Skin and Soft Tissue Infections Following Marine Injuries and Exposures in Travelers

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Background. Bacterial skin and soft tissue infections (SSTIs) in travelers often follow insect bites and can present a broad spectrum of clinical manifestations ranging from impetigo to necrotizing cellulitis. Significant SSTIs can also follow marine injuries and exposures in travelers, and the etiologies are often marine bacteria.

Methods. To meet the objectives of describing the pathogen-specific presenting clinical manifestations, diagnostic and treatment strategies, and outcomes of superficial and deep invasive infections in travelers caused by commonly encountered and newly emerging marine bacterial pathogens, Internet search engines were queried with the key words as MESH terms.

Results. Travel medicine practitioners should maintain a high index of suspicion regarding potentially catastrophic, invasive bacterial infections, especially *Aeromonas hydrophila*, *Vibrio vulnificus*, *Chromobacterium violaceum*, and *Shewanella* infections, following marine injuries and exposures.

Conclusions. Travelers with well-known risk factors for the increasing severity of marine infections, including those with open wounds, suppressed immune systems, liver disease, alcoholism, hemochromatosis, hematological disease, diabetes, chronic renal disease, acquired immunodeficiency syndrome, and cancer, should be cautioned about the risks of marine infections through exposures to marine animals, seawater, the preparation of live or freshly killed seafood, and the accidental ingestion of seawater or consumption of raw or undercooked seafood, especially shellfish. With the exception of minor marine wounds demonstrating localized cellulitis or spreading erysipeloid-type reactions, most other marine infections and all Gram-negative and mycobacterial marine infections will require therapy with antibiotic combinations.

Skin disorders are the third most common cause of morbidity in returning travelers and are usually infectious in origin and bacterial in etiology. Bacterial skin and soft tissue infections (SSTIs) in travelers often follow insect bites and can present a broad spectrum of clinical manifestations ranging from impetigo and ecthyma to erysipelas, abscess formation, necrotizing cellulitis, and myonecrosis. Significant SSTIs can also follow marine injuries and exposures in travelers, and the etiologies are often marine bacteria. The objectives of this review were to identify the most common aquatic bacterial etiologies of SSTIs following marine injuries and exposures in travelers and to familiarize healthcare providers with the epidemiology, presenting clinical features, diagnosis, treatment, and prevention of SSTIs following marine injuries and exposures.

Methods

Internet search engines were queried with the key words as MESH terms. The pathogen-specific characteristics of both superficial and deeper invasive infections caused by common and newly emerging marine bacteria were described. The immediate general management of marine wounds and the initial selection of antibiotics for marine wounds contaminated with unknown bacteria were also presented.
Results

Epidemiology of SSTIs Following Marine Injuries and Exposures

Unlike bacterial infections acquired on land while abroad and reported by travelers returning home, the epidemiology of bacterial infections following marine injuries or exposures and aquatic animal bites and stings has been less intensively investigated. Observational studies of dermatoses have documented superficial injuries, such as aquatic animal bites and stings, and skin infections as second only to sunburns in tourists visiting popular island destinations, such as Fiji, the Maldives, and Martinique. In addition to skin infections in travelers returning from tropical island destinations, skin infections among tourists who survived the Thai tsunami of 2004 have been described in detail in recent observational studies.7 After a massive tsunami struck southern Thailand on December 26, 2004, SSTIs were reported in 515 (66.3%) of the 777 patients transferred to four referral hospitals in Bangkok.7 Wound and/or purulent drainage specimens were obtained in 396 (76.9%) of these patients.7 Most of the SSTIs followed contaminated crush injuries to the legs, and most of the infections were polymicrobial in etiology (71.8%).7 The most common organisms isolated were Gram-negative bacteria, especially Aeromonas species (22.6%), Escherichia coli (18.1%), Klebsiella pneumoniae (14.5%), Pseudomonas aeruginosa (12.0%), and Proteus species (7.3%).7 Only 4.5% of the isolates were Gram-positive bacteria, most commonly Staphylococcal species.7

Many species of bacteria, especially Staphylococcus aureus and Streptococcus species, have been isolated from marine wounds and commonly cause impetigo, pyoderma, and erysipelas. In 2013, Lodde and colleagues reported a 42-year-old commercial fisherman who became incapacitated after 3 days of oral penicillin therapy for fever and exfoliative skin lesions on both hands.8 Subsequent bacteriological identification of the causative organism showed skin colonization with methicillin-sensitive S. aureus with production of Panton-Valentine leukocidin.8 The authors concluded that the marine environment was a risk factor for skin abrasion injuries that could lead to infection by highly pathogenic strains of S. aureus capable of producing Panton-Valentine leukocidin.8

In summary, observational studies in island nations frequently visited by travelers and case reports of infections following marine injuries have confirmed that skin injuries, such as fish bites, puncture wounds, abrasions, and more serious injuries, may become infected with both highly specific marine bacterial species, such as Aeromonas and Shewanella species, and highly pathogenic strains of S. aureus.

