Skin and Soft Tissue Infections and Envenomations Acquired at the Beach

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Millions of people spend vacation and leisure time on the shores of the lakes and oceans of North America. Many others are employed in water-related industries. In doing so, they potentially expose themselves to infections and envenomations that other people do not even consider when making their plans for leisure time activities or in going to work each day. This article reviews some common and uncommon beach-related infections and envenomations that can affect the skin and soft tissues of people frequenting the shores and beaches of North America. Physicians are thereby provided with a guide to such skin and soft tissue injuries and infections and will be better able to recognize and treat these maladies associated with recreational and occupational activities.

Introduction

One of America's most popular destinations for vacation and recreational activity is the beach [1,2]. Most visits to the beach are enjoyable and relaxing. However, some beach visits result in injury, infection, and/or envenomation from beach- or water-dwelling animals or microorganisms. This article reviews some of the beach-related situations resulting in skin and soft tissue injury or infection. First, this review discusses clinical scenarios caused by multicellular organisms and then reviews the skin and soft tissue infections caused by a variety of bacterial pathogens. Waterborne gastrointestinal infections may also be acquired at the beach; however, that topic is beyond the scope of this article and has been reviewed elsewhere.

Jellyfish

Jellyfish envenomation injuries occur by accidental contact with the tentacles of the jellyfish and the resultant firing of nematocyst stinging units. The severity of such injuries ranges from trivial to severe or life-threatening, depending upon the species of jellyfish, the extent (surface area) and anatomic location of contact, the duration of contact, the age and body weight of the victim, the thickness of skin at the point of contact with nematocysts, and whether the victim develops anaphylactic reaction to the venom [3•,4,5].

Symptoms may be both localized and generalized, including a stinging sensation on contact, gradually increasing intensity of the sting over the first 10 minutes after injury, erythema, pruritis, and the development of any or all of the following: papules, vesicles, pustules, and necrotic ulcers. Generalized symptoms include an increase in oral secretions, muscle spasms, respiratory distress, gastrointestinal disturbances, and cardiovascular failure [3•,6].

General principles of treatment include inactivation of the nematocytes by application of vinegar or acetic acid for approximately 10 minutes. Rinsing the area with seawater may help eliminate some of the remaining nematocysts if neither vinegar nor acetic acid is immediately available. Alcohol, freshwater, ammonia, or bleach should not be used as these will stimulate the remaining nematocysts to discharge venom. Any remaining tentacles should be lifted away from the skin. Shaving cream should then be applied to the involved area and the area should be shaved to remove any remaining nematocysts. Symptomatic therapy may include topical steroid cream, oral antihistamines, narcotics for pain, and anesthetic creams.

Anaphylaxis to the venom is unusual with the first such injury but may occur with subsequent exposures and should be treated with standard anaphylaxis protocols. Secondary infection occasionally occurs, but prophylactic antibiotic administration is not routinely recommended. If antibiotics are to be given for suspected infection, cultures should be obtained in advance if possible, since marine-related bacteria may be the pathogenic organisms in this clinical setting [7,8]. Tetanus toxoid should be administered if the patient meets current criteria from statements from the U.S. Centers for Disease Control and Prevention [9].

Stingray

An estimated 1000 to 2000 stingray injuries occur each year in North America. Most of these injuries occur when a victim accidentally steps on a stingray while wading in

sandy coastal areas. In self-defense, the stingray whips its tail upward and forward and implants a tail-spine in the victim's skin, after which the spine releases heat-labile, water-stable venom into the victim's body. Local bleeding and severe pain usually occur.

Depending on the amount of venom injected, systemic symptoms range from anorexia, nausea, vomiting, and diarrhea to such severe manifestations as muscle cramping, paralysis, seizures, increased micturition and salivation, hypotension, respiratory depression, and even cardiac abnormalities including asystole. The local lesion may eventually ulcerate and become necrotic. Secondary infection may ensue, and local marine bacterial pathogens are common as pathogenic secondary invaders [3•,10–12].

