Swimmer’s dermatoses are not uncommon in dermatological practice with the increased popularity of swimming and aquatic leisure activities. There is a wide variety of dermatological conditions related to swimming or aquatic leisure activities, the exposed aquatic environment or their equipments. These include infections such as swimming pool granuloma caused by Mycobacterium marinum or cutaneous larva migrans, and non-infectious entities such as seabather’s eruption or contact dermatitis to swimming equipment.

Keywords: Aquatic, dermatoses, swimmer, swimming

Introduction

Swimming has been recorded since prehistoric times around 7000 years ago from Stone Age painting. It is a popular leisure or sport nowadays, and was part of the first modern Olympic Games in 1896 in Athens. People swim in the swimming pool, fresh or salt water with their body immersed in water for certain duration and exposed to aquatic organisms and allergens or irritants in the water or from the equipments, which can lead to cutaneous infection, allergic or irritant reactions. Skin infections caused by faecal organisms may be more common in contaminated rivers and skin irritation can occur from blue-green algae in inland waters. The use of communal changing room facilities may increase the risk of spread of warts and tinea pedis. Occasionally, unusual skin infections such as protothecosis can be acquired. Hence, swimmer’s dermatoses are not uncommon with a wide variety of entities as shown in Table 1.
**Table 1. Classification of swimmer’s dermatoses**

<table>
<thead>
<tr>
<th>Infectious</th>
<th>Non-infectious</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Swimming pool granuloma or fish-tank granuloma caused by <em>Mycobacterium marinum</em></td>
<td>• Sea bather’s eruption</td>
</tr>
<tr>
<td>• Swimmer’s ear (acute or chronic otitis externa)</td>
<td>• Seaweed dermatitis caused by direct contact with <em>Lyngbya majuscula</em></td>
</tr>
<tr>
<td>• Swimmer’s itch (cercarial dermatitis)</td>
<td>• Sting by jellyfish, sea lice and other stinging anemones</td>
</tr>
<tr>
<td>• Seal bite (Mycoplasma infection resulting from seal bites to the hand)</td>
<td>• Coral, sea urchin injuries</td>
</tr>
<tr>
<td>• Cutaneous larva migrans</td>
<td>• Contact dermatitis</td>
</tr>
<tr>
<td>• Pseudomonas folliculitis and perioritis</td>
<td>Bathing suits, goggles, snorkel masks, life jackets, sunscreen ingredient, water plants and inhabitants of the water</td>
</tr>
<tr>
<td>• Others: cutaneous warts, tinea pedis, secondary bacterial infection of wounds (e.g. <em>Staphylococcus aureus</em>, <em>Vibrio vulnificus</em>), cellulitis, necrotising fasciitis, protothecosis</td>
<td>• Others: Sunburn, dry skin (swimmer’s xerosis), chlorine irritation and aquagenic acne, cold urticaria, aquagenic pruritus, abrasions from wetsuit folds, bikini bottom</td>
</tr>
</tbody>
</table>

**Infectious entities of swimmer’s dermatoses**

**i) Swimming pool granuloma or fish-tank granuloma (*Mycobacterium marinum* infection)**

*Mycobacterium marinum* is a slow growing mycobacterium that causes disease in freshwater and saltwater fish and sporadically in humans. The first human disease was reported in 1951, when the organism was found in granulomatous skin lesions of individuals who swam in a contaminated swimming pool in Sweden. It is the commonest atypical mycobacterial cutaneous infection.5-8

The distribution of *M. marinum* is worldwide with higher prevalence in temperate climates. Its vectors include fresh or saltwater fish, snails, shellfish, dolphins and water fleas. When cleaning fish tanks at home or in the workplace (e.g. fish markets, restaurants), trauma to the hands may result in exposure to *M. marinum*. It can also be contacted from cracks in masonry, mud and even chlorinated water, as this organism is fairly resistant to chlorine. It will grow on ordinary laboratory media in 7-10 days if cultured at 30-33°C.

Cutaneous infection with *M. marinum* requires a portal of entry through the abraded skin. The incubation period is about two to three weeks, and occasionally up to nine months. A solitary bluish-red inflammatory nodule or pustule appears at the inoculated site which then forms a crusted ulcer, supplicative abscess or verrucous nodule. Lesions are most common on the dominant hand and fingers of fish fanciers, and on the elbows, knees and feet of swimmers. *M. marinum* may present with a sporotrichoid spread in approximately 20%-33% of cases9 and occasionally causes deeper infections (e.g. tenosynovitis, septic arthritis or rarely osteomyelitis). The inhibition of *M. marinum* growth at 37°C accounts for its ability to infect the cooler body extremities; however, immunocompromised patients may have disseminated diseases.

