Streptococcus sanguis Meningitis: Report of a Case and Review of the Literature

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Abstract

Viridans streptococcus, an indigenous bacterial species of the mouth and gastrointestinal tract, is thought to be a rare cause of bacterial meningitis. The type of streptococcus involved is important because each type causes a different kind of meningitis and is associated with a different outcome. A 39-year-old previously healthy man was admitted due to the onset of acute purulent meningitis. A cerebrospinal fluid culture grew Streptococcus sanguis (S. sanguis). Although the patient was asymptomatic for dental caries, odontogenic maxillary sinusitis was found to be the cause of the meningitis. Treatment with intravenous antibiotics was successful. Following a review of the pertinent literature, we discuss the characteristics of S. sanguis meningitis.

Key words: Streptococcus sanguis, viridans streptococcus, bacterial meningitis, odontogenic maxillary sinusitis

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Introduction

Streptococcus sanguis (S. sanguis) is a viridans streptococcus that colonizes the mouth, gastrointestinal tract and female genital tract. Viridans streptococcus is the major cause of endocarditis; however, it is thought to only rarely cause meningitis (1). A close relationship between the type of streptococcus and the source of infection has been reported (2), and the source of infection greatly influences the clinical presentation and outcome. It is therefore necessary to clarify the characteristics of the kind of meningitis caused by each type of viridans streptococcus. We herein present a case of rapidly developing S. sanguis meningitis and also review the pertinent literature in order to reveal the characteristics of S. sanguis meningitis.

Case Report

A 39-year-old previously healthy man was admitted to our hospital with a chief complaint of severe headache lasting for several hours. The patient had no history of previous illness. He had been receiving orthodontic treatment involving pulpectomy for the last two years; however, he reported no previous or current toothache problems on admission.

On admission, the patient showed an impaired consciousness (Japan Coma Scale II-20) and a physical examination revealed an elevated body temperature (39.6°C), a regular pulse (100 beats/min), normal blood pressure (106/66 mmHg) and normal oxygen saturation (100%) on room air. No stiffness of the neck or Brudzinski’s sign was evident. No focal neurologic signs were detectable. An otoscopic and oral examination was unremarkable. Auscultation of the chest revealed regular heart sounds, no murmurs and no abnormalities. No palpable lymphadenopathies were noted.

The laboratory test results were as follows: white blood cell (WBC) count: 11,110/mm³ (90.3% neutrophils); C-reactive protein: 0 mg/dL (<0.5 mg/dL); aspartate aminotransferase: 26 U/L (<38 U/L); alanine aminotransferase: 17 U/L (<36 U/L); alkaline phosphatase: 199 U/L (<359 U/L); total bilirubin: 1.0 mg/dL (<1.3 mg/dL); lactate dehydrogenase: 227 U/L (<237 U/L); creatinine: 0.8 mg/dL (<1.2 mg/dL); serum glucose: 99 mg/dL (<126 mg/dL); serum sodium: 140 mEq/L (132-148 mEq/L); serum potassium: 3.8

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mEq/L (3.5-4.9 mEq/L); and serum chloride: 102 mEq/L (96-108 mEq/L). A urinary analysis was within the normal limits, and chest radiographs and computed tomography (CT) of the brain were normal. Dental radiography showed melting and inflammation of the left seventh second molar (Fig. 1) and CT of the paranasal sinuses showed left maxillary sinusitis (Fig. 2). Based on these findings, a diagnosis of odontogenic maxillary sinusitis was made.

A lumbar puncture was performed that showed cloudy cerebrospinal fluid (CSF) with an opening pressure of 260 mmH₂O. The CSF WBC count was 2,160 cells/mm³ (97% neutrophils), the protein level was 50 mg/dL and the glucose level was 55 mg/dL. The fluid was negative for Gram staining.

The patient was initially treated with intravenous vancomycin (2 g/day) and ceftazidime (3 g/day). Blood cultures were negative; however, three days after admission, the CSF culture grew Viridans streptococcus, which was subsequently identified to be S. sanguis. The isolate of S. sanguis was susceptible to penicillin, amoxicillin, cefazolincefotaxime, clindamycin and vancomycin. The therapy was changed to ceftiraxion (4 g/day), which was continued for the next 10 days. The patient fully recovered from meningitis with no apparent complications. As it was highly plausible that the source of infection was odontogenic maxillary sinusitis, the left seventh second molar was subsequently extracted to prevent a recurrence of meningitis.

