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Shewanella Algae Soft Tissue Infection in a Diabetic Patient

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Abstract

Shewanella algae infections are rare in humans. Previously approximately 65 cases of *S. algae* causing human infection have been reported and most of them have association with direct contact with sea water. We report a case of primary *S. algae* soft tissue infection in a patient with diabetic nephropathy after prolonged stay in hospital, without prior exposure to sea water/sea food.

Keywords: *Shewanella*; Soft tissue infection; Diabetes; Nephropathy; India

Introduction

Shewanella algae and *Shewanella putrefaciens* are associated with human infections that include bacteremia [1-5], cellulitis (skin and soft tissue infection) [1,2,4-9], ear infection [10], cerebellar abscess [11], wound infection [12], osteomyelitis [10] and endocarditis [13,14]. Skin and soft tissue infections caused by *Shewanella* spp. are commonly associated with chronic ulcers or infected burns of the lower extremities [1,2]. The typical predisposing factors for infection with *S. algae* or *S. putrefaciens* are exposure to a marine environment with a skin lesion or trauma [2,4,6-7,11,12,15]; other factors include the presence of a severe underlying debility, liver disease, or malignancy; prematurity and a compromised immune system [1,7,9,14]. Most infections with *Shewanella* spp. have been community-acquired, hospital-associated infections have been very uncommon [16].

We report the first case of Shewanella algae acquired causing soft tissue ulcer in a patient with diabetic nephropathy and liver involvement from India.

Case Report

A 71 year old man was admitted in Liver ICU with chief complaints of altered sensorium for 10 days, fever for 7 days. Patient was a known smoker for the last 30 years, and had diabetes (type II) and hypertension for the last 10 years and was on insulin for last 6 years. On examination, he was afebrile, had pallor, pedal edema and mild hepatomegaly. After detailed investigations patient was found to have non alcoholic fatty liver disease with diabetic nephropathy. Hyperglycemia with hypokalemia with mild increase in liver enzymes was present. Patient was started on cefoperazone-sulbactam. After two days patient was afebrile with altered sensorium so was bed ridden. He developed scrotal cellulitis and bed sore on his lower back. Blood culture showed growth of *Acinetobacter spp*. sensitive only to colistin, urine culture showed growth of yeast cells. Patient was started on colistin 1 million units 8 hourly IV. After 3 days general condition of the patient was not improving. Patient was still critically ill, with high fever, and developed bilateral chest infiltrate. His haemoglobin was 8 mg/dl, TLC 21,800/mm3, Platelet -44×1000. Patient was started with colistin, amphotericin B and vancomycin was added. A day later patient went into acute renal failure, friction ulcers developed along with increased inflammation of scrotum and chest infiltrates. Culture was sent to microbiology laboratory from infected bed sore which was considered a possible cause of sepsis in patient. His TLC was 13,400/mm^3 and haemoglobin 8.7 mg/dl. Repeated wound cultures showed growth of non fermenting gram negative bacilli which on further phenotypic and biochemical identification was identified as *Shewanella algae*. Day later patient died with cardiac arrest. No history of exposure to sea water/sea food was found. And no other predisposing factors were present.

Discussion

Out of more than 30 known *Shewanella species*, only *S. putrefaciens* and *S. algae* are considered pathogenic for humans [15]. Skin and soft tissue infections have been reported mostly in patients with exposure to sea water. *Shewanella* as an emerging pathogen in hospitals [17] has recently become a matter of concern, after an outbreak by this organism in a tertiary care center in Korea. To the best of our knowledge this is the first case of infected ulcer due to S. algae in a patient with diabetic nephropathy and decompensated liver disease, in which no predisposing factor as exposure to sea water/food was present. In recent past, a patient with decompensated liver disease presented with septicemia due to *Shewanella* [18], so our patient had a quite high risk of sepsis by this organism, but as the patient died his blood culture could not be repeated. There are no standard guidelines for treatment of Shewanella infection. Previous published studies reported that *S. algae* are susceptible to aminoglycosides, carbapenems, erythromycin, and quinolones, with variable susceptibility to penicillin and cephalosporins. However, rapid development of resistance to imipenem and piperacillin-tazobactam has been reported [15]. Thus the treatment options available are β-lactams (provided that the strain is susceptible), aminoglycosides, and quinolones [10,15]. Previous reports have shown that *Shewanella* infections should be treated aggressively with a combination of surgical therapy/debridement and appropriate antibiotics. Delay in treatment can result in unfavorable circumstances, leading septicemia/necrotizing fasciitis. The clinical manifestations of this case suggest that when it comes to the etiology for a severe soft tissue infection, in immunocompromised patients *S. algae*, should also be included in the differential diagnosis [1-5,19-23]. Only predisposing condition in this patient was diabetes which led to soft tissue infection. In literature *S. algae* in diabetic patients has not been reported. Thus, *S. algae* should also be kept in mind as a cause of soft tissue infection in diabetic patients, else a delay in treatment can lead to septicaemia/necrotizing fasciitis. This case also highlights that

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it is not always necessary to have *S. algae* infection after visit to coastal area. Other factors which are leading to emergence of this pathogen should also be looked for.

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