# Skin, Soft Tissue and Systemic Bacterial Infections Following Aquatic Injuries and Exposures

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**Abstract:** Bacterial infections following aquatic injuries occur commonly in fishermen and vacationers after freshwater and saltwater exposures. Internet search engines were queried with the key words to describe the epidemiology, clinical manifestations, diagnostic and treatment strategies and outcomes of both the superficial and the deeper invasive infections caused by more common, newly emerging and unusual aquatic bacterial pathogens. Main findings included the following: (1) aquatic injuries often result in gram-negative polymicrobial infections with marine bacteria; (2) most marine bacteria are resistant to 1st- and 2nd-generation penicillins and cephalosporins; (3) nontuberculous, mycobacterial infections should be considered in late-onset, culture-negative and antibiotic-resistant marine infections; (4) superficial marine infections and pre-existing wounds exposed to seawater may result in deeply invasive infections and sepsis in immunocompromised patients. With the exception of minor marine wounds demonstrating localized cellulitis, most other marine infections and all gram-negative and mycobacterial marine infections will require therapy with antibiotic combinations.

**Key Indexing Terms:** Aquatic infections; Marine infections; Aquatic bacteria; Marine bacteria; Marine mycobacteria; Fish pathogens; Aquaculture-related fish infections. **[Am J Med Sci 2015;349 (3):269–275.]**

Bacterial infections following aquatic injuries occur commonly and usually on the extremities in fishermen and vacationers worldwide after freshwater and saltwater exposures. Although many species of bacteria have been isolated from marine wounds, superficial soft tissue and invasive systemic infections following aquatic injuries and exposures may be caused by a small number of bacterial species, including *Aeromonas hydrophila*, *Edwardsiella tarda*, *Erysipelothrix rhusiopathiae*, *Mycobacterium marinum* and *Vibrio vulnificus*. In addition to these species, several other aquatic bacteria and mycobacteria have recently been identified as emerging or unusual causes of superficial and invasive infections following marine injuries and exposures, including *Chromobacterium violaceum*, *Shewanella* species, *Streptococcus iniae* and *Mycobacterium fortuitum*. The objectives of this review were to describe the epidemiology, presenting clinical manifestations, diagnostic and treatment strategies and outcomes of both the superficial and the deeper invasive infections caused by more common, newly emerging and unusual aquatic bacterial pathogens.

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## METHODS

Internet search engines, including PubMed, Medline, Ovid, Google and Google Scholar, were queried with the key words to meet the objectives of this review article. Because this article reviewed existing scientific publications and did not involve human or animal subjects, institutional review board approval was not required.

## RESULTS

### Epidemiology and Microbiology

#### Descriptive Epidemiology of Aquatic Infections Following the Thai Tsunami

The greatest worldwide experiences in managing acute and chronic skin and soft tissue infections (SSTIs) following aquatic injuries and exposures occurred during the ensuing years after a massive tsunami struck southern Thailand on December 26, 2004. Shortly after the disaster, Hiransuthikul et al[1] reported acute SSTIs in 515 of 777 (66.3%) patients transferred to 4 referral hospitals in Bangkok with SSTIs following crush injuries to the legs. Wound and/or purulent drainage cultures were obtained in 396 (76.9%) of these patients.[1] Most infections were polymicrobial in etiology (71.8%), and the most common organisms isolated were gram-negative bacteria, including *Aeromonas* species (22.6%), *Escherichia coli* (18.1%), *Klebsiella pneumoniae* (14.5%), *Pseudomonas aeruginosa* (12.0%) and *Proteus* species (7.3%).[1] Only 4.5% of the isolates were gram-positive bacteria, most commonly *Staphylococcal* species.[1]

Later, hundreds of vacationing Swedish tsunami survivors were transferred to hospitals in Sweden for further in-patient care.[2] Appelgren et al[2] reported 15 cases of late-onset, chronic posttraumatic SSTIs caused by rapidly growing marine mycobacteria in Swedish tsunami survivors. These mycobacterial infections occurred 20 to 105 days (median = 60 days) after the initial trauma in undamaged skin near sutured traumatic wounds or skin grafts.[2] All these cases required specific therapy with antimycobacterial medications.[2] The course of infection was protracted in all cases with healing by 12 months in most cases.[2]

The most important lessons learned from the acute and chronic management of SSTIs in these tsunami survivors with contaminated aquatic injuries included the early predominance of gram-negative causative organisms, especially aquatic *Aeromonas* species, in polymicrobial infections and the possibility of late-onset rapidly growing mycobacterial infections when conventional bacterial cultures remained inconclusive.

#### Microbiology of Skin, Soft Tissue and Systemic Infections Following Aquatic Injuries and Exposures

Although *Staphylococcus aureus*, *Streptococcus* species, *P aeruginosa* and several other bacterial species have been recovered from infected minor wounds after marine exposures, some of the more uniquely marine bacterial pathogens recovered following more severe injuries in aquatic environments

have included *Aeromonas* species, *C violaceum*, *E tarda*, marine *Mycobacterium* species, *Shewanella* species and *V vulnificus* (Table 1). Nevertheless, empiric treatment of SSTIs that develop shortly after marine water exposure should still include coverage of *S pyogenes* and *S aureus* in addition to marine water–associated organisms. Such a regimen could include a 1st-generation cephalosporin or clindamycin plus a fluoroquinolone like levofloxacin or ciprofloxacin plus doxycycline (if at increased risk for *V vulnificus* infection).

