Review of Pneumococcal Endocarditis in Adults in the Penicillin Era

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**Streptococcus pneumoniae** is an infrequent cause of bacterial endocarditis in adults, accounting for <3% of cases since the advent of penicillin in the early 1940s [1–6]. The classic presentation is acute Osler’s triad of pneumonia, meningitis, and endocarditis in an alcoholic. The usual portal of entry for infection in this setting is the lung. Less commonly, pneumococcal endocarditis occurs in other hosts or follows primary infection at other extrapulmonary sites. In such cases, the diagnosis may be delayed because of a lower index of suspicion. Despite the availability of penicillin, the mortality rate associated with this disease remains high, with case-fatality rates ranging from 28% to 60% [4, 7, 8].

The occurrence of several recent cases of pneumococcal endocarditis at our hospital (authors’ unpublished data) and in the literature [9–11] prompted us to conduct an in-depth review of the English-language literature on this subject. A review of this kind has not been done since 1990 [1]. We were able to identify 197 adult cases of pneumococcal endocarditis occurring in the penicillin era. Given that it is likely that additional cases of pneumococcal endocarditis go unreported, we believe that the prevalence of this disease is high enough to warrant a review of the relevant clinical issues, with particular attention to predisposing risk factors, clinical presentation, diagnosis, management, and anticipated outcome.

**Methods**

Using MEDLINE from 1966 to the present and reference lists from reviewed articles and textbooks in the English-language literature, we identified a total of 197 adult cases of pneumococcal endocarditis in the penicillin era. For each reported case, the following data were extracted when available: patient demographics; preexisting medical conditions, including alcoholism and valvular heart disease; length of illness before presentation; likely portal of entry and concurrent sites of pneumococcal infection (pneumonia and meningitis); valve(s) involved; and local and systemic complications (i.e., the presence and degree of valvular insufficiency, congestive heart failure, shock, or embolic events); echocardiographic findings; pneumococcal capsular serotype; antibiotic regimen; clinical outcome; surgery and/or autopsy findings; and likely cause of death.

**Results**

One hundred ninety-seven adult cases of pneumococcal endocarditis were identified from the penicillin era [1–4, 6–9, 12–50]. As shown in table 1, the male-to-female ratio was 2.5:1, and the mean age of the patients was 52 years. The most common associated medical problem was alcoholism, identified in 28.1% of cases. Thirteen percent of patients had underlying valvular heart disease. However, for 47.4% of patients, no comorbidity was identified. Patients typically presented without peripheral stigmata of infective endocarditis.

The median duration of illness before presentation was 7 days. The lung was the usual portal of entry (82.2% of cases), although an ear, nose, sinus, and/or throat source was identified in 13.7% of cases. Meningitis was a common complication (59.5% of cases). Of the cases complicated by meningitis, 29 (42%) had evidence of Osler’s triad, and 19 (28%) occurred in alcoholics. The most commonly identified capsular serotypes were 12, 1, and 8 (data on serotypes not shown).

Cardiac valve involvement and sequelae are detailed in table 2. On the basis of the 121 cases for which data are

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Table 1. Demographics and clinical characteristics of patients with pneumococcal endocarditis.

