

Case Report

Human soft tissue infection by the emerging pathogen *Shewanella algae*

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Abstract

Shewanella soft tissue infections usually occur in immunocompromised patients with a preexisting cutaneous ulcer, mostly after exposure to a marine environment or contaminated water. A 35-year-old male presented with a non-healing ulcer over the distal end of his right leg but had no predisposing factors. Cultures of exudates from the wound grew *Shewanella* on repeated occasions. Recovery was uneventful following surgical debridement and antimicrobial therapy. Early suspicion, diagnosis, and treatment with potent antibiotics are needed to prevent any further complications resulting from infection by this emerging pathogen.

Key words: *Shewanella* sp.; infections; identification

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Introduction

Shewanella spp. are found throughout the world in marine environments, and most reported human infections occur in countries with warm climates [1]. *Shewanella* spp. have been implicated in skin and soft tissue infections, bacteraemia, biliary tract infections, thoracic empyema, endocarditis, dacryocystitis, intracranial abscess, arthritis, peritonitis, ventilator-associated pneumonia, and ear infections [2,3]. *Shewanella algae* and *Shewanella putrefaciens* are the two species of *Shewanella* that are most frequently implicated in human infections, although more than 80% are reportedly caused by *S. algae* [2]. *S. algae* was introduced as a new species in 1990 and has been most frequently associated with infections of skin and soft tissues resulting from breaches of the dermis, such as ulcers or following trauma [2,4]. Although *S. algae* has been reported to be the etiological agent of distinct human infections, it is difficult to give clinical significance to the isolation of this microorganism as it may be present as a colonizer or a component of mixed bacterial flora. To date, there have been only three reports of isolation of *Shewanella* from India and these were from patients with infective endocarditis, peritonitis, and chronic obstructive pulmonary disease respectively [5,6]. To the best of our knowledge, this is the first report of skin and soft tissue infection

clearly caused by *S. algae* from India. In the present case, *S. algae* was the only bacterium isolated, on multiple occasions, and the patient responded to targeted treatment, supporting that *S. algae* was the true agent of disease.

Case report

A 35-year-old male (body weight, 72 kg), presented with a non-healing ulcer over the distal end of his right leg covering the right ankle joint and half of the dorsum of the foot and lasting for one year. He gave a history of third-degree burns on the same site 22 years ago which healed by secondary intervention after conservative treatment for one year. The patient also gave a history of trauma with his motorbike on the same site one year prior which developed into a small ulcer and gradually increased in size with a considerable amount of foul-smelling discharge. He consulted a local physician who provided him medications, the nature of which are unknown. After a period of one month there was no response to this treatment. The patient presented to a nearby tertiary care hospital for treatment and was referred to our hospital for laboratory investigations. The patient was neither diabetic, hypertensive, nor immunocompromised.

On examination there was a 20 x 15 cm cutaneous-subcutaneous wound over the distal end of

his right leg, producing an excessive and foul-smelling sero-sanguinous discharge with multiple haemorrhagic bullae. His vitals were stable and the patient was afebrile. There were no overt signs of septicemia.

