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Case Report
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Uncommon clinical presentation of a common bug: Group A *Streptococcus* meningitis

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Abstract

Group A *Streptococcus* (GAS) is a common pathogen in paediatric infections. However, it is a rare etiologic agent of bacterial meningitis. We describe a case of *Streptococcus pyogenes* meningitis complicated by sensorineural hearing loss in an immunocompetent 7-year-old boy. Clinicians should be aware of GAS as a potential cause of paediatric meningitis, especially with prominent symptoms suggestive of frontal sinusitis. Meningitis caused by GAS has been shown to be associated with significant mortality and morbidity, including neurological complications. Early screening for sensorineural hearing loss in patients with GAS meningitis can facilitate timely cochlear implant.

Keywords: *Bacterial; Hearing loss; Meningitis; Sensorineural; Streptococcus pyogenes*

Group A *Streptococcus* (GAS), also known as *Streptococcus pyogenes*, causes a wide clinical spectrum of diseases in children. Most clinicians are familiar with mild and easily treatable GAS infections, such as acute pharyngitis, impetigo, and scarlet fever. However, GAS can also cause severe invasive infections, including necrotizing fasciitis and streptococcal toxic shock syndrome (1).

In this report, we describe a case of GAS meningitis complicating acute sinusitis in an immunocompetent child. The child developed an important neurological complication, sensorineural hearing loss (SNHL), which requires time-sensitive management (2).

CASE PRESENTATION

A fully-immunized 7-year-old boy presented to a paediatric emergency department (ED) with acute left hip pain and inability to weight bear. He had a past medical history of asthma and no prior invasive infections. He had a 3-day history of fever up to 40°C and frontal headache, and a 10-day history of cough, and nasal congestion, suggestive of an upper respiratory tract infection (URTI). He had been using salbutamol inhalers at home. At presentation, he was febrile (38.9°C). He had wheezing and tenderness to palpation over the frontal sinuses. Hip examination revealed diffuse left hip pain with active range of motion but no pain with full passive range of motion. His left hip X-ray was normal. His white blood cell (WBC) count was unremarkable, erythrocyte sedimentation rate was elevated to 42 mm/hour (N: < 34 mm/hour) and C-reactive protein (CRP) level was mildly elevated to 29 mg/L (N: < 8 mg/L). After a dose of ibuprofen in ED, the hip pain resolved promptly, leading to the presumptive diagnosis of transient synovitis. He was prescribed a 3-day course of oral dexamethasone (0.3 mg/kg/day) and salbutamol for presumed viral-induced asthma exacerbation and was discharged home. Blood culture drawn at this visit was negative.

He returned to the ED 2 days later (12 days after the onset of URTI symptoms) due to persistent fever and increasingly

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severe frontal headache with photophobia. The hip pain did not recur. On exam, he appeared unwell and had meningeal signs. A lumbar puncture was performed. He received empiric IV ceftriaxone and vancomycin for presumed bacterial meningitis. He subsequently became hypotensive requiring fluid bolus, transient epinephrine infusion and admission to the paediatric intensive care unit.

Laboratory tests showed an increase in WBC count to 22.6 × 10^9/L (80% neutrophils), an erythrocyte sedimentation rate of 56 mm/hour and a CRP of 168 mg/L. He had cerebrospinal fluid (CSF) pleocytosis with a total nucleated cell count of 300 × 10^6/L (88% neutrophils), a red blood cell count of 1 × 10^6/L, CSF glucose of 3.8 mmol/L (N: 2.0 to 4.4 mmol/L) and CSF protein of 0.62 g/L (N: 0.15 to 0.60 g/L). Blood and CSF cultures both grew *S pyogenes* (*emm* type 1, subtype 1.0).

MRI head on the second and fifth days of admission showed abnormal meningeal enhancement over the left cerebral hemisphere, and substantial opacification of the left maxillary and left ethmoid sinuses, and almost complete opacification of the left frontal sinus. There were no signs of deep venous sinus thrombosis or abscess. His antimicrobials were narrowed to IV penicillin G at 400,000 units/kg/day. His cough improved within a few days and headache subsided over the first week of admission. The WBC and CRP count normalized by day 8 of admission. He completed 14 days of intravenous antibiotic therapy followed by an additional 7-day course of oral amoxicillin at discharge for presumed chronic sinusitis. He did not receive corticosteroids during his hospitalization.

At discharge, the patient appeared to have a full recovery but audiology testing 16 days later revealed profound right-sided SNHL. He underwent a successful unilateral cochlear implant 4 weeks after initial presentation.

DISCUSSION

GAS is not a classic pathogen of paediatric bacterial meningitis, accounting for about 2% of cases where the pathogen was identified (3,4). However, GAS meningitis is associated with significant mortality and morbidity (5–7). Neurological complications appear to be more common in children than in adults (7,8). In a case review of adults and children with GAS meningitis, 13 of 51 (25%) cases developed neurological complications, including nerve palsies, SNHL and motor deficits (8). The use of corticosteroids prior to antibiotics in children with bacterial meningitis has been associated with a lower rate of SNHL and other neurological sequelae, particularly in meningitis secondary to *Haemophilus influenzae* (9). However, given its low incidence, the role of corticosteroids in GAS meningitis in preventing SNHL will probably never be established. Meanwhile, early screening and identification of SNHL can facilitate timely cochlear implantation prior to labyrinthine ossification (2).

Previous case series have identified risk factors for GAS meningitis, including the neonatal period, presence of a ventriculo-peritoneal shunt and immunocompromising conditions (6,8). The definite pathogenesis of GAS meningitis remains unclear. GAS meningitis can be secondary to bacteremia or intracranial extension of head and neck infections, but cases have been reported in children with no identified continguous focus (5–7). In a literature review by Shetty et al, 12 out of 29 (41%) paediatric cases of GAS meningitis had a distant focus of infection, primarily in the ear, nose, and throat (7). Although the sinusitis pathogen was not identified in the current case, it was likely GAS given the concomitant *S pyogenes* bacteremia and meningitis. A history of new fever and worsening symptoms during recovery from a URTI should lead to consideration of a superimposed bacterial process.

The GAS surface M protein, encoded by the *emm* gene, is linked to the virulence factor of the bacteria. Over 200 *emm* types have been identified (10). *Emm* type 1 (identified in the current report) is consistently the most prevalent type in invasive GAS in North America and parts of Europe, while more diverse *emm* types cause invasive GAS infections in other parts of the world (5,6,11,12).

CONCLUSION

GAS is a rare cause of paediatric bacterial meningitis and is often associated with significant neurological complications. Clinicians should be aware of GAS as a potential cause of paediatric meningitis, especially with evidence of frontal sinusitis at presentation. Screening for hearing loss should be performed during the initial admission for GAS meningitis.

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DISCLAIMER

The views expressed in the submitted article reflect those of the authors and not an official position of the University of Ottawa or Children's Hosiptal of Eastern Ontario.

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