Streptococcus bovis meningitis

Sirs: Streptococcus bovis, a Group D non-enterococcal organism, is a common cause of endocarditis but an uncommon cause of adult meningitis. Differentiation between enterococcus and Sbovis is important because Sbovis is sensitive to penicillin alone and is positively associated with underlying gastrointestinal disease such as colonic cancer.\(^1\)\(^2\) We describe a case of adult Sbovis meningitis and review eight other cases from the literature in the English language.

A 59 year old man had a past medical history of adult-onset diabetes mellitus, excessive alcohol use, cirrhosis, and iron deficiency anaemia. In 1978, barium enema and colonoscopy had suggested a right colonic mass, but exploratory surgery revealed no neoplasm. In September 1980, ascites and personality changes had developed, both of which improved when spironolactone, hydrochlorothiazide, and neomycin were administered. In November 1980, polymyalgia rheumatica had developed; the patient responded well to prednisone therapy, 5 mg per day. Except for a change from neomycin to lactulose in 1981, medications had been the same, and his condition had remained stable. He had recently been drinking two to three ounces (60-80 g) of alcohol per day.

On 6 March 1984, the afternoon before elective cataract surgery, the patient was admitted to Kaiser Permanente Medical Center, Oakland, California. That evening, sudden, severe frontal headache developed, followed by vomiting and shaking chills. When examined, he was slightly confused without asterixis or papilloedema. His temperature was 40°C (104°F), pulse rate 112/min and regular, blood pressure 150/70 mm Hg, and respiration rate 18 per minute and unabated. His neck was supple. Results of a cardiovascular examination were normal. The abdomen was soft and not tender; the liver was firm 4 cm below the costal margin. There were no focal neurological deficits. Initial chest radiographs and urinalysis results were normal. A leucocyte count was 2,500/cu mm with 67 polymorphonuclear leucocytes (PMNs) and 18 band forms. A second physical examination nine hours later showed no change except for a stiff neck with pain on flexion. Lumbar puncture revealed an opening pressure of 27 cm H₂O and grossly cloudy cerebrospinal fluid (CSF). The CSF had a leucocyte count of 8,600/cu mm (98 PMNs, 2 lymphocytes), glucose of 110 mg/dl, protein of 610 mg/dl. Gram stain of the CSF was negative. Two of two blood cultures at 24 hr and CSF culture at 48 hr all grew Group D Streptococcus bovis. The minimal inhibitory concentration (MIC) and minimal bactericidal concentration (MBC) for penicillin were both less than or equal to 0.06 μg/ml.

The patient received a 14 day course of penicillin G, 18 million units intravenously per day. He had not clinically improved by the third day, and streptomycin 1 g intramuscularly per day was added. The MBC results were not available this time. The temperature returned to normal on the fifth day. Mental status returned to normal on the seventh day.

Repeated abdominal examinations showed no tenderness or ascites. Bone marrow aspiration, computed tomography (CT) scan of the head, two-dimensional echocardiogram, and barium enema results were all within normal limits. A dental examination showed diffuse periodontal disease, one loose left central incisor, and no evidence of abscess. At follow-up examination three weeks later, the patient was well, working, neurologically intact, and abstaining from alcohol.

Nonpneumococcal streptococcal species account for about 10% of meningitis. Group D streptococci are an uncommon cause of meningitis; few data exist on enterococcal meningitis.\(^3\) Information on nonenterococcal Group D meningitis is limited to isolated case reports.

Both enterococcal and nonenterococcal Group D streptococci cause endocarditis; Sbovis appears to cause endocarditis as frequently as enterococcus.\(^4\)\(^5\) Both infections are similar in clinical presentation but differ in two major aspects. First, Sbovis septicaemia occurs in the setting of underlying gastrointestinal disease, especially gastrointestinal neoplasms, and with oral disease. Second, Sbovis is usually susceptible to concentrations of penicillin easily achieved in the blood, whereas enterococcal endocarditis requires combined penicillin-aminoglycoside therapy for cure. Mortality is greater in enterococcal disease.\(^6\)

Differentiating between enterococcal and Sbovis meningitis has similar implications. A review of cases in the English language literature yielded eight reported cases of adult Sbovis meningitis, to which we have added our case. Three were associated with colonic disease, three with endocarditis, two with steroid use, and two with dental disease. Almost all occurred simultaneously with septicaemia. The clinical presentation included negative Gram stain of the CSF in six of seven cases and a CSF leukocyte count of less than 2,000/cu mm in five of eight specimens.

Gram stain was also negative in two adult cases of enterococcal meningitis reported by

---

Letters

Bayer et al² and in one reported by Lerner.¹

In Bayer’s review of 27 documented cases of Group D enterococcal meningitis, all but two had CSF leukocyte counts of less than 200/cu mm.

As in endocarditis, mortality is greater in enterococcal disease. Bayer et al³ reported 33% mortality in their review of enterococcal meningitis. In our review of S. bovis meningitis, six of seven patients had an excellent clinical response to antibiotics and mortality was 14%. Most cases of S. bovis meningitis (like endocarditis) respond to penicillin alone, whereas enterococcal disease may require intravenous or even intrathecal aminoglycoside therapy.³

S. bovis meningitis may be misdiagnosed as enterococcus if appropriate laboratory algorithms are not followed. Specific bacterial diagnosis allows the clinician to choose appropriate antibiotic therapy and to search for underlying gastrointestinal disease.

