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Otogenic *Fusobacterium necrophorum* meningitis

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Abstract
A case of meningitis secondary to acute suppurative otitis media in a previously healthy child is reported. The only organism isolated from blood after aerobic and prolonged anaerobic culture was identified as *Fusobacterium necrophorum*. Complete recovery followed treatment with surgery and prolonged antibiotic therapy. The role of anaerobes in the development of meningitis, the isolation and identification of *Fusobacterium necrophorum*, the clinical presentations of F. *necrophorum* infection and the choice of antibiotics in the treatment of these infections are discussed.

Introduction

Fusobacterium necrophorum may cause a number of clinical syndromes known collectively as Necrobacillosis. Examples are uncommon but the disease is potentially fatal. Diagnosis is often delayed by difficulties encountered in isolating the organism, and treatment is hampered by the uncertainty of several antibiotics in eradicating the infection in spite of *in vitro* sensitivity (Eyken, 1989). We report an uncommon case of meningitis complicating acute suppurative otitis media in which the causative organism was *Fusobacterium necrophorum*. Complete recovery followed treatment with a combination of surgery and antibiotics.

Case report

A previously healthy 5-year-old girl developed right-sided acute suppurative otitis media while on holiday in France. She made an apparently good recovery after a seven day course of an unknown antibiotic, but developed further right otalgia, headache, drowsiness and fever, a few days after stopping antibiotics. She was treated with erythromycin but within 48 hours her condition deteriorated with the appearance of profuse discharge from the right ear and symptoms of meningitis. Examination at this time confirmed signs of meningitis and the absence of any localising neurological signs. Pus was seen escaping through a small hole in the right tympanic membrane.

Lumbar puncture yielded turbid cerebrospinal fluid under increased pressure which contained 12,500 polymorphs per microlitre and a very low glucose content. No organisms were seen on microscopy of the CSF, and antigen tests against *Neisseria, Strep pneumonia, Haemophilus* and *Strep pyogenes* were all negative. No organism was isolated from CSF after aerobic and anaerobic culture. A gram-negative rod was isolated from blood after 72 hours in anaerobic culture (blood cultured in Fastidious Anaerobe Broth and subcultured in Vit K agar at one, three and seven days). The organism was identified as F *necrophorum* using standard techniques to the criteria of Holdman *et al.* (1977).

Immediately after lumbar puncture, treatment was started with intravenous penicillin and chloramphenicol in high dosage. A CT scan was carried out and was normal even after intravenous contrast. Initial improvement was soon followed by deterioration of the general condition and the development of a right lateral rectus palsy. This deterioration prompted the addition of metronidazole (125 mg intravenously eight hourly) to the treatment regimen.

In spite of adequate antibiotics the child's general condition deteriorated and within 48 hours of presentation a right cortical mastoidectomy was carried out. At operation, the mastoid mucosa was infected but there was no cholesteatoma or evidence of bony destruction. In particular the dura had not been exposed and the lateral venous sinus was healthy and patent. Following operation recovery was rapid and complete apart from the lateral rectus palsy which persisted for several months. Treatment with chloramphenicol was discontinued after 10 days, and with penicillin and metronidazole after four weeks from the time of surgery.

Discussion

Meningitis is the most frequent intracranial complication of middle ear infections, with most cases occurring after acute suppurative otitis media in children. Although the incidence of otogenic intracranial complications has fallen markedly since the arrival of antibiotics, mortality remains unacceptably high (Gower and McGuirt 1983).

The intracranial spread of infection from the middle ear may follow a number of possible routes. Infection may spread through a normal anatomical pathway such as the cochlea, or through a defect in the bony wall of the middle ear which may be congenital, traumatic in the form of an unhealed or growing fracture, following middle ear surgery, or as a result of erosion by cholesteatoma in cases of chronic suppurative otitis media. In the absence of any of these defects, infection may spread directly through necrosing bone or as a result of thrombophlebitis across intact bone. In most cases of acute otitis media infection appears to spread through necrozing bone (Ludman, 1987). In this case the bony wall of the middle ear was seen to be intact at operation. This, together with the presence of a bacteraemia suggests haematogenous spread of infection in this case.