SSTIs Following Marine Injuries and Significant Exposures

Although S. aureus, Streptococcus species, P. aeruginosa, and several other bacterial species have been recovered from infected minor wounds after marine exposures, the most commonly encountered causative and uniquely marine bacterial pathogens following more severe marine injuries have included Aeromonas species, Chromobacterium violaceum, Edwardsiella tarda, marine Mycobacterium species, Shewanella species, and Vibrio vulnificus.9

Aeromonas Species

Aeromonas species are Gram-negative rods found in warm fresh and brackish waters worldwide as aquatic animal commensals and pathogens.10 Most are capable of producing enterotoxins and hemolysins and causing acute hemorrhagic diarrhea and invasive SSTIs in both immunocompetent and immunocompromised patients following marine injuries and exposures.10,11

Aeromonas wound infections typically occur following aquatic injuries, such as alligator, fish, snake, or freshwater leech bites.10–12 Aeromonas-infected wounds usually occur on extremities or other body regions that were immersed in contaminated freshwater during warmer months.10–12 Within 24 hours, infected wounds will exhibit erythema, edema, and purulent discharge indistinguishable from streptococcal cellulitis.10–12 Fever and chills ensue in untreated or improperly treated cases and can progress to invasive infections including necrotizing fasciitis, necrotizing myositis, and osteomyelitis in immunocompromised patients.10–12

In a prospective nationwide study of Aeromonas infections in France over six of the warmest months of the year (May to October 2006), Lamy and colleagues reported 78 culture-confirmed Aeromonas infections that were predominantly wound and soft tissue skin infections (n = 34, 44%) and bacteremias (n = 20, 26%).12 The wound and soft tissue infections presented as purulent lesions (n = 25, 74%) or abscesses (n = 9, 26%) and occurred primarily in healthy males who reported high rates of environmental exposures and trauma (90+%).12 The traumatic injuries with resulting Aeromonas infections were often associated with freshwater exposures (n = 15) during recreational activities (n = 14).12 The authors also described five cases of respiratory tract infections mostly in males with comorbidities and massive aquatic exposures from near-drowning (n = 3).12 Similar to many related Vibrio species, most Aeromonas species are resistant to penicillins and first-generation cephalosporins, presumably owing to beta-lactamase production.13 In addition to wound drainage and debridement, Aeromonas wound infections should be treated initially with a combination of an aminoglycoside and either a fluoroquinolone or a third-generation cephalosporin until culture and antibiotic sensitivity results are reported and rule out Pseudomonas coinfections.11–13

Chromobacterium violaceum

Chromobacterium violaceum is an aerobic, Gram-negative bacillus and a saprophyte widely distributed in soil.
and water in tropical and subtropical regions.\textsuperscript{14–16} The organism grows rapidly on ordinary culture media and is often first identified by the violet color of its colonies. However, it is often dismissed in positive cultures as a bacterial contaminant.\textsuperscript{17} Nonpigmented strains of \textit{C. violaceum} are less commonly found than pigmented strains, but do coexist with pigmented strains and can cause mixed infections.\textsuperscript{16} Most infections are reported from temperate and tropical regions with high case fatality rates (CFRs), especially in immunocompromised patients.\textsuperscript{17,18} In 1982, Macher and colleagues reported 12 cases of \textit{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.\textsuperscript{17}

