INFECTIOUS DISEASES: BACTERIAL INFECTIONS

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OBJECTIVES

- Describe bacterial skin infections commonly seen in the outpatient setting, including presentation, diagnosis, and treatment.
- Discuss antibiotic resistance and current recommendations.
- Discuss dermatologic surgical site infections.
- Describe infections associated with cosmetic procedures.
Common Bacterial Infections
MRSA

- MC of skin and soft-tissue infections in US since 1970’s, prior was *Streptococcus pyogenes*
- 2 major subtypes of *S aureus*: Methicillin-sensitive *S aureus* (MSSA) and methicillin-resistant *S aureus* (MRSA)
- MRSA-community associated (CA): Development in individual w/out h/o MRSA isolation or if + culture obtained in outpatient setting or w/in 48 hours of hospitalization
- Health care–associated (HA) MRSA: strain isolated in pt w/in 48 hours of hospitalization w/ risk factors of resistant infection (dialysis, previous colonization, surgery in past yr, a permanent medical device or catheter, or hospital, hospice, or nursing home admission)

MRSA

- Increased resistance to methicillin due to staphylococcal chromosome cassette mec (SCC mec), specifically mecA gene.
- Panton-Valentine leukocidin (PVL): in many CA-MRSA strains, associated with increased virulence (leukocyte destruction, necrosis).
- TSST-1: Staph superantigen involved in toxic shock syndrome.
- Exfoliative toxin (ET-A, ET-B): has protease activity, splitting desmoglein 1 at granular layer and can cause staphylococcal scalded skin syndrome and bullous impetigo.

ABSCESS

• MRSA can cause varied morphologies including abscesses, cellulitis, furuncles, carbuncles, folliculitis, impetigo, or paronychia to name a few.
  • **Abscesses**: collections of pus within the dermis and deeper skin tissues
  • **Furuncle ("boil")**: hair follicle infection in which purulent material extends through dermis into subcutaneous tissue → small abscess forms
  • **Carbuncle**: coalescence of several inflamed follicles into a single inflammatory mass with purulent drainage from multiple follicles
    • back of neck, face, axillae, and buttocks are common areas of involvement

MRSA

FURUNCLE
ABSCESS

- Skin and soft tissue caused by MRSA infections do not always produce pus and abscesses.
- MC presenting symptom: inflammation and necrosis. Pain and tenderness out of proportion to clinical presentation.
- Despite appropriate diagnosis and effective tx, response to tx can exceed 6 weeks.
- DDx of lesions with necrotic papules with marked inflammation:
  - brown recluse spider bites
  - cutaneous anthrax
  - cutaneous tularemia
  - cutaneous diphtheria
  - vibrio vulnificus infections

DDX FOR ABSCESS

• Other diagnoses to consider:
  • Folliculitis
  • Hidradenitis suppurativa
  • Sporotrichosis
  • Myiasis
  • Botryomycosis
  • Blastomycosis

TREATMENT OF ABScessES

- Gram stain and culture are recommended, but treatment without these studies is reasonable in typical cases.
- Incision and drainage (I&D) is the recommended treatment for inflamed carbuncles, abscesses, and large furuncles.
- Patients with uncomplicated skin abscesses, I&D without administration of antibiotics sufficient.
  - Administration of antibiotics as an adjunct should be made based upon presence or absence of systemic inflammatory response syndrome (SIRS).
- Antibiotic coverage for MRSA is recommended for patients with abscesses/carbuncles who have failed initial antibiotic treatment, have markedly impaired host defenses, or in patients with SIRS and hypotension.

TREATMENT OF ABSCESSSES

• Recurrent abscesses:
  • I&D and culture early.
  • Treat with a 5 to 10 day course of an antibiotic effective against that pathogen.
  • Consider a 5-day decolonization regimen twice daily of intranasal Mupirocin, daily chlorhexidine washes, and daily decontamination of personal items such as towels, sheets and clothes for recurrent S. aureus infection.

