Cutaneous Manifestations of Waterborne Infections

Lucinda Elko, MD, RPh, Keith Rosenbach, MD, PhD, and John Sinnott, MD*

Address
*University of South Florida, Department of Infectious Diseases, Infectious Disease Center, Tampa General Hospital, PO Box 1289, Tampa, FL 33601, USA.
E-mail: keithrdoc@aol.com

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Although waterborne pathogens are relatively uncommon causes of cutaneous infections, these agents are being recognized with increasing frequency. Humans are exposed to water through a variety of recreational and occupational activities. Poor sanitary conditions in developing nations place the human populations at constant risk. Some aquatically acquired skin infections respond well to therapy, whereas other diseases do not require a specific treatment. However, many of these infections are rare, and effective therapy has not been defined. Gram-negative bacilli constitute the largest group of aquatic pathogens that cause skin infections. Other agents include mycobacteria, fungi, viruses, and amoeba and other parasites. Toxins from aquatic animals and parasites are associated with cutaneous diseases. Because waterborne skin infections, which are caused by a wide variety of pathogens, occur infrequently, most of the literature on the topic are case reports. This paper reviews the aquatic pathogens associated with cutaneous infections in humans and the available treatments.

Introduction

Waterborne infections can be acquired during occupational and recreational water activities. Human exposures include ingestion, inhalation, and dermal absorption of microbial organisms and toxins. More than 100 species of pathogenic bacteria, viruses, and protozoa can be found in contaminated water [1]. The quality of fresh and saltwater are frequently monitored. Key climatic variables, including precipitation and temperature, affect water quality. Increased urbanization, suburban sprawl, and pollution alter watersheds and affect freshwater and marine environments. In the treatment of sewage water, *Escherichia coli* and other fecal coliforms are indicators of level of contamination. Few studies have measured noncoliform organisms as indicators of potential disease [2••].

This article focuses on cutaneous infections caused by waterborne pathogens. These pathogens include bacteria, mycobacteria, fungi, viruses, amoeba, and other parasites (Table 1). Naturally occurring biotoxins produced by aquatic animals and parasites result in cutaneous reactions. The aquatic pathogens associated with cutaneous infections in humans and the available treatments are discussed. Infections causing systemic diseases with skin manifestations (*eg*, hepatitis A and leptospirosis) constitute a much broader topic involving numerous infectious agents and are not discussed.

Gram-negative Bacilli

Aeromonas hydrophila

Aeromonas hydrophila is a gram-negative aerobic bacillus capable of producing several virulence factors including cytotoxins, enterotoxins, hemagglutinins, and exoenzymes. *A. hydrophila* is ubiquitous in nature and is known to cause disease in fish, reptiles, and amphibians [3,4]. Infections typically occur during warm weather months. *Aeromonas* often are found in freshwater and brackish water; however, isolates have been recovered from fish tanks, swimming pools, and chlorinated tap water [3]. Medicinal leeches colonized with *Aeromonas* have been implicated in nosocomial transmission [3,4]. A case report describes a diabetic patient with chronic dermatitis developing lower extremity cellulitis caused by *Aeromonas* after repeatedly submerging his feet in a bucket of water at home [4].

Cutaneous infections are a result of freshwater exposure and trauma, such as simple abrasions, puncture wounds, propeller injuries, and alligator and water moccasin snake bites [3,5]. Most infections involve an extremity. Cellulitis develops within 48 hours of exposure to water. The disease often suppurates and may progress to bullea formation, skin necrosis, fasciitis, and myonecrosis. Other less frequent manifestations include ecthyma gangrenosum, pustular eruptions, furuncles, and subcutaneous nodules. Patients with chronic liver disease or immunologic disorders such as leukemia are at higher risk for developing sepsis [3,4]. A clinician should suspect *A. hydrophila* as the agent in a patient with a rapidly progressive cellulites and a history of freshwater exposure.

Soft tissue infections caused by *Aeromonas* often require surgical debridement and intravenous antibiotic treatment.