The portal of entry for \textit{C. violaceum} is usually a skin injury from a laceration or fish bite followed by exposure to brackish or stagnant water.\textsuperscript{15} An ulcerated skin injury from a laceration or fish bite followed by a causative agent of emphysematous putrefactive disease, \textit{Enterobacteriaceae}, is a notorious fish pathogen and \textit{Edwardsiella tarda}\textsuperscript{a} antibiotic therapy.\textsuperscript{16} Purulent abscess collections and combined intravenous infections should begin immediately with drainage of all C. violaceum at the initial injury site with regional swelling usually skin lesion with a bluish purulent discharge develops 7 months after an extremity.\textsuperscript{15} Within days, invasive septicemia may occur, especially in the immunocompromised, with high fever and disseminated macular skin lesions that progress to abscesses.\textsuperscript{18} Abscesses may also occur in bone and in the liver.\textsuperscript{14,18} The organism is susceptible to aminoglycosides, fluoroquinolones, tetracyclines, imipenem, and trimethoprim–sulfamethoxazole, but resistant to penicillins and cephalosporins.\textsuperscript{15,16} Because of high CFRs, treatment of suspected \textit{C. violaceum} infections should begin immediately with drainage of all purulent abscess collections and combined intravenous antibiotic therapy.

\textbf{Edwardsiella tarda}

\textit{Edwardsiella tarda}, a Gram-negative rod of the family Enterobacteriaceae, is a notorious fish pathogen and causative agent of emphysematous putrefactive disease of catfish.\textsuperscript{19} In 2001, Slaven and colleagues described a series of 11 cases in Louisiana with culture-confirmed extraintestinal \textit{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 fatal septicemia in a 67-year-old male with cirrhosis and hepatocellular carcinoma.\textsuperscript{20}

In 2013, Crosby and colleagues reported a case of extensive upper extremity myonecrosis resulting in transhumeral amputation in an \textit{E. tarda}-infected catfish puncture wound in a patient with hepatitis C.\textsuperscript{21} In all reported cases to date, \textit{E. tarda} was susceptible to a broad range of antibiotics including penicillins, cephalosporins, and quinolones.\textsuperscript{20,21} Most investigators concluded that extraintestinal \textit{E. tarda} infections were uncommon compared with enteric infections, but frequently presented with wound abscesses following marine exposures. Hepatic disease was the greatest risk factor for extensive myonecrosis and fatal septic shock.\textsuperscript{20,21} Intravenous therapy with a combination of antibiotics effective against Gram-negative bacteria is recommended in all cases of extraintestinal \textit{E. tarda} infections.\textsuperscript{20,21}

\textbf{Shewanella Species}

\textit{Shewanella} species are saprophytic Gram-negative bacteria and part of the normal microflora of the marine environment that are distributed in temperate and tropical regions worldwide.\textsuperscript{22,23} \textit{Shewanella} species produce yellowish-brown mucoid colonies that emit hydrogen sulfide in culture.\textsuperscript{22,23} The most common clinical manifestations of \textit{Shewanella} infections are deep ulcers associated with hemorrhagic bullae usually on the lower extremities, otitis externa, otitis media, and bacteremia.\textsuperscript{22,23} Nonhealing ulcers have resulted in necrotizing fasciitis, compartment syndromes requiring decompressive fasciotomies, and osteomyelitis.\textsuperscript{22,23} \textit{Shewanella} septicemia has been associated with endocarditis and meningitis.\textsuperscript{22,23} \textit{Shewanella} pneumonia, cholestatis, and peritonitis have been reported following aspiration or ingestion of seawater.\textsuperscript{22} Besides seawater exposure and ingestion of raw seafood, other common risk factors for \textit{Shewanella} infections have included lacerations in marine environments, preexisting lower extremity wounds or ulcers, and immunocompromise.\textsuperscript{22}

Wagner and colleagues reported a case of \textit{Shewanella algae}-infected chronic leg ulcers in a 52-year-old female with autoimmune vasculitis and myasthenia gravis 7 months after returning from a Mediterranean vacation during which she reported frequent bathing in the sea.\textsuperscript{23} Poovorawan and colleagues reported another case of severe \textit{Shewanella baliensis} soft tissue infection in the left lower leg with compartment syndrome in a 52-year-old female who had undergone orthotopic liver transplantation 6 months previously and reported handling fresh saltwater fish in fish markets.\textsuperscript{22}