## SKIN SOFT TISSUE INFECTION TREATMENT

<table>
<thead>
<tr>
<th>MSSA</th>
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<tbody>
<tr>
<td>Nafcillin or oxacillin</td>
<td>1-2 g q4h IV</td>
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<tr>
<td>Cefazolin</td>
<td>1g q8h IV</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>600 mg q8h IV or 300-450 mg qid PO</td>
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<tr>
<td>Dicloxacillin</td>
<td>500 mg qid PO</td>
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<tr>
<td>Cephalexin</td>
<td>500 mg qid PO</td>
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<tr>
<td>Doxycycline or Minocycline</td>
<td>100 mg bid PO</td>
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<tr>
<td>Trimethoprim-Sulfamethoxazole</td>
<td>1-2 DS tab bid PO</td>
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<tr>
<th>MRSA</th>
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<tbody>
<tr>
<td>Vancomycin</td>
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<tr>
<td>Linezolid</td>
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<tr>
<td>Clindamycin</td>
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<tr>
<td>Daptomycin</td>
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<tr>
<td>Ceftaroline</td>
</tr>
<tr>
<td>Doxycycline or Minocycline</td>
</tr>
<tr>
<td>Trimethoprim-Sulfamethoxazole</td>
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Stevens, D. “Practice Guidelines for the Diagnosis and Management of Skin and Soft Tissue Infections: 2014 Update by the Infectious Diseases Society of America”. Infectious Diseases Society of America. 9/1/2015.
http://www.idsociety.org/Organ_System/#Skin%20&%20Soft%20Tissue
IMPETIGO

- MC in children aged 2-5 years. MC infection in children worldwide.
- Group A strep (Streptococcus pyogenes) previously MCC, but now replaced by S. aureus.
- Nonbullous impetigo: accounts for 70% of cases → erythematous papules and thin-walled vesicles on face and extremities. Can be painful. Usually resolve without tx in 2-3 weeks.
- Bullous impetigo: thin-roofed bullae and shallow erosions. S. aureus almost always causative pathogen (phage II, type 71). Develops in areas of trauma or intertriginous areas.

NON-BULLOUS IMPETIGO

BULLOUS IMPETIGO

SECONDARY INFECTIONS

• Impetigo often complicates both acute and chronic skin diseases.
  • Atopic dermatitis
  • Psoriasis
  • Herpes Simplex Virus
  • Scabies
  • Poison Ivy
  • Pediculosis capitis
  • Insect bites
## IMPETIGO TREATMENT

<table>
<thead>
<tr>
<th>Oral</th>
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<tbody>
<tr>
<td>Dicloxacillin</td>
<td>250 mg qid PO</td>
</tr>
<tr>
<td>Cephalexin</td>
<td>250 mg qid PO</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>250 mg qid PO</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>300-400 mg qid PO</td>
</tr>
<tr>
<td>Amoxicillin-clavulanate</td>
<td>875/125 mg bid PO</td>
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<table>
<thead>
<tr>
<th>Topical</th>
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<tbody>
<tr>
<td>Retapmulin ointment</td>
<td>Apply BID</td>
</tr>
<tr>
<td>Mupirocin 2% ointment</td>
<td>Apply BID</td>
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PARONYCHIA

- Caused by breakdown in barrier between nail plate and adjacent nail fold from minor trauma to nail bed → disrupts the fingertip’s natural barrier to outside pathogens → inoculation of perionychium.

- Paronychia often related to:
  - onychophagia (ie, nail biting)
  - finger sucking
  - picking at a hangnail
  - an ingrown nail
  - manicures
  - dishwashing,
  - puncture-type trauma w/ or w/out a retained foreign body

- Noninfectious etiologies such as chemical irritants, excessive moisture, systemic conditions, and medications also can cause paronychia.
ETIOLOGY OF PARONYCHIA

• Acute form: < 6 weeks duration
  • MCC S. Aureus.
  • Other causative organisms include γ-hemolytic strep, Eikenella corrodens, group A b-hemolytic strep, and Klebsiella pneumoniae, Bacteroides, Fusobacteria species, Enterococcus faecalis, Proteus species, and Pseudomonas aeruginosa.

• Chronic: > 6 weeks duration.
  • Usually caused by a fungal infection.

PARONYCHIA PRESENTATION

- Usually presents as localized pain, redness, inflammation, and edema of lateral nail fold typically limited to a single digit often 2-5 days after initial trauma.
- +/- Fluctuance of paronychium. Patients w/ delayed presentation may develop fluctuance extending around nail, involving eponychium as well as paronychium on both the radial and ulnar sides of the digit (ie, runaround infection).
- +/- Purulence may develop underneath the nail plate, causing the nail plate to pull away from sterile matrix.


ACUTE PARONYCHIA

PARONYCHIA TREATMENT

• The most common organism causing acute paronychia is *S. aureus*, followed by *S. pyogenes*, *Pseudomonas pyocyanea*, and *Proteus vulgaris*.
• Can be treated with warm water soaks 3-4 x per day (with or without povidone or chlorhexidine) and oral antibiotics.
• If an abscess is present, I&D is recommended in conjunction with oral antibiotics.
• Cephalexin, clindamycin, or amoxicillin plus clavulanate have a wide spectrum of activity against most pathogens isolated from paronychia.
  • In areas where local methicillin-resistant *S. aureus* penetration is relatively high, clindamycin remains a better option than amoxicillin plus clavulanate.
  • Oral trimethoprim-sulfamethoxazole can also be considered as a first-line agent.
• Removal of part of the nail plate may be required.

