

## Case Report: Disseminated *Shewanella algae* Infection with Meningoencephalitis in a Traveler Secondary to Marine Injury in Madagascar

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**Abstract.** Marine microorganisms such as *Shewanella* spp., *Vibrio* spp., and *Aeromonas* spp. can cause sepsis secondary to a wound infection in the context of swimming. These microorganisms are most often susceptible to fluoroquinolones. Here, we report a unique case of *Shewanella algae* bacteremia associated with meningoencephalitis and disseminated via hematogenous spread secondary to a skin injury. The patient suffered the injury while swimming in saline water during a cruise holiday in Madagascar, and she was initially treated with amoxicillin. The neurological evolution was unsatisfactory. Better knowledge of such infections (and especially of the context in which they occur), as well as greater familiarity with the susceptibility profile of different marine microorganisms would have allowed health professionals to provide presumptive microbiological diagnosis and effective treatment earlier.

### INTRODUCTION

*Shewanella* spp. are nonfermenting gram-negative bacilli that live in specific marine environments. *Shewanella* spp. were previously classified in the *Pseudomonas* spp. family. *Shewanella* spp. infections are rare, but more and more cases are being reported in the context of swimming while traveling. Most cases affect patients with disabilities and occur in warm climates after contact with saline water.<sup>1</sup> The most frequent infections are skin and soft tissue infections,<sup>2,3</sup> pneumonia,<sup>4</sup> and hepatobiliary infections. To our knowledge, no case of disseminated *Shewanella algae* infection with meningoencephalitis has been described in the literature to date.

### CASE SUMMARY

An 83-year-old woman with no past medical history (without previously known liver disease) presented with a skin and soft tissue infection of the lower left leg, which had developed 5 days earlier. This infection was secondary to a minor injury of the hallux she had suffered while swimming in saline water during a cruise in Madagascar. The patient presented with nausea, vomiting, dehydration, and consciousness disorder. She was infused on the cruise ship and treated orally with amoxicillin. When she arrived on Reunion Island, she was brought by her family to the emergency department of a peripheral hospital, and was then quickly transferred for severe sepsis to the intensive care unit of our university hospital. On arrival, she had no fever and no hemodynamic disorder. Neurological examination reported a meningitis syndrome, a nystagmus, and a Glasgow scale score of 7, prompting orotracheal intubation. A necrotizing fasciitis of the left leg was observed, its portal of entry being a small, healing wound of the left hallux. Blood tests showed lymphopaenia (3.5 G/L), thrombopaenia (95 G/L), and an inflammatory syndrome with a C-reactive protein of 329 mg/L and a procalcitonin of 5.83 ng/mL. Cerebral computed tomography was normal, and thoracic computed tomography revealed moderate bilateral pleural effusions. Antibiotherapy with high doses of

cefotaxime and amoxicillin was immediately initiated. Amoxicillin/clavulanic acid was added to treat the skin infection. A lumbar puncture showed cerebrospinal fluid (CSF), which was turbid and under pressure with a glucose concentration < 0.1 mmol/L, a protein concentration of 6.2 g/L, a lactate concentration of 15 mmol/L, and 17 leukocytes/mm<sup>3</sup> (80% of which were neutrophil polynuclears). Direct examination revealed the presence of a mobile bacillus. A gram-negative bacillus was identified on Gram staining as well as on an aerobic blood culture (BACTEC™ FX system, Marcy-l'Etoile, Rhône, France) that became positive after 20.6 hours. Pleural effusions were drained the following day. The drained fluid was purulent with a protein concentration of 18 g/L and an lactate dehydrogenase of 3,636 UI/L. Formula analysis was impossible. Surgical exploration of the left leg revealed extensive skin and soft tissue edema without abscess as well as healthy muscles. Extensive debridement and fasciotomy were performed. Tissue cultures remained negative. CSF culture, blood cultures, and pleural fluid cultures on a chocolate agar plate + PolyviteX™ (BioMérieux, Marcy-l'Etoile, Rhône, France) macroscopically revealed the presence of small, yellow-brown, bright, and smelly colonies atypical of gram-negative bacilli. Matrix-assisted laser desorption ionization-time of flight mass spectrometry (MALDI-TOF) mass spectrometry (biotyper®, Bruker) showed *Shewanella* spp., with an identification score of 2.36. The two blood cultures sampled before antibiotic administration also tested positive for this bacteria. Finally, *S. algae* was identified by 16S ribosomal RNA gene sequence analysis.

