Cardiac device infection due to *Streptococcus pneumoniae*

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Cardiac device infections (CDIs) are recognized complications of device implantation. Most CDIs are caused by skin flora but can also result from hematogenous seeding of the device. A case involving *Streptococcus pneumoniae* CDI, which is rare, potentially vaccine preventable and may not be associated with overt antecedent pneumococcal infection, is reported.

Key Words: Cardiac device infection, *Streptococcus pneumoniae*

Une infection associée à un dispositif cardiaque causée par le *Streptococcus pneumoniae*

Les infections associées à des dispositifs cardiaques (IDC) sont des complications connues des implants cardiaques. La plupart des IDC sont causées par la flore cutanée, mais elles peuvent aussi résulter d'un ensemencement hématogène. Les auteurs présentent une occurrence rare d'IDC à *Streptococcus pneumoniae*, qui aurait peut-être pu être évitée par la vaccination et qui ne s'associerait pas à une infection pneumococcique antérieure manifeste.

CASE PRESENTATION

A 68-year-old man with chronic obstructive pulmonary disease (COPD) underwent uncomplicated insertion of a permanent, dual-chamber pacemaker for symptomatic bradycardia. Three months later, he presented to the emergency department with new-onset atrial flutter with rapid ventricular response. He had no new respiratory symptoms but was febrile with an erythematous, warm and tender pacemaker site. His white blood cell count was $17.2 \times 10^9/L$, predominantly neutrophils, with bands present. A chest radiograph showed findings consistent with COPD, but no consolidation.

The patient was empirically started on antistaphylococcal antibiotics for a pacemaker pocket infection. Two sets of blood cultures drawn on admission grew penicillin-susceptible *Streptococcus pneumoniae*, and therapy was changed to intravenous penicillin, with good clinical response. Serial chest radiographs remained unchanged, and a trans-thoracic echocardiogram did not show any valvular or pacemaker lead vegetations. Five days after antibiotic initiation, the pacemaker and leads were explanted. The pacemaker pocket contained frank pus; Gram staining revealed Gram-positive cocci in pairs, but only coagulase-negative staphylococcus grew from broth culture. A tissue specimen from around the pacemaker showed few polymorphs and no organisms on Gram stain, and grew *Propionibacterium* species. Cultures from the explanted leads were sterile. Based on the positive blood cultures and a concordant intraoperative Gram stain, the causative organism was believed to be *S pneumoniae*, and the coagulase-negative staphylococci and *Propionibacterium* were presumed contaminants. Postoperatively, the patient received four weeks of intravenous penicillin, with no further complications.

DISCUSSION

Infection remains a significant complication of cardiac device implantation and is associated with increased morbidity and mortality. The average risk of cardiac device infection (CDI) is 1% in tertiary care facilities, ranging from 0.5% for new device implantation to 2.1% for device replacement (1,2). Between 1990 and 1999, the number of cardiac devices implanted in Medicare beneficiaries in the United States nearly doubled, but the number of CDIs increased by 124%,

from 0.94 per 1000 to 2.11 per 1000 (3). This increase is likely multifactorial, due to an aging population, a greater number of indications for device insertion, higher numbers of comorbid illnesses in recipients, and heightened awareness and improved diagnosis of CDIs (3-5).

CDIs can involve the generator pocket, the transvenous portions of the device or both, and can present with local inflammation of the device pocket, wound dehiscence, cutaneous erosion by the device or isolated bloodstream infection (BSI). Device-related endocarditis may also occur. Most commonly, CDI presents as a pocket infection without bacteremia (52%), followed by lead-related endocarditis (23%) and pocket infection with BSI (17%) (6). Less frequently, CDI may present as isolated BSI (11%) (7). However, it can be challenging to discern among varying degrees of severity of CDI.

Infection can also be classified according to time of onset. Early infections present less than one month after insertion or revision, late infections one to 12 months, and delayed infections occur more than 12 months after insertion or revision (8). Early and late infections are presumed to be due to perioperative contamination of the device and should be largely preventable with good skin antisepsis, appropriate surgical antibiotic prophylaxis and proper wound care. Delayed infections are believed to be most commonly due to hematogenous seeding from another source.

Staphylococcus aureus and coagulase-negative staphylococci are most commonly implicated (5,7), consistent with perioperative surgical site contamination being the primary mechanism of infection. Less commonly, CDIs are caused by Gram-negative organisms, including *Escherichia coli* and other Enterobacteriaceae, as well as *Pseudomonas aeruginosa* (6,7).

Prolonged parenteral antibiotic therapy (two to six weeks, depending on the severity of the CDI) is required, and surgical removal of all or part of the device is usually indicated (4,7,9). Reimplantation of a new device, when required, should take place only after documented clearance of bacteremia and ideally at a remote pocket site.

Pneumococcal CDI is rare. In a cohort study of 1524 patients with cardiac devices followed for the occurrence of CDIs, four patients had *S pneumoniae* bacteremia, but none had a definite device infection (6). Only one CDI caused by *S pneumoniae* has been reported, which was

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in a patient diagnosed with pneumonia three weeks before presenting with an infected device (10). In this case, blood cultures were negative, but cultures of the pacemaker pocket revealed heavy growth of *S pneumoniae* (10).

Our patient had no symptoms or clinical or radiographical findings suggestive of pneumonia. Given his underlying COPD, we suspect that he was colonized with *S pneumoniae*. He may have contaminated his surgical site with respiratory flora postoperatively, or his device may have been seeded after a transient occult bacteremia. Although well recognized in children, occult pneumococcal bacteremia in adults is rare. In a prospective study of occult bacteremia in an urban emergency department in Israel, 4603 adults had blood cultures drawn, of which 312 were positive; only 127 isolates were considered to be 'true pathogens', and of these, only one was *S pneumoniae* (11). Similarly, a prospective study in Spain found only two cases of pneumococcal bacteremia among 110 patients with occult bacteremia (12). In a retrospective study of 1350 adult patients in an urban American emergency department, 24 had clinically significant occult bacteremia. Two patients had pneumococcal bacteremia; both had respiratory symptoms and were believed to have bronchitis and lobar pneumonia, respectively (13).

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Our patient's age and COPD place him in a population that is recommended to receive pneumococcal vaccination. His *S pneumoniae* isolate was serotype 19F, which is included in the 23-valent polysaccharide vaccine, and his CDI may have been preventable had he been immunized.

CONCLUSIONS

S pneumoniae is a rare cause of CDI. Our case underscores the need to consider unusual organisms as causative agents of CDIs and serves as a reminder of the importance of pneumococcal immunization in at-risk populations.

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