Discussion

Bacterial meningitis caused by viridians streptococcus was once presumed to be rare; however, several authors have now reported the prevalence of viridians streptococcus meningitis to not be so uncommon, accounting for 5-9% of all cases of bacterial meningitis (3-5). Furthermore, it appears to be an increasing cause of hospital-acquired meningitis and accounts for 5% of cases of nosocomial meningitis in adults (2). Most patients with meningitis caused by viridians streptococcus are reported to have underlying conditions such as infectious endocarditis, head trauma, gastroenteritis or sinusitis or to have previously undergone neurosurgical procedures (6). Several studies have reported that post-traumatic and/or post-neurosurgical states and medical procedures are major risk factors for viridians streptococcus meningitis (2, 4). Therefore, with further advances in medicine, the number of cases of streptococcus meningitis is expected to increase.

Cabellos et al. examined 28 cases of streptococcus meningitis and reported that the type of streptococcus and the source of infection are closely related (2). In their study, S. mitis was found to be associated with neurosurgical procedures or pericranial CSF fistulae, and the S. milleri group, including S. anginosus and S. intermedius, was found to be associated with brain abscesses. As the source of infection greatly influences the clinical presentation and outcome of patients with streptococcus meningitis, it is necessary that the clinical characteristics and treatment of each pathogen be determined. Two cases of S. sanguis meningitis were included in the study by Cabellos et al. and both were complicated by infectious endocarditis.

Seventeen cases of S. sanguis meningitis have been reported to date (2, 4, 7-18) and are summarized in the Table. Demographic data were available for 15 cases. These cases consisted of 11 men and 4 women with a mean age ± SD of 31±25 years (range: 0-67). Of the 15 patients, five were under 20 years of age and seven hadiatrogenic meningitis. The infections spread hematogenously in 14 patients, and three patients developed meningitis following otopharyngeal infection. All patients had underlying conditions. Two patients developed meningitis subsequent to neonatal sepsis, one after vacuum extraction (Case No.1) and the other after premature delivery (Case No. 2). S. sanguis is an indigenous species of the female genital tract; therefore, the vertical mode of transmission is possibly the cause of infection in these cases. Both obstetric manipulations, which disrupt the integrity of the skin, and premature birth, which leads to vulnerability to infection, also seem to increase the risk for sepsis and meningitis. Seven patients developed meningitis following medical procedures that may cause transient bacteremia: pantoque myelography (Case No. 3), tooth extraction (Case No. 4), esophageal dilatation (Case No. 5), epidural anesthesia (Case No. 6), percutaneous rhizotomy (Case No. 10), coagulation of the Gasserian ganglion (Case No. 11) and spinal anesthesia (Case No. 15). The other underlying conditions were CSF leakage (Case No. 7), an immunocompromised state (Case No. 8), sinusitis (Case No. 12), infectious endocarditis (Case Nos. 13 and 14) and alcoholic hepatitis (Case No. 16). The underlying condition in our patient was odontogenic maxillary sinusitis. Bacteremia, even if transient, with seeding into the subarachnoid space is the most likely cause of S. sanguis meningitis, and medical procedures, infectious endocarditis, compromised host defenses and otopharyngeal infections seem to increase the risk of this disease.

The symptoms described in all reported cases were typical of bacterial meningitis. The most common symptoms were fever and headache, followed by stiffness of the neck and an altered mental state. Two patients showed neurological deficits: abducens paresis (Case No. 11) and hearing loss (Case No. 16). The CSF data revealed purulent inflamma-

Figure 1. Dental radiograph shows pulpectomy and melting of the left seventh second molar.
Table 2. Summary of Reported Cases of Streptococcus sanguis Meningitis

