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Bacteroides fragilis Meningitis

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A fatal case of pyogenic meningitis due to *Bacteroides fragilis* in a 6-year-old boy is reported. The need for processing cerebrospinal fluid of patients with underlying conditions such as chronic otitis media for recovery of both aerobes and anaerobes is discussed.

Although anaerobic meningitis is uncommon, it is probably also overlooked on occasion because routine methods for processing of cerebrospinal fluid (CSF) do not detect anaerobes (1, 4). An illustrative case of *Bacteroides fragilis* meningitis is reported.

A 6-year-old boy came to the University of Ilorin Teaching Hospital, in October 1983 with a 5-day history of headache and fever and 2 days of vomiting. Past medical history was significant for right chronic otitis media that had not responded to unknown previous treatment. Physical examination revealed an ill-looking, confused, and febrile child with a temperature of 39.5°C. There was nuchal rigidity with positive Brudzinski's and Kernig's signs. There was no other neurological deficit, and the optic disks were normal. There was a purulent discharge from the right ear. Meningitis was suspected; CSF was obtained and found to be turbid. The patient was then put on intravenous benzylpenicillin (400,000 U/kg per day in six divided doses) and chloramphenicol (100 mg/kg per day in four divided doses). A Gram-stained smear revealed gram-negative pleomorphic bacilli, an erythrocyte count of 1,260/μl with predominant segmented neutrophils (96%), and protein content of 176 mg/dl. There was no bacterial growth on chocolate and blood agar plates that were incubated aerobically at 37°C for 48 h. The swab specimen that was taken from the right ear discharge yielded *Providencia rettgeri* susceptible to ampicillin, tetracycline, gentamicin, and cefotaxime.

On the fifth hospital day, the temperature of the child remained 39.5°C; since his condition continued to deteriorate, antibiotics were changed to intravenous gentamicin (6 mg/kg per day in three divided doses) and cefotaxime (100 mg/kg per day in two divided doses). A repeat lumbar puncture still showed the presence of gram-negative bacilli. Ziehl-Neelsen stain showed no acid-fast bacilli. Both the CSF cultured aerobically and the blood cultures were negative.

On the 13th hospital day and after 5 days of intravenous cefotaxime and gentamicin without improvement, another lumbar puncture was done.

The turbid CSF that was obtained was inoculated onto chocolate and blood agar plates, which were incubated at 37°C both aerobically and, for the first time, anaerobically. After 48 h of incubation, only the two plates incubated anaerobically yielded luxuriant growth of a bacterium susceptible to metronidazole (5-μg disk) and resistant to gentamicin (10-μg disk). The colonies were grayish. The isolate was nonhemolytic, nonpigmented, nonmotile, and catalase positive. Growth in 20% bile was positive, esculin hydrolysis was positive, and the indole test was negative. Sugar fermentation was determined by a standard method (7). The organism fermented glucose, lactose, maltose, and sucrose. It did not ferment arabinose, mannitol, rhamnose, salicin, trehalose, and xylose. The dye tolerance test (3) showed that the isolate was sensitive to taurocholate, victorian blue, and gentian violet. The diagnostic antibiotic disks method (2) showed that the organism was susceptible to erythromycin (60 μg) and rifampin (15 μg), but resistant to colistin sulfate (10 μg), penicillin G (2 U), kanamycin (1,000 μg), and vanocomycin (5 μg). The biochemical reactions were consistent with those for *Bacteroides fragilis* (2, 3, 7, 8). Positive beta-lactamase activity was detected by the starch-paper technique (6).

By the broth-disk susceptibility method (5), the isolate was susceptible to metronidazole (2 μg/ml), clindamycin (3.2 μg/ml), cefoxitin (18 μg/ml), chloramphenicol (12 μg/ml), erythromycin (3 μg/ml), and a combination of amoxicillin (16 μg/ml) plus clavulanic acid (8 μg/ml), but it was resistant to benzylpenicillin (2 U/ml), cefotaxime (18 μg/ml), tetracycline (6 μg/ml), and amoxicillin (16 μg/ml). As soon as the presence of an anaerobe was detected in the CSF, the child was put on intravenous metronidazole (22.5 mg/kg per day in three divided doses). The child died on the second day of metronidazole therapy. Autopsy was not performed.

B. fragilis, an anaerobic bacterium, has been infrequently reported as a cause of pyogenic meningitis (4). In the present case, the significant past medical history was chronic otitis media that did not improve with unknown previous treatment. It is recognized that anerobic infections of the central nervous system, including meningitis, are most likely to occur in the setting of preceding chronic infections of the head and neck. Thus, the presence of an underlying process such as chronic otitis media should prompt an evaluation for the presence of anaerobes (1, 4).

Although gram-negative, pleomorphic bacilli were observed on the CSF smear, we initially attributed failure of the organism to grow to the possible effect of antimicrobial therapy before hospitalization, a usual practice in developing countries. Furthermore, the specimen from the chronic ear discharge yielded only *P. rettgeri*. It is noteworthy that although chronic otitis media may be an underlying condition for anaerobic meningitis, the organism recovered from the ear discharge need not reflect the true etiological agent of meningitis.

Our isolate recovered from the CSF collected on the 13th hospital day was susceptible to chloramphenicol (12 μg/ml), but resistant to cefotaxime (18 μg/ml). Failure of the initial antimicrobial therapy (which included chloramphenicol) suggests the possibility of an associated parameningeal infec-

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tion. Certainly in the setting of preceding focal infection, anaerobic meningitis is often part of a more extensive intracranial infection; there may be associated brain abscess or subdural or epidural empyema. Indeed, early identification of the spinal fluid isolate as an anaerobe might have helped to suggest a focal infection. However, we were unable to investigate such a possibility because of our lack of diagnostic facilities for radionuclide scanning or computerized tomography.

Although anaerobic meningitis is rare, we believe that our case raises the possibility of its underdiagnosis in the laboratories. Therefore, we point out the need for greater awareness by both clinicians and microbiologists of the possible role of anaerobes as etiological agents of meningitis.

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