Neisseria sicca Endocarditis: Report of a Case and Review of the Literature

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A rare case of bacterial endocarditis caused by Neisseria sicca is reported. Review of the literature revealed only five other cases where sufficient data existed to confirm this particular organism as the etiological agent of bacterial endocarditis.

CASE REPORT

A 60-year-old white female was admitted to The Moses H. Cone Memorial Hospital 8 August 1975 with intermittent fever of 6 weeks' duration. She had been well until 1 July 1975 when she awoke with high fever, general malaise, and mild nausea without vomiting. The details of prehospital treatment are not known, but on at least one occasion she had received antibiotic therapy (tetracycline). Despite transient improvement, the fever, anorexia, and nausea had returned. She had experienced no weight loss and was entirely free of localizing symptoms.

She had no history of cardiovascular disease or of heart murmur. There had been no recent dental treatments or other instrumentations. Her only chronic medication had been phenytoin.

MATERIALS AND METHODS

Admission temperature was 38.9°C, blood pressure was 120/70, pulse was 88 per min and regular. A grade II/VI early systolic murmur was heard at the left sternal border without radiation. There was no lymphadenopathy, no hepatosplenomegaly, and no evidence of meningeal irritation or of peripheral embolic phenomena.

Admission laboratory data included hematocrit 34%; hemoglobin 10.8 g/100 ml; total leukocyte count 10,000 per mm³ (77% polymorphonuclears with 5% band forms); platelet count 258,000 per mm³; sedimentation rate (Wintrobe, corrected) 36 mm/hr. Urinalysis showed specific gravity 1.023, 1+ proteinuria, and three to five erythrocytes per high-power field on microscopic examination. Standard chemical studies were within normal limits; plasma protein electrophoresis revealed mild diffuse hypergammobulinemia.

Clinical and therapeutic course is depicted in Fig. 1. When six of six blood cultures were positive for a Neisseria species (vide infra), therapy was begun with continuous intravenous aqueous penicillin at 12 X 10⁶ U/day. With the appearance of an embolic lesion at the left great toe on treatment day 5, penicillin was increased to 20 X 10⁶ U/day, and streptomycin (500 mg intramuscularly every 12 h) was added. The patient subsequently became afebrile, but on treatment day 15 again spiked a temperature in association with an acute arthritis of the left wrist. Four repeat blood cultures (penicillinase added) were negative. The patient's serum was bacteriostatic and bactericidal (1, 11) at dilutions of 1:4 (studies represent peak specimens drawn 1 h after streptomycin injection), so penicillin was increased to 30 X 10⁶ U/day, and probenecid (1.0 g daily) was added. Five days thereafter, serum bacteriostatic and bactericidal levels were both 1:8. The subsequent course was one of progressive improvement with no further fever or immunological/embolic manifestations. A transient systolic click developed, followed later by prolongation and intensification of the original murmur, which became maximal at the cardiac apex. Echocardiogram demonstrated failure of the posterior mitral leaflet to completely appose the anterior leaflet during systole. At no time was there evidence of cardiac failure.

At discharge, the patient had been afebrile for 16 days and off antibiotics for 9 days. She was asymptomatic and fully ambulatory, her only discharge medications being phenytoin and chlor Diazepoxide. Electrocardiogram and radiographic cardiac silhouette were normal at discharge and have remained so since. The patient has continued free of cardiovascular symptomatology and has required no restriction of physical activity.

RESULTS

Six blood cultures with 10 ml of blood divided equally between aerobic (tryptic soy broth with sodium polyanethol sulfonate and CO₂) and anaerobic (thioglycolate with sodium polyanethol sulfonate and CO₂) media were collected over the first 24 h of hospitalization. All six blood cultures were positive, the first by hospital day 3. Diffuse turbidity was present with no evidence of hemolysis. Gram stains from either liquid or solid media showed gram-negative diplococci usually with flattening of adjacent sides. On sheep blood agar plates the colonies were dry,
adhesive, peaked, yellowish, and nonhemolytic and crumbled when removed. The organism was oxidase positive and grew on Thayer-Martin agar with no growth on nutrient agar at 22°C. Acid reactions were obtained with glucose, maltose, levulose, and sucrose with no reaction in lactose in semisolid cystine Trypticase agar. On the basis of these results, the organism was identified as Neisseria sicca, with identification confirmed by the Special Bacteriology Branch of the Laboratory Section, Division of Health Services, North Carolina Department of Human Resources, Raleigh, N.C.