Treatment should include placing the victim in supine position, raising the affected limb (if on a limb), spine extraction, saline irrigation, skin surface scrub with soap, and immersion in hot water (~ 48.8° C) for 30 to 90 minutes. Pain can be controlled with analgesia and infiltration of the affected area with lidocaine. Ten percent calcium gluconate intravenously may relieve muscle spasms. Radiographs should be obtained to look for remaining foreign bodies. The skin may be loosely closed with suture over a drain. Antibiotics may be required depending on the degree of contamination, and the victim should be closely monitored for the systemic symptoms discussed earlier, especially respiratory and cardiovascular collapse [3•,12]. Emergency personnel and physicians familiar with the intricacies of stingray injuries should be involved in the care of the patient as quickly as possible. The patient's tetanus status should be verified and appropriate tetanus preventive therapy should be included in the treatment protocol [9].

Sea Bather's Eruption

Sea bather's eruption is an aquatic dermatitis usually caused by larval forms of thimble jellyfish (*Linuche unguiculata*) and occasionally by larvae of sea anemones (*Edwardsiella lineata*). This dermatitis is one of many in the differential diagnosis of travelers returning with a new rash [13]. The very small cercarial larval forms usually become entrapped within loose swimming suits or other loose clothing worn into the water. When the bather leaves the water, pressure from the collapsing swimwear compresses the larvae against the bather's skin and the nematocyst harbored by the larval form releases venom locally into the victim's skin. Rarely are lesions seen on areas of bare skin. A pruritic rash develops and may be severe. Occasionally headache, fever, or weakness accompanies the rash. Burning of the eyes or urethra may occur if larval forms enter the eyelids or urethra, respectively.

Treatment includes removal of the swimwear, rinsing in a freshwater shower, application of vinegar or acetic acid to neutralize venom, application of hydrocortisone cream, and thorough machine washing of the swimwear to rid the swimsuit of remaining larvae [3•,14••]. Although this illness is usually self-limiting in nature, it can be quite distressing to swimmers, especially if they are frequenting beaches that are crowded and have no local shower facilities.

Swimmer's Itch

Swimmer's itch is caused by free-swimming cercariae, usually of avian species, that penetrate the epidermis of human victims swimming in waters contaminated by avian schistosomes. A recent prospective study suggests that this disease may be more common than previously reported [15]. Once the cercariae penetrate the human epidermis, they do not have the immunologic markers to complete the usual life cycle available to them in birds. They therefore die within the skin, and a local inflammatory response immediately ensues, resulting in an extremely pruritic and papular rash with the occasional development of vesicles within 24 to 48 hours.

Symptomatic disease should be treated first with topical isopropyl alcohol and then with topical calamine, topical hydrocortisone, and/or topical diphenhydramine creams. If a severe inflammatory reaction occurs, systemic corticosteroids may be required. Occasionally, secondary infected papules develop where lesions have been intensely scratched by the patient. Ivermectin may also be effective in the treatment of swimmer's itch [16]. Systemic antibiotic therapy may occasionally be necessary in these situations. Swimmer's itch is most commonly associated with freshwater beaches but may occasionally be experienced at salt water beaches [3•,17].

Cutaneous Larva Migrans

Cutaneous larva migrans results from the contact of bare skin (usually of the foot or ankle) with the larvae of dog and cat hookworms. This is most commonly seen in the southeastern United States, most frequently during the warm and rainy seasons. This occurs not infrequently on beaches on which cats and dogs roam free and/or are allowed to defecate. The most common etiologic agents are *Ancylostoma braziliense* (cat and dog hookworm) and *Ancylostoma caninum* (dog hookworm). Humans are aberrant hosts, and once the filariform larvae invade the skin, they can usually go no deeper than the epidermis. Once the epidermis is penetrated, a pruritic erythematous papule is noted, and the filariform larvae wander around aimlessly in tunnels that they burrow into the epidermis. The filariform larvae may migrate several centimeters per day, thereby creating significant inflammation and scarring in the tunnels that were burrowed by the larval form.

Infection can be avoided by wearing footwear while in infected soils and sands. Treatment with oral or topically applied thiabendazole (cream or ointment) is effective in most cases. Newer therapeutic oral antiparasitic agents include ivermectin and albendazole. Ivermectin is consid-

ered the most effective agent for treatment [18]. Secondary bacterial infection occasionally occurs and is usually treated with standard oral doses of anti-streptococcal and anti-staphylococcal antibiotics. In rare cases, waterborne organisms may predominate, and oral antimicrobial treatment should be modified based upon results of cultures when they are obtainable [19–21].

