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# Case Report: A Case of Sepsis due to *Shewanella algae* Infection in the Colombian Caribbean

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*Abstract.* *Shewanella algae* is an opportunistic Gram-negative bacillus that inhabits marine ecosystems and can cause sepsis in humans. This case report describes an 80-year-old obese woman with liver cirrhosis who presented with neurological and respiratory impairment. *Shewanella algae* were isolated in the blood cultures. Due to age and comorbidities, sepsis could be the cause of the patient's fatal outcome. *Shewanella algae* infection is a risk for immunocompromised people in the tropics.

## INTRODUCTION

*Shewanella algae* is a significant negative, nonfermenting, motile bacillus classified as *Achromobacter putrefaciens* in the 1930s and reclassified as *Pseudomonas putrefaciens* in the 1940s.[1] Subsequently, phylogenetic analysis of the bacterium formed a new family called Shewanellaceae, which has ~50 species, including *S. algae*.[1] The microorganism is an opportunistic pathogen in humans with chronic skin lesions. It has been described in immunosuppressed individuals exposed to marine ecosystems and freshwater habitats, and its presence has been reported in pig farms in Brazil.[2] Furthermore, this germ can cause clinically relevant bacteremia.[3,4]

To date, cases of sepsis due to *S. algae* have been reported in different parts of the world. However, there are no reports of infection with *S. algae* in the Colombian Caribbean. Because Shewanella is a microorganism unknown to the medical community, its clinical role goes largely unnoticed. It is also challenging to identify Shewanella with commercial systems such as API 20E, API 20 NE, and Vitek™5 (Biomerieux, Canada). Even using sophisticated equipment such as matrix-assisted laser desorption/ionization time-of-flight mass spectrometry, it is difficult to identify Shewanella.[5] This work describes *S. algae* as a pathogen of interest in the tropics.

## DESCRIPTION OF THE CASE

In October 2022, an 80-year-old woman was admitted to the emergency department of a private clinic. The patient lived in Sincelejo, Sucre, an area of the Colombian Caribbean, ~40 km from the Caribbean Sea. From the patient's account, it was not possible to establish recent trips to the sea or ingestion of seafood.

The woman was obese with a history of liver cirrhosis. She had clinical manifestations such as asthenia, adynamic dyspnea associated with irregular respiratory patterns, and use of accessory muscles. Desaturation (pulse oximetry 92%) was observed. Mild jaundice, jugular venous distention, subcostal interactions, abundant adipose tissue, grade I edema, and an altered state of consciousness were found. At the beginning of her hospitalization, two presumptive diagnoses were made. The first was sepsis of pulmonary origin, and the other was cerebrovascular disease. The initial paraclinical tests of the patient showed thrombocytopenia and leukopenia. Laboratory tests were compatible with the clinical sepsis presentation (Table 1). Increased blood nitrogen, hypoglycemia, and acute phase reactants were seen. The patient also showed hyperbilirubinemia due to increased direct bilirubin (Table 1). The electrocardiogram showed atrial fibrillation with moderate ventricular response. COVID-19 test was negative.

During hospitalization, the patient presented a hypertensive emergency with brain, heart, and lung target organs. Therefore, she required vasodilator support to attend to the emergency and high-flow oxygen with a positive response to therapy. After 6 hours, her neurological condition deteriorated. She developed a fever, for which intravenous (IV) empirical antibiotics were started (ampicillin/sulbactam and clarithromycin). The treatment was established according to autonomous management guidelines of the private clinic in Sincelejo, Sucre, where the patient was admitted. Serial blood and urine cultures were performed before the administration of empirical antibiotic therapy, and growth at 24 hours in the blood cultures was observed (Figure 1). The identification with the Vitek™ compact system (Biomerieux) reported *S. algae*, and no other microorganisms were isolated. The organism was susceptible to cefepime ($\leq$ 0.12 $\mu$g/mL), gentamicin ($\leq$ 0.1 $\mu$g/mL), meropenen (1 $\mu$g/mL), imipenen (4 $\mu$g/mL), ciprofloxacin (0.5 $\mu$g/mL), amikacin (4 $\mu$g/mL), ceftazidime (0.5 $\mu$g/mL) and resistant to cefazolin ($\geq$ 64 $\mu$g/mL). Ampicillin/sulbactam was replaced by cefepime IV because it had a better minimum inhibitory concentration.

Forty-eight hours later, the patient presented metabolic acidemia, altered tissue perfusion indices, refractory shock, and multiorgan dysfunction. Therefore, she required mechanical ventilation, vasopressor therapy, digoxin, phytomenadione, and dextrose. The patient persisted with a torpid clinical evolution and died 60 hours after admission.

