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Aquatic Sports Dermatitis: An Overview Sunil Kumar Gupta*, Deepika, Veenu Gupta and Monika

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Abstract

Cutaneous diseases are common among athletes. Skin eruptions account for almost half of all illnesses related to recreational water. The majority of cutaneous diseases are seen due to chemicals and microbes inhabiting the water bodies. Dermatological diseases commonly seen in swimmers have been described by numerous authors. These sports include swimming, scuba, diving, water polo, and snorkeling. Some of the common dermatological conditions encountered by swimmers in the aqueous medium of activity are reviewed here. Dermatologist should be familiar with these entities as this will greatly improve the physical well being of swimmers in and out of the water.

Keywords: Water; Dermatoses; Swimmers; Infection

Introduction

Aquatic sport dermatoses include a variety of skin conditions that occur in athletes who participate in sporting activities in or on the water. Cutaneous diseases are common among athletes. Skin eruptions account for almost half of all illnesses related to recreational water. The majority of cutaneous diseases are seen due to chemicals and microbes inhabiting the water bodies. In this review article, we have organized a vast number of aquatic sports dermatoses into groupings of infectious and organism related dermatoses, contact dermatoses, and miscellaneous dermatoses. Some of the common dermatological conditions encountered by swimmers in the aqueous medium of activity are reviewed here.

Infectious and Organism-Related Dermatoses

Swimmer's itch

It is also known as cercarial dermatitis, schistosome dermatitis, clam-digger's itch, caused by larvae (cercariae) from the fluke family *Schistosomatidae* occurring worldwide with the exception of Antartica [1]. It occurs in fresh water mainly. Birds & rodents are the definitive hosts who excrete ova contaminated with immature cercaria which then matures within snails. Larvae released from snails then infects humans. Release of proteolytic enzymes by cercaria helps in penetration through stratum corneum [2]. These larvae eventually die within the human skin. Sensitization to cercaria occurs in two weeks after first contact due to release of immunogenic enzymes and subsequent exposure causes lesions within hours [2].

Clinical features: The lesions appears commonly on non covered areas as 3-5 mm, erythematous pruritic papules and occasionally urticarial plaques resolving spontaneously in 3-7 days [3]. The lesions are more than 20 in number in most of the cases. Rarely, systemic symptoms including fever, chills and lymphadenopathy may co-exist [4].

Treatment: Pruritus can be controlled with antihistaminics and topical corticosteroids. In severe cases systemic steroids may be required [5]. Long exposure to stagnant, shallow water on hot summer days should be avoided to prevent swimmer's itch [6].

Swimming pool granuloma

Swimming pool granuloma, also known as fish tank granuloma or a fish fancier's granuloma is caused by atypical mycobacteria, including *Mycobacteria marinum* and *Mycobacterium scrofulaceum*.

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The infection may occur in swimming pools, lakes, beaches, rivers, and aquaria as the organism can live in both salt and fresh water. The potential vectors include fish, dolphins, shellfish, snails, and water fleas [7].

Clinical features: The infection occurs typically in trauma prone areas, including bony prominences and the lesions are verrucous nodules or plaques appearing 6 weeks after inoculation. Upper extremities particularly fingers are most commonly involved [8]. Sporotrichoid pattern is seen in 20% of cases due to proximal spread of infection to regional lymph nodes via lymphatics [9]. Ulceration may occur occasionally. For diagnosis, biopsy for histopathological examination and culture is done. Healing occurs spontaneously within months in infections confined to the skin. One patient thought to have a swimming pool granuloma actually had squamous cell carcinoma [10]. Rarely, serious complications like arthritis, osteomyelitis, bursitis, carpel tunnel syndrome, and tenosynovitis can occur from direct spread of cutaneous infections [11]. Widespread dissemination occurs in immunosuppressed patients and may lead to death.

Treatment: The affected area should be soaked in warm water for 5-10 min, three to four times per day. The first line regimen includes oral clarithromycin 500 mg twice a day for 6 weeks. Oral minocycline 100 mg twice a day can also be given but minocycline-resistant mycobacteria have been reported [5].

