Staphylococcus capitis endocarditis due to a transvenous endocardial pacemaker infection: Case report and review of Staphylococcus capitis endocarditis

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Summary

Objectives: Newer microbiologic methods to determine the species of coagulase-negative staphylococci (CoNS) have evolved which have shown that most endocarditis due to CoNS is caused by Staphylococcus epidermidis, and far fewer by Staphylococcus warneri and Staphylococcus lugdunensis.
Methods: The recent opportunity to successfully treat a patient with methicillin-resistant Staphylococcus capitis endocarditis secondary to an infected transvenous pacemaker led to a review of the literature relating to S. capitis endocarditis.
Results: Thirteen previously recorded patients were identified. Twelve (86%) patients were male. Ten had endocarditis associated with a native valve, two with prosthetic valves and one with a transvenous pacemaker. Mortality was low in all 14 cases (including this case report) with only two deaths; one in a patient with a native valve and the other with a prosthetic valve. Four of the isolates were methicillin resistant but sensitive to vancomycin, which was used in the treatment of eight patients. Those patients with prosthetic cardiac devices appear to do better when the devices are surgically removed.
Introduction

In the years prior to the availability of valve replacement surgery, infection of native heart valves by coagulase-negative staphylococci (CoNS) was stated to occur in between 1—10% of patients with infective endocarditis.\(^1\) With the development of both prosthetic valve replacement and transvenous pacemakers in the 1970s, the incidence of infective endocarditis due to CoNS has become common, constituting over 50% of such infections at the present time\(^4\),\(^5\) and may at times consist of several genetically different clones.\(^6\)

For many years CoNS were grouped together as a single species, \textit{Staphylococcus albus}. Recent studies have suggested that certain species of CoNS such as \textit{S. epidermidis}\(^7\) and \textit{S. lugdunensis}\(^1\),\(^8\)\(^–\)\(^10\) are more frequent causes of infective endocarditis. The authors recently observed a patient with \textit{S. capitis} endocarditis secondary to an implanted pacemaker. Thirteen previously-reported patients with \textit{S. capitis} endocarditis were also reviewed.\(^11\)\(^–\)\(^21\)

Case report

An 80-year-old white male underwent transvenous pacemaker placement in 1987 and again in February 2002. Aside from previous orthopedic surgery 20 years earlier, he had not undergone any other surgical procedures nor did he take medication regularly. He claimed he had not felt well since his last pacemaker insertion. For the last 3 to 4 months he noted occasional episodes of fever and experienced persistent fatigue and malaise. He also lost ten pounds. As a result of these complaints the patient was admitted to the Eisenhower Medical Center on 20 May 2002. Aside from a grade 2/6 apical systolic murmur and a pallid appearance, physical examination was unremarkable. Laboratory studies showed only a normocytic, normochromic anemia and modest elevations of the sedimentation rate and C-reactive protein. Three blood cultures drawn on admission to the hospital grew \textit{S. capitis} after 72 hours of incubation. The colonies on blood agar were noted to be tiny. Sensitivity studies showed the organism to be methicillin resistant but sensitive to vancomycin and rifampin.

The patient was begun on 1 g of vancomycin intravenously daily, and peak and trough blood levels drawn 15 minutes before and 60 minutes periodically after drug infusion were consistently therapeutic. Within 48 hours he felt better and his temperature elevation, which had been as high as 38.5 °C, resolved. A transthoracic echocardiogram was non-diagnostic but a transesophageal echocardiogram was suspicious for vegetations on the pacemaker leads. On 1 June 2002 the patient underwent removal of the implanted transvenous pacemaker and pacemaker leads, and placement of new epicardial leads with an abdominal pacemaker implant.

At surgery he was found to have an infected pacemaker lead with vegetations extending into the tricuspid valve. The valve appeared severely damaged with gross destruction of the chordae to both the septal and anterior leaflets necessitating porcine valve replacement. Cultures taken during surgery from the transvenous pacemaker lead later grew \textit{S. capitis}. His postoperative course was uneventful. He was continued on vancomycin at the same dosage for a total of six weeks and follow-up blood cultures carried out monthly for six months were sterile.

Discussion

Less than 25 years after William Osler\(^22\) described endocarditis in his Goulstonian Lectures, Lenhartz\(^23\) in 1901 recorded a case of \textit{S. albus} endocarditis. Thirty years later, Thayer\(^24\) noted an incidence of \textit{S. albus} endocarditis in 1.96% of 306 cases of infective endocarditis observed over 40 years at the Johns Hopkins Hospital. In 1951 Cates and Christie described a 1.0% incidence of \textit{S. albus} endocarditis in 1.96% of 306 cases of infective endocarditis observed over 40 years at the Johns Hopkins Hospital. With the advent of mitral valvulotomy in the 1950s and later with prosthetic heart valve replacement and transvenous pacemakers, CoNS became major pathogens causing endocarditis in these patients.\(^4\),\(^5\),\(^26\)