**CELLULITIS**

- Infection of deep dermis and subcutaneous tissue presenting as ill-defined area with erythema, swelling, and tenderness. +/- fever, chills
- Caused by disruption in skin barrier in immunocompetent patients
- Predisposing factors: previous attack of cellulitis, older age, obesity, venous insufficiency, saphenous venectomy in CABG patients, edema, and a skin surface disrupted by trauma, ulceration, or inflammatory diseases of the skin, such as allergic contact dermatitis, atopic dermatitis, and venous eczema


ETIOLOGY OF CELLULITIS

• MCC: group A strep (GAS), often residing in interdigital toe spaces; less commonly S. Aureus.
  • Purulent cellulitis usually caused by MRSA
• Erysipelas: specific type of cellulitis involving more superficial dermal structures and distinguished clinically by raised borders and clear demarcation between involved and uninvolved skin.
  • Predominantly due to beta-hemolytic streptococci
• Infection with GAS causes antistreptolysin O (ASO), anti-hyaluronidase, and anti-Dnase-B antibody positivity
• S. pyogenes erythrogenic exotoxins: SPE-A, SPE-B, SPE-C

CELLULITIS MIMICKERS

- Infectious
  - Necrotizing fasciitis
  - Erysipelas
  - Cutaneous abscess
  - Herpetic whitlow
  - Erythema migrans
- Dermatologic
  - Stasis dermatitis
  - Hypersensitivity reaction
  - Fixed drug reaction
- Inflammatory
  - Acute arthritis (gout)
  - Acute bursitis

Can be difficult to diagnose....

**74% of in-patient dermatology consults for cellulitis were pseudocellulitis**

CELLULITIS TREATMENT

- Cultures of blood, cutaneous aspirates, biopsies, and swabs are not routinely recommended.
  - Blood cultures however should be taken if systemic signs are present
  or
  - In patients with malignancy on chemotherapy, neutropenia, severe cell mediated immunodeficiency, immersion injuries, and animal bites.
- Typical cases of cellulitis should receive treatment against streptococci. Many clinicians also include coverage for MSSA.
- For coverage of streptococci and MRSA, use clindamycin or TMP-SMX w/ a B-lactam.

• When systemic signs of infection are present, inpatient treatment with intravenous antibiotics are indicated.
• For patients whose cellulitis is associated with penetrating trauma, MRSA infection elsewhere, IVDA, or SIRS, use vancomycin or another antimicrobial effective against both MRSA and streptococci.
• In severely compromised patients broad-spectrum antimicrobial coverage considered. Vancomycin plus either piperacillin-tazobactam or imipenem/meropenem is recommended for severe infections.

CELLULITIS TREATMENT

• Necrotizing infections require emergent debridement with IV antibiotics.
• The recommended duration of antimicrobial therapy is 5 days, but treatment should be extended if the infection has not improved.
• Hospitalize if: there is a concern for deeper or necrotizing infection, cases of poor compliance, for those severely immunocompromised, or if outpatient treatment is failing.
• In lower extremity cellulitis, carefully examine the interdigital toe spaces because treating fissuring, scaling, or maceration may eradicate colonization with pathogens and reduce the incidence of recurrent infection.
• Prophylactic antibiotics, such as oral penicillin or erythromycin bid for 4-52 weeks, or IM benzathine penicillin every 2-4 weeks should be considered in those who have 3-4 episodes of cellulitis per year despite attempts to control predisposing factors.

Antibiotics are among the most commonly prescribed drugs, however, up to 50% of the time antibiotics are not optimally prescribed, often done so when not needed, incorrect dosing or duration. (CDC)

Staphylococcus epidermidis (S. epidermidis) is completely resistant to erythromycin and partially resistant to clindamycin and tetracycline after 12 weeks of treatment.

Evidence suggest the use of topical erythromycin and clindamycin – the most commonly used topical antibiotics in acne – has contributed to the gradual increase in resistance over the last 20 years. (Humphrey)

ANTIBIOTIC RESISTANCE

- Dermatologist prescribed 9.5 million antibiotic prescriptions in 2009
  - Tetracyclines 69%
  - Cephalophorins 11%
  - TMP-SMX 7.5%
  - Aminopenicillins 5.1%
  - Macrolides 3.3%

ANTIBIOTIC RESISTANCE

- Tetracycline-resistant and erythromycin or clindamycin-resistant strains of *P. acnes* were found in 20 and 50% of patients, respectively, in a European study.
- Resistance has also been found in other pathogens commonly associated with dermatology:
  - Macrolide-resistant *S. pyogenes* and *S. aureus*
  - Mupirocin-resistant *S. aureus*
  - Vancomycin-resistant *S. aureus*
  - Quinolone-resistant *S. aureus, P. aeruginosa*, and mycobacteria

ANTIBIOTIC RESISTANCE

- Sub-antimicrobial doses doxycycline (20 mg BID) compared with antimicrobial doses (100 mg QD) in patients with moderate facial acne, both treatments significantly decreased inflammatory lesion counts
  - 20 mg dose led to an 84% and 90% reduction in the number of papules and pustules, respectively.
- Sub-antimicrobial dosing should be considered when possible to decrease the incidence of resistance and is being used in areas of medicine other than dermatology.