Isolates of *A. hydrophila* are universally resistant to penicillin, ampicillin, carbenicillin, and cefazolin [3]. *Aeromonas* produce at least three β-lactamases, but antibiotic resistance to third-generation cephalosporins has not been a major problem [6••]. More than 90% of isolates are susceptible to ceftriaxone, ceftazidime and cefepime, amoxicillin/clavulanate, ampicillin/sulbactam, piperacillin, piperacillin/tazobactam, ticarcillin, ticarcillin/clavulanate, and imipenem, meropenem, and aztreonam. *Aeromonas* are susceptible to fluoroquinolones, trimethoprim/sulfamethoxazole, tobramycin, and amikacin.

Chromobacterium violaceum

Chromobacterium violaceum is characterized as a long gram-negative bacillus that produces a violet pigment, violacein. It is a saprophyte found in soil and water in tropical and subtropical areas. Although it rarely causes disease in humans, the mortality rate is high and includes healthy patients without underlying medical conditions [7,8]. Similar to *Aeromonas*, *C. violaceum* cellulitis is associated with warm weather months and a history of skin trauma and freshwater exposure. In the United States, *C. violaceum* has a higher prevalence in the southeast, especially in Florida. Patients with the immune disorder chronic granulomatous disease are at higher risk for infection with *C. violaceum*.

Skin infections often include pain at the inoculation site, cellulitis, pustules, and lymphadenitis. The cellulitis can be rapidly progressive, ulcerate with a central black necrotic base, and a purulent discharge. Occasionally, the local infection may disseminate resulting in visceral abscesses and sepsis [7]. One case report includes a healthy young male in Florida who sustained a knee injury at a beach, which developed into a draining abscess for more than 2 months and eventually progressed to sepsis, disseminated disease, and death [9]. Another case report includes two young and healthy female flight attendants diagnosed with *C. violaceum* cellulites after sustaining multiple lacerations and abrasions during a jet-liner crash on a tropical Pacific island [8].

The optimal antibiotic regimen for the treatment of patients with this infection is not well-described. *C. violaceum* is resistant to cephalosporins and ampicillin in vitro [7]. Skin infections often disseminate and cause sepsis; thus, treatment usually involves multiple intravenous antibiotics, such as ciprofloxacin, gentamycin, and piperacillin. Relapse is common, and treatment with oral trimethoprim/sulfamethoxazole, ciprofloxacin, or doxycycline for weeks to months after intravenous therapy may be needed.

Erysipelothrix rhusiopathiae

Rosenbach's disease, or erysipeloid, is caused by *Erysipelothrix rhusiopathiae*, a thin pleomorphic, nonsporulating, gram-positive bacillus. *E. rhusiopathiae* is found worldwide in a variety of animals and fish. It does not cause disease in fish but can persist for long periods of time in the mucoid exterior slime layer of these animals. Infection frequently is seen in persons handling fish. Most human infections occur through occupational exposure in fisherman, fish handlers, butchers, and veterinarians [10]. Common names for erysipeloid include fish handler's disease, seal finger, and whale finger.

Abrasions and puncture wounds in the skin are the portal of entry. *Erysipelothrix* causes a subacute cellulitis and most often is located on the fingers. Infection may progress to bacteremia and endocarditis. The lesion is well-defined, slightly raised, and violaceous in the cutaneous form [10]. It usually resolves without treatment after 3 to 4 weeks. Oral antibiotics such as penicillin, clindamycin, or ciprofloxacin are effective if therapy is needed.

Francisella species

Francisella species are small, gram-negative cocco-bacilli that cause tularemia. In the United States, *Francisella tularensis* is strongly associated with wild rabbits and transmission by ticks and deerfly bites. However, *F. tularensis* has been isolated from streams, rivers, and lakes in Europe and Asia. It occasionally is isolated from contaminated water in the United States. Other *Francisella* strains, including *F. novicida* and *F. philomiragia*, can be isolated from water during warm weather months [11,12].