<table>
<thead>
<tr>
<th>Finding</th>
<th>Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (n = 26)</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>90 (71.4)</td>
</tr>
<tr>
<td>Female</td>
<td>36 (28.6)</td>
</tr>
<tr>
<td>Age (y) (n = 126)</td>
<td></td>
</tr>
<tr>
<td>Mean (range)</td>
<td>52 (18–82)</td>
</tr>
<tr>
<td>Underlying medical condition(s) (n = 135</td>
<td></td>
</tr>
<tr>
<td>Alcoholism</td>
<td>38 (28.1)</td>
</tr>
<tr>
<td>Valvular heart disease</td>
<td>18 (13.3)</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>5 (3.7)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>4 (3.0)</td>
</tr>
<tr>
<td>Asplenia</td>
<td>4 (3.0)</td>
</tr>
<tr>
<td>Malignancy</td>
<td>3 (2.2)</td>
</tr>
<tr>
<td>Prosthetic heart valve</td>
<td>3 (2.2)</td>
</tr>
<tr>
<td>Chronic lung disease</td>
<td>2 (1.5)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1 (0.7)</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>1 (0.7)</td>
</tr>
<tr>
<td>Renal failure</td>
<td>1 (0.7)</td>
</tr>
<tr>
<td>Corticosteroid therapy</td>
<td>1 (0.7)</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>1 (0.7)</td>
</tr>
<tr>
<td>Chronic otitis media</td>
<td>1 (0.7)</td>
</tr>
<tr>
<td>None</td>
<td>64 (47.4)</td>
</tr>
<tr>
<td>Duration of illness before presentation (d) (n = 69)</td>
<td></td>
</tr>
<tr>
<td>Median (range)</td>
<td>7 (1–150)</td>
</tr>
<tr>
<td>Timing of diagnosis (n = 117)</td>
<td></td>
</tr>
<tr>
<td>Before death</td>
<td>103 (88.0)</td>
</tr>
<tr>
<td>After death</td>
<td>14 (12.0)</td>
</tr>
<tr>
<td>Portal of entry (n = 73)</td>
<td></td>
</tr>
<tr>
<td>Lung</td>
<td>60 (82.2)</td>
</tr>
<tr>
<td>Sinuses</td>
<td>6 (8.2)</td>
</tr>
<tr>
<td>Ear</td>
<td>3 (4.1)</td>
</tr>
<tr>
<td>Oral cavity</td>
<td>1 (1.4)</td>
</tr>
<tr>
<td>Skin</td>
<td>1 (1.4)</td>
</tr>
<tr>
<td>Genitourinary tract</td>
<td>1 (1.4)</td>
</tr>
<tr>
<td>Gastrointestinal tract</td>
<td>1 (1.4)</td>
</tr>
<tr>
<td>Peripheral stigmata (n = 78)</td>
<td></td>
</tr>
<tr>
<td>Absent</td>
<td>69 (88.5)</td>
</tr>
<tr>
<td>Present</td>
<td>9 (11.5)</td>
</tr>
<tr>
<td>Petechiae</td>
<td>7</td>
</tr>
<tr>
<td>Splinter hemorrhages</td>
<td>2</td>
</tr>
<tr>
<td>Osler’s nodes</td>
<td>1</td>
</tr>
<tr>
<td>Roth’s spots</td>
<td>1</td>
</tr>
<tr>
<td>Pneumonia (n = 101)</td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>61 (60.4)</td>
</tr>
<tr>
<td>Absent</td>
<td>40 (39.6)</td>
</tr>
<tr>
<td>Meningitis (n = 116)</td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>69* (59.5)</td>
</tr>
<tr>
<td>Absent</td>
<td>47 (40.5)</td>
</tr>
</tbody>
</table>

* Unless stated otherwise, data are no. (%) of patients.
¹ Twelve patients had two or more conditions.
² Twenty-nine (42%) of 69 had Osler’s triad; 19 (28%) of 69 were alcoholics.

available, the aortic valve was most commonly involved (74.4% of cases). In 13.2% of cases, at least two valves were affected. The rate of local and systemic complications was high. Surgery- or autopsy-confirmed valve perforation and paravalvular abscess occurred in 34.7% and 33.7% of cases, respectively; congestive heart failure, in 48.6%; and embolization, in 24.3%.

Echocardiography was performed in 38 (19%) of 197 cases [1–4, 8, 9, 14, 17, 19, 38, 41] and in 19 (32%) of the 59 cases for which surgery or autopsy data were available [1–4, 7, 8, 17, 41]. Most patients (95%) underwent transthoracic echocardiography (TTE). Valvular vegetations were identified in 50% of cases, and perforation was identified in 2.6%; in a substantial number of cases (28.9%), ‘‘destruction’’ was noted without additional descriptions. On the basis of surgery- or autopsy-confirmed data, the sensitivity of TTE for identification of valvular destruction and/or perforation was 58%. TTE failed to detect each of four similarly confirmed cases of paravalvular abscess.

On the basis of the 182 cases of pneumococcal endocarditis for which outcome data were available, the overall mortality rate was 63%. On the basis of the 68 cases for which cause of

Table 2. Cardiac findings for patients with pneumococcal endocarditis.