The histologic features range from non-specific inflammation in the first few months to well-formed tuberculoid granulomas in older lesions with
fibrinoid masses rather than caseation. Langhans’ giant cells are not always present, and intracellular acid-fast bacilli (longer and broader than tubercle bacilli) are detectable in only about 10% of cases. Epidermal changes including ulceration and pseudoepitheliomatous hyperplasia may occur in chronic lesions.

Cultures are important to exclude other cutaneous sporotrichoid infections including leishmaniasis, sporotrichosis and other atypical mycobacterial infections (e.g. M. kansasii, M. chelonei). A positive culture of M. marinum can be obtained in 70-80% of cases if grown between 30 and 33°C. Imaging should be considered to rule out deep infection especially in treatment-refractory cases.

M. marinum is usually sensitive to clarithromycin, minocycline, doxycycline, amikacin, ciprofloxacin, moxifloxacin and trimethoprim-sulphamethoxazole. Based on in-vitro susceptibility and clinical response in a 16 culture-positive case series, clarithromycin is the drug of choice for confirmed or suspected cases. Two agents should be considered for serious infections (e.g. clarithromycin and ciprofloxacin). The antibiotic(s) should be continued for one to two months after resolution of symptoms, typically for a total of three to four months. Treatment failure is more usually related to involvement of deeper structures than to the antibiotic regimen.

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**iii) Cutaneous larva migrans**

This is a parasitic skin manifestation caused by hookworm larvae that usually affect cats, dogs and other animals. Humans can be infected by walking barefoot, sitting or lying unprotected on sandy beaches that have been contaminated with animal faeces. It is also known as creeping eruption as the larvae migrate under the skin’s surface and cause itchy red lines or tracks. The wandering thread-like line is about 3 mm wide, commonly on the feet, hands and buttocks, in a bizarre and serpentine pattern.

Causes of creeping eruption include Ancylostoma brasiiliense, A. caninum, A. ceylonicum, Uncinaria stenocephala and Bunostomum phlebotomum. These are all hookworms of various animals, of which the dog hookworm, A. brasiiliense is the commonest cause of creeping eruption in human. The larvae advance at a rate of a few millimetres to a few centimetres each day, and are somewhere in front of the head of the track.

The disease can be clinically diagnosed and is self-limiting. Larva currens (the urticarial weal caused by subcutaneous larvae of Strongyloides stercoralis migrating several centimeters per hour), migratory myiasis (tortuous, thread-like red line that ends in a terminal vesicle due to larvae of penetration. If the individual is not sensitised to the cercariae, lesions subside within 12 hours. Otherwise, lesions may evolve over 10-20 hours in sensitised patients into intensely itchy papules, which can coalesce into plaques and last 1-2 weeks. In severe cases with repeated exposure to cercariae, lesions may evolve into vesicles and pustules, and fever and headache can occur.

Mild corticosteroid creams, calamine lotion or oral antihistamine can be beneficial for symptomatic relief. Cool compresses, baking soda or colloidal oatmeal bathing have been recommended. Antibiotics may be used to treat secondary infections and oral steroids can be considered in severe reactions.

**ii) Swimmer’s itch (cercarial dermatitis)**

This follows penetration of the skin by cercariae of avian schistosomes for which humans are not the primary host. Swimmer’s itch develops on an exposed area of skin 12-24 hours after contact with these larvae forms, usually in fresh water, when they mistakenly penetrate the person’s skin instead of its usual host (e.g. duck). Outbreaks are more common in temperate climates in summer. The risk is increased by the time spent in the water and when there is an onshore wind.

The initial symptom may be a prickling sensation soon after leaving the water. Erythematous macules appear 10-15 minutes after larval penetration. If the individual is not sensitised to the cercariae, lesions subside within 12 hours. Otherwise, lesions may evolve over 10-20 hours in sensitised patients into intensely itchy papules, which can coalesce into plaques and last 1-2 weeks. In severe cases with repeated exposure to cercariae, lesions may evolve into vesicles and pustules, and fever and headache can occur.