MARK A JACOBSON
ELIZABETH T ANDERSON
The Department of Medicine,
Kaiser Permanente Medical Center,
Oakland, California, USA.

Recurrent vertigo: cochlear-vestibular interaction

Sir: Recurrent vertigo is a commonly encountered clinical problem. Fortunately most of these patients can be satisfactorily classified even if no specific treatment is available.¹ There are, however, patients who do not comfortably fit established diagnostic categories leading to suggestions for additional “disease entities” such as benign recurrent vertigo, recurrent peripheral vestibulopathy, and disabling positional vertigo.⁴ The last of these is believed to be due to a specific aetiology of microvascular compression causing “hyperactivity” of the vestibular nerve. Surgical therapy has been suggested for this latter condition.

A patient seen recently did not fit any of the above categories but did have a syndrome suggesting abnormal conduction between auditory and vestibular nerves. The possibility of microvascular compression had been considered but surgery was deferred. The patient was subsequently treated with phenytoin with excellent resolution of his symptoms. This case suggests that anticonvulsant therapy is indicated in some patients with recurrent vertigo.

A 72 year old man noted the onset of tinnitus in the left ear 6 years previously and in the right ear 3 years previously. An acute attack of imbalance lasting half an hour occurred in 1979, followed by recurrent symptoms of a sensation of imbalance daily since that time. The sensation of imbalance was not rotational nor was it a sensation of fainting. There was a definite sensation of movement that might produce a fall but without a consistent direction. Symptoms of imbalance were not related to movement or position but were predictably produced by specific sounds. The sound of chewing lettuce for example would produce symptoms chewing other food would not. Sounds of similar frequency composition would also produce symptoms; however, loud sounds of other frequencies would not. Coughing, sneezing and pressing the ear had no effect. A left ear plug provided partial relief from symptoms produced by external sounds. There was a history of chronic atrial fibrillation but not other heart disease, hypertension nor diabetes. The only medication being used was 10 grains (650 mg) of aspirin per day.

Neurological examination was within normal limits including Hallpike tests. Fis-tula testing with Frenzel’s lenses was negative. Studies obtained in 1981 showed bilateral mild high frequency hearing loss with good discrimination and caloric studies showed a 29% left unilateral canal paresis. BAER showed a prolonged latency of wave III with an increased I-III interval with right ear stimulated and no reproducible responses with left-sided stimuli. CT scanning with posterior fossa emphasis with and without contrast was normal as was a posterior fossa pantopaque study.

Therapy was begun with phenytoin 300 mg/day and within 2 weeks symptoms were markedly improved with decreased dizziness and tinnitus and ear plugs were no longer required. The aspirin was continued in light of the patient’s age and history of atrial fibrillation even though transient ischaemia was not considered a likely explanation for his symptoms. Repeated attempts to decrease phenytoin dosage resulted in an increased frequency of attacks.

This case appears to be unique. The history does not fit the common syndrome of benign positional vertigo.5 nor are there sufficient features to suggest a Meniere’s syndrome.⁵ The absence of spontaneous vertigo since 1979 also makes diagnosis of benign recurrent vertigo⁶ or recurrent peripheral vestibulopathy⁷ unlikely. The major symptom of sound induced vertigo suggests an abnormal cochlear vestibular interaction. This effect is often labelled as “Tullio’s phenomenon”, although there is some inconsistency as to whether this term should include only a movement of the visual environment induced by sound or include any cochlear vestibular interaction. The usual implication, however, is that the effect is mechanical rather than electrical coupling occurring as a result of trauma or scarring in the middle ear.⁷ In this case the effect was clearly pitch or tone related and not related to volume of sound, making a mechanical explanation unlikely. It is more likely that the interaction was electrical at either the nerve or the brainstem level. Similar nerve to nerve ephaptic transmission has been demonstrated in “dystrophic” mice⁸ and postulated as a mechanism in the pain of causalgia.⁹

It cannot be determined with certainty in this case whether the interaction was at the level of the 8th nerve or brainstem. The absence of any central signs or symptoms, the increased I-III interpeak BAER latency on the right as well as a unilaterally depressed caloric would however favour a peripheral (nerve) origin for this patient’s symptoms.

Recently microvascular compression of the 8th nerve has been postulated as a cause of both vertigo and tinnitus and indeed surgical therapy has been reported to improve symptoms in many of these patients.⁴ Unfortunately there is no mention of trials of anticonvulsant therapy for these patients.

References


Accepted 19 January 1987

Reprint requests to Mark A Jacobson, MD,
San Francisco General Hospital, Ward 84,
Bldg 80, 995 Potrero Avenue, San Francisco, CA 94110, USA.
Streptococcus bovis meningitis.

M A Jacobson and E T Anderson

*J Neurol Neurosurg Psychiatry* 1987 50: 940-941
doi: 10.1136/jnnp.50.7.940

Updated information and services can be found at:
http://jnnp.bmj.com/content/50/7/940.citation

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Notes**

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/