The diagnosis of \textit{Shewanella} infections can be established by positive blood or lesion aspirate cultures, but the speciation of \textit{Shewanella} causative strains will require molecular characterization by polymerase chain reaction (PCR).\textsuperscript{22,23} \textit{Shewanella algae} is resistant to penicillins and first- and second-generation cephalosporins.\textsuperscript{22,23} Most species are sensitive to a broad range of antibiotics including aminoglycosides, third-generation cephalosporins, and fluoroquinolones.\textsuperscript{22,23} For invasive infections, especially in immunosuppressed patients, most authorities recommend 2 weeks of intravenous antibiotic therapy with third-generation cephalosporins combined with either aminoglycosides or quinolones followed by 2 to 4 weeks of oral antibiotic therapy.\textsuperscript{22}

\textbf{Vibrio vulnificus}

\textit{Vibrio vulnificus} has emerged as a highly virulent bacterial pathogen that can cause three types of infections: (1) acute gastroenteritis from eating raw or undercooked shellfish; (2) invasive septicemia following ingestion of raw or undercooked shellfish, especially oysters; and (3) necrotizing wound infections following marine injuries and exposures.\textsuperscript{24,25} \textit{Vibrio vulnificus} is a halophilic, Gram-negative, curved rod-shaped
bacterium that prefers temperatures above 18°C, and is free living in marine environments with low to moderate salinities. Increasing seasonal temperatures and decreasing coastal salinity levels appear to have favored a greater concentration of Vibrio bacilli by the filter-feeding shellfish of the US Atlantic seaboard and the Gulf of Mexico, especially oysters (Crassostrea virginica).26

Vibrio vulnificus can be detected in the gut of oysters and other shellfish and in the intestines of fish that inhabit oyster reefs.26,27 Septicemia comprises 58% of V. vulnificus infections, but causes more than 80% of the fatalities.25,28,29 Nearly all patients with V. vulnificus septicemia consumed raw oysters (96%) and 61% died, mostly men.25,28,29 Necrotizing wound infections comprise another third of the cases of V. vulnificus infection, but cause only 8% of the deaths.25,28,29

Men are uniquely predisposed to V. vulnificus infections for several reasons including occupational and recreational exposures to fish and shellfish, higher serum iron levels, increased rates of alcoholism and liver disease, and lower levels of protective estrogens.30 Other predisposing, non-gender-related host risk factors for V. vulnificus infections include all hematological conditions that increase serum iron levels (hemochromatosis and thalassemia major); chronic liver disease (cirrhosis, hepatitis, and hepatoma) or liver transplant; diabetes mellitus; end-stage renal disease; and immune suppression by steroid therapy, cancer chemotherapy, splenectomy, and acquired immunodeficiency syndrome (AIDS).25,30

Necrotizing skin infections or septicemia following marine injuries, or ingestion of, or exposure to, raw seafood or seawater, especially during the warmer spring and summer months, should prompt suspicion of V. vulnificus infections.31 Gram stains on aspirates from bularious lesions or discharges from necrotic ulcers may demonstrate the characteristic Gram-negative rods.32 Blood cultures will also be positive for V. vulnificus in 30% of wound infections with secondary septicemia and in 70% to 100% of cases of primary invasive septicemia.32 Antibiotic therapy should be instituted immediately as delays in the initiation of antibiotic therapy for 24 hours have been associated with a 33% CFR and delays greater than 72 hours with a 100% CFR.33 The US Centers for Disease Control and Prevention (CDC) has recommended a third-generation cephalosporin, specifically cefazidime, plus doxycycline, as initial empiric antibiotic combinations for suspected V. vulnificus infections.34 In pediatric cases where doxycycline and quinolones are contraindicated and in cases of antibiotic sensitivities, aminoglycosides and trimethoprim–sulfamethoxazole may be substituted for cefazidime and doxycycline.34 Early surgical consultation for wound debridement and monitoring for compartment syndromes are also indicated as early surgical management of V. vulnificus wounds has also been demonstrated to decrease high mortality rates.34,35

SSTIs Following Seafood Ingestion, Seafood Preparation and Handling, and Other Marine Exposures

Erysipelothrix rhusiopathiae

Erysipelothrix rhusiopathiae is a Gram-positive, non-sporulating rod frequently misidentified microscopically as other nonsporulating rods, such as Lactobacillus species and Listeria monocytogenes.36 Erysipelothrix rhusiopathiae is alpha-hemolytic on blood agar and is frequently misidentified in culture as Streptococcus viridans.36 Microbiological differentiation of E. rhusiopathiae from other Gram-positive bacilli usually requires a positive test for hydrogen sulfide on triple sugar iron agar or DNA detection by PCR.36 In fish, the organism can persist for long periods in the exterior slime without causing cutaneous infections.36

