CASE REPORT

Group B streptococcal meningitis in a previously healthy man

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SUMMARY

Group B *Streptococcus* (GBS) is an infrequent cause of meningitis in adults, usually affecting elderly patients and those with serious underlying disease. It is more commonly recognised as one of the leading aetiological agents of neonatal sepsis following maternally derived infection during pregnancy. We report a case of a previously healthy 26-year-old man who presented with fevers, confusion and headache. Lumbar puncture results were consistent with bacterial meningitis, and blood cultures grew GBS. To the best of our knowledge, our patient represents one of the few reported cases of GBS meningitis in a previously healthy young man. Interestingly, our patient had a significant family history of central nervous system infection including a son with herpes simplex virus encephalitis, a sister with meningococcal meningitis and a great-uncle with meningitis of unknown cause. We discuss genetic factors that may predispose certain people to develop meningitis with normally harmless microorganisms such as GBS.

BACKGROUND

Group B *Streptococcus* (GBS), or *Streptococcus agalactiae*, is an infrequent cause of meningitis in adults, usually affecting elderly patients and those with serious underlying disease.¹ It is more commonly recognised as one of the leading aetiological agents of neonatal sepsis following maternally derived infection during pregnancy. We report a case of a previously healthy 26-year-old man with GBS meningitis, and an interesting family history of central nervous system (CNS) infections.

CASE PRESENTATION

A 26-year-old man with no significant comorbidities, presented with a 4 h history of worsening severe headache, photophobia and confusion. He denied preceding bowel, urinary or chest symptoms, and did not report any history of recent skin or soft tissue injuries.

Interestingly, the patient had a significant family history of CNS infection. His newborn son developed herpes simplex virus (HSV) type 1 encephalitis 8 days following birth, passing away on day 12; his sister had meningococcal B septicaemia at 2 years of age; and a great-uncle was diagnosed with meningitis of unknown origin.

On initial presentation, the patient was febrile at 40°C, tachycardic and extremely agitated. There was no visible rash, and examination of the chest and abdomen was unremarkable. Physical examination did not identify any focal neurology or signs of raised intracranial pressure; however, the patient

was drowsy, with a Glasgow Coma Score varying between 10/15 and 13/15.

INVESTIGATIONS

Lumbar puncture revealed cloudy cerebrospinal fluid (CSF) with a high white cell count of 2140/mm³ (90% polymorphs), high protein of 4.7 g/L and low glucose of 2.5 mmol/L; results consistent with bacterial meningitis (figure 1). Viral PCR for HSV, varicella zoster virus and enteroviruses was negative. Initial microscopic examination failed to detect any bacterial organisms and CSF culture was negative at 48 h incubation. This may have been attributed to CSF sterilisation following the administration of the first dose of antibiotics approximately 12 h prior to lumbar puncture. Blood culture, however, was positive for GBS after <24 h and, as such, this was deemed to be the causative bacterium.

MRI of the brain was normal. Chest X-ray was unremarkable and there was no evidence of endocarditis clinically or on transthoracic echocardiogram. Serum immunoglobulins and complement were normal and HIV testing was negative. No source for GBS infection was identified.

TREATMENT

The patient was initially treated with intravenous aciclovir, ceftriaxone and dexamethasone, as both viral encephalitis and bacterial meningitis were considered differential diagnoses. He was managed under close observation on an acute medical ward, and intensive care support was required in the form of sedation for CT and lumbar puncture due to the patient's severe agitation at that point in time. Antimicrobial treatment was changed to ceftriaxone and low-dose gentamicin 3-day later, when blood cultures grew GBS and viral PCR was negative. Gentamicin was stopped after a further 3 days once endocarditis had been excluded.

OUTCOME AND FOLLOW-UP

The patient survived and made a rapid improvement within 48 h of antibiotic treatment. He completed a 14-day course of intravenous ceftriaxone. Fortunately, he suffered no neurological sequelae on discharge, likely attributable to the fact that he was young, and previously fit and healthy, with no significant underlying medical conditions. He is due for follow-up audiology testing and a repeat echocardiogram.

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CSF variable	Result	Normal range
Physical appearance	Cloudy	
Gram stain	+++ WBC (90% polymorphs) No organisms seen	
White blood cells/cmm	2140	
Red blood cells/cmm	470	
Xanthochromia	Negative	
CSF protein	4.7g/L	0.15 – 0.8g/L
Serum protein	65g/L	66 – 83g/L
CSF glucose	2.5mmol/L	2.2– 3.9mmol/L
Serum glucose	7.1mmol/L	<11.1mmol/L

Figure 1 CSF results (CSF, cerebrospinal fluid; WCC, white cell count).

DISCUSSION

The rate of GBS infection in all adult age groups is thought to be increasing, with an estimated twofold to fourfold increase in the incidence of invasive GBS infections in non-pregnant adults reported in the past two decades.² However, reports of adult meningitis secondary to GBS remain rare, with an estimated incidence of 0.15 cases per 100 000.¹ The majority of reported cases outside the peripartum period have been in elderly patients or those with serious underlying comorbid conditions, such as diabetes mellitus and other immunosuppressive diseases.³ To the best of our knowledge, this patient represents one of the very few reported cases of GBS meningitis in a previously healthy young man.

Clinically, GBS meningitis is indistinguishable from meningitis caused by other pyogenic bacteria, with an acute onset and a high incidence of neurological dysfunction. However, GBS meningitis is frequently rapidly progressive with a higher proportion of patients with GBS presenting within 24 h of the onset of symptoms than those with other forms of bacterial meningitis.³ In addition, the case fatality rate associated with GBS meningitis is considerably higher than that associated with meningococcal meningitis, at around 27–34%.^{4 5}

Although our patient's MRI scan did not demonstrate any gross anatomical abnormality that would predispose to meningitis, his concerning family history raised the possibility of an underlying inherited genetic susceptibility. Colonisation with GBS is common—it is estimated that up to 31% of young men are colonised,⁶ and its presence does not generally result in disease in healthy adults.

Several studies have investigated the association between genetic factors and the risk of developing meningitis. Abnormalities in complement pathways have been implicated in susceptibility to bacterial meningitis,⁷ and genetic polymorphisms in toll-like receptors involved in pathogen recognition have been identified in patients who have suffered from bacterial infections and viral encephalitis.^{8 9} Greater awareness of the genetic factors that influence an individual's susceptibility to meningitis may facilitate early recognition and treatment of the

disease as well as extending vaccination programmes to at-risk groups.

Interestingly, we were unable to identify the focus of GBS infection in our patient, despite reports in the literature describing the most frequent distant foci to be the endometrium, respiratory tract and the endocardium.² However, with our patient's family history of bacterial meningitis and viral encephalitis, and the fact that many in the population are harmlessly colonised with GBS, it remains to be answered whether there is a specific component in this patient's genetic makeup that has made it more likely for what is ordinarily a harmless bacteria to cause life-threatening meningitis.

Learning points

- ▶ Group B *Streptococcus* (GBS) is an infrequent cause of adult meningitis, but it carries a high case fatality.
- ▶ GBS meningitis must not be considered exclusive to the peripartum infectious period or to patients with comorbid illnesses.
- ▶ Further research is required to characterise a genetic predisposition to bacterial and to viral central nervous system infections.

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Competing interests None declared.

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