Meningitis Due to Gemella haemolysans in a Pediatric Case

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Gemella haemolysans is a rare pathogen in cases of bacterial meningitis. We present a case of meningitis due to G. haemolysans in a 17-month-old boy. This is the first reported case of Gemella meningitis in a child. The patient completely recovered following intravenous therapy with linezolid and chloramphenicol.

A 17-month-old boy who was diagnosed at the age of 2 months with complex congenital heart disease (a perimembranous ventricular septal defect and a small patent ductus arteriosus) was admitted to the hospital with complaints of fever, vomiting, and loss of appetite for 2 days. A physical examination revealed an ill-appearing baby with a head circumference of 46.5 cm, a weight of 10,500 g, and a height of 77 cm. His body temperature was 39.2°C, his heart rate was 164/min, and his respiratory rate was 28/min. The anterior fontanelle was closed, and he had a grade 2/6 systolic murmur at the left sternal border. There was no hepatomegaly or splenomegaly, and he had no sick contacts or trauma. He had received three doses of hepatitis B vaccine, as well as polio, diphtheria, tetanus toxoid, pertussis, and first Bacille Calmette-Guerin (recommended in the childhood immunization schedule in Turkey) vaccines, and had not received Haemophilus influenzae type b vaccine (recommended in the childhood immunization schedule in Turkey for 3 months). His medications included digoxin and spironolactone because of heart disease. There was no family history of invasive bacterial infection. Blood, cerebrospinal fluid (CSF), and urine samples were taken and sent to the laboratory for culture and biochemical analyses. The complete blood count showed a white blood cell count of 21,830/mm³, of which 74% were neutrophils, 8% were bands, 16% were lymphocytes, and 2% were monocytes; a hemoglobin level of 11.9 g/dl; and a platelet count of 170,000/mm³. His serum basic chemistry was normal, except for a glucose level of 183 mg/dl. The level of C-reactive protein was 30.4 mg/dl, and the erythrocyte sedimentation rate was 56 mm/h. Serum immunoglobulin and subclass levels were normal, and the serum was negative for anti-HIV antibody. Urine analysis and a chest radiograph were normal. Transthoracic echocardiography was negative for endocarditis. CSF analyses showed a protein concentration of 102 mg/dl, a glucose concentration of 11 mg/dl, a red blood cell count of 250/mm³, and a white blood cell count of 4,500/mm³, of which 88% were neutrophils and 12% were lymphocytes. The CSF was negative by gram staining. The diagnosis was suspicion of bacterial meningitis, and empirical antibiotic treatment was started with ampicillin (300 mg/kg/day) and cefotaxime (200 mg/kg/day) as part of our clinical protocol. Dexamethasone (0.6 mg/kg/day, 4 days) was given intravenously before the first administered antibiotic dose. The CSF specimen was cultured on 5% sheep blood, eosin-methylene blue, and chocolate agars at 35°C in 5 to 10% CO₂ for 48 to 72 h. Because of a suspicion of endocarditis, three pairs of aerobic and anaerobic blood cultures, with each bottle containing 1 to 2 ml of the patient’s blood, were obtained prior to initiation of antimicrobial therapy. The blood cultures were incubated in a BACTEC 9120 instrument (Becton Dickinson and Company, Sparks, MD). The urine specimen was cultured on 5% sheep blood eosin-methylene blue at 35°C in 5 to 10% CO₂ for 24 to 48 h. Blood and urine cultures were negative, but in CSF cultures on sheep blood and chocolate agars, small, grayish colonies, 10⁶ CFU/ml, grew after 72 h. Colonies were weakly alpha-hemolytic on sheep blood agar. Gram staining of the colonies showed gram-variable cocci. The standard conventional biochemical method (15) and the API 20 STREP identification system (bioMérieux, Marcy l’Étoile, France) were used to identify these colonies. The organism was non-motive and oxidase, catalase, and bile esculin reaction negative and failed to grow in broth containing 6.5% NaCl. In the API 20 STREP system, the isolate gave biotype number 040011011000 with a probability of 99.5%, which was interpreted as “very good identification.” All tests showed that Gemella haemolysans was present in the CSF culture. Antimicrobial susceptibility testing was performed by the Kirby-Bauer disk diffusion method with Mueller-Hinton agar supplemented with 5% sheep blood and incubation in 5% CO₂ at 35°C (20). The susceptibility of the isolate to antimicrobial agents was determined by applying the Clinical and Laboratory Standards Institute susceptibility criteria used for viridans group streptococci (4). The isolate was resistant to penicillin, ampicillin, ceftazidime, ceftriaxone, clindamycin, levofloxacin, and vancomycin (zone diameter, 14 mm) and susceptible to linezolid and chloramphenicol (Oxoid Limited, Hampshire, England) by the disk diffusion method. After the antimicrobial susceptibility test, intravenous administration of linezolid (100 mg/kg/day) and chloramphenicol (20 mg/kg/day) was started on day 4 as a substitute for ampicillin and cefotaxime. An Etest was performed while disk diffusion results were obtained. Later, we determined the vancomycin MIC by the Etest method according to the manufacturer’s (AB Biodisk, Solna, Sweden) recommendations and found that the organism was susceptible (MIC, 1 μg/ml) but we did not change the treatment protocol.

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The patient’s clinical status progressively improved. Forty-eight hours after initiation of linezolid and chloramphenicol treatment, the patient became afebrile. A subsequent CSF culture on day 6 was negative. After antibiotic treatment for 10 days, the patient’s clinical status was excellent, his inflammatory indexes were normal, there was no evidence of neurologic sequelae, and his hearing was normal.