The six classic forms of tularemia include ulceroglandular, glandular, oculoglandular, pharyngeal, typhoidal, and pneumonic. Cutaneous exposure to *F. tularensis*, *F. novicida*, or *F. philomiragia* result in the ulceroglandular or glandular form of tularemia [11,13]. Patients infected with *F. philomiragia* commonly have underlying conditions such as chronic granulomatous disease, history of near-drowning in sea or estuary water, and myeloproliferative disease [11,12]. *F. tularensis* is difficult to grow in culture and is diagnosed often by clinical history and serology. *F. novicida* and *F. philomiragia* can be cultured.

Antimicrobial treatment of *F. novicida* and *F. philomiragia* is similar to treatment of *F. tularensis*. Most strains are susceptible to ciprofloxacin, streptomycin, gentamycin, tetracyclines, ceftriaxone, ceftazidime, and imipenem. Isolates produce β-lactamases and are resistant to ampicillin and cefazolin [12,13]. The drug of choice for treatment of tularemia is streptomycin.

Halomonas venusta

Halomonas venusta is a mildly halophilic gram-negative bacillus initially found in the marine environment surrounding Oahu, Hawaii. There has been one case in the literature describing *Halomonas* infection in a healthy female bitten by a fish of unknown species while diving at the Maldive Islands. Symptoms initially manifested as pain, with cellulitis and a watery discharge. The patient was treated with imipenem/cilastatin and then oral ciprofloxacin. The lesion healed without complication [14].

Table 1. Bacterial, fungal, and mycobacterial causes of cutaneous manifestations of waterborne infections

Organism	Epidemiology and associations	Clinical cutaneous manifestations	Diagnosis	Treatment
Aeromonas hydrophila	Gram-negative rod; associated with warm months, mainly freshwater and brackish water, leeches, alligator bites	Soft tissue infection with subcutaneous abscess, bullous lesions, myonecrosis, ecthyma gangrenosum, furuncles, necrotizing fasciitis	Microbiologic cultures	All fluoroquinolones, amikacin, ceftriaxone, ceftazidime, cefepime, tobramycin, aztreonam, imipenem, amoxicillin/clavulanate, meropenem, ampicillin/sulbactam, piperacillin, ticarcillin, piperacillin/tazobactam, ticarcillin/clavulanate, trimethoprim/sulfamethoxazole. May require surgical incision and drainage. Antibiotic duration usually 2 to 4 weeks.
Chromobacterium violaceum	Gram-negative rod, found in soil and water in tropical and subtropical areas	Ulceration with central black eschar, purulent discharge, and surrounding cellulites. Relapse occurs after initial response to therapy.	Microbiologic cultures	Antibiotic therapy is not well-documented. Successful treatment with ciprofloxacin, gentamicin, and piperacillin, or imipenem. May require surgical incision and drainage. Antibiotic duration usually 2 to 4 weeks. Then prolong treatment with oral doxycycline, ciprofloxacin, or trimethoprim/sulfamethoxazole for weeks to months to prevent relapse.
Erysipelothrix rhusiopathiae	Thin, pleomorphic, nonsporulating gram-positive rod. Main host is swine. Lives in the mucoid exterior slime layer of fish.	Subacute cellulitis most commonly on the fingertips. It is slightly raised and violaceous.	Microbiologic cultures, clinical diagnosis	Resolves in 3 to 4 weeks without treatment. If treatment is needed, most strains susceptible to penicillins, cephalosporins, clindamycin, imipenem, and ciprofloxacin. Antibiotic duration usually 2 weeks, if necessary.
Francisella species	Small, gram-negative cocco-bacilli; associated with northern hemisphere, rabbits. In Europe and Asia, isolated from streams and lakes. Most infections occur May through October.	Typically ulceroglandular appearance but can appear with lymphadenopathy alone	*Francisella tularensis* is difficult to grow in culture. Diagnosed often by clinical history and serology. Other *Francisella* strains grow on routine culture.	Streptomycin. Most strains are susceptible to quinolones, gentamycin, tetracyclines, ceftriaxone, ceftazidime, cefepime, imipenem, and meropenem. Antibiotic duration usually 2 weeks.
Loboa loboi	Fungus, mainly in South and Central America; associated with dolphin exposure	Skin nodules and plaques resembling keloid scars	Tissue fungal culture	Treat with cryotherapy and surgical excision. Poor response to antifungal therapy.
Mycobacterium fortuitum	Rapidly growing *Mycobacterium* associated with foot spas; increased risk if patient has razor-shaved legs before spa treatment	Multiple furuncles	Biopsy of tissue showing AFB and AFB culture positive	Normally resolves without treatment. Can administer single therapy, but dual therapy is preferred with ciprofloxacin, doxycycline, clarithromycin, or minocycline for 6 to 12 months.

