Abstract

Seabather’s eruption is an important differential diagnosis when a patient who has recently swum in a subtropical ocean presents with a pruritic rash in the distribution of their swimwear. Treatment with systemic corticosteroids is indicated in severe cases and can successfully reduce symptoms. Oral steroid therapy in general has proven to be an effective treatment for many acute and chronic diseases but has long been associated with increased risk for infections. In this report, we present an atypical case of cutaneous larva migrans and discuss its clinical unmasking after systemic steroid treatment was given for an initial diagnosis of seabather’s eruption.

Introduction

Seabather’s eruption is a benign, superficial reaction to toxins from marine-animal larvae. It is the most common marine-related problem in the waters south of the United States. It was reported in Florida as early as 1903 as a “rash which set up an intense itching” shortly after bathing in ocean water. In 1949, Sams postulated the eruption was caused by “some living, microorganism, in the nature of nematocysts from some form of coelenterate which is free floating.” Substantiating Sams’ initial claim, the thimble jellyfish, Linuche unguiculata, is now thought to be the cause of seabather’s eruption in the southeastern United States, Mexico and the Caribbean. The skin reaction occurs when jellyfish stinging cells called nematocysts get trapped underneath swimwear and inject venom there, causing an immune response and succeeding rash. The condition is often exacerbated with persistent use of the contaminated swimwear, causing friction on nematocysts, or when the swimmer later bathes in fresh water, inducing osmotic irritation and subsequent envenomation.

Seabather’s eruption is diagnosed clinically as a constant, pruritic and erythematous rash, papular, macular or urticarial, most commonly located underneath swimwear. Differential diagnoses include animal schistosomiasis (swimmer’s itch), scabies, insect bites, varicella zoster, contact dermatitis, folliculitis and almost any marine-origin dermatosis. A skin biopsy is not required for diagnosis of seabather’s eruption but most commonly shows a superficial and deep perivascular and interstitial infiltrate consisting of lymphocytes, neutrophils, and eosinophils. The syndrome is not considered contagious and is self-limiting, usually lasting an average of three to seven days. Treatment of seabather’s eruption consists mainly of supportive therapy with topical corticosteroids and antihistamines, with systemic steroid use reserved for severe cases.

Case Report

A 52-year-old female presented to her dermatologist complaining of an itchy rash on her groin and upper leg for one week. The patient stated she recently traveled to Mexico, where she spent several days on the beach and swimming in the ocean. Physical exam revealed erythematous, edematous papules on her lower abdomen and groin, assuming a location directly beneath her swimsuit (Figure 1). A biopsy was collected, and the patient was discharged with a topical corticosteroid and antihistamine regimen.

Less than 24 hours after the initial visit, the patient called the office complaining she could not tolerate the constant itch. She was prescribed a methylprednisolone dose pack and was encouraged to follow up with the office in four to five days or earlier if symptoms worsen. Biopsy results were received (Figures 2 and 3) and revealed a “papular urticaria/arthropod assault” consistent with an initial clinical diagnosis of seabather’s eruption.

Five days after the initial visit, the patient returned for follow-up stating the rash was spreading. Physical exam showed a worsening of symptoms with urticarial papules and serpiginous plaques radiating from the initial site (Figure 4). The patient was diagnosed with cutaneous larva migrans. She was instructed to discontinue the oral steroid and was given a single dose of 12 mg oral ivermectin. One week after treatment, the patient’s rash was resolved.

Discussion

Corticosteroids and immune function

Corticosteroids play a critical role in treating common diseases like rheumatoid arthritis, chronic obstructive pulmonary disease and a host of mild- to- severe inflammatory disorders. Since their advent in the late 1940s, both short- and long-term use of oral corticosteroid therapy has
been associated with increased risk of infections.7 The mechanism of action of these biologically active steroid hormones is spread across the entire immunological-response spectrum. Lionakis reported that glucocorticoids affect virtually every cell type involved in immunity and inflammation.8 Klein observed that a single dose of corticosteroids caused neutrophilic leukocytosis, monocytopenia, and eosinopenia within four to six hours of dosing.9 These and other reports have shown that steroid therapy is associated with decreased migration of neutrophils to target tissues, a reduction of inflammatory cytokines, and an inhibition of hydrogen peroxide production in lysosomes.6,10 Together, these inhibitions decrease microorganism killing and subsequent infection elimination.

**Cutaneous larva migrans**

Cutaneous larva migrans (CLM), or creeping eruption, is the most common skin disease among travelers returning from tropical destinations.11 CLM is caused by a penetrating parasite, most commonly the Ancylostoma braziliense, which flourishes in the gastrointestinal tracts of cats and dogs. Commonly found along tropical beaches where animal feces gets deposited, parasite larvae can remain viable and infectious for months in a warm and humid environment.12 With 95% of patients with CLM reporting recent exposure to a beach, prevention is focused on limiting contact with feces-contaminated sand or soil.1 The best suggested community prevention of CLM has been to ban dogs from beaches, a difficult task especially in developing countries. More individual forms of prevention include wearing shoes while walking, and lying on a mattress or sand washed with the ocean tide when touring a beach frequented by dogs.

In a human host, larvae migration is confined within the epidermis, causing the classic presentation of incessantly pruritic, erythematous, edematous, serpiginous tracks.13 The irregular track pattern advances at an average rate of 2.7 millimeters per day and can often be used to estimate infection duration.12 CLM is diagnosed clinically by recognition of these raised, red-purple, linear or serpiginous tracks, which usually occur along the feet. Other common sites of involvement include the buttocks, thighs, elbows, back and face.

For many clinicians, the disease may mimic scabies, schistosomiasis, tinea corporis, or contact dermatitis. But as outlined by Heukelbach, these can be easily ruled out once features of CLM are understood, leaving a differential diagnosis comprising dermatoses with serpiginous, migratory lesions.14 These can include strongyloides stercoralis (larva currens), fascioliasis, varicella zoster, a serpiginous ganglion cyst, and hair growing horizontally in the skin.

Skin biopsy is not recommended for diagnosis of CLM, as larvae advance ahead of the visible tracks, usually resulting in a negative biopsy. Eosinophilia is present in only 30% of cases, proving this test to be inadequate as a diagnostic study.12 First-line treatments of CLM include oral ivermectin and albendazole. Caumes reported that a single 12 mg oral dose of ivermectin achieved a cure rate of 81% to 100%, and a single 400 mg oral dose of albendazole achieved a cure rate of 46% to 100%.11 Oral steroids are generally avoided in parasitic or other occult infections due to their immunosuppressing effects.

**Conclusion**

As discussed in this case report, we submit that our patient had an atypical presentation of cutaneous larva migrans most likely from lying on a sandy beach contaminated with dog feces. With the original erythematous, papular rash appearing directly beneath the patient’s swimwear and the absence of any cutaneous tracks, seabather’s eruption was initially diagnosed. Not until treatment of a severely pruritic eruption with an oral steroid regimen did identifiable serpiginous and linear tracks appear, warranting a modification of the clinical diagnosis and treatment. Once therapy was changed to an indicated treatment of cutaneous larva migrans, the patient’s symptoms resolved.

**References**


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