<table>
<thead>
<tr>
<th>Finding</th>
<th>No. (%) of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valve(s) involved (n = 121)</td>
<td></td>
</tr>
<tr>
<td>Aortic</td>
<td>90 (74.4)</td>
</tr>
<tr>
<td>Mitral</td>
<td>38 (31.4)</td>
</tr>
<tr>
<td>Tricuspid</td>
<td>10 (8.3)</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>1 (0.8)</td>
</tr>
<tr>
<td>Two or more valves</td>
<td>16 (13.2)</td>
</tr>
<tr>
<td>Vegetation(s)</td>
<td>19 (50.0)</td>
</tr>
<tr>
<td>‘‘Destruction’’</td>
<td>11 (28.9)</td>
</tr>
<tr>
<td>Perforation</td>
<td>1 (2.6)</td>
</tr>
<tr>
<td>Abscess</td>
<td>0</td>
</tr>
<tr>
<td>Valve fluttering</td>
<td>4 (10.5)</td>
</tr>
<tr>
<td>Early valve closure</td>
<td>2 (5.3)</td>
</tr>
<tr>
<td>Pericardial effusion</td>
<td>4 (10.5)</td>
</tr>
<tr>
<td>Abnormal (unspecified)</td>
<td>9 (23.7)</td>
</tr>
<tr>
<td>Complication(s) (n = 144)</td>
<td></td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>70 (48.6)</td>
</tr>
<tr>
<td>Embolization</td>
<td>35 (24.3)</td>
</tr>
<tr>
<td>Specified</td>
<td></td>
</tr>
<tr>
<td>Unspecified</td>
<td>26</td>
</tr>
<tr>
<td>Brain</td>
<td>6</td>
</tr>
<tr>
<td>Large vessel</td>
<td>3</td>
</tr>
<tr>
<td>Shock</td>
<td>3</td>
</tr>
<tr>
<td>Other metastatic sequelae</td>
<td>4 (2.8)</td>
</tr>
<tr>
<td>Septic arthritis</td>
<td>3</td>
</tr>
<tr>
<td>Paraspinal abscess</td>
<td>1</td>
</tr>
<tr>
<td>Surgery or autopsy finding(s) (n = 95)</td>
<td></td>
</tr>
<tr>
<td>Perforation</td>
<td>33 (34.7)</td>
</tr>
<tr>
<td>Abscess</td>
<td>32 (33.7)</td>
</tr>
<tr>
<td>Vegetation(s)</td>
<td>30 (31.6)</td>
</tr>
<tr>
<td>‘‘Destruction’’</td>
<td>26 (27.4)</td>
</tr>
<tr>
<td>Pericardial involvement</td>
<td>3 (3.2)</td>
</tr>
<tr>
<td>Infected thrombus</td>
<td>1 (1.1)</td>
</tr>
</tbody>
</table>

* Thirty-six of 38 underwent transthoracic echocardiography.
death was determined, the attributable mortality rate was 65%. Among the 37 patients whose cases were managed with a combined medical-surgical approach [1–4, 7, 8, 13, 14, 17, 23–26, 41, 42], the mortality rate was 32%, as compared with 62% among the 91 patients treated with antibiotics alone.

Discussion

Pneumococcal endocarditis is considered a rare entity. However, the true prevalence and incidence of this disease are unknown since the literature primarily consists of case reports and small case series. In the preantibiotic era, *S. pneumoniae* was responsible for ~15% of all cases of infective endocarditis [51]. From review of the available data since the advent of penicillin, the prevalence appears to have significantly declined and is now estimated to range from 1% to 3% [1–6]. This decline is presumably due to early administration of appropriate antimicrobial therapy, resulting in either prevention or rapid control of bacteremia and thus a decreased incidence of endocardial seeding. Despite this overall decline in prevalence, however, we identified 197 adult cases of pneumococcal endocarditis occurring in the penicillin era.

The risk factors that predispose individuals to pneumococcal endocarditis remain incompletely defined, but likely these factors parallel those that predispose individuals to invasive pneumococcal disease in general. In the immunocompetent host, following establishment of localized *S. pneumoniae* infection, an array of host immune responses are normally elicited that act to contain infection and rapidly clear bacteremia when it does occur. These responses are both humoral and cellular in nature and local and systemic in distribution; they include induction of IgG and IgA antibodies that are directed primarily at the pneumococcal capsule, activation of the complement pathways, and mobilization and activation of various phagocytic effectors [52]. A variety of conditions that impact on host immunity may thus predispose individuals to sustained bacteremia and/or serious invasive pneumococcal disease, including those associated with impaired antibody generation, quantitative or qualitative neutrophil defects, and/or defective function of the reticuloendothelial system.

Identified risk factors for invasive pneumococcal disease include conditions that affect local clearance mechanisms (such as chronic tobacco use, recent respiratory infection, and chronic pulmonary disease) and conditions characterized by impaired systemic host immune responses (such as extremes of age, malnutrition, chronic glucocorticoid therapy, various chronic disorders [cirrhosis, renal insufficiency, and diabetes mellitus], HIV infection, other congenital or acquired immunodeficiencies, and alcoholism) [12, 53–56].