AFB–acid-fast bacilli.

Table 1. Bacterial, fungal, and mycobacterial causes of cutaneous manifestations of waterborne infections *(Continued)*

Organism	Epidemiology and associations	Clinical cutaneous manifestations	Diagnosis	Treatment
Mycobacterium marinum	Before 1962, most cases associated with swimming pools. After 1962, most cases associated with aquarium cleaning, crab pinches, and contact with sea-urchin spines.	Ulcers, nodules, or nodular lymphangitis. Rarely, warty nodule or plaque on bony prominence.	Biopsy of tissue showing AFB and AFB culture positive	Therapy for 3 to 12 months. Regimens include doxycycline, minocycline, clarithromycin, trimethoprim/sulfamethoxazole, or rifampin plus ethambutol.
Mycobacterium ulcerans	Swamp water in Africa, Asia, Australia, and South America	Buruli ulcer (nodule develops into necrotic ulcer). May involve bone.	Biopsy of tissue showing AFB and AFB culture positive	Difficult to treat. Surgical debridement and dual antibiotics (rifampin, clarithromycin, dapsone, streptomycin, clofazimine, and/or trimethoprim/sulfamethoxazole) for weeks to months.
Plesiomonas shigelloides	Gram-negative rod; associated with fresh and brackish water	Cellulitis	Diagnosis by clinical history and microbiologic culture	Ciprofloxacin. Also, ceftriaxone, ceftazidime, cefepime, amoxicillin/clavulanate, ampicillin/sulbactam, piperacillin, ticarcillin, and piperacillin/tazobactam, ticarcillin/clavulanate, imipenem, meropenem, aztreonam, and trimethoprim/sulfamethoxazole. Antibiotic duration usually 2 weeks.
Pseudomonas aeruginosa	Gram-negative rod; associated with hot tubs, loofah sponges, and wetsuits	Papulovesicular folliculitis in adults and hot-foot syndrome with painful erythematous plantar nodules in children and adolescents	Microbiologic cultures, clinical diagnosis	Resolves spontaneously without treatment
Pseudomonas (Shewanella) putrefaciens	Gram-negative rod from soil, water, rotten meat, and dairy products. Predilection for tissues with poor circulation.	Otitis media in divers. Soft tissue infections such as cellulitis in patients with chronic infection/stasis of the lower extremities and liver disease.	Microbiologic cultures. Produces massive amounts of hydrogen sulfide in triple sugar agar.	Ciprofloxacin, ceftriaxone, ceftazidime, cefepime, piperacillin, ticarcillin, piperacillin/tazobactam, ticarcillin/clavulanate, imipenem, meropenem, amikacin, and tobramycin. Antibiotic duration usually 2 weeks.
Vibrio vulnificus	Curved, gram-negative rods, require saline environment to live. Infection occurs April through October, associated with fins of tilapia fish and common carp.	Cellulitis with bullous manifestations occur within 7 days of exposure to seawater. Predilection for patients with liver disease.	Diagnosis by clinical history and microbiologic culture	First-line antimicrobial agents include cefotaxime or ceftazidime, plus doxycycline. All quinolones, ceftriaxone, cefepime, piperacillin, ticarcillin, piperacillin/tazobactam, ticarcillin/clavulanate, imipenem, and meropenem. Requires surgical incision and drainage. Antibiotic duration usually 4 weeks or longer.

AFB–acid-fast bacilli.

