastases, which potentially could have an effect on exposing epitopes for the development of BP.

A diagnosis of lichen planus pemphigoides instead of BP is another consideration given that BP-180 is elevated in lichen planus pemphigoides and lichenoid eruptions are a common cutaneous adverse effect to immune checkpoint inhibitors. However, none of the patients had coexisting lichen planus or a lichenoid infiltrate on pathology.

Given that all the patients have been elderly and the majority displayed the characteristic distribution of BP, it is possible that some of the patients previously could have had low titer autoantibodies, yet clinical significance of their disease was precipitated by the PD-1/PD-L1 inhibitors.

Conclusion

This case is novel because it demonstrates the potential for rapid and sustained improvement in nivolumab-induced BP with plasma exchange and rituximab for targeted immunomodulation in the increasing population of patients presenting with unique autoimmune phenomenon with checkpoint inhibition.

References


Figure 1: Coadjacent bullous eruption covering approximately 75% of body surface.

Figure 2: Post-treatment with healing erosions and post-inflammatory hyperpigmentation.

Figure 3: Subepidermal bullae with few eosinophils.

Figure 4: Coadjacent bullous eruption covering approximately 75% of body surface.

Figure 5: Post-treatment with healing erosions and post-inflammatory hyperpigmentation.

Figure 6: Subepidermal bullae with few eosinophils.
A Rare Etiology of Flagellate Erythema: A Case Report & Review

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INTRODUCTION
• Flagellate erythema is a dermatosis comprised of hyperpigmented, pruritic, linear, & erythematous streaks.
• It has been described in association with bleomycin use1, dermatomyositis2, adult-onset still disease3, & shiitake mushroom consumption6.
• The recognition of this rare diagnostic clue is paramount in discovering its underlying condition as it may have significant health implications for the patient.

CASE PRESENTATION
• 45 y/o female with a hx of migraines & excessive sun exposure
• Presents with complaints of a very pruritic rash on her abdomen, buttocks, & lower extremities.
• 2 days prior, took acetaminophen-butalbital-caffeine & malanga (Ipomoea batatas) & boniato (Xanthosoma sagittifolium) & butalbital
• Pt denies any prior occurrences or any other associated symptoms.
• On exam, patient presented with multiple erythematous, hyperpigmented linear streaks scattered on bilateral legs, buttocks, & inferior abdomen consistent with flagellate erythema. Excoriations were diffusely present.
• Histology: a dense, perivascular lymphocytic infiltrate with very few eosinophils & marked dermal edema. Melanin diffusely scattered within epidermal basal layer but not within the dermis. No iron dermal deposition.

DISCUSSION
• Flagellate erythema is a rare cutaneous phenomenon described as linear erythematous streaks with pruritus & hyperpigmentation. Known etiologies are bleomycin, dermatomyositis, adult-onset still disease, & shiitake dermatitis. Our patient did not fall into any common etiological category & historically was newly exposed to Butalbital-acetaminophen-caffeine, malanga, & boniato prior to onset. A thorough literature search on these three compounds showed no evidence of flagellate erythema as an adverse reaction.
• Bleomycin, an antitumor medication, is used as treatment with certain malignancies. Flagellate erythema has been reported as an adverse effect of bleomycin with an incidence rate of 10-20%. The precise mechanism remains unclear although some speculate that bleomycin induces generalized pruritus leading to scratching. The scratching allows for the drug to exit blood vessels & react with the skin.
• Dermatomyositis is an inflammatory myositis with cutaneous manifestations. Well characterized cutaneous manifestations are heliotrope rash, Gottron’s papules, periungal telangiectasia, & shawl sign. Flagellate erythema has been reported in association with disease activity & may precede muscle symptoms. Dermatomyositis has a 15-25% increased risk for malignancy.
• Adult-onset Still’s disease is an inflammatory disease comprised of high spiking fevers, arthralgia, hyperferritinemia, hepatosplenomegaly & rash. The characteristic rash is a salmon maculopapular erythema that appears during high fevers. Persistent erythematous plaques suggesting flagellate erythema have been reported in few cases.7
• Shiitake dermatitis, AKAs tocoerderma, is caused by the consumption of undercooked shiitake mushrooms. Hypersensitivity is highest in China & Japan where the mushroom is commonly grown & consumed. Flagellate erythema originates from significant pruritus & the Koebner phenomenon leading to linear grouping of non-pigmented papules. The rash improves on its own within two weeks.7

