**ABSTRACT**

A 66-year-old man with a history of apical variant hypertrophic cardiomyopathy, heart failure with preserved ejection fraction (HFrEF), severe pulmonary hypertension, and prior Group B streptococcal mitral valve endocarditis four months before, presented with generalized body shakes and urinary incontinence. Computed tomography angiography revealed an acute left M1 occlusion. The patient underwent mechanical thrombectomy. Within 24 hours of presentation, he developed hypotension, tachycardia, and fever. Infectious workup revealed a leukocytosis. One out of two sets of blood cultures revealed bacteremia with *Shewanella algae*. A transthoracic echocardiogram revealed a large mitral valve vegetation with multiple mobile components portending a high thromboembolic risk, as evidenced by his acute presentation with multiple embolic infarcts. He was diagnosed with infectious endocarditis caused by *Shewanella algae*, a rare marine environment pathogen. He was treated with ciprofloxacin 750 mg twice daily orally and meropenem 2 g every eight hours intravenously with an initial decrease in the mitral valve vegetation size. He was discharged on ceftriaxone 2g and ciprofloxacin 750mg every 12 hours for a total of six weeks from his first negative blood cultures. He was monitored through transthoracic echocardiography as he continued medical management with levofloxacin 750 mg daily. Six months after his discharge from the hospital, he developed worsening heart failure and elected to pursue comfort measures only.

**KEYWORDS:** *Shewanella algae*; Infective Endocarditis, Marine plankton Endocarditis

**INTRODUCTION**

*Shewanella* spp. are hydrogen sulfide-producing, gram-negative, oxidase and catalase-positive, bacilli found in marine environments.¹ ² *Shewanella* has been isolated in foods, sewage, fresh water, and salt water.³ The most common etiology is infection of skin or soft tissue following trauma that leads to a breach in the integrity of the skin.² It has been found in body wounds, feces, conjunctiva, urine, cerebrospinal fluid, bile, ascitic fluid, pleural fluid, and stored blood.³ Major risk factor associated to these organisms are hepatobiliary disease, peripheral vascular disease with chronic leg ulcers, and poor hygiene.³

Infective endocarditis (IE) is an infection of the native valves, prosthetic valves, or endocardium.⁴ IE mortality estimates are as high as 25%.⁴ Common pathogens causing IE include *Staphylococci* and *Streptococci*. Only a few cases of *Shewanella* infective endocarditis have been reported with treatment options not clearly delineated.

