Case Report

Necrotizing fasciitis caused by *Shewanella putrefaciens* in a uremic patient

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Shewanella putrefaciens rarely causes infections in humans. This report describes a case of necrotizing fasciitis caused by *S. putrefaciens* in a uremic patient who recovered in spite of inadequate antibiotic treatment. *S. putrefaciens* is a possible causative organism of necrotizing fasciitis, and the absence of any sign of systemic infection cannot rule out the possibility of invasive infection in uremic patients. Surgical intervention is important in such cases.

Key words: *Shewanella putrefaciens*, necrotizing fasciitis, uremia

Introduction

Shewanella putrefaciens, first described and named by MacDonell and Colwell in 1985 [1], is the only non-fermentative Gram-negative rod that produces hydrogen sulfide. It was first isolated from tainted butter by Derby and Hammer in 1931 and classified as *Achromobacter putrefaciens* [2]. *S. putrefaciens* had been described by other investigators under different names, including *Pseudomonas putrefaciens* [3], *Pseudomonas rubescens* [4], group "Ib" bacteria [5], *Flavobacterium* group 4 [6], and *Alteromonas putrefaciens* [7]. It is a ubiquitous saprophyte that has been isolated from a variety of sources including fresh water, stagnant water, seas, lakes, rivers, sewage, oil emulsions, natural gas, petroleum brines, milk, cream, ground beef, and frozen poultry carcasses [8]. We report a case of soft-tissue infection caused by *S. putrefaciens* in a uremic patient that rapidly progressed to necrotizing fasciitis in spite of presenting no sign of systemic infection.

Case Report

A 67-year-old woman was admitted to a regional hospital with painful swelling of the left thigh that had persisted

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for 1 day. She had a history of chronic renal failure and bilateral renal stones and was undergoing regular hemodialysis for the past 10 years. On June 9, 2002, while shopping at a local market, her left lateral thigh was punctured just above the knee by the fin of a fresh water fish. Pain and swelling around the punctured site were noted the next day; she was admitted to the emergency room and was administered antibiotics along with cefazolin and gentamicin. Blood collection or wound culture was not carried out.

On admission, the patient was afebrile and showed stable vital signs — temperature, 36°C; pulse rate, 78/min; respiratory rate, 18/min; blood pressure, 136/70 mm Hg. Although her left thigh was tender, only mild swelling was noted. There was no erythema or local heat. There was no sign of liver cirrhosis. Leukocyte count was 13,200/mm^3 with left shift (91.3% neutrophil), hemoglobin count was 11.0 g/dL, and platelet count was 133,000/mm^3. Other abnormal laboratory findings included C-reactive protein (35 mg/dL) and creatinine (6.3 mg/dL). On hospital day 2, the patient was shifted to ceftazidime 2 g/day and minocycline 100 mg every 12 h for better coverage of *Vibrio vulnificus* and *Aeromonas hydrophila*. Two sets of blood cultures were collected before change of antibiotics. On hospital day 3, ecchymosis and hemorrhagic bullae were noted around the puncture site (Fig. 1), and the patient complained of worsening of symptoms. Computed

Fig. 1. Puncture site showing ecchymosis and hemorrhagic bullae.

tomography imaging of the lower limb revealed invasion of the deep tissue. The patient received fasciotomy and debridement. Gram stain of the surgical specimen showed Gram-negative bacilli (Fig. 2). The patient was transferred to another regional hospital soon after surgery at the request of the family. Antibiotics given in the first hospital included cefazolin and gentamicin for 1 day, followed by ceftazidime and minocycline for 2 days. Oxacillin was prescribed at the second hospital for 10 days, and the patient received no further surgical debridement other than change of dressings. The patient survived and received a split-thickness skin graft 10 days after fasciotomy. No obvious sign of systemic infection such as hypotension, tachycardia, hyperventilation, or change in mentation was noted during the entire course. The maximal body temperature was 37.6°C. Pathologic finding was compatible with that of necrotizing fasciitis. Blood culture was negative for bacterial growth. Culture of surgical specimen at the first hospital finally yielded pure growth of *S. putrefaciens* that was confirmed by the Vitek (bioMérieux, Durham, NC, USA) and BBL Crystal (Becton Dickinson, Sparks,
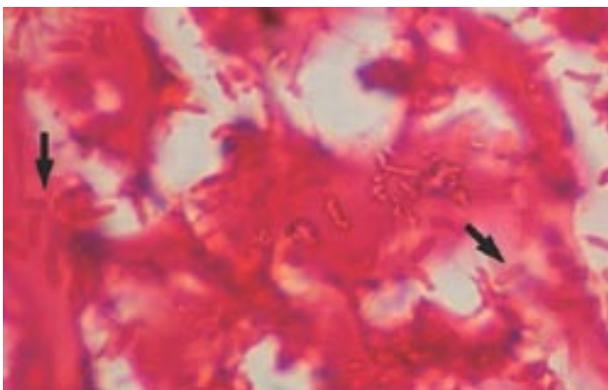

Fig. 2. Gram stain of the surgical specimen showing Gram-negative bacilli (arrows).