CLINICAL & HISTOPATHOLOGICAL IMAGES

DISCUSSION
• Flagellate erythema is not a sufficient cause to terminate cancer therapy
• Erythematous papules from shiitake consumption & lipids

REFERENCE INFORMATION

REFERENCES
Introduction
Basaloid follicular hamartoma (BFH) is a rare, benign, neoplasm of the hair follicle, characterized by multiple brown papules typically involving the face, scalp, and trunk. Diagnosis is made histologically via biopsy, which is important in order to distinguish BFH from basal cell carcinoma (BCC) or other epithelial neoplasms with malignant potential. Correct diagnosis allows for avoidance of unnecessary surgeries to remove benign lesions and prompt management of potential malignancies.

Case Description
A 68 year old male presented to Dermatology seeking cosmetic treatment for hundreds of homogenous, waxy, verrucoid, brown papules on his face. The patient stated they had been present for years and had recently been increasing in number. The patient had not sought any prior treatment but found them cosmetically bothersome. There was no history of myasthenia gravis and no physical exam findings such as palmar pitting or alopecia to indicate generalized type. (Figure 1)

Figure 1 Right temple before treatment with PDL.

Figure 2 – Right temple at 2-month follow-up

Discussion
Clinically, most cases of BFH present with multiple 1-2mm tan to brown colored papules located on the face, scalp, neck, axilla, trunk and pubic area.1,4 Five types of BFH have been described in the literature:
1) Solitary or multiple papules
2) Localized linear papule/plaque
3) Localized papule with associated alopecia;
4) Generalized papules associated with myasthenia gravis and alopecia
5) Generalized autosomal dominant familial type without associated disorders.3,4,5,6

The patient in this case was deemed to have the multiple papules type, as he had no other relevant signs or symptoms and no family history that might place him in another subtype.

BFH arises due to a mutation in the patch (PTCH) gene located on chromosome 9q23, the same gene thought to cause nevoid basal cell carcinoma syndrome.3 Expression of the mutation is thought to be milder in BFH, as these are benign tumors.6,8

Conclusions
Treatment options for BFH are limited and no standard of care has been determined. There are reports of 5-aminolevulinic acid plus photodynamic therapy as a safe and mildly effective cosmetic treatment for BFH, and this is the treatment of choice in children.1 Our patient was treated with Pulsed Dye Laser (PDL). The right temple had 1 treatment with pulse duration of 1.5msec and 10J cryogen. At 2-month follow-up (Figure 2), the patient had significant cosmetic improvement.

PDL has traditionally been used to treat vascular lesions such as port-wine stains, but has also been used for acne, scars, and photorejuvenation. While the mechanism is unknown, PDL may work secondary to its destructive effect on the surrounding vasculature supplying the neoplasm, thereby reducing the nutrients available to the affected cells. It also may target the chromophore of melanin, causing selective photothermolysis of BFH cells.9,10,11

More research is needed to find effective treatments for BFH, as these lesions can be cosmetically bothersome to affected individuals. However, the authors postulate that PDL may be a safe and effective treatment for BFH, and possibly other adnexal neoplasms.

References
Worsening indurated pink translucent nodules and severe hyperkeratosis of the lower extremities: A Case of Elephantiasic Pretibial Myxedema.

A 61 year old white woman presented with bilateral lower extremity dermatis, swelling and skin thickening that began 5 years ago; shortly before she was diagnosed with Graves disease (Figures 1-3).