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flies of the genera Gasterophilus and Hypoderma) and gnathostomiasis (intermittent, migratory, subcutaneous swellings) must be distinguished. Eighty-one percent of A. brasiliense lesions disappear in four weeks, while some persist for many months.

Ivermectin in a single dose of 200 µg/kg body weight is the main treatment, and albendazole 400 mg/day by mouth for three days or topical application of 10% thiabendazole are alternatives.

iv) Pseudomonas folliculitis and periporitis
Inflatable swimming pools, Jacuzzis and hot tubs may represent a particular hazard, even when the water inside is adequately disinfected. Pseudomonas aeruginosa is able to withstand relatively high temperatures and high chlorine levels, and colonises the over-hydrated stratum corneum of the follicular ostia.2

Symptoms usually occur 8-48 hours after exposure. The rash is usually itchy and polymorphic, with erythematous macules, papules, pustules and vesicles and mostly affects the bathing-suit area. There may be associated conjunctivitis, pharyngitis, and occasionally swollen and painful breasts, abdominal symptoms and lymphadenopathy.

‘Pseudomonas hot foot syndrome’ had been described as a distinctive painful nodular eruption on the soles of children who had been exposed to P. aeruginosa in a heated wading pool with a grit-coated floor.14

Treatment is symptomatic in uncomplicated cases. The lesions usually clear spontaneously over 7-10 days.

v) Swimmer’s ear
Otitis externa in swimmers is common in tropical and subtropical areas, especially among white people. Swimming, high temperature and high relative humidity encourage maceration and secondary bacterial or fungal infections of the external auditory canal. Freshwater swimming appears to be a particular risk factor. Failure to dry the ears completely after swimming, shampooing or showering may be contributory factors.15

vi) Seal finger
Seal finger is a finger infection caused by Mycoplasma species (e.g. M. phocacerebrale) through direct contact with seals, sea lions or walruses. Traditionally an occupational hazard of seal hunters, but it is increasingly common in wildlife workers. Tetracycline for two to six weeks is the treatment of choice.3,16,17

Non-infectious entities of swimmer’s dermatoses

i) Seabather’s eruption
First described in 1949 as a pruritic papular eruption occurring in bathers off the eastern coast of Florida, this condition is believed to be a hypersensitivity reaction to the larval form of thimble jellyfish, Linuche unguiculata or certain anemones (e.g. Edwardsiella lineata) trapped underneath the swimwear, in intertriginous areas or in the hair of the bather.3 It has also been reported in Mexico, the Caribbean and in many tropical and sub-tropical waters. In Florida it is called ‘Pica-Pica’, the Spanish for ‘Itchy-Itchy’.3,18-21

It is a seasonal dermatitis from March through August and affects swimmers, snorkelers, or divers soon after getting out of the water. The onset ranges from few hours to up to 24 hours since the sea bathing. Most patients (~98%) would have pruritic papules over the bathing suit area, which are usually concentrated in tight-fitting areas and last for one to two weeks. Itching can be quite severe and the eruption may become painful. Occasionally, 10-18% patients may have fever, malaise or other systemic symptoms (but up to 40% with age <16). Children younger than 16 years old and surfers have a higher risk for seabather’s eruption.
Wearing bathing suits for prolonged periods after swimming, showering in fresh water and mechanical stimulation (e.g. rubbing with a towel) may provoke the discharge of venom by the larvae and make the eruption worse. Hence, bathers are advised to take off the bathing suits before showering in high prevalent areas.

Topical corticosteroid (e.g. 1% hydrocortisone cream), oral antihistamines and topical antipruritics (e.g. calamine lotion or 0.5%-1% menthol) may improve the pruritus, while ice-pack or nonsteroidal anti-inflammatory drugs may reduce the pain and inflammation. Oral prednisolone can be considered in severe reaction.

**ii) Seaweed dermatitis**

It is a skin rash caused by direct contact with a poisonous type of seaweed, most commonly the blue-green alga, *Lyngbya majuscula*. It produces two toxins called *lyngbyatoxin A* and *debromoaplysiatoxin*. Fragments of seaweed are caught under the swimwear with its toxin rubbing into the skin. The reaction may start a few minutes to a few hours after the swimmer leaves the water. An itching and burning sensation followed by a red, sometimes blistering rash occurs, sometimes in an entire swimsuit pattern. It often affects the scrotum in men and inframammary folds in females, depending on the type of swimwear used. Symptoms typically last for four to 48 hours. Sometimes, the affected person can have periorbital and perioral swelling, with or without the rash.