**CASE REPORT**

A 66-year-old man suddenly developed generalized body shakes and urinary incontinence. He subsequently collapsed with sudden onset facial and body weakness that quickly progressed into seizure-like activity. This event was witnessed by the patient’s family who called emergency medical services. The patient’s past medical history included apical variant hypertrophic cardiomyopathy, heart failure with preserved ejection fraction, severe pulmonary hypertension, Group B streptococcal mitral valve endocarditis, hypertension, chronic obstructive pulmonary disease (COPD), and diabetes mellitus type 2. Notably, he had a recent admission to an outside hospital for Group B streptococcal mitral valve endocarditis four months before. Transthoracic echocardiogram (TTE) at that time showed a possible calcified vegetation on the posterior leaflet of the mitral valve with mild mitral regurgitation. At that time, he was treated with ceftriaxone 2 grams intravenously for a total of six weeks for presumed subacute endocarditis. He was discharged with outpatient follow-up and had a repeat TTE that showed no significant change. He also subsequently had an admission to an outside hospital six weeks prior to this presentation for left, lower extremity cellulitis after a clamming accident in which he sustained a puncture wound to his calf; he was initially treated with ceftriaxone 2 grams daily and vancomycin per infectious disease guidance and later transitioned to doxycycline 100mg and augmentin 875–125mg twice daily for seven days. Review of his outpatient wound care follow-up appointments showed improvement of his lower extremity injuries. On physical examination, vital signs revealed a heart rate of 73 beats/minute, blood pressure of 120/50, a temperature of 97.5°F and oxygen saturation of 99%. Cardiopulmonary examination revealed a regular rate and rhythm with a loud systolic murmur consistent with...
mitral regurgitation. On neurological examination, he was unable to follow commands, was globally mute, exhibited a right-sided facial droop, and had complete motor weakness of the right extremities. He had 1-plus pitting edema to his lower extremities; his left lower leg was dry with granulation tissue; compared to prior media images his left lower extremity had significantly improved. History was obtained through family as detailed information from him was limited by his complete aphasia. Laboratory evaluation was only remarkable for hs-troponin I of 1991 (3–57 ng/L). A complete blood count (CBC) showed white blood cells (WBCs) of 9.0 and a basic metabolic panel showed creatinine of 1.18mg/dL. Electrocardiogram revealed sinus rhythm with T-wave inversions in the lateral leads I, aVL, V5, and V6. Computed tomography angiography for emergent large vessel occlusions (CTA ELVO) revealed an acute left M1 occlusion. An electroencephalogram (EEG) was performed and did not reveal epileptiform activity. He was taken for thrombectomy and was subsequently managed for acute stroke in the neurology intensive care unit. Within 24 hours of presentation, he developed hypotension. A CBC revealed a leukocytosis to 18.7. Sepsis was suspected and blood cultures were obtained. He was initially treated with vancomycin and meropenem. One out of two sets of blood cultures revealed bacteremia with Shewanella algae, pan-susceptible. Transthoracic echocardiogram demonstrated a large vegetation [25mm x 23mm] on the posterior mitral leaflet with mobile components, mild mitral insufficiency, patent
foramen ovale, and an EF of 56% [Figures 1,2]. He was diagnosed with infective endocarditis. He met Duke’s major criteria of echocardiographic findings of endocarditis and minor criteria of positive blood cultures with *Shewanella*, had a predisposing heart condition of previously infected heart valve, with fevers, and embolization. As his aphasia improved, he was able to provide a more detailed history; he recalled injuring himself with a clamping rake while clamming in a coastal marine environment a few weeks prior which, he stated, caused a wound in his calf. Based on susceptibility analysis for antibiotic therapy, he was transitioned to meropenem 2g every eight hours intravenously and oral ciprofloxacin 750 mg twice daily orally for 12 days. With antibiotic treatment for two weeks, the mitral valve vegetation decreased in size (14mm x 11mm) [Figures 3,4].

On hospital day 19, the patient had clinically improved. He was discharged home with a minimum six-week antibiotic treatment of ceftriaxone 2g and ciprofloxacin 750mg every 12 hours from his first negative blood cultures with outpatient follow-up and plans for surgery of the mitral valve as outpatient. After six weeks of treatment, a transthoracic echocardiogram was performed which was unable to visualize any mitral valve vegetations but did reveal a nonspecific thickening of the mitral valve, mitral annular calcification, and moderate regurgitation. He had a follow-up visit with infectious disease and was transitioned to levofloxacin until definitive surgical intervention. That same week, he had his follow-up with cardiothoracic surgery, but his multiple comorbidities made him a high-risk surgical candidate. After risk and benefit discussions, the patient determined he did not want to pursue aggressive surgical interventions. A month later, he had a repeat follow-up transthoracic echocardiogram which again revealed severe mitral annular calcifications, but this time also showed a small linear, mobile vegetation. Within six months of his presentation, the patient follow-up and plans for surgery of the mitral valve as outpatient. Unfortunately, over the next several weeks he developed severe fatigue, worsening shortness of breath, progressive orthopnea, and lower extremity edema. A repeat TTE showed moderate to severe mitral regurgitation. Within six months of his presentation, he succumbed to his infection secondary to progressive heart failure. No follow-up echocardiogram was performed prior to his death as he had decided to pursue comfort measures only.