Mycobacterium marinum

Mycobacterium marinum is a nontuberculous, free-living mycobacterium first isolated in 1926 and first documented as the cause of human disease in 1951. Infections are related to swimming and water contact, from both freshwater and salt water exposure [22]. Many patients have contact with fish or aquariums that house fish. However, a significant number of patients with documented *M. marinum* disease may not give a history of contact with aquatic environments [23].

Usually some type of skin injury occurs, followed by the slow development of papular or nodular lesions after approximately 2 to 4 weeks of incubation [24,25]. The primary skin lesions may be plaques, encrustations, ulcerations, or verrucous lesions and usually spread proximally along the lymphatic system. Eventual maturation to fluctuant lesions may also occur. Lesions are most commonly seen on the extremities, probably reflecting better growth of *M. marinum* at 37° C, that is, in the cooler areas of the body. The lymphatic spread mimics the behavior of sporotrichosis and can be easily confused with this syndrome caused by *Sporothrix schenckii*. In general *M. marinum* causes localized disease, but dissemination and bacteremia with *M. marinum* have been reported [26]. History of water exposure can often be elicited in patients with disease due to *M. marinum*. The diagnosis is most dependably made by tissue biopsy for histological appearance and for culture. Acid-fast organisms are rarely seen on tissue biopsy, but cultures of the tissue when incubated at 37° C are usually positive. The microbiology laboratory should be alerted to your consideration of this as a possible pathogen so that cultures can be incubated at 37° C. *Mycobacterium tuberculosis* grows best between 30° C and 33° C [25].

Drug therapy for disease due to *M. marinum* is clarithromycin, minocycline, doxycycline, or a combination of rifampin plus ethambutol. Some patients with early disease may respond to treatment with trimethoprim-sulfamethoxazole. Antibiotic regimens should be given for at least 3 months [27]. Deep fluctuant lesions may require surgical excision for cure.

Soft Tissue Bacterial Infections Related to Water Exposure

Any type of abrasion, laceration, or trauma sustained at the beach may lead to microscopic or macroscopic breaches in the integrity of the skin. This loss of cutaneous integrity may allow pathogenic bacteria to bypass the body's primary protective barrier (ie, the skin itself). The most common pathogens isolated from cultures obtained from patients with non–water-associated cellulitides are the ß-hemolytic streptococci and *Staphylococcus aureus* [28]. This section will discuss some aquatically acquired bacteria, which should be included in the differential diagnosis of etiologic agents in water-contaminated wounds that lead to wound infections, cellulitis, and occasionally to more severe soft tissue infections such as necrotizing fasciitis [29].

***Aeromonas* species**

The *Aeromonas* spp are gram-negative bacilli found in fresh water, brackish water, surface water, drinking water, polluted waters, waste water, and even in marine environments, especially those that interface with fresh waters. Aeromonas can survive throughout a broad range of temperatures and in all but the most extreme salt concentrations. There are seven mesophilic, motile organisms in the Aeromonas group that cause human disease and produce a broad range of toxins and other substances that contribute to their pathogenicity: *A. hydrophila*, *A. veronii biovar sobria*, *A. caviae*, *A. veronii biovar veronii*, *A. jandaei*, *A. schubertii*, and *A. trota*. These organisms cause wound infections and may cause the sepsis syndrome in the absence of a definitive visible wound [30]. A recently described outbreak of skin and soft tissue infection caused by *A. hydrophila* was highly associated with a game of "mud football" in Australia [31]. Gastrointestinal infection due to *Aeromonas* spp has presented with a wide variety of symptom complexes. Most are self-limited and are beyond the scope of this paper. Most of the human *Aeromonas* pathogens are very susceptible to broad-spectrum penicillins, carbapenems, second- and third-generation cephalosporins, trimethoprim-sulfamethoxazole, the tetracyclines, and the fluoroquinolones [30].

