Successful Treatment of Postoperative Meningitis Due to Haemophilus influenzae Without Removal of an Expanded Polytetrafluoroethylene Dural Graft

Artificial dural grafts are frequently used in cranial and spinal operations. The question of whether such grafts must always be removed in cases of infection has not been resolved. Factors that may influence the decision to remove the graft include the site, duration, and source of infection; prior use of antibiotics; host response to the implanted material; and the characteristics of the infecting organism [1]. We describe a patient in whom antibiotic therapy resulted in the successful treatment of postoperative meningitis in the presence of a synthetic dural graft implanted after suboccipital craniectomy and tumor resection.

A 30-year-old woman with a recent history of headaches and difficulty with balance was found to have a well-demarcated, predominantly intracranial lesion (3.5 cm in diameter) in the right jugular fossa area. Radiographic studies indicated that the lesion was a schwannoma of the ninth or tenth cranial nerve. The patient underwent elective suboccipital craniectomy and tumor resection.

On the basis of our experience in this case, peritoneal antibiotic sulfamethoxazole in combination with use of one or two brief lavages is clinically efficacious by laparoscopy. Gastrointest Endosc 1994;40:357–9.

References
As shown in Table 1, the patient had no specific signs or symptoms, and the results of physical examinations were unremarkable. Follow-up radiographic studies did not show any fluid collection or abnormality in the area of the operation; the right mastoid process was normal. The patient was given cefazolin prophylactically at the beginning of the operation and for 2 days afterward.

No fluid collection or related abnormalities were observed. A lumbar puncture was performed; the opening pressure was 30 mm Hg. Results of assessments of the CSF are shown in Table 1. A gram stain did not reveal any microorganisms. The patient was admitted to the hospital and treated empirically with ceftriaxone, metronidazole, and vancomycin pending the results of CSF cultures. Two days after presentation, cultures yielded *Haemophilus influenzae* type b. Results of blood cultures and other assessments for infection were normal. In accordance with the results of susceptibility testing, the patient continued to receive ceftriaxone, but therapy with metronidazole and vancomycin was discontinued. During hospitalization, the patient had episodes of fever; her maximum temperature was 38.8°C. She was discharged 8 days after presentation, after she had been afebrile for 48 hours. She declined repeated lumbar puncture at the time of discharge.

Intravenous ceftriaxone therapy (2 g every 12 hours) was continued after discharge, for a total course of 10 weeks, guided partly by results from analyses of CSF obtained by serial lumbar punctures (table 1). Throughout treatment, the patient had no specific signs or symptoms, and the results of physical examinations were unremarkable. Follow-up radiographic studies did not show any fluid collection or abnormality in the area of the operation; the right mastoid process was also normal. The patient has remained unremarkable. Follow-up radiographic studies did not show any.

The operation in our patient involved the mastoid air cells, and we suspect that despite efforts to occlude these cells and isolate them from the CSF, the meningeal infection occurred as a result of exposure to and subsequent contamination from the mastoid area. This hypothesis is supported by the fact that the pathogen isolated from the CSF is commonly found in the mastoid. It appears unlikely that the infection originated from the expanded PTFE substitute.

The length of the therapy in this case was partly determined by the results of CSF analyses. A shorter course may be possible. We do not know whether the same results would have been possible with other pathogens or dural substitutes, since bacteria that cause meningitis vary with respect to their adherence to foreign bodies as well as susceptibility to antimicrobial agents, and prosthetic materials vary with respect to their infectivity [1, 9]. *H. influenzae* type b is an encapsulated pathogen that commonly colonizes the upper and lower respiratory tracts, causes bacteremia and meningitis, most commonly in children, but has become less common in the United States since the introduction and widespread use of the *H. influenzae* type b protein–polysaccharide vaccine [10]. In comparison with other types of bacteria that adhere readily to prosthetic devices, such as coagulase-negative staphylococci, little is known about the adherence of *H. influenzae* to prosthetic devices. The polysaccharide capsule of *H. influenzae* type b, which is like those of other encapsulated CNS pathogens such as *Neisseria meningitidis* and *Streptococcus pneumoniae*, is probably less adherent than that of many other organisms [1]. Therefore, specific data on the treatment of infections due to various organisms in the presence of prosthetic devices are of paramount importance, particularly when the rates of such infections are low and the spectrum of potential infectious agents is high.