Patient’s symptoms progressively worsened post thyroidectomy and achievement of euthyroid state with levothyroxine. Previous diagnoses included cellulitis and myxedema treated with multiple failed attempts of oral antibiotics. No family history of related conditions. No previous biopsy was obtained.

**PHYSICAL EXAM:** Indurated, 1-2cm thick violaceous plaques with indurated pink translucent nodules; associated deep fissures with active serous drainage and overlying yellow-white crust on bilateral pretibial areas, ankles and dorsal feet. (Figures 1-3) Plantar surface was covered by thick scale.

Other physical exam findings included proptosis, exophthalmos and surgical scar on the anterior neck.

**DX:** After obtaining informed consent 2 biopsy specimens were obtained for hematoxylin-eosin and other special stains (Figures 3-6).

Elephantiasis pre tibial myxedema was diagnosed based on these clinical and histological findings.

**CASE PRESENTATION**

**HPI:** A 61 year old white woman presented with bilateral lower extremity dermatitis, swelling and skin thickening that began 5 years ago; shortly before she was diagnosed with Graves disease (Figures 1-3).

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**LEARNING OBJECTIVES**

- Recognizing elephantiasis pre tibial myxedema (PTM)
- Understanding pathophysiology of elephantiasis PTM.
- Creating treatment plan for patient suffering from elephantiasis PTM.
- Ddx for elephantiasis PTM.

**DISCUSSION**

**EPIDEMIOLOGY:** Elephantiasis pre tibial myxedema (PTM) is the most severe variant of non-filarial myxedema occurring in only 1% of patients with Grave’s disease.

**PATHOPHYSIOLOGY:** It is theorized that T-cells stimulate shared antigens between the thyroid and pre tibial tissue and release TGF-B and IL1-alpha that stimulate fibroblasts to produce and deposit mucin-like glycosaminoglycans in tissues. The pre tibial fibroblasts may be more sensitive to this stimulation. The pretibial area is favored secondary to hydraulic forces, decreased lymphatic cytokine clearance and dependent position.

**CLINICAL:** Grossly enlarged and disfigured appendage, usually with functional restriction and cosmetic concerns for the patient. Cutaneous changes include non-pitting edema of lower extremities that does not resolve with cessation. The initial cobblestone appearance later becomes mossy and verrucous. Because hair follicles are prominent, it produces the characteristic papules d’orange appearance.

**TREATMENT:** Complete decompressive lymphatics with surgical scar on the anterior neck. Therapeutic modalities like complete decompressive lymphatics with surgical scar on the anterior neck. Therapeutic modalities like complete decompressive therapy, topical corticosteroids with inclusive dressing, povidone-iodine, sclerode and weight reduction have proven beneficial.

**REFERENCE**

7. Largo Medical Center, Largo, Florida
Abstract

Severe cutaneous adverse reaction to drugs (SCARs) affect approximately 2% of hospitalized patients and are associated with morbidity, mortality, health-care cost, and drug and diagnosis challenges. Diagnosis criteria has been established for each of these entities; however, there are overlapping features which pose a diagnosis challenge. For example, AGEP may present with facial edema and present with blisters or even mucosal involvement. DRESS may have pustules and even have a TEN-like presentation. It is important for health care providers to be aware of these overlapping features in order to avoid pitfalls in diagnosis and treatment.

Dress syndrome has up to 20% mortality and carries a risk of autoimmune conditions including autoimmune thyroiditis, while AGEP tends to be benign and carries no sequela.

