BRIEF COMMUNICATION

Travel-Related Chronic Hemorrhagic Leg Ulcer Infection by *Shewanella algae*

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*Shewanella algae* is an emerging seawater-associated bacterium. In immunocompromised patients, infections may result in bacteremia, osteomyelitis, and necrotizing fasciitis. Our patient, suffering from autoimmune vasculitis and myasthenia gravis, developed typical hemorrhagic bullae and leg ulcers because of *S algae*. She was treated efficiently with a combination of ciprofloxacin and piperacillin.

*Shewanella algae* is a seawater-associated mesophilic emerging bacterial pathogen.1 Most reported infections occur in countries with warm climates and result from contact of contaminated water with disintegrated skin.2,3 The clinical disease spectrum ranges from skin and soft tissue infections after breaches of the dermis, such as ulcers or following trauma,2,4,5 to septicemia, meningitis, endocarditis, and pericarditis.2,3 An increasing number of infections are described in immunocompromised patients after contact with seawater.3,5 Here, we report a severe *S algae* skin infection after bathing in the Mediterranean Sea in an immunosuppressed patient with underlying vasculitis.

Case Report

A 52-year-old female Croatian immigrant was admitted to our hospital in Germany in June 2011 for deep ulcers with hemorrhagic bullae on both lower limbs (Figure 1), which had developed over the last 3 months. Previously, on an outpatient basis, an immunosuppressive treatment with prednisolone and mycophenolate-mofetil had been increased to 80 and 1,500 mg daily, respectively, as the patient’s past medical history had included an autoimmune vasculitis, sensomotoric polyneuropathy, and myasthenia gravis. However, the ulcers had worsened increasingly despite the intensified iatrogenic immunosuppression.

The skin lesions had appeared approximately 7 months after the patient had returned from a journey to Croatia where she had visited relatives. During her stay in Croatia and the last 2 years no apparent skin lesions had been noticed. Previous cutaneous ulcers due to the vasculitis primarily diagnosed in 2005, which had never been hemorrhagic, had relapsed a few times before, and she had been treated successfully lately with mycophenolate-mofetil and prednisolone. In 2005, approximately 1 month after the initiation of the first immunosuppressive treatment, a pulmonary tuberculosis had developed, which had been treated successfully with tuberculosis medication.

As there was no improvement during 6 weeks of intensified immunosuppression as an outpatient, we further increased the dose of mycophenolate-mofetil up to 2,000 mg daily at the beginning of her hospital stay. At the same time a biopsy taken from the lesion revealed perivascular inflammation, predominated by neutrophil infiltration. A bacteriological swab taken at our hospital on admission showed monomicrobial growth of gram-negative rods with brownish-mucoid appearance in large quantities after incubation on blood agar, chocolate agar, and MacConkey agar.
The organism was catalase and oxidase positive and exhibited weak beta hemolysis. The isolate was identified as *S. algae* by VITEK 2 and susceptible to piperacillin-tazobactam, ceftazidime, cefotaxime, imipenem, ciprofloxacin, and aminoglycosides. In the light of an *S. algae* infection, the patient was further interviewed about seawater exposure. The patient reported that she had bathed frequently in the Mediterranean Sea near Orebiz during her stay in Croatia. Treatment with mycophenolate-mofetil was stopped and the dose of prednisolone was reduced to 20 mg/day. The patient received piperacillin 0.5 g tid and ciprofloxacin 500 mg bid for 20 days, and on X-ray films an intact corticalis and no signs of osteolysis could be detected on both lower limbs. Magnetic resonance imaging could not be performed because of residual metallic structures left in situ after a former bone fracture. During antibiotic therapy the ulcers healed continuously and the immunosuppressive treatment was further reduced to 15 mg prednisolone.

**Discussion**

Chronic cutaneous ulcers of the legs have been identified as the most common risk factor for *S. algae* skin infections. Bacteremia is rare and has been described in patients with leg ulcers and immunosuppression or other underlying medical conditions. Severe cases of osteomyelitis after trauma and contact to stagnant water, and myonecrosis and necrotizing fasciitis leading to amputation after seawater exposure have been reported. In skin infections, hemorrhagic bullae are characteristic, as seen here. Cutaneous infections with *Vibrio vulnificus* and *Aeromonas hydrophila*, other seawater-associated bacterial pathogens, closely resemble the clinical picture. In temperate regions, *S. algae* can be found during the summer months in seawater as well. The organism had often been misidentified as *Shewanella putrefaciens* based on biochemical characteristics and lack of microbial database entries in the past.

It was shown retrospectively that most infections were in fact caused by *S. algae* and 16SrRNA gene polymerase chain reaction amplification following by sequencing for correct identification was performed by several investigators. *Shewanella algae* is considered to be more pathogenic than *S. putrefaciens*. Infections should be treated aggressively with a combination of surgery or drainage and antibiotic therapy; however, there is little clinical experience. *Shewanella algae* is resistant against penicillins and first- and second-generation cephalosporins, and development of resistance to piperacillin-tazobactam and imipenem under therapy has been described. However, naturally occurring derepressed Ambler class D beta-lactamases have been accused for that effect. Most often, *S. algae* is susceptible to ceftazidime, cefotaxime, aminoglycosides (especially amikacin), and quinolones, although *S. algae* has been described to harbor quinolone resistance progenitor genes. In the light of this unusual resistance pattern, third-generation cephalosporins should be best combined with quinolones or aminoglycosides in an antibiotic treatment regimen for *S. algae* infections.

In conclusion, besides *A. hydrophila* and *V. vulnificus*, *S. algae* should be taken into account if a skin and soft tissue infection after marine exposures is evident. Third-generation cephalosporins and ciprofloxacin empirically cover all three seawater-associated pathogens in an antibiotic treatment. As described here, extensive cutaneous ulcers, besides hemorrhagic bullae, can be caused by *S. algae* in immunosuppressed individuals. *Shewanella algae* infections primarily arise from colonization of nonhealing wounds, chronic ulcers, or by penetrating traumas with the microorganisms from environmental sources.

**Declaration of Interests**

The authors state that they have no conflicts of interest.

**References**

7. Nozue H, Hayashi T, Hashimoto Y, et al. Isolation and characterization of *Shewanella algae* from human clinical...

