FATAL GROUP A STREPTOCOCCAL MENINGITIS IN AN ADULT

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Abstract

Despite the recent resurgence in reports of invasive Group A Streptococcal (GAS) infections worldwide, it remains a rare cause of pyogenic meningitis both in children and adults. We report a case of fatal GAS meningitis in a healthy adult emphasizing the need for clinicians to be aware of its fulminant course, prompting early diagnosis and treatment. There is also a need to consider postexposure chemoprophylaxis in close contacts of such cases.

Key words: Group A streptococcal meningitis, Streptococcus pyogenes

Group A streptococcal (GAS) invasive disease is being increasingly reported worldwide in recent years and has reemerged as a public health threat. However pyogenic meningitis caused by GAS is uncommon both in children and adults accounting for less than one percent of all cases of meningitis in the antibiotic era. Recently, fulminant cases of pyogenic meningitis caused by GAS in children and adults have been reported. However, only a few cases of GAS meningitis with a favourable outcome in the paediatric age group, have been reported from India.

We report a case of GAS meningitis in a previously healthy adult with a fulminant course and fatal outcome.

Case Report

A previously healthy, 18-year-old male was admitted in our hospital with a two-day history of fever and headache associated with vomiting and four episodes of generalized tonic clonic seizures prior to admission. Each episode of seizure lasted for about five minutes and was associated with uprolling of eyeballs, frothing from the mouth and urinary incontinence. After the last episode of seizure the patient lapsed into an altered sensorium. There was no history of an upper respiratory tract infection, ear discharge, head injury or any cranial surgery. His past medical history was unremarkable and there was no family history suggestive of any significant illness, including tuberculosis.

On admission, the patient was comatose, with a pulse of 89/minutes, blood pressure of 110/70 mm of Hg and a temperature of 100°F. Pupils were equal and sluggishly reacting to light. Bilateral subconjunctival haemorrhages were noted. The respiratory, cardiovascular and abdominal examination was non-contributory. No skin rashes were detectable. Neurological examination revealed the patient to be in altered sensorium. There was nuchal rigidity, absence of spontaneous movement and hypotonia of all four limbs. A glucometric random blood sugar level was 132 mg/dL. A provisional clinical diagnosis of status epilepticus with viral meningoencephalitis was considered. Empirical treatment with parenteral mannitol, ceftriaxone and phenytoin was instituted but his condition continued to deteriorate necessitating intubation and assisted ventilation. Despite these supportive measures, he succumbed within one hour of admission.

In view of the fulminant course in an apparently healthy adult, consent for a partial autopsy confined to examination of the brain alone was obtained from close relatives of the deceased, to ascertain the nature of illness and cause of death. An immediate postmortem lumbar puncture revealed a turbid cerebrospinal fluid (CSF) with 450 cells/cumm (60% lymphocytes and 40% polymorphonuclear cells). The brain at autopsy revealed a thick yellowish green exudate that covered the superolateral surfaces, coursing along the cortical veins to dip into the sulci. The coronal slices of the brain did not reveal any focal lesions, abscesses or ventriculitis. The brain stem and cerebellum were normal. Histological examination of sections taken from the cortex revealed dense infiltrate of polymorphs admixed with mononuclear cells flooding the subarachnoid space suggestive of a pyogenic meningitis. The entrapped cortical vessels were deeply congested. No infiltration of parenchyma by inflammatory infiltrate was noted. The histological section of the brain also showed grampositive cocci in the midst of acute inflammatory exudate in the subarachnoid space.

Gram stain of the postmortem CSF as well as of the exudate covering the brain revealed numerous gram-positive cocci in chains. Culture on blood agar yielded β haemolytic streptococci identified as GAS (Streptococcus pyogenes) by standard bacteriological methods and confirmed by latex agglutination test using Streptex kit (Remel, USA). The isolate was sensitive to penicillin, ceftriaxone, erythromycin, vancomycin, ciprofloxacin, amikacin and tetracycline.
Contact A. Superolateral surface of brain showing thin purulent exudates along parasagittal area obscuring superficial veins over frontal lobes (arrows), B. Photomicrophotograph reveals dense acute inflammatory exudates within subarachnoid space (SAS) (haematoxylin and eosin, x80), C - Gram stain of CSF showing gram positive cocci in chains (x480)

Discussion

Although GAS are frequent colonisers of the oropharynx, meningitis due to GAS usually occurs in association with upper respiratory tract infections, otitis media, sinusitis, head-injuries or cranial surgery. The exact source of infection in our patient could not be ascertained. Although children may develop a fulminant form of GAS meningitis, unresponsive to prompt institution of appropriate antibiotics, adults with GAS meningitis tend to have a more favorable outcome. However, recently a few cases of GAS meningitis in adults associated with a rapid course and fatal outcome have been reported. Our patient had a similar fulminant course and died even before an antemortem diagnosis could be made.

In a nation-wide retrospective review of 41 adults with GAS meningitis over a 14-year period in Netherlands a high rate of seizures (32%), focal neurologic deficits (36%) and hyponatremia (58%) apart from fever and neck-stiffness as the common clinical manifestations have been reported. A high mortality (27%) and neurological sequelae in 36% of the survivors was documented.

Despite the emergence of resistance to several antibiotics, GAS are still sensitive to most antibiotics, with penicillin being the first choice of treatment. Studies among household contacts suggest that GAS causing severe disease in one person is more likely to cause disease of similar severity if transmitted to others. Public health guidelines in Canada where invasive GAS disease is nationally notifiable since 2000, recommend prophylaxis for household contacts and close contacts of certain invasive GAS diseases, including GAS meningitis, since several clusters of invasive GAS infections have been described in various settings.

Enhanced surveillance and documentation of GAS meningitis and other invasive infections can help assess the disease burden in India, define the risk to close contacts and aid in formulating guidelines for management of such cases and prophylaxis for contacts.

Though GAS is a rare cause of meningitis, the present report underscores the need for clinicians to be aware that GAS meningitis can have a fulminant course in adults, warranting prompt diagnosis and aggressive therapy. There is also a need to consider post exposure antibiotic prophylaxis in close contacts of such cases.

References


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