## Pathogen-Specific Clinical Manifestations

### *Aeromonas* Species

*Aeromonas* species are gram-negative rods found in warm soil and fresh and brackish waters worldwide as aquatic animal commensals and pathogens.[3] Most are capable of producing enterotoxins and hemolysins and causing acute hemorrhagic diarrhea and invasive SSTIs in both immunocompetent and immunocompromised patients following aquatic injuries and exposures, including near-drowning.[3] *Aeromonas* wound infections may occur following freshwater traumatic injuries, such as alligator, fish, snake and leech bites.[3] *Aeromonas*-infected lesions usually occur on extremities or other body regions with open wounds that were immersed in contaminated freshwater during warmer months.[3,4] Within 24 hours, infected wounds exhibit erythema, edema and purulent discharge indistinguishable from streptococcal cellulitis.[3] Fever and chills will ensue in untreated or improperly treated cases and can progress to invasive infections, especially in the immunocompromised patients, with necrotizing fasciitis, necrotizing myositis and osteomyelitis.[3,4]

Although most *Aeromonas* SSTIs occur following aquatic immersions, Vally et al[4] were the 1st to report *Aeromonas* infections following mud exposures in an outbreak of *A hydrophila* wound infections in 26 mud football players in 2002. None of the players reported any immunocompromising illnesses, and all recovered uneventfully with antibiotic therapy.[4] In general, *Aeromonas* isolates encountered in human infections are susceptible to fluoroquinolones, tetracyclines, aminoglycosides, carbapenems, monobactams and 3rd- and 4th-generation cephalosporins; variable levels of susceptibility have been reported for trimethoprim-sulfamethoxazole.[5] In addition to wound drainage and debridement, *Aeromonas* wound infections should be treated initially with either a fluoroquinolone or a 3rd-generation cephalosporin with the possible addition of an aminoglycoside until culture and antibiotic sensitivity results are reported and rule out *Pseudomonas* coinfections.[5,6]

### *Chromobacterium violaceum*

*Chromobacterium violaceum*, an aerobic, gram-negative bacillus, is an ubiquitous saprophyte found in soil and water in tropical and subtropical regions worldwide, including the southeastern United States.[7,8] Although widely distributed geographically, *C violaceum* is a low-grade pathogen that causes few infections in immunocompetent persons and is often dismissed as a bacterial contaminant in positive cultures.[7] The organism grows rapidly on ordinary culture media and is typically identified visually by the violet color of its colonies.[7,8] Nonpigmented strains of *C violaceum* are less commonly found than pigmented strains but do co-exist with pigmented strains and can cause mixed SSTIs.[8,9] Most cases are reported from temperate and tropical regions with high case fatality rates (CFRs) in the immunocompromised patients.[10] In 1982, Macher et al[10] reported 12 cases of *C violaceum* infections in the United States in patients with chronic granulomatous disease, 7 of whom died of invasive septicemia 7 days to 15 months after initial infections. The possibility of neutrophil dysfunction should be considered when evaluating patients who present with fulminant infections secondary to this organism.

The portal of entry for *C violaceum* is usually a skin injury from a laceration or fish bite followed by exposure to brackish or stagnant water. An ulcerated skin lesion with a bluish purulent discharge develops at the initial injury site with regional swelling usually on an extremity. Within days, hematogenous dissemination may occur, more commonly in the immunocompromised patients, with high fevers and disseminated macular skin lesions that progress to abscesses. Abscesses may also occur in bone, liver, lung and spleen. Because of the rarity of this infection, treatment recommendations are limited. The organism is generally susceptible to aminoglycosides, fluoroquinolones, tetracyclines, carbapenems, chloramphenicol and trimethoprim-sulfamethoxazole but resistant to most penicillins and cephalosporins.[8,9] Because of high CFRs, treatment of suspected *C violaceum* infections should begin immediately with drainage of all purulent abscess collections and empiric administration of combination intravenous antibiotic therapy, which can be narrowed once susceptibilities become available.[9] Treatment length is typically prolonged when abscesses are present.

### *Edwardsiella tarda*

*Edwardsiella tarda*, a gram-negative rod of the family Enterobacteriaceae, is a notorious aquaculture pathogen and causative agent of emphysematous putrefactive disease of catfish.[11] In 2001, Slaven et al[12] described a series of 11 cases in Louisiana with culture-confirmed extraintestinal *E tarda* infections during the period, 1993 to 1999, with 5 wound infections (3 with marine exposures), 5 abscesses requiring surgical drainage and 1 case of bacteremia.

The investigators concluded that extraintestinal *E tarda* infections were uncommon compared with intestinal infections, frequently presented with wound abscesses requiring surgical incision and drainage in patients with marine injuries or exposures and could cause extensive myonecrosis and fatal septic shock in the immunocompromised patients, especially in patients with chronic liver disease.[12] The authors recommended therapy with antibiotics that are effective against gram-negative bacteria (ie, ampicillin, cepahalosporins such as cefazolin and ceftazidime, aminoglycosides, fluoroquinolones) in all cases of extraintestinal *E tarda* infections.[12]

### *Shewanella* Species

*Shewanella* species are saprophytic gram-negative bacteria that are distributed in warm and temperate regions worldwide and are part of the normal microflora of the marine environment. There are more than 50 species of *Shewanella*, all of which produce yellowish brown mucoid colonies that emit hydrogen sulfide in culture. Several *Shewanella* species have been recently recognized as emerging causes of soft tissue and invasive infections after seawater exposures, including *Shewanella algae* (most common), *Shewanella haliotis*, *Shewanella putrefaciens* and *Shewanella xiamenensis*.[13,14] The most commonly reported clinical manifestations of *Shewanella* infections are deep ulcers associated with hemorrhagic bullae usually on the lower extremities, otitis externa, otitis media, biliary tract infection and bacteremia.[13–15] Non-healing ulcers have resulted in necrotizing fasciitis, compartment syndromes requiring decompressive fasciotomies and osteomyelitis.[15] *Shewanella* sepsis has been associated with endocarditis and meningitis.[13]