Plesiomonas shigelloides

Plesiomonas shigelloides is a ubiquitous gram-negative bacillus, isolated from freshwater and brackish water in temperate and tropical climates. It usually is associated with oysters but rarely causes cellulitis after trauma and water exposure. Ciprofloxacin is the drug of choice for treating infections. Other susceptible antibiotics include other fluoroquinolones, ceftriaxone, ceftazidime, cefepime, amoxicillin/clavulanate, ampicillin/sulbactam, piperacillin, piperacillin/tazobactam, ticarcillin, and ticarcillin/clavulanate and imipenem, meropenem, and aztreonam. *P. shigelloides* is susceptible to trimethoprim/sulfamethoxazole but often is resistant to the aminoglycosides and tetracyclines [15].

Pseudomonas aeruginosa

Pseudomonas aeruginosa is a gram-negative bacillus frequently associated with pneumonia, bacteremia, and skin lesions (icthyma gangrenosum) in burn and neutropenic patients. *P. aeruginosa* was recognized as the causative agent for hot tub folliculitis in the early 1970s. Three environmental conditions predispose to infection: length of water exposure, increased number of bathers, and hypochlorination [16]. Cases have been associated with loofah sponges, wetsuits, and after skin depilation [17]. A new manifestation known as *Pseudomonas* hot-foot syndrome has been recognized in children and teenagers who used a community wading pool [18].

Hot tub folliculitis appears 1 to 3 days after hot tub exposure. Lesions are papulovesicular on an erythematous base, 2 to 5 mm in diameter, and some lesions will develop into pustules [19]. The distribution usually is on the trunk, buttocks, proximal limbs, and where apocrine glands are most abundant, such as the ear, nipple, and areola areas. The neck, soles, palms, and mucous membranes are not involved. Other symptoms may accompany the rash and include low-grade fever, malaise, headache, axillary adenopathy, and breast tenderness in both sexes [16]. The syndrome resolves spontaneously without treatment over the next 7 days and is not contagious.

Pseudomonas hot-foot syndrome was first diagnosed in 1998 in children and adolescents. The syndrome included painful erythematous plantar nodules that appeared 40 hours after the patient used a wading pool with a floor coated with abrasive grit to prevent slippage [18]. During physical examination, there were diffuse, dusky erythema of the soles, with deep 1- to 2-cm nodules on the weight-bearing plantar surfaces. Similar to hot tub folliculitis, it has a benign, self-limited course, and all patients recovered by day 14 with or without treatment.

Pseudomonas (shewanella) putrefaciens

Pseudomonas putrefaciens, a gram-negative bacillus, varies in length and produces a characteristic water-soluble red, tan, or pink pigment. It has been isolated from water, soil, shellfish, and petroleum brines. *P. putrefaciens* originally was described as a cause of otitis media in divers and is associated with cellulitis and soft tissue infections in patients with poor circulation and chronic leg dermatitis [20,21]. Chronic lower extremity infections and liver disease have been identified as possible risk factors for *P. putrefaciens* sepsis [22]. In one case report, an elderly male with prior chronic dermatitis had been swimming at two local beaches on Long Island where shellfish were frequently harvested. He had also ingested raw clams from one of the beaches. A few weeks later, the patient was diagnosed with an exacerbation of lower extremity dermatitis/cellulitis and *P. putrefaciens* bacteremia [20].

Isolates are routinely resistant to ampicillin and cephalosporins. *P. putrefaciens* is susceptible to ceftriaxone, ceftazidime, cefepime, piperacillin, piperacillin/tazobactam, ticarcillin, ticarcillin/clavulanate and amikacin, tobramycin, and ciprofloxacin.

