Group A Streptococcal Meningitis

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Group A β-haemolytic streptococci are usually associated with skin infections and pharyngitis. Rarely they may cause serious infections like septicaemia or meningitis. We describe an infant who presented with meningitis caused by Group A β-haemolytic streptococci and had serious complications.

Group A β-haemolytic streptococci (GABHS) are commonly associated with cutaneous infections and pharyngitis. Although a frequent cause of bacteremia in the preantibiotic era, presently they are rarely associated with bacteremia or meningitis. After many years of low prevalence the non-suppurative sequelae such as rheumatic fever and glomerulonephritis are on the rise again in Western countries.1-3 We describe a case of meningitis caused by GABHS with severe illness and a complicated course characterized by hydrocephalus and seizures.

Case Report
A 2½-month-old Saudi male child was admitted to Suleimania Children’s Hospital, Riyadh with a history of fever, excessive crying, irritability and poor sucking for 1 day. There was no history of vomiting, diarrhoea or any significant illness. He was the product of a full-term, normal vaginal delivery. There was no history of any significant illness in the past. He was immunized with BCG only. On examination his temperature was 38.6 °C, heart rate 160/min, and respiratory rate 28/min. His weight was 6.3 kg, height 62 cm and occipitofrontal circumference 42 cm. He was a well-hydrated but extremely irritable child. The anterior fontanelle was full. Examination of the ears, nose, throat, lungs, heart and abdomen was essentially unremarkable. There was no nuchal rigidity. Kernig’s and Brudzinski’s signs were negative. Laboratory findings on admission included haemoglobin 9.4 g/dl, white blood cells 15.8 × 10^9/l with 79% polymorphs, 18% lymphocytes and 3% monocytes. Serum sodium was 134 mmol/l, potassium 4.4 mmol/l, glucose 8.3 mmol/l and urea 4.5 mmol/l. Lumbar puncture revealed a turbid cerebrospinal fluid (CSF). The CSF cytology showed white blood cells 29.2 × 10^6 cells/l, polymorphs 94% and lymphocytes 6%. CSF glucose was 0.11 mmol/l, protein 65.6 mg/l. The culture of the CSF grew GABHS. Blood culture showed no growth. He was started on treatment with ceftriaxone and penicillin. On the second day he started to have generalized convulsions which were controlled with anticonvulsants. He became afebrile after 4 days and remained so until the 15th day. He was given antibiotics for 14 days. Follow-up CSF examinations revealed progressively reducing pleocytosis and no growth on culture. There was no change in the occipitofrontal circumference but ultrasound examination of brain showed the lateral, third and fourth ventricles increasing in size. A CT scan of brain revealed normal ventricles with bifrontal atrophy initially but follow-up CT scan showed gross dilatation of the ventricles suggesting the presence of a communicating hydrocephalus. A ventriculoperitoneal shunt was inserted on day 30. His stay in the hospital was also complicated by a nosocomial infection with Serratia marcescens septicaemia which responded to a course of cefotaxime and amikacin.

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Brainstem auditory evoked response performed after completion of treatment was normal. He has been developing normally on follow-up after 1 year. He has started walking with support, and speaks a few single words. The occipitofrontal circumference is 48 cm and the hearing is normal. The ventriculoperitoneal shunt is functioning normally.

Discussion

The group A β-haemolytic streptococcus has never been a common cause of childhood meningitis, but is much less frequent now than in the first half of this century. Nyhan & Fouke recorded 16 GABHS isolates among 37 isolates from blood culture of neonates in 1933 and 1934, but only five of 69 such isolates in 1944–1957. Until approximately 30 years ago GABHS was the leading cause of puseral and neonatal sepsis but was then replaced by Gram negative organisms and more recently by group B streptococci. Nursery outbreaks or sporadic neonatal cases of infection with GABHS still occur on occasion, but most are mild illnesses with cutaneous involvement. Infrequently, illness may be in the form of sepsis and meningitis. Tripoli in 1936 found that 5% of cases of bacterial meningitis in New Orleans were caused by streptococci, the specific group not being known. In 1938 Neal reviewed 3502 cases of bacterial meningitis in all age groups in New York City; 8% were streptococcal, presumably group A. Murphy in a retrospective review documents the severity of meningitis caused by this aetiological agent.

Prior to the availability of sulphonamide preparations the case fatality rate with streptococcal meningitis was 95%. An increasing number of bacteremias, suppurative and non-suppurative GABHS infections have been reported recently from various countries. Yagupsy & Giladi reported 20 children with GABHS bacteremia over a 7-year period; two of these had meningitis and recovered after 12 weeks of antibiotic therapy. Harnden & Lennon reported from New Zealand 13 patients with severe GABHS infection over a 4-year period; 11 of them had bacteremia, whilst one had meningitis.

Two previous reports from Riyadh and Tabuk describing childhood bacterial meningitis do not include any cases caused by GABHS. Abomelha & Uduman et al. from the eastern province of Saudi Arabia reported 105 cases of bacterial meningitis including two caused by GABHS but did not give any details.

In summary GABHS can cause a severe meningitis with frequent complications. Our report in conjunction with others suggests that a worldwide change in the virulence of GABHS may have occurred recently.

References


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