MD, USA) identification system. Susceptibility test by disk diffusion method performed in accordance with the National Committee for Clinical Laboratory Standards [9] revealed that the isolate was sensitive to cefuroxime, ceftriaxone, ceftazidime, cefepime, aztreonam, imipenem/cilastatin, piperacillin/tazobactam, ciprofloxacin, gentamicin, and amikacin, but resistant to cefazolin, piperacillin, and trimethoprim/sulfamethoxazole.

Discussion

S. putrefaciens has been recovered from a variety of human clinical specimens. However, the significance was difficult to determine since it was often isolated with other pathogens. Four different syndromes had been identified in the past. The first syndrome was bacteremia with a fulminant course associated with liver disease, malignancy, or other severe underlying debilitating conditions. The second syndrome was bacteremia with a relatively benign course associated with chronic ulcer or infected burn wound of the lower extremities [10]. The third syndrome was bacteremia associated with prematurity and congenital pneumonia [11]. The fourth syndrome was skin and soft tissue infection with a wide variety of clinical manifestations. Other rare manifestations included otitis media, arthritis or osteomyelitis, pneumonia, empyema, meningitis, intra-abdominal infection, infective endocarditis, and ophthalmic infection [12].

The major risk factors of *S. putrefaciens* infection are hepatobiliary disease, malignancy, severe debilitating disease, peripheral vascular disease with chronic leg ulcer, poor hygiene, and low socioeconomic status. *S. putrefaciens* seldom causes lethal infection in immunocompetent hosts. In most cases, the bacteria reside in devitalized tissues or denuded skin and serve as a nidus for opportunistic infection. Soft tissue infections have various clinical manifestations including infected leg ulcer, cellulitis, abscess formation, and wound infection, which are often preceded by chronic ulceration of the lower limbs, trauma, burn wound, or sea water exposure [12,13]. The clinical course was often benign, except in rare cases [12,14]. Four cases of peritonitis in continuous ambulatory peritoneal dialysis patients [12,15], one case of bacteremia in a peritoneal dialysis patient [16], and another in a hemodialysis patient [10] had been reported in the past. Our case presented an atypical course of necrotizing fasciitis. The absence of clinical signs of a cytokine storm in such an invasive infection is of special interest. Uremic

patients present with immunologic dysregulations that may be attributed to renal failure, poor nutrition, iron overload, or dialysis [17,18]. There is often a coexistence of defects in both nonspecific and specific host defense mechanisms. This may explain why the patient did not mount a remarkable systemic inflammatory response. *S. putrefaciens* is often resistant to penicillin and first-generation cephalosporins, but more sensitive to quinolones, third- and fourth-generation cephalosporins, and aminoglycosides. Susceptibility to tetracycline and trimethoprim/sulfamethoxazole is less predictable [10-12]. In spite of inadequate antibiotic treatment, our patient recovered after surgery, which further highlights the importance of surgical intervention in such cases.

Recently, many cases of *S. putrefaciens* infection were proven to be caused by *Shewanella algae,* which is now considered to be the predominant human pathogen in this genus [13]. Important differential characteristics between these two species include the ability of *S. algae* to produce mucoid colonies with beta-hemolysis on sheep blood agar, to grow at 42°C and in 6.5% sodium chloride, to reduce nitrite, and an inability to produce acid from maltose. Because *S. algae* was not included in our automated identification system and the pathogen was no longer available for further identification, we cannot exclude the possibility that *S. algae* was the true pathogen in this case.

V. vulnificus and *A. hydrophila* were the most common pathogens associated with necrotizing fasciitis in Taiwan, particularly in patients with liver cirrhosis. *S. putrefaciens* (or *S. algae*) is also a possible pathogen, and uremic patients may present little or no systemic sign of infection. In addition, timely surgical intervention is important.

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