\textit{Staphylococcus epidermidis} is the CoNS species most often isolated; in recent series it constituted up to 91% of the isolates.\(^1\),\(^5\),\(^7\) \textit{Staphylococcus warneri} and \textit{S. lugdunensis}\(^1\),\(^7\)\(^–\)\(^10\) have also been recorded etiologies of infective endocarditis caused by CoNS. Together with coryneforms, CoNS consti-
tute the major portion of the skin flora of man and other mammals. 28 Staphylococcus capitis is part of the normal flora of the skin of the scalp, face, ears and neck. 29 Staphylococcus capitis is divided into two subspecies: subspecies capitis and subspecies ureolyticus. Staphylococcus capitis accounts for ~5% of pathogenic isolates of CoNS, and has been shown to cause pneumonia, urinary tract infection, catheter-related bacteremia and cellulitis. 28,30

Infective endocarditis due to S. capitis has thus far been described in 13 patients. All but three have involved native valves with two infecting prosthetic valves and a single case of infection in an implanted transvenous pacemaker. 11–25 Twelve of all 14 (86%) including this patient were male. These cases are summarized in Table 1. Only two deaths were recorded, one in a patient with a prosthetic valve and the other in a patient with a native valve. Although the number of cases of endocarditis due to S. capitis is small, early removal of either a prosthetic valve or infected pacemaker would appear prudent, 21 while native valve endocarditis could successfully be treated with appropriate antimicrobials alone.

Pacemaker infections occur in 15% of implanted permanent pacemakers but the reported incidence of pacemaker-related endocarditis varies from 0.13–7%. 32–36 The large majority are due to CoNS. 26,28,29 Obviously, pacemaker-associated infection does not always indicate simultaneous endocarditis. Cacoub et al. 37 reported a large series of 33 patients with definite pacemaker endocarditis based upon surgery, autopsy or bacteriological findings of valvular vegetations or electrode-tip wire vegetation. Seventy-five percent of patients were ≥60 years of age. Although pouch hematoma or inflammation was common (58%) other predisposing factors for endocarditis were rare. Early infection (<3 months) occurred in about 30% of patients and late (>3 months) in the remainder. Fever was present in 36% of patients and 24% manifested evidence of chronic debilitating disease as in this case report. Transthoracic echocardiography was a poor technique to evaluate vegetations while transesophageal echocardiography identified lead or valvular vegetations in 96% of patients that were tested. Staphylococcus epidermidis was found in >50% of patients and most often in early rather than late endocarditis. Overall mortality was 24%.

Only a single case of a pacemaker-related endocarditis caused by S. capitis has been previously described. 20 In that patient, as well as in the one described in this report, the pacemaker was promptly removed with a successful outcome.

In the current patient, the clinical course could be termed subacute, while in others it can be acute, resembling S. aureus infections. 1 Endocarditis associated with CoNS has been noted to present with a clinical picture of an acute endocarditis in 25% of patients, 1 perhaps secondary to certain exotoxins it shares with S. aureus. However, the majority of patients with endocarditis due to CoNS usually

<table>
<thead>
<tr>
<th>Case</th>
<th>Ref</th>
<th>Age</th>
<th>Sex</th>
<th>Pacemaker</th>
<th>Treatment</th>
<th>Outcome</th>
<th>Complications</th>
<th>MRSA</th>
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<td>72</td>
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<td>M</td>
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<td>35</td>
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<td>Aortic</td>
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<td>Vancomycin, valve replacement, pacemaker removed</td>
<td>Survived</td>
<td>None</td>
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</table>

MRSA = methicillin resistant S. capitis; PR = present report.
present with a subacute picture more reminiscent of endocarditis due to the viridans streptococci.\(^1,17\) In the current patient due to \textit{S. capitis} endocarditis, the colonies that grew on blood agar were very tiny and required 72 hours to grow. Although auxotyping studies were not performed on the isolated organism, its slow growth and small colony appearance suggest that they are small colony variants (SCVs). It is generally assumed that \textit{S. capitis} and the SCVs respond to nafcillin, cephalosporins and vancomycin with or without rifampin. However, methicillin (and thus nafcillin) resistance, although uncommon with \textit{S. capitis} isolates, has been reported\(^11,18\) and was present in this patient. Methicillin-resistant strains of CoNS are most often found in patients with urinary tract infections, meningitis and endocarditis.\(^28\) Of all 14 patients with endocarditis due to \textit{S. capitis}, eight received vancomycin and only one died. Of the remainder who received penicillins and or cephalosporins, only one died. Four strains (24%) were methicillin-resistant.

With the current ability to establish specific species of CoNS according to criteria defined by Kloos and Schleifer,\(^29\) it is now possible to specifically categorize infection by these organisms in patients with endocarditis. Not only will the epidemiology, clinical presentations and therapeutic options be elicited, but the microbiologic mechanisms involved in the initiation and progression of endocarditis will likely be further defined.

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References


