

Pruritic Urticarial Rash in a 17 Year Old Male

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Abstract

Primary care pediatricians see a wide spectrum of children with rashes and searching for a cause can be challenging. We describe a 17-year-old boy with sudden onset of widespread pruritic, urticarial rash. A dermatologist was consulted and he was diagnosed with cercarial dermatitis, otherwise known as swimmer's itch.

Keywords: Pruritic; Urticarial rash; Swimmer's itch

Case

A 17-year-old male with an unremarkable past medical history developed a pruritic, urticarial rash a few minutes after showering. The symptoms progressed over the next few hours and spread to his trunk, upper extremities, neck and groin areas. He had no fever, cough, coryzal, facial, tongue swelling or shortness of breath. He was taking no medications. His past medical and surgical history were unremarkable and he had no known allergies. He mentioned that the family had returned from a camping trip at a local lake two days previously. His physical examination was notable for an erythematous maculopapular rash with wheals ranging from 1-10 centimeters in diameter located predominantly on exposed areas of his trunk and extremities (Figure 1,2 and 3). The patient was referred to a dermatologist and diagnosed with cercarial dermatitis or swimmer's itch. The symptoms gradually improved over the next few days but his symptoms waxed and waned and were exacerbated by exposure to heat (warm showers, exercises and outdoor activities). He was treated with diphenhydramine, 25 mg by mouth every 6 hours as needed for itching. The pruritus resolved but the rash persisted. One week later the rash resolved. Due to resolution of symptoms, the patient declined further laboratory testing.



Figure 1: Erythematous maculopapular rash



Figure 2: Erythematous maculopapular rash



Figure 3: Erythematous maculopapular rash

Discussion

Cercarial dermatitis, or swimmer's itch is an allergic rash that can occur after swimming or wading in freshwater ponds, lakes or other bodies of slow moving water that have been infested with flukes from the Schistosomatidae family [1-9]. The flukes can be from avian or non-avian (snails, rodents) hosts [1-3]. Adult schistosomes reside in the mesenteric blood vessels of birds and mammals, and following passage from the blood to the intestines, eggs are deposited in the water with the host feces [1-5]. Subsequently, miracidiae hatch and penetrate the intermediate host, usually snails [4-7]. Within the snail they develop into cercariae, which leave the snail and reside in the upper water layer of lakes [7]. While awaiting a definitive host the cercariae are capable of penetrating and burrowing into the skin of human swimmers [1,2,4,7]. Although the swimmer is a non-compatible dead-end host for the larvae, penetration leads to a local inflammatory immune reaction which, as in our patient, may take place 12-48 hours after exposure [2,5].

Humans are not primary hosts for the larvae of the Schistosomatidae [2-5]. The most common genera associated with swimmer's itch in humans are *Trichobilharzia* and *Gigantobilharzia* (avian) or *Schistosomatium douthitti* (snails and rodents) [2,3,5]. Most outbreaks are related to snail maturation, a time when the larvae are shed [4]. The longer the duration of swimming in infested area, the higher the risk for reaction [8]. Children are often exposed to longer time in shallow waters where the concentration of cercariae larvae are highest, predisposing them to infestation [1,5,6,8,9]. Once the larvae penetrate the human host they die and sensitization or an inflammatory reaction ensues [2,4]. Often, the initial infestation does not result in a reaction but causes sensitization, which results in the clinical eruption following subsequent exposures [2,3,9]. The affected areas are generally present on exposed areas of skin and an erythematous pruritic urticarial rash develops within hours of exposure in cases with prior sensitization. Each papule represents the site of parasitic penetration [2,5]. Symptoms peak within two to three days, and generally resolve within a week [2]. Management is symptomatic. There are five Schistosomatidae spp. which utilize humans as a definitive host [10]. The majority of cases are caused by *S. mansoni*, *S. japonicum*, and *S. haematobium*. *S. mansoni* and *S. japonicum* are causative agents of Katayama fever [10]. Similar to other Schistosomatidae spp., free-swimming cercariae penetrate host skin in infested water bodies [10]. In the acute phase, penetration causes an urticarial rash, fever, and fatigue; this progresses to chronic constitutional symptoms, non-pruritic rash, liver damage, diarrhea, and pulmonary infiltrates occurring two to ten weeks post-exposure [10,11]. Bilharziasis cutanea tarda is a rare cutaneous presentation of schistosomiasis. Patients develop pruritic, erythematous, indurated papules months to years after initial infestation [12,13]. Lesions most commonly occur in the groin or anogenital area [13]. Mature forms of Schistosomatidae spp., for which humans are definitive hosts, mate in the inferior mesenteric vein tributaries and release eggs into the venous system [13]. It is hypothesized that some eggs reach this region of the body via the rectal venous plexus, instead of passing through vessels into the intestinal lumen [13]. Ectopic ova encysted in the dermis cause inflammation and t-cell mediated granuloma formation [12,13]. Diagnosis is made by histologic identification of encysted ova [12,13]. Patients with Katayama fever and bilharziasis cutanea tarda generally respond to treatment with a short course of oral praziquantel [12,13]. Patients who present with these dermatologic manifestations, as well as cercarial dermatitis may develop chronic schistosomiasis. Morbidity from chronic schistosomiasis is predominantly due to granuloma formation from recurrent ova deposition in the venous systems of the bladder, intestines, spleen, and liver [14]. Symptoms include hematuria, hematochezia, hepatosplenomegaly, portal hypertension, and malnutrition [14]. The most life-threatening consequence of schistosomiasis is gastroesophageal bleeding from varices secondary to portal hypertension [14].

The diagnosis of swimmer's itch is made by clinical observation and history [7]. The differential diagnosis includes contact dermatitis, impetigo (bacterial dermatitis), sea bather's eruption, and aquagenic pruritis [4]. Typically patients will have had exposure to an infested water source less than 96 hours prior to onset of symptoms [4]. Patients develop an erythematous maculopapular or pustular rash on exposed areas of skin [7]. Skin biopsies can be diagnostic for cercariae in the epidermis however they are rarely identified [4]. Patients are treated symptomatically, in severe cases systemic antihistamines or corticosteroids may be used. First-generation antihistamines (H1-receptor antagonists) such as oral diphenhydramine are used to mediate the allergic response of swimmer's itch; generally the condition is self-limited. Complications such as sloughing of the skin and secondary bacterial infection may occur as well as systemic symptoms such as fatigue, fever, swelling, and chills [2]. Treatment of secondary infection is specific to the bacterial pathogen, however, generally consist of topical antibiotic ointments and if necessary systemic antibiotic therapy [15]. Swimmer's itch may be prevented by avoiding slow-moving water bodies with vegetation (Table 1).

Symptom & signs	Cercarial dermatitis (Swimmer's Itch)	Sea bather's Eruption	Aquagenic Pruritus
Skin	Itchy urticarial rash (first exposure); pruritic papules with subsequent exposure. Affects all exposed areas.	Pruritic papules in covered skin or hair.	Intense itching without observable skin lesions.
Site	Slow moving ponds/lakes or other bodies of water. Sometimes in ocean	Ocean	Contact with water
Cause	Hypersensitivity reaction to cercarial stage of water borne Schistosomatidae.	Hypersensitivity reaction to immature nematocysts of larval stage thimble jellyfish, sea anemones and other larval cnidarians.	Unknown

Table 1: Differential Diagnosis of Swimmers Itch

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