Group B streptococcal meningitis in a previously healthy adult

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Group B streptococcus (GBS; *Streptococcus agalactiae*) has emerged in recent years as an important cause of invasive infection in non-pregnant adults. However, GBS meningitis in this group is rare. The majority of cases reported previously have been in patients with underlying immunosuppression. Here we report a case of GBS meningitis in a previously healthy adult.

DISCUSSION

First recognized as an important cause of human infection in the early 1970s, GBS classically causes meningitis and septicaemia in neonates and infants less than 12 weeks of age. Approximately 3 infants per 1000 live births are affected. Pregnant women are also at risk with a ten-fold increased incidence of invasive GBS disease (Schwartz et al, 1991).

In recent years it has become increasingly apparent that GBS is also an important cause of disease in non-pregnant adults, this group now accounting for approximately one-third of the reported cases of invasive GBS disease. In this group, the incidence is highest in people above the age of 60 years. In 1997 there were 481 cases of GBS septicaemia in people over the age of 15 years in England and Wales (including pregnant women). This represents an approximate 50% increase compared to 1990 (personal communication, Communicable Disease Surveillance Centre, April 1998).

In non-pregnant adults, GBS disease usually presents as skin and soft tissue infection or septicaemia with no identified source. Other manifestations include osteomyelitis, urosepsis, pneumonia, peritonitis, endocarditis and meningitis.

In the majority of cases one or more predisposing factors are identified. The commonest of these is diabetes (seen in one third of affected patients). Malignancy, urological abnormalities, renal disease, cirrhosis and human immunodeficiency virus (HIV) infection are also associated (Farley et al, 1993).

GBS is an invasive organism but seldom causes acute inflammation at the portal of entry. Its appearance is therefore usually unheralded. While the

CASE REPORT

A 38-year-old male was admitted with a 12-hour history of headache, drowsiness and vomiting. He had no past medical history of note apart from an episode of otitis media that affected the right ear 8 years previously. On examination he was semi-conscious and pyrexial (temperature 39.0°C) with mild nuchal rigidity. There were no focal neurological signs. There was no rash. There were no other significant findings. Blood cultures were drawn and he was given 2 g of intravenous cefotaxime.

Full blood screen including glucose was normal apart from a white cell count of 11.5 $x10^9$/litre (range: 4–11$x10^9$/litre). Chest radiograph was normal. Urinalysis was normal (culture was subsequently negative).

A computed tomography (CT) scan of the head was normal. Lumbar puncture revealed purulent CSF at raised pressure. CSF analysis was as follows: 8500 white cells/mm^3 (all neutrophils), protein 7.8 g/litre, glucose <0.1 mMol/litre (plasma glucose 6.5 mMol/litre). Gram-positive cocci in pairs were identified on the gram stain. The appearance was not typical of *Streptococcus pneumoniae* in that the streptococci were not lanceolate in shape, although it was considered that the action of the antibiotics given before the lumbar puncture may have accounted for this. A diagnosis of probable pneumococcal meningitis was made and he was started on intravenous benzylpenicillin (1.2 g 2-hourly). However, group B streptococcus was subsequently cultured both from the CSF and blood (sensitive to penicillin). Given the history of otitis media and the possibility of an active source of sepsis arising from the middle ear, cefotaxime (2 g intravenously 8-hourly) and gentamicin (5 mg/kg intravenously per day) were added in. Following the lumbar puncture he was transferred to intensive care where he was intubated in view of his declining level of consciousness.

He was extubated 48 hours later. At this time, it was noted that he had developed mild right-sided weakness. A thorough ear, nose and throat examination was performed: no parameningeal focus for infection could be identified. A repeat CT scan of the head and sinuses at this stage was normal.

His clinical progress was rapid. Within 1 week he was walking unaided and had no residual signs. Three weeks after admission, he was clinically well, although he continued to have a low grade fever. No relevant clinical signs, particularly those of endocarditis, were identified. Repeat blood cultures, urine cultures and chest radiograph were normal. Transthoracic echocardiography was normal. Magnetic resonance imaging of the brain, sinuses and cord was normal, with no evidence of a subdural or epidural collection of pus. A repeat lumbar puncture was within normal limits.

At this stage a decision was made to discontinue the antibiotics and take multiple further cultures. However, within 72 hours defervescence occurred and the patient remained apyrexial. An antibiotic-associated fever was therefore considered to be a likely cause of the low grade pyrexia. He was discharged 4 weeks after admission, having made a full recovery. A human immunodeficiency virus test was negative. Immunoglobulin and complement levels were normal.

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majority of cases in infants are acquired vertically, the source of infection in adults is less clear. GBS is found in the genital tract in up to 25–35% of healthy non-pregnant women (Dillon et al, 1987) as well as in the perineum, rectum and oropharynx in men and women and in the male urethra. It is therefore likely that the genitourinary tract is the reservoir and the primary site for dissemination of infection in many cases (Dunne and Quagliarello, 1993).

GBS meningitis accounts for only 3–4% of cases of invasive GBS disease in non-pregnant adults (Farley et al, 1993). Only two cases of GBS meningitis were reported in adults in England and Wales in 1997 (personal communication, Communicable Disease Surveillance Centre, April 1998). Males and females are equally affected. There is a bimodal distribution with peaks of incidence in the third and eighth decades (Dunne and Quagliarello, 1993).

The majority of patients affected either have classical risk factors for GBS infection or a distant focus of infection. Although post-neurosurgical cases and spread from parameningeal foci have occasionally been reported, haematogenous spread to the meninges is thought to occur in the majority, as evidenced by the high incidence of positive blood cultures (in 94% of cases). A primary extracranial source of infection (e.g. endocarditis, soft tissue infection) is found in 30% of cases (Sarniento et al, 1993).

The clinical presentation and CSF findings are similar to those found in other causes of bacterial meningitis (Dunne and Quagliarello, 1993). The mortality of meningitis is similar to that for other invasive GBS syndromes and is higher in the elderly (18% in those over the age of 60 years; Schuchat et al, 1997) and those with co-morbid disease (Dunne and Quagliarello, 1993).

The treatment of choice is high dose (20–30 mega units/day) intravenous benzylpenicillin for at least 14 days. Although uniformly sensitive to penicillin, minimum inhibitory concentrations are four to eight times higher for GBS than group A streptococci. Penicillin may be combined with gentamicin for antimicrobial synergy if the strain of GBS is penicillin tolerant (although this is unusual) or in those with significant co-morbid disease. In penicillin allergic individuals, third generation cephalosporins or vancomycin may be used.

CONCLUSIONS

A case of GBS meningitis in a previously healthy adult is reported. No evidence of immunosuppression nor of a distant or parameningeal focus of infection could be identified. The distant history of otitis media is unlikely to be relevant: no evidence of active middle ear or sinus disease could be identified. Cases such as this appear to be extremely rare (Sarniento et al, 1993).

Although initially misdiagnosed as pneumococcal meningitis, this did not affect the outcome as the early treatment for both conditions is similar.

Although the importance of GBS infection in neonates and infants is well known, its importance as a cause of invasive infection in adults is under-appreciated. Clinicians are likely to become increasingly aware of GBS as an important cause of morbidity and mortality in adults, particularly the elderly and those with chronic illness. HM

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