DERMATOLOGIC SURGERY AND WOUND INFECTIONS

• Post-surgical wound infections are the most common adverse effect, but are not that common.
• In a large, multicenter, prospective study of Mohs procedures, there were 83 (0.4%) reported infections out of 20,821 cases.
  • Similar low rates have been reported in smaller, multicenter prospective studies (0.07-0.9%).

PREVENTING SURGICAL SITE INFECTIONS

Antibiotic prophylaxis or not?

• Antibiotic prophylaxis in dermatologic surgery: advisory statement 2008 (JAAD) states antibiotics may be indicated for:
  • Procedures on the lower extremities or groin
  • Wedge excisions of the lip and ear
  • Skin flaps on the nose
  • Skin grafts
  • For patients with extensive inflammatory skin disease
  • For patients with high-risk cardiac conditions & a defined group of patients with prosthetic joints when the procedure involves breach of the oral mucosa

• Recent survey study sent to Mohs surgeons concluded dermatologic surgeons overuse perioperative antibiotics for prevention of surgical site infection, infective endocarditis, and prosthetic joint infection based on current recommendations.

Prepping of the skin

- A 2015 Cochrane review of current evidence found some evidence that preoperative skin preparation with 0.5% chlorhexidine in methylated spirits was associated with lower rates of surgical site infections following clean surgery than alcohol-based povidone iodine paint, however this data was from a poor single study.
- A similar review of literature found that while there are few well-controlled studies demonstrating superiority of a given regimen, alcohol-based iodophor and chlorhexidine products seem to exhibit greater efficacy than their aqueous counterparts.
- Both concluded ultimately it is up to the surgeon to choose and that future studies are needed.

PREVENTING SURGICAL WOUND INFECTIONS

How sterile do we need to be?

• In a prospective comparison study of 1,255 Mohs cases, infection risk was the same between high-cost (n = 5, 0.9%) and low-cost groups (n = 5, 0.7%).
  • High cost=
    • Sterile gloves for all stages & closure
    • Sterile half-drape for closure
    • Sterile knee length gown for closure
  • Low cost=
    • Sterile gloves for closure only
• Concluded that it may be possible to further reduce costs without altering infection rate by using clean, nonsterile gloves during reconstruction as well.

PREVENTING SURGICAL WOUND INFECTIONS

Sterile gloves vs non-sterile gloves

- In 2,025 Mohs cases, there was no increase in prevalence of infection using sterile glove for both excision and reconstruction (0.5%) compared to the using non-sterile gloves (0.49%)
  - The cost of gloves was $5.66 for 1 sterile glove case and $1.63 for 1 non-sterile case
- Similar results were seen in previous smaller studies


PREVENTING SURGICAL WOUND INFECTIONS

Topical antibiotics vs petrolatum/paraffin
• In a recent systematic review and meta-analysis, there was no statistically significant difference in incidence of postsurgical wound infections between the use of topical antibiotics and petrolatum/paraffin.
• There was also no significant difference between applying and not applying ointment to surgical wounds.
• Wounds at increased risk of developing surgical site infections
  • wounds in diabetics
  • wounds located in certain anatomic regions
  • wounds created by some surgical procedures

COSMETIC PROCEDURES AND INFECTIONS

Dermal Fillers

• According to a recent JAAD CME article, infections associated with cosmetic procedures are rare
  • 0.2% infections with soft tissue filler injections
  • 4 cases of cellulitis with calcium hydroxylapatite filler out of 2,089 soft filler injections

• Rare infections can occur:
  • 3 cases of mycobacterium chelonae from one plastic surgery clinic
  • Source was tap water from clinic ➔ pts used nonsterile ice pre-procedure

Lasers

- Reported bacterial infection rates post-CO2 procedures range from 2.2%-8.2%, with some evidence that antibiotic prophylaxis may decrease infection rates.

- Rare infections have been reported:
  - M. chelonae skin infection has been associated with ablative laser procedures such as CO2 and fractional CO2 laser resurfacing.
  - M. chelonae skin infection developed 1 week after a hair removal session with variable-pulsed alexandrite laser performed on both legs at a beauty center.

REFERENCES

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Thank You!