An antibiogram was performed on a Mueller–Hinton agar plate with a 0.5 McFarland inoculum, incubated at 35°C in normal atmosphere, and interpreted according to European Committee on Antimicrobial Susceptibility Testing guidelines (2016 v1) for *Pseudomonas* spp. The strain was susceptible in vitro to piperacillin/tazobactam, all third- and fourth-generation cephalosporins (minimal inhibitory concentrations measured by E-test were 0.5 mg/L for cefotaxim and 0.064 mg/L for cefepime), amikacin, gentamicin, ciprofloxacin, and cotrimoxazole. The strain was resistant to amoxicillin, amoxicillin/clavulanic acid, ticarcillin, piperacillin, ticarcillin/clavulanic acid, aztreonam, and meropenem. After bacterial identification, antibiotherapy was modified with the introduction of cefepime (6 g/day) and ciprofloxacin (400 mg/8 hour).

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The evolution was marked by septic shock requiring hemodynamic support with noradrenaline as well as by renal failure necessitating 2 days of continuous hemofiltration. Control lumbar punctures were performed on days 5 and 8 of hospitalization. They showed, respectively, protein concentrations of 5 and 3.4 g/L, glucose concentrations of 0.4 and 1.2 mmol/L, and lactate concentrations of 15.7 and 12.7 mmol/L. CSF formulas contained, respectively, 3,100 leukocytes/mm<sup>3</sup> (60% of which were neutrophil polynuclears) and 420 leukocytes/mm<sup>3</sup> (85% of which were neutrophil polynuclears). All control blood cultures sampled after effective treatment came back negative. The neurological evolution was initially unfavorable: examination showed spontaneous eye opening and disoriented response to nociception. A cerebral magnetic resonance imaging performed on day 5 showed meningoencephalitis with ventriculitis that was treated with corticosteroids; it also revealed multifocal ischemic lesions of the right precentral and post-central cortex related to an infectious vasculitis. The neurological condition eventually improved. The patient was extubated on day 30 and transferred to a medical unit on day 36. On neurological examination, she presented with complete left hemiplegia, but also with preserved consciousness and orientation alternating with periods of drowsiness.

## DISCUSSION

Our patient is the only reported case of disseminated community-acquired *S. algae* infection with meningoencephalitis. The literature mentions only one case of nosocomial meningitis caused by *Shewanella putrefaciens*, which was secondary to a cerebral hemorrhage treated by neurosurgery.<sup>5</sup> Two cases of cerebral abscess secondary to purulent otitis<sup>6,7</sup> have also been described (including a *Klebsiella pneumoniae* coinfection). However, other instances of community-acquired *Shewanella* spp. infection with meningoencephalitis may have gone unreported, as this microorganism was previously classified as *Pseudomonas putrefaciens*, *Alteromonas putrefaciens*, and *Achromobacter putrefaciens*. Moreover, *Shewanella* species are difficult to differentiate from other bacteria through conventional approaches (MALDI-TOF mass spectrometry).<sup>8</sup> In our study, *S. algae* was finally confirmed by 16S RNA gene sequence analysis.

Based on the initial description of the skin and soft tissue infection, and given that *Shewanella* spp. strains often enter the skin in the context of swimming<sup>9</sup> we can confidently state that the portal of entry was cutaneous. We can also assume that hematogenous spread was responsible for the meningoencephalitis and bilateral pleural effusions. Antibiotherapy was adapted to the susceptibility profile of the bacteria described in the literature (resistance to penicillins, carbapenems, piperacillin/tazobactam, as well as susceptibility to third- and fourth-generation cephalosporins, ciprofloxacin, and aminoglycosides).<sup>9</sup> Specifically, we decided on a combination therapy of cefepim (due to its lower minimum inhibitory concentration) and ciprofloxacin (due to its good diffusion in CSF). The microbiological evolution was favorable under effective treatment, and all microbiological analyses (blood cultures and CSF) came back negative. However, the neurological evolution was unsatisfactory because of the delay in treating this case of sepsis with meningoencephalitis<sup>10</sup> caused by amoxicillin-resistant bacteria.

Infections caused by aquatic bacteria (*Shewanella* spp., *Vibrio* spp., *Aeromonas* spp., etc.) are potentially severe and

relatively frequent in warm climates. Studies have shown that fluoroquinolone susceptibility is almost constant across aquatic bacteria, whereas beta-lactam susceptibility varies widely according to the isolated bacteria.<sup>11</sup>

## CONCLUSION

Severe infections acquired while swimming are rare and mainly associated with a small number of microorganisms (*Shewanella* spp., *Vibrio* spp., *Aeromonas* spp., etc.). This unique case of severe disseminated *S. algae* infection associated with meningoencephalitis illustrates the importance of clinical history for accurate microbiological diagnosis. It also shows that knowledge of the antibiotic susceptibility profile of aquatic bacteria is essential for proper therapeutic management.

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