Of the aforementioned factors, alcoholism is one of the strongest risk factors for pneumococcal endocarditis. The triad of pneumonia, meningoitis, and endocarditis following infection with pneumococcus was first described by Heschi [57] in 1862 and subsequently by Osler [58] in 1881 (the year that pneumococcus was discovered). Since that time, a number of investigators have linked pneumococcal endocarditis with alcoholism [7, 13–16, 59], transforming Osler’s original triad into a tetrad [16].

Precisely why alcoholism predisposes individuals to serious invasive pneumococcal disease, including endocarditis, remains unclear. An array of immunologic defects have been described in alcoholics, any of which might contribute to sustained bacteremia and risk of endocardial infection; these defects include leukocyte defects such as impaired chemotaxis, a dysfunctional reticuloendothelial system due to impaired expression of Fc receptors, and impaired delayed-type hypersensitivity [16, 60–62]. However, it remains unclear which, if any, of these abnormalities can be directly attributed to alcohol. Most researchers believe that the effect of alcohol and alcoholism on host immunity is multifactorial, stemming not only from various immunologic defects as described but also from malnutrition and the propensity to aspiration.

Failure to receive the pneumococcal vaccine has also been proposed as a risk factor for invasive pneumococcal disease, when controlling for vaccinated host populations [63, 64]. The aim of pneumococcal vaccination is to prevent the development of systemic disease due to any of the vaccine’s 23 pneumococcal serotypes. However, it is unknown whether vaccination affords protection against pneumococcal endocarditis. Previous reports of pneumococcal endocarditis often failed to provide adequate data on either the infecting pneumococcal serotype [1, 2, 4, 7–9, 12, 13, 16–37] or patients’ vaccination histories [1, 7–9, 12, 15, 16, 18–50]. Although a number of series described pneumococcal endocarditis in patients who did not receive the pneumococcal vaccine [2, 4, 13, 17], there are little data regarding infection occurring in the setting of prior vaccination.

In one series of nine Alaskan natives with pneumococcal endocarditis [14], two patients had received the 14-valent pneumococcal vaccine 4 to 5 years previously. One patient had no known medical problems and developed endocarditis due to serotype 23 that was represented in the vaccine, while the other patient was an alcoholic with chronic pulmonary disease who developed infection due to a strain that was not serotyped. The investigators noted the apparent inability of previous vaccination to prevent infection in their patients and suggested that certain serotypes may be poorly immunogenic. They did not comment on the fact that most people who receive the vaccine have an antibody response to only 75% of the antigens included. Furthermore, elderly patients and those with alcoholism and/or chronic pulmonary disease are likely to respond suboptimally to vaccination and have a more rapid decline in antibody titer to nonprotective levels over time [63–67].

In terms of clinical presentation, patients typically develop pneumococcal endocarditis following pneumococcal infection elsewhere, at a site usually within the upper or lower respiratory...
tract [3, 4]. Early reviews of pneumococcal endocarditis described older patients who had the typical Osler’s triad. These patients presented with pneumococcal pneumonia following which, after a brief apparent recovery, they developed fever, sepsis, and cardiac failure due to acute valve infection and destruction. Most of these patients died of purulent pneumococcal meningitis within 30 days of the onset of pneumonia. Our review confirms that the lung continues to be the most common portal of entry for pneumococcal endocarditis.

Much less commonly, adult cases of pneumococcal endocarditis have followed sinusitis, mastoiditis, otitis media, tooth extraction, and septic abortion [3, 7, 9, 20]. Although some of these patients were presumably receiving oral antibiotic therapy prior to developing endocarditis, most investigators failed to provide the details of therapy; thus, the explanation for the occurrence of endocarditis remains unclear, with possible contributors being predisposing host risk factors, antibiotic resistance, and/or suboptimal serum drug concentration. Three prior reports described pneumococcal endocarditis developing in patients being treated empirically for a preexisting condition (i.e., urinary tract infection, recurrent fever and chills, and pneumonia) with an antibiotic to which S. pneumoniae either was [38] or should have been [1, 4] susceptible. Further details are needed, however, to better understand why oral antibiotic therapy failed to prevent bacteremia in these particular cases.

