Streptococcus agalactiae Meningitis in Adults: Report of Two Cases

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Abstract

Two cases of *Streptococcus agalactiae* meningitis in adults are reported. The first patient was a 40-year-old man who presented with acute fever, headache, stiffness of the neck and confusion. During treatment, he developed left hemiparesis from cerebral infarction and bilateral deafness. The other was an 80-year-old man who presented with acute confusion and stiffness of the neck. During treatment, he developed septic shock and generalized tonic-clonic convulsions. Diagnosis was established by latex agglutination of streptococcus B-antigen and confirmed by cerebrospinal fluid-culture later on. The first patient survived but continued to have deafness whilst the other died from septic shock.

Key word : *Streptococcus agalactiae,* Meningitis, Adults

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Streptococcus agalactiae or group B streptococcus (GBS) is a major cause of meningitis and septicemia in neonates, but meningitis caused by this organism rarely occurs in adults[1]. The authors reported two additional cases of GBS meningitis in adults.

CASE REPORT

Case 1:

A 40-year-old man was admitted to Srinagarind Hospital in December 1994 with the chief complaint of severe headache with vomiting for 10

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hours and confusion for 2 hours. He had been well until one day prior to admission; he experienced mild headache, which was relieved by acetaminophen. There was no history of recent common cold, sinus, ear, or genitourinary infection.

Physical examination showed a stuporous man with body temperature of 38.5°C and stiffness of the neck. The tympanic membranes were clear. Other findings were unremarkable.

The peripheral white cell count (WBC) was 10,800 cells/mm^3 with 82 per cent polymorphonuclear (PMN) cells. Serum glucose, BUN and creatinine, electrolytes, urinary analysis and chest X-ray were within normal limits. Serum VDRL and TPHA and anti-HIV antibody were non-reactive. A mildly traumatic lumbar puncture showed cloudy cerebrospinal fluid (CSF) with a protein level of 772 mg/dL and a glucose level of 10 mg/dL (simultaneous serum glucose level of 116 mg/dL). The red blood cell count was 2,020 cells/mm^3 and the WBC count was 9,600 cells/mm^3 with 98 per cent PMNs. Gram's stain revealed moderate intracellular Gram-positive cocci and latex agglutination test for bacterial antigen was positive for GBS.

The patient was treated with intravenous penicillin G 24 million units/day. On the next day, he developed mild left hemiparesis. CT scan of the brain showed a small brain infarction at the right high parietal region. Three days later, the results of hemocultures and CSF culture grew group B beta-hemolytic streptococcus. His mental status gradually improved within 5 days after treatment and body temperature returned to normal on the 7^{th} day of treatment, but we then found that he was unable to hear. Audiogram revealed bilateral deafness. He received penicillin for 14 days. On follow-up 2 months later, his motor weakness had returned to normal power but he continued to have bilateral deafness.

Case 2:

An 84-year-old man was admitted to Srinagarind Hospital in July 1998 because of disorientation for 5 hours. Significant history revealed abdominal surgery for peptic ulcer perforation, which occurred about 13 years ago.

Physical examination showed an old man in a confused state with normal body temperature and stiffness of the neck. Other findings were unremarkable.

The peripheral WBC count was 8,100 cells/mm^3 with 62 per cent PMN cells with toxic granules and 20 per cent band forms. The platelet count was 66,000 cells/mm^3. Serum BUN and creatinine was 49.2 and 3.4 mg/dL respectively. Serum bicarbonate was 15.5 mEq/L. Liver function test showed a total bilirubin level of 2.3 mg/dL, direct bilirubin level of 1.2 mg/dL and SGOT level of 66 mg/dL. Serum glucose, urinary analysis and chest X-ray were within normal limits. Abdominal sonography demonstrated gallstones. CT scan of the brain revealed some degree of brain atrophy. Lumbar puncture revealed normal opening pressure. The WBC count was 77 cells/mm^3 with 85 per cent PMNs. Protein level was 246 mg/dL and glucose level was 10 mg/dL (serum glucose level of 91 mg/dL). No bacteria were seen with Gram's stain. Latex agglutination test for bacterial antigen was positive for GBS.

The patient was treated with intravenous ceftriaxone 4 g/day. On the next day, he developed septic shock and generalized tonic-clonic convulsions and finally, he died on the 6^{th} day of hospitalization. The result of the CSF culture grew group B beta-hemolytic streptococcus.

DISSCUSSION

The most common source of GBS is probably the vagina[2], which accounts for the mechanism of neonatal sepsis and meningitis and a portion of the disease in adults. Also, this organism can be found in the rectum and the throat[2]. In adults, clinical manifestations caused by this pathogen include skin, soft-tissue or bone infection; bacteremia; urosepsis; pneumonia; peritonitis; pharyngitis; endometritis; endocarditis; arthritis and meningitis. In adults, GBS meningitis rarely occurs. The clinical manifestations of GBS meningitis and CSF findings are indistinguishable from other forms of acute pyogenic meningeal infection. Domingo et al[1] revealed that 86 per cent of the patients had comorbid conditions and 50 per cent had a distant focus of infection. The mortality rate was 34 per cent. Factors associated with a poor prognosis were advanced age and the presence of complications: neurological complications such as coma or focal neurological signs and/or extraneurological complications such as shock, acute respiratory failure, acute renal failure or consumption coagulopathy, as in case 2. In general, most of the survivors have complete recovery. Neurological sequelae include transient facial palsy,

tetraplegia, asymmetric hearing impairment and bilateral deafness.

In case 1, who developed bilateral deafness and cerebral infarction, the mechanism of cerebral infarction should be secondary from vasculitis[3]. In the patient with GBSM and deafness that was previously reported, the symptoms occurred in the early course of the disease and did not improve with dexamethasone therapy[4].

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เยื่อหุ้มสมองอักเสบจากเชื้อ*สเตร็พโตคอคคัส อะกาแลคเทีย*ในผู้ใหญ่: รายงานผู้ป่วย 2 ราย

วีรจิตต์ โชติมงคล, พ.บ., อร่าม พูนศรีอร่าม, พ.บ.**

รายงานผู้ป่วยเยื่อหุ้มสมองอักเสบจากเชื้อ*สเตร็พโตคอคคัส อะกาแลคเทีย*ในผู้ใหญ่ 2 ราย รายแรกเป็นผู้ป่วยชายอายุ 40 ปี มีอาการ ไข้ ปวดศีรษะ ต้นคอแข็ง และสับสนอย่างรวดเร็ว และระหว่างการรักษา เกิดอัมพาตครึ่งซีกจากสมองขาดเลือดและหูหนวกทั้งสองข้าง รายที่สองเป็นผู้ป่วยชายอายุ 80 ปี มีอาการสับสนและต้นคอแข็งอย่างรวดเร็ว และระหว่างการรักษาเกิดภาวะความดันโลหิตต่ำจากการติดเชื้อและชักทั้งตัว ตรวจพบแอนติเจนของเชื้อได้ด้วยวิธี latex agglutination และการเพาะเชื้อของน้ำไขสันหลังในผู้ป่วยทั้งสองราย ผลการรักษาพบว่าผู้ป่วยรายแรกรอดชีวิตแต่หูหนวก ส่วนรายที่สองเสียชีวิต

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