Case

A 37-year-old male with history of seizure disorder on Levetiracetam for several years presented to the ED with an itchy rash for 4 days. Phenytion was added to his seizure treatment 4 weeks prior. Initial work up demonstrated leukocytosis with elevated eosinophils, and elevated liver enzymes. Patient had confluent erythema involving trunk, upper extremities, thighs and face. There were hundreds of tiny non-follicular sterile pustules on the upper chest and face. He had facial edema and desquamation of the upper chest and neck. Punch skin biopsy showed pustular dermatitis with eosinophils. Patient met the diagnostic criteria of DRESS under the EuroSCAR and Japanese’s group criteria. He also has a possible diagnosis of AGEP under the EuroSCAR study. He had facial edema and desquamation of the upper chest and neck. Punch skin biopsy showed pustular dermatitis with eosinophils. Patient met the diagnosis of DRESS under the RegiSCAR and Japanese’s group criteria. He also has a possible diagnosis of AGEP under the EuroSCAR study. He had facial edema and desquamation of the upper chest and neck. Punch skin biopsy showed pustular dermatitis with eosinophils.

Discussion

Drug reaction with eosinophilia and systemic symptoms (DRESS), usually begins 2-6 weeks after exposure to certain drugs. DRESS has a complex natural course and very diverse clinical presentation. Anticonvulsant medications tend to be the most common triggers, however, other drugs including antibiotics, osteoporosis medications, and kinase inhibitors have been reported. Dermatologic findings include facial edema, exanthematous morbilliform eruption, pustules and sometimes mucosal involvement. Systemic involvement is thought to result from organ infiltration of eosinophils or lymphocytes. The organs typically involved include liver presenting as hepatic cytolyis to fulminant hepatitis; kidney, lung and heart involvement.

Cardiac involvement leading to myocarditis or pericarditis with elevation of cardiac enzymes can be fatal.

Acute generalized exanthematous pustulosis (AGEP) is an adverse drug reaction which typically begin within 48 hours of drug exposure but can be as long as 11 days post exposure. Most common associated medications include aminopenicillins, quinolones, hydroxychloroquine, sulfonamides, terbinafine, diltiazem, ketocanazole, and fluconazole; however, viral infections, dietary supplements, and hypersensitivity to mercury, radiation and spider bites have been reported as triggers for AGEP.

Dermatologic features of AGEP include hundreds of nonfollicular sterile tiny pustules on an erythematous base. It favors intertriginous region and often begins on the trunk and it can be pruritic.

There has been randomized controlled studies looking into management of DRESS and AGEP. Systemic corticosteroids have been used for the management of DRESS but there is no standardized assessment of outcomes. Retrospective studies have shown the use of potent topical corticosteroids to be helpful in mild to moderate DRESS with less side effects that systemic corticosteroids. Prompt withdrawal of the offending drug for AGEP my be adequate with topical corticosteroids as adjunctive therapy.

Conclusion

Severe cutaneous adverse drug reactions (SCARs) in hospitalized patients carry increase morbidity, mortality and health-care cost. SUS/TEEN are considered the most severe types of SCARs; however, DRESS syndrome carries up to 20% mortality. Patients with DRESS syndrome can have multiple organ involvement including cardiac involvement which can be fatal. Additionally, patients with DRESS syndrome carry a higher risk of autoimmune thyroiditis.

Here we presented a case of DRESS syndrome associated with use of phenytion. Our case was interesting because this case also showed AGEP features including multiple tiny non-follicular pustules on an erythematous base involving the trunk and face.

This case shows that SCARs have overlapping features and therefore, practitioners must be aware of such features as to avoid delay in diagnosis and treatment. DRESS syndrome carry a much higher mortality than AGEP and there is increased risk of autoimmune thyroiditis and therefore close follow up is required.

References

Introduction

Erosive pustular dermatosis (EPD) is a superficial skin disorder usually arising on the scalp of the elderly. It was first reported in Britain in 1977.1 It lacks specific clinical and histopathological findings. It is characterized as chronic intermittent crusted papules or plaques, pustules and erosions developed in local traumatized skin. High potency topical steroids have the most success in curing the condition. At some point during the clinical course, a scarring alopecia develops that can also involve adjacent areas. EPD of the scalp is diagnosis of exclusion after neoplastic, infectious or other inflammatory conditions are ruled out. The predisposing factors include age and a history of various types of local skin trauma, thus, the incidence of EPD of the scalp is most likely underestimated in American literature.2