Clinical experience with expanded PTFE membranes used for duraplasty has been documented in published articles [3–6] and in company files at W. L. Gore & Associates, manufacturer of the devices. Five cases of postoperative infections have been reported.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>At presentation</th>
<th>At 5 w</th>
<th>At 8 w</th>
<th>At 12 w</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appearance</td>
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<td>Clear, colorless</td>
<td>Clear, colorless</td>
<td>Clear, colorless</td>
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<tr>
<td>WBC count (mm³)</td>
<td>4,753</td>
<td>11</td>
<td>5</td>
<td>6</td>
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<tr>
<td>PMNs (%)</td>
<td>81</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
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<td>Lymphocytes (%)</td>
<td>NA</td>
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<td>RBC count (mm³)</td>
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<td>20</td>
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<tr>
<td>Glucose level (mg/dL)</td>
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<td>50</td>
<td>55</td>
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<tr>
<td>Protein level (mg/dL)</td>
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<tr>
<td>Culture results</td>
<td>Positive</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
</tr>
</tbody>
</table>

NOTE. NA = data not available; PMNs = polymorphonuclear cells.

* Blood glucose concentration, 118 mg/dL.
although information concerning the specific bacteria involved are not available. Meningitis in conjunction with skin infections occurred in two of those cases. One case resolved with conservative treatment, whereas the other required reoperation and removal of the membrane. The other three cases were characterized by both skin infections and extradural collections. Antibiotic treatment was effective in two of these cases; the third required removal of the membrane. Our patient had no evidence of wound infection or breakdown, CSF leakage, or a collection at the operative site or dural graft interface, as determined with use of postoperative radiographs. For patients in whom such complications are present, removal of the graft may be necessary.

Jules M. Nazarro and Donald E. Craven

Epidemiological Study

Pasteurella multocida Endocarditis: A Molecular

Human infections due to Pasteurella multocida often occur after an animal bite or scratch and usually involve soft-tissue sites. Although a variety of organs may be affected, reports of endocarditis due to P. multocida are rare [1–6]. We describe a fatal case of endocarditis due to P. multocida in a cirrhotic patient without a history of an animal bite or scratch. Several P. multocida strains were isolated from the oral cavities of the patient’s dog and cat, but various phenotypic and DNA fingerprint tests pinpointed the dog as the likely source of infection. A 65-year-old man who owned a healthy dog and cat presented with confusion and increasing swelling of the legs. Although he denied any history of animal bite or scratch or exposure to other animals, he reported that his dog occasionally licked his edematous legs and feet. His medical history included cryptogenic cirrhosis, chronic lower-extremity lymphedema, and mild aortic stenosis. He was febrile and hypotensive and lethargic, but he did not have any focal neurological deficits. His lower extremities were markedly edematous, but there was no evidence of recent trauma or inflammation. A previously described systolic ejection murmur was heard in the aortic area, but there were no signs of peripheral septic embolism, and findings of the remainder of the examination were normal.

References


Laboratory studies showed the following values: WBC count, 10,900/mm³; hemoglobin, 10.1 g/dL; platelet count, 55,000/mm³; creatinine, 2.4 mg/dL; total bilirubin, 2.3 mg/dL; and aspartate aminotransferase, 100 IU/L (normal level, 15–46 IU/L). Urinalysis showed microhematuria. There were no infiltrates on a chest radiograph. Despite supportive therapy with fluids, vasopressors, and intravenous ampicillin/sulbactam, he died 16 hours after admission. Autopsy revealed a 10-mm vegetation composed of clumps of gram-negative organisms, but the vegetation was not cultured. Blood obtained for culture on admission yielded P. multocida subspecies gallicida.

Three P. multocida strains (D1, D2, and D3) were recovered from the oral cavities of the patient’s dog, and three strains (C1, C2, and C3) were recovered from his cat. Table 1 shows the phenotypic features of these strains, the strain (P) isolated from the patient’s blood, four additional P. multocida strains (SCD1, SCD2, SCD3, and SCD4) obtained from four other local domestic dogs, three P. multocida isolates (H1, H2, H3) previously recovered in unrelated cases of bite wounds in humans, and a reference strain (American Type Culture Collection 29977, P. multocida subspecies gallicida). Only one of the strains isolated from the oral cavity of the patient’s dog (D1) was identical to that isolated from the patient’s blood (P).

Figure 1 shows the DNA fingerprint profiles of 13 P. multocida strains obtained by using HhaI restriction [7]. Only the D1 strain had a DNA profile similar to that of the P strain. Systemic complications due to P. multocida infection are rare, except in immunocompromised patients, especially those with chronic liver disease [8], such as our patient. His chronic lymphedema might also have facilitated the entrance of the organisms into the bloodstream after his dog licked his lower extremities. A similar case was reported by Hombal and Dincsoy [6]. Endocarditis due to P. multocida is uncommon and, according to diagnostic criteria for infective endocarditis [9], only three definite cases [4–6] and three possible cases [1–3] have been published. These