Once endocardial infection is established, the course of pneumococcal endocarditis is typically aggressive and is associated with high morbidity and mortality rates. This poor outcome stems from a variety of factors. First, the presentation is generally acute. Patients typically lack peripheral stigmata of endocarditis that may contribute to a delay in diagnosis and initiation of appropriate antibiotic therapy. Second, for reasons that remain incompletely defined but likely pertain to the host as well as the organism itself, rapid destruction of endothelial tissue generally occurs followed by acute valvular insufficiency, congestive heart failure, and, in a smaller percentage of cases, early death [3, 4, 13]. A subset of patients have been described, however, who manifest a more subacute presentation [4, 19].

Third, cardiac and extracardiac sequelae are common, in particular hemodynamic instability secondary to valve perforation, abscess formation, and systemic embolization. This occurrence is in part due to the fact that, although S. pneumoniae may infect either native or prosthetic valves, it appears to have a predilection for the aortic valve [2–4, 6, 8, 14, 16, 20, 39, 59] (which, as compared with other infected heart valves, is associated with the highest rate of local complications). In addition, the vegetations that form tend to be large, predisposing individuals to systemic embolization [68, 69]. Our review confirms that for pneumococcal endocarditis in general, as well as for those cases associated with the highest rate of complications, the aortic valve is most commonly affected.

The diagnosis of infective endocarditis is in large part clinical. Echocardiography, however, plays an important role in confirming the diagnosis, particularly in cases where either the infecting pathogen and/or the host involved makes the diagnosis challenging. In addition, echocardiography provides critical noninvasive information regarding function and status of valves and paravalvular structures and, as a result, helps determine the need for as well as timing of surgical intervention. Recent studies have demonstrated that the sensitivity and specificity of transesophageal echocardiography (TEE) for detecting valvular vegetation and associated complications are superior to those of TTE [70]. Our review confirms the limited diagnostic sensitivity of TTE for pneumococcal endocarditis in that potentially life-threatening complications such as valvular perforation and paravalvular abscess were undetected in 42% and 100% of cases, respectively.

By comparison, there are only limited data on the diagnostic utility of TEE for this particular infection. From our review, we identified only two cases in which patients with pneumococcal endocarditis underwent TEE [9, 25]. In one case, TEE disclosed a mobile mass on the insertion ring of a prosthetic valve, which was associated with paravalvular reflux; this patient survived without surgical intervention. In a second case, TEE was employed to evaluate the possibility of a ring abscess in a woman with a TTE-confirmed aortic valve vegetation and prolongation of the PR interval on electrocardiogram. No abscess was detected, and the patient did well with medical therapy alone. These cases illustrate the major role TEE can play in guiding the need for surgical vs. medical therapy in known cases of pneumococcal endocarditis. Given the superior sensitivity of TEE over TTE, its use in such cases will likely translate into improved outcome due to earlier detection of potentially fatal complications.

Patients with pneumococcal endocarditis may be treated either medically or via a combined medical-surgical approach. With regard to antimicrobial therapy, the optimal regimen remains unclear in the era of penicillin-resistant S. pneumoniae. Parenterally administered penicillin has traditionally been the antibiotic of choice for treatment of serious pneumococcal infections; however, worldwide rates of multidrug-resistant S. pneumoniae are increasing, and in the United States, the rate of multidrug resistance ranges from 0 to 28% depending on the area and age group [71]. Although there are data [72] related to the use of high doses of penicillin for the treatment of pneumonia due to either intermediate- or high-level penicillin-resistant strains of pneumococcus, data are currently lacking for similar cases of either endocarditis or meningitis. In such cases, there is an increased inoculum of organisms, and successful outcome mandates achievement of bactericidal concentrations of drug. In the setting of penicillin resistance, even intermediate-level resistance, high doses of intravenous penicillin may not achieve an adequate tissue concentration of antibiotic. Accordingly, recommenda-
tions regarding empirical therapy for pneumococcal endocar-
ditis should likely parallel those recently adopted for pneu-
mcoccal meningitis [73].

There are only two case reports [9, 10] that detail the out-
come of pneumococcal endocarditis caused by penicillin-
resistant \textit{S. pneumoniae} isolates. The first case [10] was a
2-year-old boy who received cefteram and cefpodoxime for
treatment of a febrile illness 2 weeks before being hospitalized
because of pneumococcal endocarditis involving the mitral
valve. His blood isolate was highly resistant to penicillin (MIC,
6.25 \mu g/mL) and ceftriaxone (MIC, 6.25 \mu g/mL) but was sus-
ceptible to imipenem (MIC, 0.2 \mu g/mL) and cefuzonam (MIC,
0.39 \mu g/mL). The child responded successfully to medical
therapy with imipenem, cefuzonam, and ampicillin. The
second case [9] was a 63-year-old woman who received 2 days
of an unspecified oral antibiotic for treatment of otitis media
2 weeks before being hospitalized because of pneumococcal
endocarditis involving the aortic valve. Her blood isolate was
highly resistant to penicillin (MIC, 6 \mu g/mL) and ceftriaxone
(MIC, 6 \mu g/mL) but was susceptible to rifampin (MIC,
1 \mu g/mL) and vancomycin (MIC, 0.75 \mu g/mL). The patient
was treated successfully with a 6-week course of vancomycin
and rifampin.