The strain grew rapidly enough for susceptibility testing by the Kirby-Bauer disk diffusion method, and it was found to be susceptible to penicillin, streptomycin, tetracycline, erythromycin, chloromycetin, kanamycin, ampicillin, and cephalothin and resistant to clindamycin and methicillin. Minimum inhibitory concentrations by broth tube dilution (14) of penicillin and cephalothin were 0.78 and 3.124 μg/ml, respectively. Minimum bactericidal concentrations (1) of penicillin and cephalothin were 1.56 and 6.25 μg/ml, respectively (end point, 100% kill).

DISCUSSION

A review of the literature for possible \( N. \) sicca endocarditis revealed only five previously reported cases caused by the organism (6, 7, 12, 13, 15). Another report (8) cannot be evaluated adequately from a bacteriological point of view, since the method of identification of the strain is not described and the strain could possibly represent two or three currently recognized species within the genus. This latter case was, however, referenced elsewhere (15) as representing a case of \( Neisseria \) pharyngis sicca endocarditis. Bacteriological data in the other five case reports did, however, include Gram stain, colony morphology, and other testing to indicate with extremely high probability that the organism was \( N. \) sicca in each. Testing for oxidase activity was, strikingly, lacking in all except the oldest reference (12).

Data from previously reported cases and the current case are summarized in Table 1. The patient reported here was older than those previously reported, whose ages ranged from 14 to 27 years. Onset of symptoms was sudden in all cases, but diagnosis was delayed 10 or more weeks in two patients, including the current case. Four of the seven patients had no known history of valvular heart disease, and in only one was there apparent relationship to a manipulative procedure known to result in transient bactere-
<table>
<thead>
<tr>
<th>Patient</th>
<th>Onset</th>
<th>Previous heart disease</th>
<th>Heart murmur</th>
<th>Immunological/embolic manifestations*</th>
<th>Positive blood cultures</th>
<th>Leukocyte count</th>
<th>Duration of illness (days)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Sex</td>
<td></td>
<td></td>
<td>Petechiae</td>
<td>Meningeal (spinal fluid)</td>
<td>Hematuria</td>
<td></td>
<td></td>
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<tr>
<td>25</td>
<td>M</td>
<td>Sudden, with headache, fever, generalized achiness</td>
<td>None</td>
<td>Rough, apical systolic, transmitted to axilla</td>
<td>Skin, mucous membranes, serosal surfaces</td>
<td>Irrational CSF: &quot;pus and globulin&quot;</td>
<td>Occasional</td>
<td>2</td>
</tr>
<tr>
<td>27</td>
<td>M</td>
<td>Sudden, with cramping lower abdominal pain, painful fingers and toes</td>
<td>Rheumatic, with mitral stenosis and aortic insufficiency</td>
<td>Systolic and diastolic murmurs at both apex and base</td>
<td>Skin, mucous membrane, serosal surfaces, two Osler nodes</td>
<td>None</td>
<td>RBC increase in Addis count</td>
<td>1</td>
</tr>
<tr>
<td>21</td>
<td>F</td>
<td>Sudden, with headache, chills and fever, generalized achiness</td>
<td>Rheumatic, with mitral stenosis</td>
<td>Diastolic rumble at apex</td>
<td>Skin over chest</td>
<td>Stiff neck; CSF: 450 WBC, 96% PMN; culture +</td>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td>26</td>
<td>M</td>
<td>Chills and fever for 6 weeks</td>
<td>None</td>
<td>Rough, apical systolic, transmitted to axilla</td>
<td>None; embolic occlusion of right brachial and femoral arteries</td>
<td>Frequent</td>
<td>&quot;Many&quot;</td>
<td>16,000</td>
</tr>
<tr>
<td>14</td>
<td>F</td>
<td>Sudden, with headache, fever, blurred vision</td>
<td>None</td>
<td>Rough, apical systolic, transmitted to axilla</td>
<td>Mucous membranes, retinae, skin</td>
<td>Occasional</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>60</td>
<td>F</td>
<td>Sudden, with malaise, nausea, fever</td>
<td>None</td>
<td>Systolic, left sternal border and apex</td>
<td>None</td>
<td>On admission only</td>
<td></td>
<td>6 of 6</td>
</tr>
</tbody>
</table>

* CSF, Cerebrospinal fluid; WBC, leukocytes; RBC, erythrocytes; PMN, polymorphonuclear leukocytes.
* Illness terminated in death. Diagnosis proved by postmortem examination.
* Onset of illness to completion of antimicrobial therapy.
mnia (6). All patients manifested embolic phenomena. Mortality was high (67%), with the four deaths equally divided between patients with and without antecedent valvular heart disease. All fatalities occurred before the advent of antimicrobial therapy, with the remaining cases having been successfully treated with sulfapyridine or penicillin (with or without streptomycin).

LITERATURE CITED