Erysipelothrix rhusiopathiae

This organism is a straight or slightly curved, aerobic or facultatively anaerobic, gram-positive bacillus that may occasionally appear gram-negative on the Gram-stained smear because of its easy decolorization during the Gram stain process. The organism is found worldwide in many vertebrate and invertebrate animal species. Its primary reservoir is domestic swine.

Risk factors for infection include abrasions and puncture wounds at the site of entry and an occupation that exposes the wound to the organism. Those at greatest risk for this disease are fishermen, butchers, fish handlers, farmers, slaughterhouse workers, and veterinarians. The clinical manifestations of human disease are divided into three categories: 1) localized cutaneous lesion, 2) diffuse eruption/cellulitis with systemic symptoms, and 3) severe bacteremic infection often associated with infective endocarditis [32].

Strains of *Erysipelothrix rhusiopathiae* are highly susceptible to penicillin, ciprofloxacin, imipenem,

clindamycin, and most cephalosporins. Imipenem and penicillin G appear to have the highest level of activity against this organism. *E. rhusiopathiae* is usually resistant to vancomycin, trimethoprim-sulfamethoxazole, and other sulfonamides. The length of antimicrobial therapy depends upon whether or not the disease has progressed to infective endocarditis. Usually the duration is 2 weeks of therapy for nonendocarditis illnesses and 4 to 6 weeks for patients with infective endocarditis. Prevention is imperative for those in high-risk occupations and should combine almost obsessive hand-washing with the wearing of protective attire, especially gloves, when dealing with potentially infectious animals and inanimate surfaces contaminated by animal tissue [33].

Vibrio vulnificus

Vibrio vulnificus is a marine halophilic *Vibrio* species with highest organism concentrations in warm coastal waters. Infections due to this organism are most prevalent from April to October. It is the most virulent of the non-cholera *Vibrio* species and its clinical disease is primarily manifested as a severe, distinctive, and potentially life-threatening cellulitis. The clinical illness is usually abrupt in onset with severe fever and chills, followed within 36 hours by metastatic skin lesions that begin as cellulitis and rapidly develop into hemorrhagic bullous lesions, vesicles, and eventually necrotic ulcers. Septic shock is often present, and the mortality rate of patients with bacteremia approaches 50%.

Severe disease is usually associated with consuming raw oysters within the previous 7 days. Organism entry is usually via the gastrointestinal tract or via wound contamination by waters infested with the organism. Severe disease is not unusual, but surprisingly little is known about the pathogenesis of this organism [34]. Risk factors for the disease include liver disease, iron overload states such as hemochromatosis, hemolytic anemia, chronic renal failure, malignancy, HIV infection, and immunosuppressive medications. Persons developing wound infections almost always have some type of pre-existing minor wound or a wound sustained while engaging in water-related activities in coastal waters [35]. The wound can range from a crab bite or a minor laceration sustained from a fishing hook to stingray envenomation or contact with aquatic animals such as mussels [36]. In studies looking at wound infections due to *Vibrio vulnificus*, approximately 50% to 60% of patients have some predisposing illness other than the wound, whereas the other infected patients were otherwise healthy [37]. The presence of an underlying illness, especially cirrhosis, predicted a shorter incubation period and a more rapid progression from mild to life-threatening disease. Fatality rates also increase as the duration of time from onset of symptoms to initiation of appropriate antimicrobial therapy increases [8].

Other Waterborne Illnesses and Envenomations

This review concentrates on envenomations and infections of the skin and soft tissues that result from exposure to animals and microorganisms on the beaches of North America. A review of waterborne gastrointestinal infections and other venom-producing animals is beyond the scope of this article. The reader is referred to several excellent reviews for detailed information on these topics [3•,10,15,38, 39••,40••].

Conclusions

The beaches of North America provide the destination of choice to many people seeking rest and relaxation for leisure activities and vacation travel [1,2]. Occupational exposures may also occur, especially related to employment in the fishing industry. Some of the common envenomations and infections causing skin and soft tissue injury that may be encountered at the beaches and shores of North America have been reviewed in this article. It is my hope that this information will allow physicians to be more aware of the potential exposures to pathogens and toxins that are inherent in vacation visits to the beach or employment in water-related industries. Physicians will then be able to recognize these not-so-unusual skin and soft tissue infections and envenomations more quickly and effectively.

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