In humans, E. rhusiopathiae infection usually occurs following skin injury while handling or preparing colonized fish with an incubation period of 1 to 2 days.36,37 Most human infections are of two cutaneous forms, localized cutaneous (erysipeloid) and generalized cutaneous; both of which are characterized by painful, throbbing erythematous and intensely pruritic lesions.36,37 Unlike cutaneous infections, invasive E. rhusiopathiae infections are unusual with less than 100 cases reported and characterized by septicemia and subacute bacterial endocarditis (SBE), which can be easily misidentified as S. viridans SBE.36 Erysipelothrix rhusiopathiae SBE more commonly involves the aortic valve and has a higher CFR (40%) than other types of SBE.36 The organism is sensitive to penicillins, first-generation cephalosporins, and carbapenems; but resistant to vancomycin and aminoglycosides.36–38 As vancomycin is often administered empirically for the treatment of presumed SBE, rapid microbiological differentiation of E. rhusiopathiae from other Gram-positive causative organisms of SBE is critical.36

Mycobacterium Species

The aquatic, atypical mycobacteria are the acid-fast pathogenic agents of piscine mycobacteriosis and can cause external and solid organ granulomas in over 150 fish species.39 Aquatic mycobacteria thrive at temperatures of 30°C to 33°C in fresh and saltwater environments, and are chlorine- and iodine-resistant.39 Mycobacterium marinum is the most common cause of external granulomas in fish handlers and aquarium workers.39,40 Mycobacterium marinum infections typically begin as localized areas of red-violet verrucous or crusted plaques at inoculation sites 7 or more days (mean = 21 days) after puncture wounds or minor lacerations in marine environments on the cooler, distal regions of the extremities.39,40 Solitary or multiple granulomatous nodules will develop later in the inoculation site and may ulcerate with a yellowish, purulent discharge.40 In some cases, metastatic nodular lesions can develop in a sporotrichoid manner along the proximally draining lymphatic routes, ulcerate, and become secondarily infected.41
Deeper, invasive infections, including septic arthritis, bursitis, tenosynovitis, and osteoarthritis, may occur in indolent or untreated cases and, less often, in immunocompromised patients. Diagnostic techniques include acid-fast stains and culture of nodule discharges, aspirates, or biopsies, and PCR identification of mycobacterial nucleic acids in confusing cases. In a 2012 case discussion of a seashell collector with an indolent hand infection and initially negative acid-fast stains and cultures, Safdar and colleagues recommended narrowing the differential diagnosis quickly while awaiting cultures by confirming a mycobacterial infection with recent tuberculin skin test conversion, excluding pulmonary tuberculosis by negative chest radiograph, and obtaining a negative interferon-gamma-release assay test, such as the QuantiFERON-TB Gold test, with less cross-reactivity to atypical mycobacteria. In all suspected cases of *M. marinum* infection, clinicians should pursue confirmation by culture and begin combined antibiotic therapy with clarithromycin and ethambutol, the currently recommended two first-line tuberculostatic therapy with penicillins, and trimethoprim–sulfamethoxazole. The isolates were sensitive to a broad range of antibiotics including aminoglycosides, cephalosporins, macrolides, penicillins, and trimethoprim–sulfamethoxazole. The investigators concluded that most patients had been inoculated with *S. iniae* in association with minor injuries received during preparation of fresh fish, especially tilapia, and recommended that precautionary measures should be taken, especially by immunocompromised elderly patients, when handling or filleting whole, uncooked fish to prevent *S. iniae* infections.

### General Management of Marine Injuries

Even minor abrasions and lacerations sustained in marine environments should be considered potentially contaminated with common marine microbes, such as *Vibrio* species. All wounds should be irrigated initially with a sterile diluent solution, if possible, such as normal saline. Crushed or devitalized tissues should be excised by sharp dissection under local anesthesia or peripheral nerve blocks. Foreign bodies should be removed. Diagnostic imaging is often indicated, especially in puncture wounds, to exclude retained foreign bodies. Potential constriction bands, such as bracelets, rings, and watches, should be removed from the injured extremity and baseline extremity circumference measurements taken in the event of swelling from necrotizing fasciitis and compartment syndromes. Sequential surgical debridements will be indicated in many cases, in all cases of necrotizing fasciitis, and following fasciotomies for compartment syndromes. Most wounds should be left open or packed open to heal by secondary intention. Delayed primary closures may be indicated for potentially disfiguring facial wounds. Tetanus prophylaxis is indicated for all marine wounds.

