Soonchunhyang Medical Science 24(1):85-87, June 2018 pISSN: 2233-4289 | eISSN: 2233-4297 SMS

CASE REPORT

Case of *Staphylococcus schleiferi* Vascular Access Infections with a Hemodialysis Patient

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Staphylococcus schleiferi has been infrequently reported as a human pathogen. We experienced the case of vascular access infection by *Staphylococcus schleiferi* in a 65-year-old hemodialysis patient. Metastatic infection occurred despite the removal of infected focus and appropriate antibiotic therapy. This case highlights the importance of considering *Staphylococcus schleiferi* as a true pathogen in vascular access infection with hemodialysis patients.

Keywords: *Staphylococcus schleiferi*; Persistent bacteremia; Renal dialysis

INTRODUCTION

Staphylococcus schleiferi was first described in 1988 by Freney et al. [1], and it has been considered as a member of persistent or transient human pre-axillary skin flora [2]. Although the virulence factors such as deoxyribonuclease, lipase, esterase, protease, beta-hemolysin, and adherence have been described, there were only limited reports implicating it as the causative pathogen of several human infections [2,3]. However, distinguishing between infection and contamination presents an important challenge for clinicians. Here, we report the case of vascular access infection caused by *S. schleiferi*.

CASE REPORT

A 65-year-old man on hemodialysis was admitted with sensation of heat and pain at the insertion site of the left femoral tunneled cuffed catheter. He previously received kidney transplantation 25 years ago, but is on hemodialysis due to the graft rejection after 4 years. He had been repeatedly operated thereafter for vascular accesses because of recurrent infections. Eight months earlier, a left axillo-right jugular necklace type arteriovenous bridge graft (AVBG) was performed while using the left femoral tunneled cuffed catheter for hemodialysis. On the day of admission, the initial physical examination revealed redness at the insertion site of the left femoral tunneled cuffed catheter. His temperature was 40° C, the blood pressure was 160/90 mm Hg, and the heart rate was 110 beats/min. Laboratory findings were leukocyte 23,400 cells/μL (neutrophil 91%), hemoglobin 13.3 g/dL (normal range, 12–16 g/dL), platelet count 134,000/μL (normal range, 130,000–450,000/μL), C-reactive protein 5.5 mg/dL (normal range, 0–0.5 mg/dL), and procalcitonin 35 ng/mL (normal range, 0–0.5 ng/mL). Methicillin-resistant *S. schleiferi* were isolated by MicroScan WalkAway-96 plus system (Siemens, Munich, Germany) in all three pairs of blood culture. Femoral tunneled cuffed catheter was removed on the day of admission, and *S. schleiferi* was isolated from the femoral catheter tip culture. Vancomycin was started empirically on the day of admission. There was no vegetation on transesophageal echocardiography and there was no evidence of endophthalmitis on ophthalmologic consultation, no evidence of hematogenous vertebral osteomyelitis on computerized tomography 7 days after admission. There was no evidence of pneumonia on

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Received: May 29, 2018 / **Accepted after revision:** Jun. 7, 2018

chest X-ray. After 14 days of vancomycin therapy, blood cultures remained positive despite the fact that adequate vancomycin trough levels were maintained (15–23.9 μg/mL). Fifteen days after admission, a sensation of heat and pain were experienced at the left axillo-right jugular AVBG site. We therefore considered it as a sign of metastatic infection and infected graft, and bypass graft excision was done on that day. *S. schleiferi* was isolated on graft material and pus obtained during surgery. The blood culture became negative 6 days after surgery and he was maintained on vancomycin (5 mg/kg) for 2 weeks following resolution of bacteremia. A follow-up 6 months after discharge revealed that the infection has not recurred.

DISCUSSION

Infections are the second leading cause of mortality (11.9%) among hemodialysis patients [4], and arteriovenous vascular graft is more commonly associated. In a retrospective study on vascular access infections in 224 hemodialysis patients in Korea, 79.7% had an arteriovenous vascular graft and 12.5% had a tunneled cuffed catheter. The most common causative organism was Staphylococcus aureus (62.5%; methicillin-susceptible, 35.2%; methicillin-resistant, 27.3%) [5]. To our knowledge, this is the first reported case of *S. schleiferi* causing vascular access infection in hemodialysis patients and was successfully treated with complete foci removal.

S. schleiferi has two distinct subspecies, which are known as *S. schleiferi* subsp schleiferi and *S. schleiferi* subsp coagulans. They can be distinguished by activity of tube coagulase and urease. *S. schleiferi* subsp schleiferi is tube coagulase and urease negative, whereas *S. schleiferi* subsp coagulans is tube coagulase and urease positive [6].

S. schleiferi has shown diverse human involvement including infection of pacemaker wires, endocarditis, extradural abscesses, and meningitis [6,7]. A retrospective study on 28 patients with *S. schleiferi* infection found that this pathogen was most commonly involved with surgical wound infections. The majority of patients in this study were older men (mean age of 64 years), and the most common underlying co-morbidity was malignancy. Infection was nosocomial in 22 cases (78.6%). Two patients became infected after aorto-femoral bypass grafting and femoro-popliteal bypass grafting for peripheral vascular disease [8].

S. schleiferi subsp *coagulans* infection in humans is a rare condition. Leung et al. [6] described a case with endocarditis, and Thibodeau et al. [9] reported a patient with a left ventricular assistance device infection awaiting heart transplantation.

The actual occurrence of *S. schleiferi* infections is underreported due to the erroneous identification of *S. schleiferi* as *S. aureus* in routine laboratory testing. Both strains exhibit beta-hemolysis and are morphologically similar on blood agar. Moreover, *S. schleiferi* subsp. *schleiferi*, like *S. aureus*, produces both a clumping factor and thermonuclease [10]. In one study of *S. schleiferi* infection, due to previous use of MicroScan identification, 20 cases of *S. schleiferi* infection might have been misdiagnosed, because their corresponding isolates were previously identified as *S. aureus* [8].

In our case, the tunneled cuffed catheter and AVBG probably acted as a pathogenic factor. In this regard, it is tempting to speculate that specific adhesins and slime produced by this organism could have favored its growth as a biofilm adherent to the graft, starting and maintaining the *S. schleiferi* infection. On the other hand, growth as a biofilm could have protected the staphylococci from antibiotics.

Although we performed an appropriate removal of the tunneled cuffed catheter in the femoral vein and administered appropriate antibiotic therapy in our patient, persistent bacteremia caused metastatic infection of the prepared left axillo-right jugular necklace type AVBG. It was not until the prepared AVBG was removed that *S. schleiferi* bacteremia resolved. Therefore, clinicians should consider vascular access infection in hemodialysis patients in the differential diagnosis of *S. schleiferi* bacteremia and the removal of infected foci is critical for a good prognosis.

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