CASE REPORT

An unusual case of *Streptococcus agalactiae* meningitis in a patient with systemic lupus erythematosus

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Abstract

Background: *Streptococcus agalactiae* (group B Streptococcus) is a major cause of sepsis and meningitis in neonates and an important cause of invasive disease in adults.

Case description: We describe an unusual case of fatal bacterial meningitis caused by *Streptococcus agalactiae* in a young man suffering from systemic lupus erythematosus for over 20 years. The young man was transferred intubated in AHEPA University Hospital in a coma; twenty-four hours upon arrival and despite intense invasive treatment, he died from multiple organ failure.

Conclusion: The risk of serious infections in patients with systemic lupus erythematosus even under treatment with moderate doses of corticosteroids is high. Hippokratia 2015; 19 (4):372-373.

Keywords: *Streptococcus agalactiae*, meningitis, immunocompromised patient, systemic lupus erythematosus

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Introduction

Streptococcus agalactiae, also referred as Lancefield group B Streptococcus (GBS), is a leading cause of bacterial meningitis and sepsis in neonates and an important cause of disease among pregnant women. In recent years, GBS has also been recognized as an emerging cause of invasive bacterial infections in adults. Their majority suffer from significant underlying medical conditions, notably diabetes mellitus, cancer, cirrhosis, previous administration of glucocorticoids, immunological and neurological impairment[1,2].

Patients with systemic lupus erythematosus (SLE) are more prone to develop infections compared to the general population. The most common sites of infections are lungs, skin and genitourinary tract[3]. Bacterial meningitis is rare in patients with SLE and is most commonly caused by *Cryptococcus neoformans, Mycobacterium tuberculosis, Listeria monocytogenes* and *Streptococcus pneumoniae*[4,5].

Case report

We report an unusual case of a young patient with SLE, who developed fatal community-acquired GBS meningitis, pointing out the necessity of timely proper therapy in this patient group.

A 41-year-old Italian tourist was transferred to the intensive care unit (ICU) of AHEPA University Hospital after being intubated elsewhere with clinical signs of central nervous system (CNS) infection. He had a known 20-year history of SLE under fludrocortisone medical treatment (10 mg/24h) without any recent hospitalization.

During the preceding week, while he was in his country he presented with atypical gastrointestinal disorders and was reported to have received oral nimesulide. Five days before his admission to AHEPA Hospital, while traveling to another country, he developed high fever (>39.5^{o}C) and went to a state hospital where doctors prescribed ciprofloxacin (500 mg x 2). After showing some clinical improvement, he continued his journey towards Greece in good mental status but soon developed an episode of febrile diarrhea accompanied by drowsiness, headache, confusion and cervical stiffness. He was transferred to the nearest district general hospital where he presented mydriatic and in atrial fibrillation and immediately was intubated with Glasgow Coma Scale (GCS): 6/15.

He was transferred intubated to the ICU of AHEPA for hospitalization. Brain computed tomography imaging revealed ventricular dilatation accompanied by effacement of subarachnoid spaces and irregular parenchymal contrast enhancement of the brain in the left parietal region. These findings were consistent with brain edema

and meningitis. Because of high cerebral pressure, a lumbar puncture could not be performed at that time. Blood and urine were sent to the laboratory for analysis and culture. Laboratory investigations revealed a white blood cell (WBC) count of 7.6 x 10^3/mm^3 (with 82.6 % granulocytes), a C-reactive protein level at 22 mg/dl (normal range 0.0-0.8 mg/dl) and D-Dimers at 7235 ng/ml (normal range <500 ng/ml). Cultures of all samples were negative for growth. Empirical therapy with vancomycin (500 mg x 4), ceftriaxone (2 gr x 2), acyclovir (250 mg 2 x 3) and adrenaline were initiated as per ICU protocol. The following day the patient's status deteriorated and despite intense invasive treatment with adrenaline, hydrocortisone, mannitol, furosemide, antimicrobials and antivirals he died from multiple organ failure one day after admission to ICU.

After full authorization and informed consent, postmortem lumbar puncture was performed. The cerebrospinal fluid (CSF) analysis showed neutrophilic WBC of 1800 cells/mm^3, glucose concentration of 43 mg/dl, protein level of 500 mg/dl, while Gram staining identified gram-positive cocci. CSF cultures were positive 24 hours later. Bacterial identification, performed with the VITEK2-automated system (bioMérieux, France), identified *Streptococcus agalactiae*. Antimicrobial susceptibility testing performed by disc diffusion method on Mueller-Hinton agar with 5% sheep blood showed that the isolate was susceptible to penicillin, ceftriaxone, and vancomycin.

Discussion

GBS is a gram positive, beta-hemolytic streptococcus that colonizes the urogenital and gastrointestinal tract of healthy adults. Isolates can be categorized into ten different serotypes based on the bacteria's capsular polysaccharide. Five serotypes (Ia, Ib, II, III, V) account for the majority of diseases[2]; among them, serotype type III, especially the "hypervirulent" clonal complex ST17, was found to be the most prevalent in cases of bacterial meningitis among neonates[3]. Though pregnant women are at higher risk of invasive GBS infections; a significant increase has been reported among non-pregnant adults during the past two decades[1,6,7]. The rise in GBS infections was partly attributed to aging and underlying chronic diseases. GBS clinical manifestations include bacteremia, skin and soft tissue infection, pneumonia and osteomyelitis[2,8]. Among them, meningitis is uncommon accounting for about 4% of all cases of bacterial meningitis in adults. The majority of them had a serious underlying disease, and the risk of meningitis was higher among the elderly ones. GBS meningitis is a severe infection with poor outcome[8,9].

Among SLE patients, CNS bacterial infections are rare comprising only up to 3% of all SLE infections[4]. There is only one report in the literature of GBS meningitis in a patient with SLE, who achieved full clinical recovery[10]. In the reported case, the young man was not seriously immunocompromised and had no signs of active SLE. There is evidence that even moderate doses of corticosteroids might increase the risk of severe infections in SLE patients[11]. In the reported case, the delay in establishing the diagnosis of CNS infection played a crucial role in the outcome, and this was attributed to his initial presentation with gastrointestinal symptoms.

Even though there has been an improvement in the survival of SLE patients over the past decades, infections are one of the main causes of morbidity and mortality[3]. Clinicians must be aware of the susceptibility of these patients towards infections, even if treated with moderate doses of corticosteroids. A careful follow-up is recommended, as the early diagnosis of bacterial infections and appropriate management is critical for a positive outcome.

Conflict of interest

Authors declare no conflict of interest.

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