The affected individual should remove the swimsuit immediately and wash the skin vigorously with soap and water. The eruption can be treated as a sunburn using wet towels and soothing creams (e.g. calamine). Mild topical corticosteroids can be considered but in severe reactions, systemic steroids are needed.

**iii) Jellyfish stings**

Jellyfish commonly have stinging nematocysts. They consist of a bell-shaped body with tentacles, within which the nematocysts reside. Stings are delivered when contact is made with the tentacles and nematocysts discharge into the skin.

Jellyfish stings result in immediate pain as well as delayed and recurrent cutaneous reactions. Erythematous, urticarial or haemorrhagic streaks may be noticed. Confirmation of envenomation can be obtained by tape-stripping of nematocysts from the skin. A lichen planus-like eruption has been reported. *Chironex fleckeri*, the Pacific box jellyfish is generally considered the most dangerous species, and stings commonly result in shock. Storms often drive the jellyfish into shallow water in great numbers and sea-bathing or surfing should be discouraged during extreme weather.

Avoidance is the best means of preventing injury. When envenomation occurs in a swimmer, the victim should be immediately removed from the water to prevent drowning. Soaking the site in hot but not scalding water (~40°C) will denature some of the venom proteins and sea water can be used to remove jellyfish tentacles. Antivenom is available for some of the more toxic species. Treatment with topical corticosteroids and calcineurin inhibitors may be helpful for delayed reactions. Tropical jellyfish sting inhibitors, including lotions combined with sunscreens, are available as over-the-counter products to inactivate jellyfish stinging cells.

**iv) Sea urchin injuries**

Envenomation by sea urchins produces immediate burning pain, which can be very intense and lasts for several hours. The degree of local swelling is sometimes severe. Immediate treatment consists of careful removal of spines and pedicellariae and immersion of the affected area in hot water to relieve the pain. Erbium-yttrium-aluminium-garnet (Er:YAG) laser ablation has proved effective to
remove the spines and can be considered in difficult cases.24

The puncture wounds heal within one to two weeks if there is no secondary infection. Implantation epidermoid cysts may develop from fragments of epithelium driven into the wounds by the sea urchin spines.

Delayed granulomatous reactions (foreign-body or sarcoideal type) usually develop several months after the injury with bluish papules or nodules appearing at the site of penetration by the spines. These lesions are very persistent if untreated, and treatment by intralesional steroids can be considered. It has been suggested that M. marinum may play a pathogenic role in some case of sea urchin granuloma and that this cause should be ruled out.21

v) Coral injuries
Envenomation by fire corals usually produces immediate burning or stinging pain, followed by urticarial lesions at the contact site. These may evolve into a localised vesico-bullous eruption, and subsequently to a chronic granulomatous, lichenoid lesion.21

The lesions should be rinsed with seawater. Fresh water will increase the pain and therefore should be avoided. Topical acetic acid (vinegar) or isopropyl alcohol can then be applied to inactivate the venom, followed by removal of the nematocysts with tweezers or tape, and immobilisation of the extremity. Topical steroids can be used as needed for itching.

vi) Contact dermatitis
Allergic contact dermatitis can occur, for example to the elastic or dyes of swimming costume, goggles, snorkel masks or mouthpieces. Rarely, a toxic leucoderma has been reported from the use of goggles.2

vii) Physical trauma
Physical trauma to the skin include ‘surfer’s knee injury’, rope burns on the extremities of water skiers, ‘purpura gogglorum’ caused by the effects of pressure and suction, water-slap injuries on the anterior thighs of speed swimmers, and ‘swimmer’s shoulder’-an erythematous rough plaque caused by friction from the unshaven chin while swimming freestyle.2

viii) Bikini bottom
A nodular folliculitis of the inferior buttocks, probably caused more by follicular occlusion from not changing out of a damp swimsuit than by specific pathogens.2

Conclusion
The type of hobbies and travel are important but commonly neglected clues in history taking in daily clinical practice. Although most swimmer’s dermatoses are not life-threatening and some are self-limiting, we should complete our history taking with hobby and recent travel to achieve a prompt diagnosis and appropriate treatment. For example, with increasing international travel, sea bather’s eruption may still be encountered in the traveler returning from Florida or the Caribbean.

References