Blood investigations were within normal limits with mild leucocytosis and elevated C reactive protein (CRP) and erythrocyte sedimentation rate (ESR) (CRP: 32 mg/l; Normal range 0-8 mg/l, ESR 62 mm/hr; normal < 10 mm/hr). A skin biopsy was sent for histopathological examination and revealed suppurative necrosis without any evidence of skin cancer. Surgical debridement with skin grafting was planned. An exudate was collected from the raw area with the help of sterile swabs and subjected to microscopy and culture. Gram stain of the swab showed Gram-negative rods along with pus cells. No fungal elements were seen. Another swab was inoculated on routine culture media for bacterial and fungal growth. After 24 hours, small brown mucoid colonies were observed on blood agar, macconkey agar, and chocolate agar in pure growth which exhibited beta haemolysis on sheep blood agar. The cultured isolate was a Gram-negative rod that was subsequently identified as *S. algae* by a battery of conventional biochemical reactions. Identification of the isolate as *S. algae* was confirmed by VITEK 2 system (bioMerieux Vitek, Inc, Marcy l'Etoile France). By Kirby-Bauer disk diffusion, the microorganism was susceptible to ampicillin, amoxicillin-clavulanic acid, ceftazidime, ceftriaxone, cefepime, cefotaxime, imipenem, meropenem, aztreonam, gentamicin, amikacin, netilmicin, piperacillin-tazobactam, co-trimoxazole and ciprofloxacin [7]. A second sample was taken, which revealed the same microorganism with the same susceptibility profile. Blood, urine and stool cultures collected on this occasion remained negative. On the assumption that this microorganism was a colonizer rather than a pathogen, no antibiotic was started and the patient was treated symptomatically with wound debridement, repeated dressings, and multivitamins. The patient presented to the laboratory after three weeks and was found to have no improvement in the wound infection; therefore, a repeat sample was obtained, which again grew *S. algae* in pure culture and with the same susceptibility pattern.

The patient was started on ampicillin 500 mg every six hours and gentamicin 80 mg every 12 hours I/V for 14 days, and then switched to oral ampicillin 500 mg three times daily for 10 days. Discharge from the wound site decreased and a repeat culture

obtained after three weeks was negative. Surgical debridement with skin grafting was subsequently performed. Local signs of infection started reducing with the treatment and the wound took two months to completely heal.

Discussion

Shewanella comprises of a group of Gram-negative oxidative and non-oxidative bacilli whose important phenotypic attribute is production of H₂S [8]. These Gram-negative bacilli are motile, catalase positive, oxidase positive, ornithine decarboxylase positive, and hydrolyse gelatin. *S. algae* show mucoid colonies with weak haemolysis on sheep blood agar, grow at 42°C and in 6% NaCl, and have the capacity to reduce nitrite [1,9].

S. algae have been implicated in more infections in human beings than *S. putrefaciens* possibly because of the production of bacterial hemolysin by *S. algae* [10]. In a recent publication, M.S. Tsai *et al.* reviewed soft tissue infections caused by *Shewanella* spp.; they reported 27 cases of soft tissue infection and identified a chronic ulcer over the leg as the most common risk factor for soft tissue infection by *Shewanella* sp. [2]. Infections caused by *Shewanella* usually develop in immunocompromised patients with a preexisting cutaneous ulcer after contact with contaminated water [9]. However, the current case patient was neither immunocompromised nor gave any history of recent contact with water. Of note, a large number of published studies of infection associated with *Shewanella* could not establish seawater exposure as the source of infection [2,6].

In this case, no antibiotic therapy was started even after repeated isolation on two separate occasions because the microorganism was assumed to be a colonizer or a laboratory contaminant. The isolation of the same microbe in pure growth on a third occasion, even after debridement and repeated wound dressing, raised the possibility of an infective etiology leading to a non-healing of ulcer. Response to antibiotic treatment was dramatic, with the disappearance of local signs of inflammation and discharge within 14 days. A repeat culture obtained at the end of antibiotic therapy yielded no growth. However, the wound took a considerable time to completely heal.

There are no standard guidelines for treatment of *Shewanella* infection. Previous published studies reported that *S. algae* is 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 [1]. Thus the treatment options available are β -lactams (provided that the strain is susceptible), aminoglycosides, and quinolones [1,3,10]. 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 to amputation [1,2].

In this case, treatment was not started on initial isolation of *Shewanella*, as it was not suspected to be the etiological agent. This case highlights the need to consider *Shewanella* as a potential emerging pathogen, and utmost microbiological vigilance is required to identify this unusual agent of disease. Early suspicion, diagnosis, and treatment with potent antibiotics are needed to prevent further complications.

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