Swimmer's ear

Otitis externa or swimmer's ear is a bacterial infection commonly seen in swimmers with five times increased frequency as compared to non-swimmers [12]. Epithelial maceration occurs on prolonged exposure to water and dissolves sebum in the ear canal. Also, the protective pH of the ear canal is altered by the dilutional effect of water. Bacteria then proliferate, generate debris, and invade the ear canal lining leading to symptoms of otitis externa, the most common

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pathogens being *Pseudomaons aeruginosa* or *Staphylococcus aureus* [5]. Minor trauma and anatomic anomalies also predispose to invasion by bacteria [13].

Clinical features: Patient complains of moderate pain and pruritus in the ear, but in severe cases otorrhea, pronounced pain, fever and malaise may be disabling. Hearing loss can occur due to edema and secretions in the ear canal. Infection of temporal and adjacent bones can cause necrotizing (malignant) otitis externa in immunocompromised patients especially in elderly diabetics often complaining of otorrhea and severe otalgia worsening at night [14].

Treatment: Liquid antibiotic ear drops in combination with topical steroids, commercially available in a combined form, should be applied twice a day [5]. Long term systemic antibiotics and occasionally surgical debridement may be needed in cases of malignant otitis externa [14]. Gentle irrigation of the ear canal after swimming may prevent infection along with prophylactic instillation of an acidic solution. For recurrent episodes, acetic acid 2% in propylene glycol may be helpful.

Pseudomonas foot syndrome

Pseudomonas sp. can cause pseudomonas foot syndrome. It was first reported as an outbreak in a community wading pool [15].

Clinical features: It presents as extremely tender nodules on soles typically developing within 48 hours after exposure to contaminated water. The differential diagnosis includes palmoplantar eccrine hidradenitis (PEH) but the lesions are sterile in this case and there is history of usage of cold, damp footwear for long term [16,17].

Treatment: Symptomatic treatment is provided to the patient. Reducing the abrasiveness of pool floors, and super chlorinating the pool water may help in prevention [15].

Hot tub folliculitis

Hot tub folliculitis is also known as "*Pseudomonas aeruginosa folliculitis*" and "splash rash". It occurs after exposure to contaminated water in hot tubs, swimming pools, showers, baths, whirlpools by *Pseudomonas aeruginosa* which survives easily in alkaline warm water [18,19]. The infection may also occur in women after shaving legs and beneath diving suits [20].

Clinical features: The lesions appear 8-48 hours after exposure to contaminated water as follicular papulopustules on submerged surfaces. Pale green fluorescence is seen under Wood's lamp early in the course of the disease. Systemic symptoms including fever, nausea, diarrhea, sore throat and lymphadenopathy may be experienced by the patient. Lesions usually heal without scarring but rarely hyperpigmented macules or subcutaneous abscesses may form. *Ecthyma gangrenosum* or subcutaneous nodules and cellulitis may develop in immunosuppressed patients [21].

Treatment: Spontaneous resolution without treatment occurs in less than 2 weeks in immunocompetent patients. For symptomatic relief, 5% acetic acid compresses may be given.

Bikini bottom

A deep bacterial folliculitis of the inferior buttocks or bikini bottom is seen mainly in swimmers who wear damp, tight fitting swimwear for long period of time. Maceration and poral occlusion appears to be the major contributing factors for this infection caused by *Streptococcus* or *Staphylococcus*.

Clinical features: The firm, inflamed deep nodules over the inferior

gluteal crease appearing 3 to 5 days after spending prolonged exposure to water at the beach or lake in tight fitting, wet swimsuit [22].

Treatment: A ten day course of oral cephalexin helps in treating these patients along with prompt removal of wet swimwear and avoiding them for atleast 10 days [22]. Topical antibiotics used for treating acne like clindamycin and erythromycin solutions may be helpful. Recreational swimmers should be advised to remove the swimsuit and apply an absorbent powder in between.

Diving suit dermatitis

Diving suit dermatitis have been described by many authors in scuba divers caused by *Pseudomonas aeruginosa* O:10 and O:6. It results from superhydration of epidermis due to occlusive diving suits.

Clinical features: The patient usually presents with diffuse, erythematous papules on the trunk and extremities and fever, malaise, and headache rarely occurs.

Treatment: Treatment includes oral antibiotics, such as ciprofloxacin 500 mg twice a day. Divers should immediately shower after diving and diving suits should be cleaned with 0.45% lactic acid.