Vibrio species

Vibrio bacteria characteristically are curved, halophilic gram-negative bacilli inhabiting marine and estuary environments. Infections are associated with warm weather months, shellfish, and recreational water activities. *Vibrio* infections are most common in states bordering the Gulf of Mexico and the Chesapeake Bay. A voluntary *Vibrio* Surveillance System involving Florida, Alabama, Louisiana, and Texas reports 84% of all *Vibrio* cases in the United States [23•]. *Vibrio vulnificus*, *Vibrio parahaemolyticus*, and *Vibrio alginolyticus* account for 86% of *Vibrio* cases. *Vibrio* infections generally occur as one of three syndromes: wound infection, gastroenteritis from the ingestion of undercooked shellfish, or primary septicemia without the history of wound infection or gastroenteritis. Almost all cases of wound infection involve trauma and exposure to shellfish or seawater 7 days before illness [23•,24]. Most infections occur during April through October when the Gulf water is warmer and the salinity of the water is higher—ideal conditions for *Vibrio* growth. In the Jordan Valley of Israel, there have been two seasonal outbreaks of *V. vulnificus* associated with the fins of tilapia fish bred in artificial ponds [25]. Other outbreaks in Israel have been associated with handling of common carp, a pond-cultivated fish, after finning injuries to the hand from bones or spine of fish [26].

Wound infections have a rapidly developing cellulitis with hemorrhagic bullae, ulceration, and necrotizing fasciitis. Fifty percent of patients with wound infection sustained the wound at time of exposure, 21% reported a pre-existing wound, and 29% were unsure of timing of injury [23•]. Many injuries were work-related and occurred during occupational exposure of dock workers, commercial fishermen, and oyster shuckers. The mean age of infection is 40 years and the mortality rate is 9% to 17% [23•,24,27]. Patients with pre-existing liver disease are at increased risk for sepsis and have a higher fatality rate.

Because of the severe nature of the disease, treatment of wound infections and cellulitis (especially *V. vulnificus*) involves emergent surgical debridement and multiple intravenous antibiotics. Blood cultures are routinely performed during the treatment of patients because of the high rate of septicemia. The treatment regimen of choice is ceftazidime or cefotaxime, plus doxycycline, with or without ciprofloxacin. Other useful treatments include the other bactam antibiotics: ceftriaxone, cefepime, piperacillin, piperacillin/tazobactam, ticarcillin, ticarcillin/clavulanate, imipenem, meropenem, and all other fluoroquinolones. *V. vulnificus* often is resistant to the aminoglycosides tobramycin and amikacin, which are usually considered for the treatment of other gram-negative bacillary sepsis syndromes.

Mycobacteria

Mycobacterium marinum

Mycobacterium marinum is naturally found in fresh and saltwater and causes swimming pool granuloma, fish tank granuloma, or sea urchin granuloma. Infections are associated with trauma and water exposure, followed by a prolonged incubation period ranging from 5 and 270 days, with a mean incubation period of 21 days [28•]. The skin lesion develops into a large granulomatous ulcer. Before 1962, most cutaneous infections involved swimming pool injuries. The epidemiology has changed with the chlorination of swimming pools, and today this pattern accounts for less than 5% of cases. The infection is diagnosed more often in home aquarium owners. Vectors include fresh or saltwater fish, snails, shellfish, dolphins, and crustaceans, including pinches from crab claws in coastal South Carolina and contact with sea urchin spines in the Mediterranean and Atlantic coastal waters [28•,29, 30].

Mycobacterium marinum usually manifests as a single cutaneous lesion of the upper extremity. Cutaneous ulcers and nodules, and nodular lymphangitis (sporotrichoid pattern), occur after exposure to *M. marinum* with lacerations or abrasions exposed to fresh or saltwater or after injuries related to fish spines [28•]. The infection may appear as a warty nodule or plaque on a bony prominence [29]. Deep infections are rare; however, a chronic infection may develop into osteomyelitis if not treated appropriately.

Typical to mycobacterial infections, treatment includes multiple antibiotics administered for 3 to 12 months. Recommended regimens include clarithromycin, doxycycline, or minocycline, trimethoprim/sulfamethoxazole, or rifampin plus ethambutol [28•,29].