**DISCUSSION**

*Shewanella algae* is an emerging pathogenic marine plankton that has rarely been found in the clinical setting. Only a few cases of *Shewanella* infections have been reported and *Shewanella IE* is extremely rare. In clinical cases, *S. algae* and *Shewanella putrefaciens* are the most commonly isolated *Shewanella* organisms. *Shewanella algae* accounts for approximately 80 percent of infections. In mice studies, it has been suggested that *S. algae* has the highest pathogenicity among *Shewanella* species. *Shewanella algae*’s virulence factors potentially include its hemolytic activity. *Shewanella spp* can form biofilms. Most cases have been reported in countries with warm or temperate climates. *Shewanella species* are found in marine environments and exposure to bodies of water is a likely source for patients with *Shewanella infections*.

Prior literature suggests that 53–80% of patients had mucosal or skin portal of entry and 44% had been exposed to marine environments. As a clammer, the patient spent his time in marine environments. A breach in his skin integrity could have allowed *Shewanella algae* entry. We presume the injury our patient sustained with the clamping rake in a saltwater habitat prior to presentation was the origin of his infection, as *Shewanella species* are present in saltwater.

Given the rarity of this organism, treatment options for severe infections are not well elucidated. Early surgery is considered in patients with severe valvular pathology, heart block, annular abscess, signs of heart failure, fungal or highly resistant organisms, and in recurring emboli or enlarging vegetations, or a size of >10mm vegetations. The patient did meet these criteria, but his comorbidities and his acute stroke presentation made him a poor surgical candidate. Medical management was pursued based on prior literature on *Shewanella* and susceptibility profiles. *Shewanella spp.* are susceptible to cefotaxime, piperacillin and tazobactam, gentamicin, and ciprofloxacin. They are also typically susceptible to carbapenems, erythromycins, and quinolones. *Shewanella spp* can have resistance to imipenem due to their secretion of oxacillinase. Ampicillin and cephalosporin susceptibility is variable, third- and fourth-generation cephalosporins have been used for treatment. For *S. algae* there is resistance to colistin. In one study, 87% of the patients recovered from a general infection of *Shewanella* species, 28% developed bacteremia, and 13% died.

Endocarditis associated with this organism is extremely rare with only a few cases of *Shewanella* species reported. In two reported cases of *Shewanella putrefaciens* endocarditis, one patient was treated with gentamicin and penicillin, resulting in an uncomplicated hospital course, and the other patient was treated with cefepime and gentamicin but required valve surgery. Only one case has previously been reported on *Shewanella algae* endocarditis. That patient was treated with high-dose ceftriaxone and oral ciprofloxacin. He improved medically by week two, but by week five of treatment he developed new-onset heart failure. He died within two weeks following discharge to a palliative care unit due to progression of his infection.

Similar to prior cases, this case of *Shewanella endocarditis* also affected the mitral valve. This case is unique as it is the only *Shewanella algae* endocarditis to affect a native valve and the second reported case worldwide of *Shewanella algae* endocarditis.
CONCLUSIONS
IE can have an insidious onset only to manifest suddenly with severe findings late in the disease course as shown through this case. This case also highlights the importance of a heightened clinical index of suspicion and detailed history are necessary to make the correct diagnosis.

References

Authors
Jessica M. Gonzalez, MD, Warren Alpert Medical School of Brown University, Rhode Island Hospital, Department of Medicine, Providence, RI.
Anshul Parulkar, MD, Warren Alpert Medical School of Brown University, Rhode Island Hospital, Department of Cardiology, Providence, RI.
Gabriel Lowenhaar, MD, Warren Alpert Medical School of Brown University, Rhode Island Hospital, Department of Emergency Medicine, Providence, RI.
Tasnim F. Imran, MD, MPH, Warren Alpert Medical School of Brown University, Rhode Island Hospital, Department of Cardiology, Providence, RI; Providence VA Medical Center, Lifespan Cardiovascular Institute.

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Correspondence
Jessica M Gonzalez, MD
Rhode Island Hospital, Department of Medicine
595 Eddy Street
Providence, RI 02903
jlowenhaar@gmail.com