Molluscum contagiosum

Molluscum contagiosum, also known as water warts are caused by Pox virus in 2-10% swimmers [23]. The association between molluscum contagiosum and swimming was first reported by Walker in 1910 and he referred to molluscum as "the itch of the bath" [24]. Molluscum occurred in 7.5% of swimmers and in 3.6% of nonswimmers in a Japanese study [25]. In another study, link between molluscum and use of an outdoor swimming pool was demonstrated in 75% of patients [26].

Clinical features: Lesions present as pearly white or skin colored papules with central umblication in infected athletes. Spontaneous resolution without scarring occurs in majority of cases. Occasionally "Molluscum dermatitis" due to host immune response to viral antigen can occur.

Treatment: Spontaneous resolution within one year occurs in most cases, but may last longer. Treatment options include trichloroacetic acid, curettage, liquid nitrogen, 5-Flourouracil or imiquimod.

Warts

Warts or Verrucae occur in swimmers 1.8 times more commonly as compared to non swimmers [27]. Another study demonstrated development of warts in 27% of swimmers who used communal showers [28].

Clinical features: Warts presents as well defined, verrucous papules with a rough surface. Plantar warts presents as endophytic papules on the soles. Black dots over the lesion, representing thrombosed capillaries, are diagnostic of warts and help differentiate them from a corn or callus.

Treatment: Treatment modalities include mechanical destruction with liquid nitrogen, curettage, and laser or chemical destruction with application of trichloroacetic acid, salicylic acid, and cantharidin. Self application with agents like imiquimod, 5-FU, squaric acid dibutyl ester (SABDE), and diphenylcyclopropenone (DPCP) under occlusion are also helpful.

Athlete's foot

Tinea pedis or athlete's foot is extremely common cutaneous

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infection transmitted through swimming pools, shower floors, and pool decks [29]. Fungal elements are present over shed skin on these skin surfaces. Athletes with impaired skin barrier are at increased risk of infection. One group found a prevalence of superficial fungal infections of 13.2% on day 1 compared to 22.2% on day 12 among students in swimming courses [26]. Another group reported that 63.6% of 559 students were carriers of dermatophytes who were enrolled in swimming class [29].

Clinical features: Three different clinical patterns are seen: interdigital, inflammatory/bullous, and moccasin. Recurrence is commonly seen.

Treatment: Topical antifungals twice a day for one to several months with systemic antifungal agents, such as terbinafine 250 mg daily or itraconazole 200 mg daily for 2 weeks in cases of extensive lesions or recalcitrant infections are beneficial. To prevent infection sandals should be worn while on pool decks or public shower floors.

Contact Dermatoses

Goggles

Swim goggles can cause allergic contact reactions after exposure to rubber accelerators, such as dibutylthiourea, used in the assembly of black neoprene rubber padding [30]. Exposure to phenolformaldehyde resin and benzoyl peroxide in swimming goggles can also cause contact allergies [31].

Clinical features: Patient may develop periorbital erythema and vesicles associated with itching and in severe cases yellow exudates may occur.

Treatment: This condition can be treated effectively with short course of mid potency corticosteroids with topical immunomodulators for mild and chronic cases. Systemic steroids may be required for severe cases.

Wet suit dermatitis

It occurs in divers sensitized to dibutylthiourea, diethylthiourea, diphenylthiourea, para-tertiary-butylphenol-formaldehyde resin or ethylbutylthiourea [32]. These allergens exist as glues that attach nylon linings or as rubber accelerators.

Clinical features: Patient presents with vesicular and eczematous pruritic eruption on the neck, trunk, and extremities.

Treatment: Sensitized athlete should find wet suits without thiourea accelerators.

Swim cap dermatitis

Swim cap dermatitis occurs due to mercaptobenzothiazole which is a component of rubber bathing and swim caps [33].

Clinical features: Lesions develop over skin covered with swim caps as well-defined, erythematous, scaly plaques.

Treatment: To prevent this reaction, affected individuals should use swim caps composed of silicone [34].

Nose clip and earplug dermatitis

Rubber accelerators used to make nose clips and earplugs can cause allergic reactions in swimmers.

Clinical features: Depending on the type of equipment used,

patient develops well defined scaly erythematous plaques ove nose or ears.

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Treatment: Silicone nose clips and ear plugs should be used.

Pool dermatitis

Chemicals such as chlorine and bromine used to control microbial growth in pools can cause irritant contact dermatitis. Brominated pools cause severe and moderate eruptions as compared to chlorinated pools [35].

Clinical features: It presents as urticarial or eczematous plaques on uncovered areas.