Case Presentation

A 92 year old Caucasian female presented to a dermatology office with the complaint of intermittent dry areas and pimples on the top of her head for over a year. The patient had a history of androgenic alopecia affecting the vertex and apical portions of the scalp. Upon initial presentation she had several yellow crusted papules and pustules located in the thinning portion of her apex (Figure 1). A presumptive diagnosis of folliculitis and impetigo was made and she was placed on topical clindamycin and mupirocin twice a day. Her lesions did not improve and she returned in two months for reevaluation. Upon examination she had several rough scaly papules on an erythematous base, a few erosions and a dark crusted hyperkeratotic plaque (Figure 2). She was thought to have actinic keratosis and treated with cryotherapy. A biopsy was done on the larger plaque to rule out neoplastic behavior. The histopathology revealed a mixed inflammatory infiltrate without any atypia (Figures 5-8). A diagnosis of EPD was considered and the patient was placed on Clobetasol 0.5% ointment. The patient showed clinical improvement, although throughout the following year she continued to have flares. Over time she developed fibrosis of her scalp (figures 3-4).

Histology

• Recognizing characteristic histological patterns with microscopy with staining or immunofluorescence assist in ruling out other possible diagnoses.2

• Histological findings of erosive pustular dermatosis are non-specific with link to neutrophil-stimulating cytokines and chemokines.6,8

• There is epidermal atrophy without evidence of atypical keratinocytes.

• In longstanding lesions there is also a loss of hair follicles and sebaceous glands with dermal fibrosis associated with resultant scarring alopecia.3

Images

Figure 1 shows the patient’s initial clinical presentation. There are multiple yellow crusted papules and pustules.

Figure 2 depicts a hyperkeratotic plaque concerning for neoplastic behavior that was biopsied.

Figures 3 and 4 demonstrates erosions, crusted papules and plaques in the setting of worsening scarring alopecia two years later.

Histology

• Figures 5-8 depict extensive inflammatory infiltrate, lack of atypical keratinocytes, and decreased density of hair follicles and sebaceous glands.

Discussion

Symptoms & Clinical Course: Lesions appear as superficial yellow-brown crusts with underlying erythema. The borders are irregular and may have smaller areas of preserved skin. The number of lesions and surface area it covers varies from case to case.3 Primary lesions are usually sterile, but samples from the area may be positive for bacterial or mycological colonization. Lesions are generally asymptomatic but can be associated with pain or pruritus.2 There is often a scarring alopecia with destruction of the hair follicles and follicular stem cells due to chronic inflammation. Due to the chronic nature of EPD, a clinical suspicion of neoplasm formation should be considered during future evaluations of the area.4

Populations at Risk: EPD is typically seen in elderly patients, with women more commonly diagnosed than men.1 Ultradamage along with other etiologies and modalities causing skin injury are predisposing factors. There is also association in patients with autoimmune disorders such as Hashimoto’s thyroiditis, autoimmune hepatitis, rheumatoid arthritis, myelodysplastic syndrome, and myasthenia gravis.6,8

Pathogenesis: EPD is believed to be due to local skin trauma.4 The pathogenesis of the disorder is poorly understood. Published cases include patients with a history of sun exposure, CO2 laser therapy, topical imiquimod muUBL, topical tretinoin, hair transplantation, surgery for coelom.2 Scars, gynecomastia, cryotherapy, radiation, contact dermatitis from a prosthesis hair piece, and post-herpetic zoster.10 Immunosenescence, an age-associated decline in the immune system, is a hypothesized mechanism that leads to an abnormal immune response to wound healing.10,11 An additional association with autoimmune disorders is supported with a link to neutrophil-stimulating cytokines and chemokines.6,8

Diagnosis: The differential diagnosis for EPD includes squamous cell carcinoma, basal cell carcinoma, localized cicatricial pemphigoid, chronic discoid lupus erythematosus, folliculitis decalvans, ulcerative pustular dermatosis and bacterial or fungal infections.1 EPD is a diagnosis of exclusion.