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age(Sex)</th>
<th>Mode of Underlying Infection Condition</th>
<th>Symptoms</th>
<th>CSF data</th>
<th>Blood Culture</th>
<th>Susceptibility to Penicillin</th>
<th>Antibiotic Therapy (days)</th>
<th>Outcome</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Leucocytes (mm^3)</td>
<td>Glucose (mg/dL)</td>
<td>Protein (mg/dL)</td>
<td>Gram stain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>0M Hem</td>
<td>Vacuole extraction, VMT</td>
<td>F</td>
<td>108</td>
<td>ND</td>
<td>271</td>
<td>+</td>
<td>Sen</td>
<td>GM–PCG(21)</td>
</tr>
<tr>
<td>2</td>
<td>0M Hem</td>
<td>Premature, VMT</td>
<td>F</td>
<td>0</td>
<td>&lt;5</td>
<td>&gt;1,500</td>
<td>+</td>
<td>ND</td>
<td>ABPC–GM(4)</td>
</tr>
<tr>
<td>3</td>
<td>67M Hem</td>
<td>Panuonie myelography F, H, N</td>
<td>9,860</td>
<td>37</td>
<td>179</td>
<td>-</td>
<td>ND</td>
<td>ND</td>
<td>MIPC–CTX–PCG(8)</td>
</tr>
<tr>
<td>4</td>
<td>32M Hem</td>
<td>Tooth extraction F, H, N</td>
<td>700</td>
<td>56</td>
<td>140</td>
<td>ND</td>
<td>-</td>
<td>ND</td>
<td>PCG (ND)</td>
</tr>
<tr>
<td>7</td>
<td>1M Oto</td>
<td>CSF leak</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>8</td>
<td>5F Hem</td>
<td>ALL, Chemo</td>
<td>1,955</td>
<td>27</td>
<td>155</td>
<td>ND</td>
<td>+</td>
<td>Sen</td>
<td>CTX–CLDM–APC(19)</td>
</tr>
<tr>
<td>9</td>
<td>15M Hem</td>
<td>V–P shant</td>
<td>4,500</td>
<td>8</td>
<td>250</td>
<td>ND</td>
<td>-</td>
<td>ND</td>
<td>VCM–CAZ (ND)</td>
</tr>
<tr>
<td>10</td>
<td>62M Hem</td>
<td>PRZ</td>
<td>1,750</td>
<td>5</td>
<td>376</td>
<td>ND</td>
<td>-</td>
<td>Int</td>
<td>PCG+CP(14)</td>
</tr>
<tr>
<td>11</td>
<td>59F Hem</td>
<td>CGG</td>
<td>287</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>-</td>
<td>Sen</td>
<td>AMPC–CP–PCG(10)</td>
</tr>
<tr>
<td>12</td>
<td>65M Oto</td>
<td>Sinusitis</td>
<td>15,630</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>+</td>
<td>Sen</td>
<td>AMPC(14)</td>
</tr>
<tr>
<td>13</td>
<td>ND Hem</td>
<td>IE</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>-</td>
<td>Sen</td>
<td>ND</td>
</tr>
<tr>
<td>14</td>
<td>ND Hem</td>
<td>IE</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>16</td>
<td>35F Hem</td>
<td>Alcoholic hepatitis F, H, N, C, hearing loss</td>
<td>6,400</td>
<td>40</td>
<td>218</td>
<td>-</td>
<td>ND</td>
<td>CTRX (10)</td>
<td>PR 18</td>
</tr>
</tbody>
</table>


The CSF leukocyte count was elevated in 14 patients with a 60-97% polymorphonuclear predominance. The mean leukocyte count was 3,537±4,454/mm³ (range: 0-15,630). The protein concentration ranged from 50 to 1,500 mg/dL (mean: 320±382) and the glucose level was 5-127 mg/dL (mean: 37±35). Gram staining was performed in seven patients and gram-positive cocci were observed in three cases (43%). In cases of S. sanguis meningitis, a nega-
tive stain does not seem to rule out this infection. Blood cultures were performed in 13 patients and were positive in only four patients (31%), two of whom died. Although hematogenous spread was the main source of \( S. \text{sanguis} \) meningitis, transient bacteremia and limited invasiveness are possible reasons for the relatively low positive rates observed in the blood cultures.

Initial antibiotic treatments were obviously targeted to cover a broad spectrum of organisms, and treatment was switched to penicillin, ampicillin or amoxicillin in most cases where streptococcus was identified. Antibiotic susceptibility was demonstrated in seven patients, the strains of six patients showed sensitivity to penicillin and the strain of one patient (Case No. 10) showed reduced sensitivity to penicillin (minimum inhibitory concentration 0.5 mg/L). Studies of antimicrobial susceptibility of isolated \( S. \text{sanguis} \) have revealed resistance and reduced sensitivity to penicillin in approximately 10-26% of cases and to cefotaxime and ceftriaxone in 4-6% of cases (19, 20). In cases of bacterial meningitis, failure of initial antibiotic therapy can lead to death. It is more appropriate to start patients on cefotaxime or ceftriaxone until the results of antibiotic susceptibility are obtained in cases where \( S. \text{sanguis} \) could be the cause of meningitis.

At the end of treatment, two of the 15 patients (Cases No. 2 and 8) died. Of the 13 patients who survived, 12 recovered fully, while one (Case No. 16) continues to have impaired hearing.

Notably, in the present case, the patient was asymptomatic for dental caries due to having undergone pulpectomy. The lower part of the maxillary sinus, which is the main causative site for odontogenic maxillary sinusitis, is not routinely imaged on head CT. Therefore, unless suspected, odontogenic maxillary sinusitis may often be overlooked. Clinicians should therefore be mindful that asymptomatic odontogenic maxillary sinusitis can sometimes be the cause of meningitis, especially in patients with a recent history of orthodontic treatment.

The authors state that they have no Conflict of Interest (COI).

References