Mycobacterium fortuitum

Mycobacterium fortuitum are distributed ubiquitously in soil and water. In 2002, an outbreak of *M. fortuitum* lower extremity furunculosis was associated with footbaths at a nail salon in California. Risk factors for the 110 cases included a history of trauma (shaving and pedicure) with water exposure (the 10 footbaths). Patients typically had multiple furuncles on the lower extremities. Some lesions ulcerated but others regressed with spontaneous healing and substantial scarring. At the end of the investigation, 32 patients were culture-positive for *M. fortuitum* and two patients were positive for unidentified mycobacterium [31]. All 10 footbaths at the spa yielded *M. fortuitum*. Many of the lesions healed spontaneously. Treatment included single or dual therapy with the oral agents ciprofloxacin, clarithromycin, doxycycline, and minocycline.

Mycobacterium ulcerans

Mycobacterium ulcerans is found naturally in the swamp waters of Africa, Asia, Australia, and South America and causes Buruli ulcer. The lesion develops after trauma to the lower extremity from swamp vegetation. A nodule develops and evolves into a necrotic ulcer. The infection is more virulent than *M. marinum* and results in deformity of the limb. *M. ulcerans* responds poorly to antibiotic therapy. Treatment often involves surgical debridement and dual therapy with dapsone, streptomycin, rifampin, clarithromycin, clofazimine, and trimethoprim/sulfamethoxazole [32].

Acanthamoeba

Acanthamoeba are ubiquitous, small, free-living amebas isolated from soil, water, hot tubs, and air conditioners. *Acanthamoeba* exist in cyst and trophozoite form. Trophozoites are the infectious and invasive form of the organism [33]. The amoeba often is associated with keratitis in contact lens wearers. Skin lesions may appear as tender or nontender nodules, papules, pustules, or ulcers. In patients with AIDS, *Acanthamoeba* can disseminate to the central nervous system and appear as chronic or subacute encephalitis [34].

Definitive therapy has not been established. Several agents have variable efficacy, including pentamidine, 5-flucytosine, ketoconazole, rifampin, sulfadiazine, neomycin, kanamycin, and paromomycin [33].

Cnidarian Larvae and Seabather's Eruption

Cnidarians are marine animals with stinging structures known as nematocysts. Cnidarians range in size from microscopic jellyfish and large Portuguese man-of-war and corals and sea anemones. Cases occur during the warmer weather months. An outbreak in Long Island was traced to sea anemone larvae, whereas in Florida and the Caribbean, the larvae of the thimble jellyfish can cause seabather's eruption [35,36]. The microscopic larvae are trapped in the bathing suits of swimmers. The rash usually begins 4 to 24 hours after seabathing, although most patients feel a prickly sensation while in the water. The lesions are found underneath the clothing/swimsuit and at pressure points such as the beltline. Those who try to relieve the stinging by showering or jumping into a swimming pool usually

report an exacerbation of symptoms. The exposed area progresses to a pruritic, macular, papular, or urticarial rash for 3 to 7 days [35]. Treatment includes antihistamines, topical steroids, and systemic steroids in severe cases.

Dracunculus medinensis

Guinea worm disease or dracunculiasis is caused by *Dracunculus medinensis*. The pond-water parasite was endemic to the developing nations in Africa, the Middle East, and the Indian subcontinent. In 1986, the World Health Organization adopted a resolution calling for the eradication of the Guinea worm in an estimated 3.5 million people afflicted with the disease in 20 countries [37]. The two remaining major endemic foci are in the Sudan and northern Ghana. Patients are initially infected after drinking water that has been contaminated with crustaceans carrying the immature forms of the parasite. A year after entering the human host, the meter-long worm emerges through a chronic skin lesion, usually on the lower extremity. No effective treatment exists and the worm has to be slowly extracted by rolling the worm on a stick 1 cm a day until removed.

Herpes Simplex

Herpes simplex commonly causes recurrent ulcers. A specific risk factor occasionally can be identified for recurrent orolabial herpes episodes. In 1995, a case report described a young male physician who had 10 to 11 episodes of recurrent oral herpes per year. No cultures were performed, and the lesions only partially responded to acyclovir. After keeping a journal for 3 years, he noted that the episodes were related to swimming in chlorinated water. After discontinuation of swimming in chlorinated pools, the patient was episode-free for 25 months [38]. He continued to swim in freshwater and saltwater without episodes of oral herpes.