Treatment: EPD is treated with the most success with high potency topical steroids. Other pharmacological treatments include topical tacrolimus, isotretinoin, calcipotriol, and dapson.21 Even though EPD could result from photodynamic therapy, there has been success with its use.2 Surgical excision of the lesions is another treatment option; however, it may cause recurrence due to its traumatic nature.3 Scarring alopecia is usually a permanent consequence of the disorder no matter the treatment modality.

Conclusion

EPD presents as superficial crusts and ulcerations in epithelial areas with a history of local trauma typically in elderly females. Because of these non-specific symptoms, diagnosis of EPD is delayed and a diagnosis of exclusion. EPD resolves with a topical steroid regime and heals with scarring alopecia in the affected area.

References

Paronychia with excessive granulation tissue as a side effect of isotretinoin treatment

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Hackensack Meridian Health Palisades Medical Center

ABSTRACT
Isotretinoin has been successfully used to treat many patients, however, not without numerous adverse effects. Various adverse effects have been reported, including teratogenicity, myalgias, hypertriglyceridemia and transaminitis.1 Less commonly reported as a side effect is excessive granulation tissue resulting in acute paronychia. Presented here is a patient treated with isotretinoin who developed painful paronychia of the great toe with excessive granulation tissue.

CASE PRESENTATION
Patient is a 19-year-old female who presented with severe nodulocystic, scarring acne. After failing a topical regimen, she was initiated on isotretinoin at 0.5 mg/kg/day with a goal of 120 mg/kg cumulative dose. Her initial laboratory evaluation did not reveal any abnormalities. Her treatment course was complicated by hypertriglyceridemia requiring treatment with fenofibrate and simvastatin. She also developed a transaminitis involving both her ALT and AST. She completed a six-month course with significant improvement in her acne. She returned to the clinic three weeks after completion with complaint of a red, swollen and significantly painful right great toe that had developed over 3 days. She denied systemic symptoms. The patient was initiated on cefadroxil 500 mg po twice a day for seven days and topical mupirocin ointment twice daily for ten days. She was seen in clinic four days later and noticed decreased erythema and tenderness.

DISCUSSION
The adverse effects of isotretinoin treatment are common and vary in severity. Although this adverse event has been reported for many years, the mechanism remains unknown. Current theory is there is a hyperactive inflammatory and wound healing response at the sites of involvement. The isotretinoin causes exacerbation of epithelial function in the nail matrix, leading to a local exfoliative dermatitis with accumulation of scales in the nail folds.1,2 The scales are introduced into the periungual tissue and lead to a foreign body reaction. Similar reaction has been seen in patients receiving etretinate for the treatment of psoriasis.3 Due to the lack of data on this condition, treatment is not well documented. It remains controversial as to whether isotretinoin needs to be discontinued; reports state either decreasing dosage or discontinuing the medication may be necessary. de Almeida Figueiras et al. recommend starting treatment with a two to three week course of topical steroids and topical antibiotics. If the patient fails this regimen, oral antibiotics may be required, and surgical treatment remains the last resort.1,2

CONCLUSION
Many of the more common side effects of isotretinoin are well documented in regards to their prevalence and management. Paronychia with excessive granulation tissue has been reported for many years, however, both its exact prevalence and its pathogenesis remain unknown. It is important to be aware of these less common side effects and familiarize with their management.

REFERENCES

CONFLICTS OF INTEREST
No relevant conflicts of interest to disclose.
Upcoming Meetings:

2018 AOCD Fall Meeting
Westin San Diego - Gaslamp Quarter
San Diego, CA
October 9 - October 13, 2018

2019 AOCD Spring Meeting
JW Marriott Orlando
Orlando, FL
April 7 - April 13, 2019

2019 AOCD Fall Meeting
Omni Nashville Hotel
Nashville, TN
September 24 - September 28, 2019