### The Clinical Manifestations of Marine Infections and the General Management of Contaminated Marine Wounds

Although the causative pathogens and outcomes may differ, the initial clinical manifestations of the SSTIs following marine injuries and exposures are no different from those acquired after terrestrial exposures and may be classified as impetigo, erysipelas, cellulitis, and necrotizing infections. Impetigo is initially characterized by bullous lesions and is usually caused by *S. aureus* or *Streptococcus pyogenes*. Any empirically prescribed antimicrobials should be effective against both causative pathogens. *Streptococcus iniae* infections are also characterized by either impetigo or cellulitis. Erysipelas is characterized by fiery red, tender, painful plaques with well-demarcated edges and is usually caused by penicillin-sensitive *S. pyogenes*. Like erysipelas, cellulitis is usually caused by *S. pyogenes*. *Erysipelothrix rhusiopathiae* infections typically cause erysipeloid manifestations. All these superficial infections may be treated empirically with single antibiotics, specifically penicillins or macrolides in cases of penicillin allergy, until culture and antibiotic susceptibility results are reported.

Furuncles, carbuncles, and abscesses are usually caused by *S. aureus*, and should be treated initially with penicillinase-resistant semisynthetic penicillins or first-generation cephalosporins. Marine necrotizing infections may be monomicrobial and caused by
V. vulnificus, Aeromonas hydrophila, E. tarda, or other marine bacteria, but may also be polymicrobial. Both monomicrobial and polymicrobial necrotizing infections may develop rapidly to necrotizing fasciitis and myonecrosis, especially in patients with preexisting chronic wounds, such as venous stasis ulcers, or chronic dermatoses, or in immunocompromised patients on steroid therapy or with liver disease, diabetes mellitus, AIDS, or cancer. These cases should be treated initially with antimicrobials that are effective against Gram-positive aerobic bacteria, Gram-negative bacteria, and anaerobes, especially in cases of penetrating trauma, until confirmed positive cultures from drainage or biopsies are reported. People who have cirrhosis, hepatitis, uremia, hemosiderosis, or other iron-retaining conditions are at higher risks for invasive Vibrio infections with the highest CFRs and should be empirically covered with combinations of third-generation cephalosporins (ceftazidime) and tetracyclines (doxycycline), fluoroquinolones and tetracyclines, or aminoglycosides and trimethoprim–sulfamethoxazole, in pediatric cases where doxycycline and quinolones are contraindicated.

Grossly contaminated or infected wounds and all puncture wounds should be cultured or biopsied immediately, and the microbiology laboratory should be notified in advance that special culture media or biopsies are required. Such as sodium-ascorbate, sodium-pyruvate, or other media. In all suspected cases of M. marinum infection, clinicians will need to pursue confirmation by culture and begin combined antibiotic therapy with clarithromycin and ethambutol. All definitive antibiotic therapy should be based on precise pathogen identification by culture or molecular signature and antibiotic susceptibility testing. With the exception of minor marine wounds demonstrating localized cellulitis or spreading erysipelas-type reactions, most other marine infections and all Gram-negative and mycobacterial marine infections will require therapy with antibiotic combinations.

Conclusions and Recommendations
Travelers with well-established risk factors for increasing severity of marine infections, including those with open wounds, suppressed immune systems, liver disease, hemochromatosis, alcoholism, diabetes mellitus, hematological disease, chronic renal disease, AIDS, and cancer, should be cautioned about the risks of marine infections through exposures to marine animals, seawater, the preparation of live or freshly killed seafood, and the ingestion of seawater or consumption of raw or undercooked seafood, especially oysters. Travel medicine practitioners should maintain a high index of suspicion regarding potentially catastrophic bacterial infections following marine injuries and exposures, especially V. vulnificus in the Gulf of Mexico, C. violaceum in the Western Pacific, and Shewanella infections in the Mediterranean and Western Pacific. Initial antibiotic therapy in cases of unknown bacterial etiologies should be based on the initial clinical manifestations of impetigo, erysipelas, cellulitis, pyodermas, or necrotizing soft tissue infections.

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Declaration of Interests
The author states that he has no conflicts of interest.

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