Loboa loboi

Lobomycosis is caused by the fungus *Loboa loboi*, previously classified as *Paracoccidioides loboi*. The disease occurs mainly in South and Central American coastal areas. It forms skin nodules and plaques resembling keloids and involves the earlobes, distal parts of the extremities, and buttocks [39]. The disease has been acquired in dolphins off the coast of Florida [40]. It is difficult to cure with antifungal medications. Cryotherapy and surgical excision have been curative.

Protetheca

Prototheca species are achlorophyllic unicellular algae in the slime-flux of trees and freshwater environments. The group rarely causes human disease except as a rare cause of postoperative wound infection and cutaneous infections involving patients with underlying immunosuppression [41]. The most common agent is *Prototheca wickerhamii*, which grows on routine agar. Most cutaneous disease has been reported from temperate areas and produces a hyperplastic and granulomatous response. Lesions are painless vesicules, bullae, and ulcers with purulent discharge and crusting. The lesions may have an "apple-jelly" hue. Treatment has been with amphotericin B because 4% of the algae wall is composed of ergosterol. In 1995, there was a case report of an elderly female with a diffuse, ill-defined erythema of the arm, forearm, and hand. There were 4- to 6-mm flesh-colored papules within the same area. She responded to oral itraconazole and the lesions were resolved after 2 weeks of therapy [41].

Schistosoma haematobium

Cutaneous disease is rare for human *Schistosoma* species. A recent case report documents a women with a 2-month history of eruption in her groin, which was occasionally pruritic after she rode a bike. Examination revealed firm, red papules, some in a linear fashion. Her physicians initially were concerned about cutaneous larvae migrans, and a punch biopsy was completed, which showed *Schistosoma* ovum in the dermis surrounding a hair follicle. A centrifuged urine sample contained *Schistosoma* ova. The patient was treated with two doses of praziquantel, 6 hours apart. One month after treatment, cystoscopy was performed and no abnormalities were found. Three years before the diagnosis, the patient had been canoeing in Africa [42].

Nonhuman *Schistosoma* and Cercarial Dermatitis

Cercarial dermatitis is a pruritic inflammatory response to the penetration of human skin by nonhuman *Schistosoma* larvae, contracted while swimming or wading in freshwater lakes. In a case report from 2001, the disease was acquired after aquarium exposure to snails infected with the cercariae [43]. The appearance is maculopapular rash in uncovered skin areas exposed to the water. Antihistamines and topical corticosteroids are the main treatment.

Conclusions

Although uncommon, cutaneous infections can be acquired through contact with water. The infections are associated with superficial trauma before or during the water exposure. Most of these infections are caused by gram-negative bacilli. Other pathogens include mycobacteria, gram-positive bacteria, fungus, virus, and other organisms. Certain conditions, such as *P. aeruginosa*, hot tub folliculitis, *E. rhusiopathiae* (Rosenbach's disease), herpes simplex virus, and seabather's eruption, are benign with a self-limited course and often do not require specific anti-

microbial therapy. Other pathogens, including *Francisella* species, *H. venusta*, *P. shigelloides*, *P. putrifaciens*, *Acanthamoeba*, *Protetheca*, and if necessary *E. rhusiopathiae*, result in cellulitis that requires treatment with oral or intravenous antibiotics. These infections respond to a short course of treatment; usually 2 weeks is adequate. A few gram-negative bacilli, such as *A. hydrophila*, *C. violaceum*, and especially *V. vulnificus*, cause an acute rapidly progressing cellulitis, often associated with bacteremia, sepsis, and death. Treatment is urgent to emergent, requiring surgical debridement and multiple intravenous antibiotics for 3 to 4 weeks or longer. The patient's clinical response often dictates the duration of therapy. The chronic skin infections associated with water exposure are caused by mycobacteria such as *M. fortuitum*, *M. marinum*, and M. *ulcerans*, and the fungus *L. loboi*. Prolonged antibiotic treatment for 3 to 12 months often is necessary and ineffective, requiring adjunctive surgical debridement.

References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

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