*G. haemolysans* was first described by Thjotta and Böe as *Neisseria haemolysans* in 1938. Berger further showed that it was catalase and oxidase negative and attacked carbohydrates fermentatively, unlike neisseriae. He considered that it should be allocated to a new genus, *Gemella* (little twin), within the family *Neisseriaceae*, with a single species, *G. haemolysans*. Nucleic acid hybridization studies showed no relatedness to members of the family *Neisseriaceae*. On the basis of these findings, the genus *Gemella* was transferred to the family of *Streptococcaceae* (7, 13, 14, 21). *G. haemolysans* is a commensal organism of the upper respiratory, gastrointestinal, and genitourinary tracts of humans (7, 14). However, as an opportunistic pathogen, *G. haemolysans* occasionally causes severe localized and generalized infections. Endocarditis (7, 8), central nervous system infections (1, 9, 12, 13, 16), eye infections (17), spondylodiscitis (11), thorax empyema (6), and bacteremia (22) have been described. Endocarditis is the most common infection caused by this organism (7, 8). However, we did not find any clinical signs associated with endocarditis or any sign of endocarditis on echocardiography. Also, all of the blood cultures were negative. Interestingly, the infection site was the central nervous system, not the heart, in our case. Central nervous system infection due to *G. haemolysans* is very rare. To date, worldwide, there have been six documented cases of meningitis and a case of brain abscess due to *G. haemolysans*.

*G. haemolysans* infections due to *G. haemolysans* are summarized in Table 1. The most common primary septic sites in the cases of intracranial *Gemella* sp. infections were the upper respiratory tract and the oral cavity (9, 10, 12, 16, 19). In the present case, the portal of entry of the infecting organism is uncertain.

Infections caused by *G. haemolysans* in the pediatric age group are rare. In a Medline research, we found only three pediatric cases and none of them involved the central nervous system (Table 2).

Generally, human infections caused by *Gemella* species are associated with underlying conditions, including an immunocompromised state, cancer, heart disease, sinusitis, or poor dental health, as well as with previous invasive medical procedures. Some cases have been reported in immunocompetent patients and have even caused life-threatening conditions in previously healthy people (7, 11). In our case, the patient had an underlying chronic heart disease without a source of infection.

Infections due to *G. haemolysans* have been observed infrequently. During gram staining, cells are easily decolorized and may therefore appear gram variable or even gram negative; also, identification of the organism may be delayed owing to its slow growth and fastidious requirements. It is likely that gram-staining abnormality and morphological polymorphism are responsible for the misidentification of *Gemella* spp. They may be alpha-hemolytic on blood agar and catalase negative and appear as gram-positive cocci. Therefore, it can be initially misidentified as a viridans group streptococcus and reported as a part of the normal flora. This may explain why so few cases are reported (8, 18, 20, 21).

In most cases, the infections were successfully treated with antibiotic therapy, usually penicillin or amoxicillin associated with gentamicin (8). Species isolated from clinical specimens in the past were usually sensitive to penicillin G and ampicillin. However, recent data suggest an emerging resistance. In 1993, a strain of *G. haemolysans* that was recovered from a blood culture was found to be sensitive to penicillin G, ampicillin, gentamicin, and cefuroxime and resistant to vancomycin, teicoplanin, erythromycin, and tetracycline (21). Our isolate was highly resistant to most of the antibiotics tested but sensitive to vancomycin, linezolid, and chloramphenicol.

In conclusion, although *G. haemolysans* is a rare pathogen in humans it may occasionally cause severe infections, including meningitis, endocarditis, and bacteremia, and while *G. haemolysans*

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**Table 1. Characteristics of previously reported cases of intracranial infections due to *G. haemolysans***

<table>
<thead>
<tr>
<th>Reference</th>
<th>Primary septic site</th>
<th>Intracranial involvement</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Paranasal sinus</td>
<td>Meningitis</td>
<td>Good</td>
</tr>
<tr>
<td>9</td>
<td>Oral cavity</td>
<td>Brain abscess</td>
<td>Good</td>
</tr>
<tr>
<td>12</td>
<td>Paranasal sinus</td>
<td>Meningitis</td>
<td>Good</td>
</tr>
<tr>
<td>13</td>
<td>Unknown</td>
<td>Meningitis</td>
<td>Good</td>
</tr>
<tr>
<td>16</td>
<td>Oral cavity</td>
<td>Meningitis</td>
<td>Good</td>
</tr>
</tbody>
</table>

**Table 2. Case reports of *G. haemolysans* infections in children**

<table>
<thead>
<tr>
<th>Reference</th>
<th>Age</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>Underlying condition or source of infection</th>
<th>Culture site</th>
<th>Therapy</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>6 yr M</td>
<td>M</td>
<td>Endocarditis</td>
<td>Congenital truncal arteriosus, valvular surgery</td>
<td>Blood</td>
<td>Amoxicillin and gentamicin</td>
<td>Good</td>
</tr>
<tr>
<td>3</td>
<td>10 yr M</td>
<td>M</td>
<td>Wound infection</td>
<td>Rheumatic heart disease</td>
<td>Blood</td>
<td>Cephalexin</td>
<td>Good</td>
</tr>
<tr>
<td>5</td>
<td>13 yr M</td>
<td>M</td>
<td>Pneumonia leading to renal failure</td>
<td>Operative repair of pectus excavatum, transtracheal fixating devices</td>
<td>Blood</td>
<td>Cefotaxime and hemodialysis</td>
<td>Good</td>
</tr>
</tbody>
</table>

This report 17 mo M Meningitis Complex congenital heart disease CSF Linezolid and chloramphenicol Good

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*a* M, male.
*Lysans* is highly susceptible to many antibiotics, our data may show a possible increasing resistance rate.

REFERENCES


