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FIRST REPORT OF A LEFT VENTRICULAR ASSIST DEVICE INFECTION CAUSED BY STAPHYLOCOCCUS SCHLEIFERI SUBSPECIES COAGULANS: A COAGULASE POSITIVE ORGANISM

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Case Report

A fifty-five year old female was diagnosed with non-ischemic cardiomyopathy four months prior to presentation. She had no past medical history and was taking no medications prior to her diagnosis of non-ischemic cardiomyopathy. At the time of diagnosis of heart failure, a Heartmate II left ventricular assist device (LVAD) was placed, and shortly after an intra-cardiac defibrillator (ICD) was placed. Three months after the LVAD was placed she developed purulent drainage at the driveline exit site. Culture of the exudate grew abundant coagulase negative staphylococcal species. The patient was treated with ten days of oral trimethoprimsulfamethoxazole 160/800 mg twice daily. Seven weeks later, she again re-developed drainage at the LVAD exit site. On this second occasion, there was surrounding erythema and the patient felt systemically fatigued with description of subjective chills. Her medications at the time of presentation included aspirin, carvedilol, warfarin, lisinopril, aldactone, omperazole, colace, senna, simethicone, and tylenol. She had a pet cat and pet dog. On physical exam, the patient was afebrile with a heart rate of 84, and normal LVAD flow rates. Her exam was remarkable for erythema and tenderness around her driveline site with purulent drainage. Her laboratory values were significant for a white blood cell count of 13.9 per mm^3 with 79% neutrophils. All other laboratory data were normal including negative blood cultures. CT scan of the abdomen showed a small amount of fluid surrounding the LVAD outflow tubing, however no large fluid collections were seen (Hannan et al., 2011). Gram stain from her LVAD driveline site drainage showed no white blood cells but gram-positive cocci in clusters with a positive tube coagulase test. The species was initially identified as *S. aureus* and the patient was started on vancomycin therapy. Later, the isolate was identified by Vitek 2 as *S. schleiferi* subspecies *coagulans*. The identification was confirmed by phenotypic tests for sugar fermentation and showed the

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following: mannitol negative, maltose negative, lactose negative, sucrose positive, trehalose negative. The Vitek 2 system was used to test susceptibility. The organism was susceptible to the following antibiotics: ciprofloxacin, clindamycin, erythromycin, gentamicin, moxifloxacin, oxacillin, rifampin, tetracycline, trimethoprim-sulfamethoxazole, and vancomycin. Therapy was switched to intravenous oxacillin. After 2 weeks of intravenous therapy the regimen was switched to dicloxacillin. Due to slight elevation in liver function tests, the regimen was changed to oral cephalexin, which continued until time of transplantation two months after her initial presentation. She underwent successful heart transplantation, without evidence of active infection intra-operatively, and experienced no infectious complications post-operatively.

Staphylococcus schleiferi was first described in 1988 and termed subspecies *schleiferi* with the majority of case reports in dogs (Freney et al., 1988). Various human cases have been described including wound infection, prosthetic infections, and bacteremia (Celard et al., 1997; Da Costa et al., 1998; Freney et al., 1988; Hernandez et al., 2001; Igimi et al., 1990; Kumar et al., 2007; Latorre et al., 1993; Vandenesch et al., 1994). A review of 28 cases in the literature in 2001, reported that all isolates were coagulase negative: 50% of cultures were from wound infections, 19.4% were found in blood cultures, 13.8% from catheter tips, 8.3% from ear exudates, and 5.5% from cerebrospinal fluid. Other sites, each with one case, included pleural fluid, corneal exudate, biliary drainage and urine (Hernandez et al., 2001). *S. schleiferi* is often mistaken for *S. aureus* as both express clumping factor and heat stable DNase. While the subspecies *schleiferi* does not produce a staphylocoagulase, it can produce a pseudocoagulase and therefore is sometimes described as coagulase positive. However, protease inhibitors and anticoagulants can frequently inhibit clotting activity and therefore it is usually reported as coagulase negative (Vandenesch et al., 1994). However, the subspecies *coagulans*, which was first described in 1990 from infections of dogs with otitis externa, is truly coagulase positive as it can clot plasma even in the presence of such inhibitors (Igimi et al., 1990; Vandenesch et al., 1994). The first human case of *S. schleiferi* subspecies *coagulans* was described in 1994 in a patient with a surgical wound infection of the finger (Kumar et al., 2007; Vandenesch et al., 1994). A second human case, reported in 2007, described a 58 year old male liver transplant recipient who developed *S. schleiferi* aortic valve endocarditis with multiple other sites involved including blood, ascitic fluid, synovial fluid, and evidence of paravertebral abscess on MRI (Kumar et al., 2007). This current case, to our knowledge, is the third case of *S. schleiferi* subspecies *coagulans* described in humans. This is the first case of a device infection. This case demonstrates that this bacterium can often be erroneously categorized as *S. aureus*; and while physicians often rely on the tube coagulase test to determine if their patients are infected with *S. aureus*, it is important to recognize that there are other gram-positive bacteria that can have a positive tube coagulase test. While no series have been performed on antibiotic susceptibilities of *S. schleiferi* in humans, our isolate displayed susceptibility to many antibiotics and this information may help clinicians in the initial choices available to treat this species. Given this organism is most commonly found in dogs, this case exemplifies the importance of obtaining a social history and contact with animals. We suspect this patient became infected with *S. schleiferi* through contact with her dog but we were unable to culture the pet.

Transparency Declaration

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