Treatment: Avoidance of offending agent in sensitized swimmers helps prevent this condition.

Pool water dermatitis

Pool water dermatitis represents an allergic contact dermatitis to chlorinated or brominated as compared to pool dermatitis which is an irritant contact dermatitis.

Clinical features: It manifests as erythematous scaly plaques associated with itching.

Treatment: Sensitized individuals should avoid halogenated water.

Miscellaneous Dermatoses

Purpura gogglorum

Purpura gogglorum appears as periocular purpura have been described by many authors [36]. The various etiologies proposed for this disorder include:

- Under negative pressure suction trauma due to frequent pulling away of goggles [37]
- Direct collision forces or goggles snapping back into the eye [38]
- Tightening of strap of poorly fitting goggles [39].

Clinical features: Athletes presents with periocular purura.

Treatment: Spontaneous resolution of lesions occur. In case of extensive swelling, tenderness or visual changes, patient should be evaluated for fracture of facial bones.

Swimmer's shoulder

First reported by Koehn [40] this condition occurs when an unshaven chin rubs against the shoulder in swimmers during the crawl stroke while turning the head to breathe resulting in irritant dermatitis [40].

Clinical features: Lesions appear shortly after swimming as erythematous, rough plaques appear on the anterior aspect of shoulder and disappearing within hours.

Treatment: Petrolatum jelly may alleviate pain.

Green hair

Green hair occurs in swimmers with natural or tinted bond, gray or white hair. It is a reversible pigmentary change of cosmetic concern. It affects children commonly as compared to adults [41]. The discoloration is due to copper ions either occurring naturally in water source, or released from copper pipes used in construction of some older swimming pools [41].

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Clinical features: Athletes presents with the obvious green tint in the hair easily detected in bright light and becomes more pronounced when they are wet [42]

Treatment: Application of 2-3% hydrogen peroxide for 30 minutes will remove the color. Use of commercial chelating agents after swimming is effective.

Aquagenic pruritus

The term aquagenic pruritus was first described by Steinman [42] created a set of criteria for the diagnosis of aquagenic pruritus. These criteria include:

- 1) severe pruritus, prickling, stinging or burning sensation after contact with water;
- 2) discomfort that develops within minutes of water contact;
- 3) no visible skin changes;
- no concurrent skin disease, internal disorder or medication that can explain the discomfort;
- 5) exclusion of aquagenic, cholinergic, cold, heat, and vibratory urticaria and symptomatic dermatographism;
- 6) Exclusion of polycythemia rubra vera.

Clinical features: Patient complains of pruritus or a burning, tingling or stinging sensation without skin lesions at sites of contact with water and the symptoms last for 10 min to 2 hours [43].

Treatment: Pruritus may be relieved with phototherapy, particularly PUVA and narrow-band UVB [44,45].

Pool palms

Repetitive rubbing of skin surfaces mainly fingers, palms, and toes against the rough surfaces of pool causes frictional dermatitis and numerous cases have been reported [46,47].

Clinical features: Lesions occur on convexities of palmar hands and fingers as symmetrical erythematous plaques [48].

Treatment: The condition resolves spontaneously when the athlete stops rubbing.

Aquagenic acne

Increased sebum production after prolonged (3 to 4 hours) hyperhydration of skin have been demonstrated in various studies [49]. Also there is occlusion of pilosebaceous orifices and obstruction of sebum outflow producing further overcompensation by the sebaceous gland [50]. Chlorine by irritating follicular orifices stimulate poral occlusion which also contribute to development of acne [50].

Clinical features: Patients develops erythematous popular lesions on face mainly the lateral areas of chin, nasolabial folds, and the medial cheeks, along with increased oiliness.

Treatment: Milder antibiotic acne creams may be tried initially followed by introduction of systemic antibiotics if response is inadequate. Low dose isotretinoin may be introduced gradually if no response occurs. Patient should be instructed to avoid harsh soaps, cleansers, or astringents as they dissolve the sebum.

Conclusion

Cutaneous dermatoses related to aquatic sports are common.

Certain conditions are specifically associated with freshwater or saltwater, while others may occur in both settings. The etiologies of water-related dermatoses are usually infectious and organism related or result from contact dermatitis. Our review highlights the key physical findings that are most consistent with particular aquatic sports dermatoses, first-line treatment guidelines and preventive measures that help in the management of these water-related skin conditions.

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