Bacterial meningitis of whatever cause is rarely caused by anaerobic organisms (Finegold, 1977), but sporadic reports and small series of cases of meningitis due to anaerobic organisms have appeared in the literature (Tarnvik *et al.*, 1986; Tarnvik, 1986).

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The organisms most commonly responsible for acute otitis media, and consequently for otogenic meningitis, are *Haemophilus influenzae* and *Streptococcus pneumoniae* (Ludman, 1987). Anaerobic organisms are recognized pathogens in chronic middle ear infections but are not commonly associated with acute infections. F *necrophorum* was the only organism isolated from blood culture and must be presumed to be the organism causing acute otitis media and meningitis in this patient.

F *necrophorum* produces a severe septicaemic illness in previously healthy children and young adults. This most commonly follows a sore throat but, as in our case may follow middle ear infection. In a recent study of 45 cases of F *necrophorum* septicaemia (Eyken, 1989), 29 followed a sore throat, while in seven cases septicaemia followed acute middle ear infection. Although uncommon, F *necrophorum* infections are an important cause of morbidity and mortality which may be overlooked because of clinicians' unfamiliarity with the condition coupled with the difficulties encountered in isolation and identification of the organism.

F *necrophorum* is a non-spore forming, non-motile, strictly anaerobic gram-negative rod belonging to the family Bacteroidaceae which is normally resident in the gastro-intestinal tract, oral cavity, and female genital tract. Isolation of Fusobacteria in culture requires strict anaerobic conditions. Identification relies on the demonstration of the obligate anaerobic nature of the characteristic fusiform rods, the failure to take up Gram stains, the absence of spores, and most reliably the determination of fermentation products using gas liquid chromatography. Fusobacteria produce n-butyric acid, allowing differentiation from other members of the family Bacteroidaceae. (Barnes, 1979) F *necrophorum* is distinguished from other species by its production of lipase and by its production of propionic acid from lactate (Eyken, 1989).

Although F *necrophorum* shows an *in vitro* sensitivity to penicillin, metronidazole, and a number of other antibiotics except aminoglycosides, the correlation between *in vitro* sensitivity and clinical response is not straightforward. In our case the initial otitis media improved with antibiotic treatment although the general condition did not return to normal. Erythromycin, to which the organism was sensitive *in vitro*, did not prevent the development of meningitis even though it was used immediately upon reappearance of symptoms. This uncertain response to antibiotics is not peculiar to our case and reference to a similar progression of infection despite apparently appropriate antibiotics has been noted previously. (Moore-Gillon *et al.*, 1984; Tarnvik *et al.*, 1986; Eyken, 1989).

The choice of treatment in cases of F *necrophorum* infections is not yet certain. Several reports including ours indicate a role for metronidazole. Our patient improved after surgery and the addition of metronidazole. Moore-Gillon *et al.* (1984) refer to five cases of F *necrophorum* infection recovery in three of whom seemed to coincide with the addition of metronidazole. Tarnvik *et al.* (1986) obtained good results with cephuroxime and metronidazole. Eyken (1989) suggests a treatment regimen of metronidazole and penicillin.

Conclusion

Meningitis is still the commonest intracranial complication of middle ear infection. It is most commonly caused by aerobic organisms but can result from infection with anaerobic organisms. F. *necrophorum* must be considered in a previously healthy child with meningitis in whom bacterial culture under normal conditions fails to grow any organisms, and in whom the infection progresses in spite of appropriate antibiotics. Selection of antibiotics based on *in vitro* sensitivity, while helpful, does not always lead to the expected clinical improvement. Metronidazole appears to have an important role in the treatment of infections caused by this organism.

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