Although no formal recommendations can be made at this
time regarding the optimal empirical therapy for patients with
suspected or documented pneumococcal endocarditis, on the
basis of the aggressive nature of this disease as well as the risk
of associated meningitis (pending formal susceptibility pat-
terns for the isolate), it seems reasonable to provide treatment for
such patients similar to the current recommendation for those
with presumed pneumococcal meningitis (i.e., ceftriaxone plus
vancomycin). Depending on the organism’s final susceptibility
pattern, this regimen should be modified accordingly. Even
though there are animal data suggesting that the use of penicil-
lin at doses that yield blood concentrations consistently three-
to fourfold above the MIC for pneumococcus results in sterili-
zation of cardiac vegetations [74], data on outcomes for
humans that support this approach are required before it can
be confidently adopted.

From our review of the literature, it appears that the prefer-
able treatment of patients with pneumococcal endocarditis
may be a combined medical-surgical approach. The mortality
rate among the 37 patients whose cases of pneumococcal
endocarditis were managed with a combined medical-surgi-
cal approach was 32%, as compared with 62% among pa-
tients treated with antibiotics alone. Although susceptibility
data were typically not provided for cases with a fatal out-
come, it is doubtful that antibiotic resistance contributed
significantly to the poorer outcome for the latter group, be-
cause most cases occurred before the emergence of penicil-
lin-resistant pneumococci. The improved outcome for pa-
tients treated with a combined medical-surgical approach
does, however, mandate the need for highly sensitive diag-
nostic modalities, such as TEE, to guide the timing of surgi-
cal intervention.

In conclusion, although the incidence of pneumococcal
endocarditis has decreased since the advent of penicillin,
cases of pneumococcal endocarditis still occur and are asso-
ciated with a poor outcome. The poor prognosis of this dis-
ease has been attributed to both host and microbial factors,
including older age, alcoholism, diabetes, and other concom-
itant diseases as well as the organism’s propensity to infect
and rapidly destroy left-sided heart valves. Although the
disease occurs most commonly in alcoholics, other host pop-
ulations may be affected, and their presentation may be
somewhat unusual. The aggressive nature of this infection
makes early diagnosis and treatment crucial. TEE serves as
a rapid means of assessing valve status (including competi-
tency, level of destruction, and presence or absence of para-
valvular abscesses) and thus determines the need for as well
taking of surgical intervention. Given the widespread and
variable prevalence of \beta-lactam-agent-resistant \textit{S. pneumo-
niae}, empirical therapy should likely consist of one of the
more reliable higher-generation cephalosporins (i.e., ceftri-
axone, cefotaxime, or cefepime) with or without vancomy-
cin, depending on the prevalence of cephalosporin-resistant
\textit{S. pneumoniae} in one’s geographic area. A combined med-
cal-surgical approach, employing a prolonged course of par-
terental antibiotics (4–6 weeks) plus early valve replacement,
appears to result in a lower rate of attributable mortality and
should therefore be strongly considered.

References
1. Bruyn GAW, Thompson J, Van Der Meer JWM. Pneumococcal endocardi-
2. Clark R, Carlisle JT, Valainis GT. \textit{Streptococcus pneumoniae} endocarditis pre-
4. Powderly WG, Stanley SL Jr, Medoff G. Pneumococcal endocarditis: report of
5. Bayliss R, Clarke C, Oakley CM, Somerville W, Whitfeld AGW, Young
6. Straus AL, Hamburger M. Pneumococcal endocarditis in the penicillin
8. Ugolini V, Pacifico A, Smitherman TC, Mackowiak PA. Pneumococcal
endocarditis update: analysis of 10 cases diagnosed between 1974 and
9. Whitby S, Pallera A, Schaerig DR, Bronze MS. Infective endocarditis
caused by \textit{Streptococcus pneumoniae} with high-level resistance to peni-
penicillin-resistant \textit{Streptococcus pneumoniae}: successful treatment

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