## DISCUSSION

*Shewanella algae* is an opportunistic bacterium that can cause severe infections with a high mortality rate, particularly in immunosuppressed patients with chronic skin lesions of the lower limbs or underlying liver disease, as is the case in

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TABLE 1
Laboratory tests of the patient

| Laboratory tests | Day 1—admission | Day 2 | Day 3 | Day 4 |
|---|---|---|---|---|
| Hemoglobin (g/L) | 10.6 | 11.5 | 10.2 | 8.4 |
| Hematocrit (%) | 31% | 34.9% | 30.1% | 26.1 |
| White blood cells ($mm^3$) | 2.63 | 1.46 | 5.64 | 9.34 |
| Neutrophil (%) | 63% | 70.6 | 81 | 80 |
| Platelet ($mm^3$) | 35.000 | 30.000 | 20.000 | 12.000 |
| Prothrombin time (seconds) | 19.4 | 19 | 21 | 45 |
| Thrombin time (seconds) | 51.40 | 51.7 | 57 | 87 |
| Total bilirubin (mg/dL) | 2.97 | 4.44 | 5.52 | 5.16 |
| Direct bilirubin (mg/dL) | 1.34 | 1.99 | 2.58 | 2.11 |
| Indirect bilirubin (mg/dL) | 1.63 | 2.45 | 2.94 | 3.05 |
| Procalcitonin (ng/mL) | – | 9.51 | – | – |
| Creatinine (mg/dL) | 2.80 | 3.1 | 3.0 | 3.54 |
| Urea (mg/dL) | 88.14 | 100 | 102 | 92 |
| Blood urea nitrogen (mg/dL) | 41.43 | 47 | 47 | 43 |
| Aspartate aminotransferase (U/L) | 33.90 | 37.3 | 69.4 | 6413.4 |
| Alanine aminotransferase (U/L) | 18.12 | 19.09 | 27 | 1207.0 |
| Acid lactic (mmol/L) | – | – | 16 | 20.3 |
| Semiquantitative CRP (mg/L) | 96 | | | |

CRP = C-reactive protein.

the patient in this study study.[6,7] *Shewanella algae* is more frequent in tropical environments near marine coasts.

This case could be associated with the hematogenous spread of *S. algae* to the central nervous system and the lungs. Those findings are similar to those of Yu et al.,[5] who described that *S. algae* is mainly found in ear, nose, throat, central nervous system, and cardiopulmonary infections. However, reports of pneumonia and neuroinfection by *S. algae* are scarce in the literature and demonstrate the bacterium's high pathogenic and dissemination capacity.[8,9]

It is essential to note that climate change may be associated with increased cases of *S. algae* infection. The increase in temperature of the marine ecosystem could promote the growth, pathogenic potential, and expansion of this microorganism to subtropical areas.[3,10] There are no guidelines or consensus for the antimicrobial treatment of *S. algae*. In general, the treatment of *S. algae* is carried out based on results obtained in the antibiogram. Thus, it is necessary to generate management guidelines for these opportunistic microorganisms, particularly in immunocompromised individuals in tropical areas.[5]

*Shewanella algae* is a pathogen that should be considered in travel medicine and by health personnel, especially those who work in areas near an ocean. *Shewanella algae* should be considered in immunosuppressed individuals with a history of hepatobiliary disease with sepsis of unknown origin and in tourists with underlying diseases returning from tropical marine ecosystems.

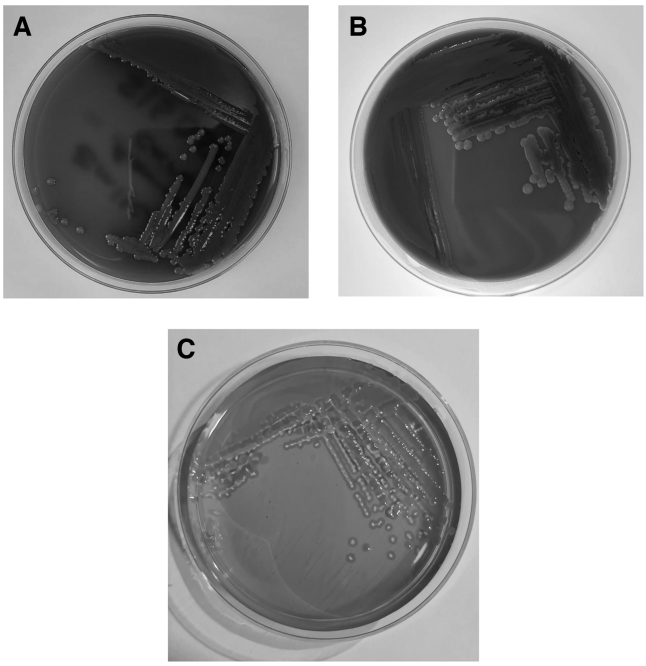


FIGURE 1. Growth of *Shewanella algae* on blood agar (**A**), chocolate (**B**), and McConkey (**C**). This figure appears in color at www.ajtmh.org.

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