

Meningoencephalitis in splenectomized patient caused by concurrent *Streptococcus pneumoniae* and Herpes simplex virus infection

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In this paper we have described a meningoencephalitis in splenectomized patient caused contemporaneously by *Streptococcus pneumoniae* and Herpes simplex virus. The unusual course of pneumococcal meningitis accompanied with worsening of the patient's condition and repeated comatous status directed clinical diagnosis to the new etiologic agent of meningoencephalitis. After antibiotic and antiviral therapy the patient fully recovered.

Key words: Concurrent, Meningoencephalitis, Herpes, Pneumococcal.

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Introduction

Simultaneous infections of the central nervous system caused by different pathogens are very rarely seen even in immunocompromised patients. In the adult population up to 60 years of age, *Streptococcus pneumoniae* is responsible for about 60% of cases of acute bacterial meningitis (1). Splenectomy is a predisposing factor for bacterial infections especially caused by *Streptococcus*

pneumoniae (2, 3) Herpes simplex encephalitis is acute necrotising encephalitis with the highest mortality rate and is the most common encephalitis occurring in immunocompetent patients (4, 5). In this paper we have described meningoencephalitis in a splenectomized patient caused contemporaneously by *Streptococcus pneumoniae* and Herpes simplex virus. There is no similar report in the current medical literature.

Case report

M.M. 41-year old man was referred to our hospital after 24 hours of fever, sudden frontal headache, nausea, vomiting and progressive mental confusion. On admission, he was unconscious, with high fever and nuchal rigidity. There were no neurologic signs of lateralisation but skin abdominal reflex function was absent. Lung and heart findings were normal, TA 100/70 mmHg, puls 96/min. He had a history of splenectomy sixteen years ago, and he was not received pneumococcal vaccine. First lumbar puncture performed on the day of admission showed purulent cerebrospinal fluid (CSF) with 151 cells/mm^3 , predomination of neutrophils, undetectable level of glucose and high protein level of 2,2 g/l. Blood and CSF cultures have shown presence of *Streptococcus pneumoniae*. Cultivation of microorganisms was performed in Bacteriology Department of Military Medical Academy. Blood culture was performed on blood agar and CSF culture on chocolate agar. Patient treatment was started immediately with ceftriaxone 4g/day intravenously along with the antiedematous therapy. On the second day of the therapy, the patient become afebrile, and he was in good condition, conscious, but vesicles appeared on patient lips. On the sixth day, the patient became febrile again, he had difficulties with speech and his state of consciousness was worsened resulting in coma again. Repeated lumbar puncture has showed blood in CSF with 1827 cells/mm^3 (predominantly lymphocytes), decreased glucose level of 1.9 mm/L (glucose in blood 5.7 mmol/L) and 1.6g/L of proteins. Cultivation CSF for presence of Herpes simplex group viruses was performed in Virology Department of Military Medical Academy and were positive after six days in both CSF samples.

Acyclovir (1,5 g daily) was introduced into the therapy simultaneously with continuous administration ceftriaxon and anti-

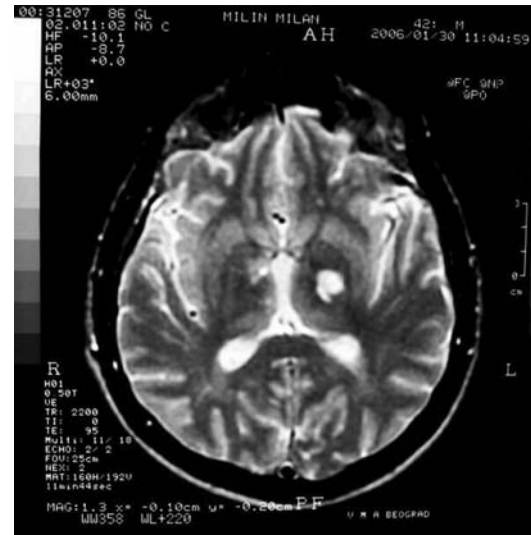


Figure 1 Typical focal lesions in basal ganglia

edematous drugs. Two days after introduction of antiviral therapy the patient became conscious, his neurologic findings revealed-motor aphasia, ptosis of the right eyelid, hemiparesis of the right facial nerve and pyramid deficit of the right side of the body.

Magnetic resonance imaging of brain has showed typical focal lesions in basal ganglia (Figure 1).

After two week successive medication and the physical rehabilitation, the patient was able to walk by himself and speak much better.

Discussion

Different types of immunodeficiencies are predictive for infections even of central nervous system. Defects in humoral immunity could lead to recurrent infections with encapsulated bacteria such as *Haemophilus influenzae* or *Streptococcus pneumoniae* (6). Immunodeficiency of T cells is found most commonly accompanying HIV infection, neoplastic disease, corticosteroids and cytotoxic agents use and is mainly associated with infection with intracellular pathogens (*M. Tuberculosis*, Herpes zoster virus, Herpes simplex virus, Cytomegalovirus *Cryp-*

tococcus etc.) (6). There was no evidence of congenital and acquired immunodeficiency in our patient except splenectomy.

Splenectomized patients are likely to develop addiction to septic conditions caused by encapsulated microorganisms, in the first place by *S. pneumoniae*. According to some reports splenectomized patients have twelve times higher risk for developing severe sepsis comparing to patients with spleen (7). A sepsis caused by *S. pneumoniae* in splenectomized patients occurs abruptly with possible development of acute meningitis (8). Due to the rapid development of meningitis, the cerebrospinal fluid could be clear, without cells or with a small number of cells, causing diagnostic confusion.

The viral genome of Herpes simplex virus usually persists as a latent infection in the trigeminal ganglion, and reactivation could be caused by a number of stimuli including febrile illnesses, menstruation, sunlight, stress and immunosuppression (9). There is experimental evidence of latent herpes simplex reactivation after pneumococcal pneumonia in mice (10). The virus has a predilection for temporal lobes of the brain, but extra temporal involvement could be found in as many as 55% of patients (11). Focal changes are identified especially in older patients (12). Concurrent infections of the central nervous system with microorganisms of quite different taxa are extremely rare. Infections have been described with two bacterial species (13), but CNS infections caused by concurrent bacterial and viral microorganisms have not been reported so far. Unusual course of pneumococcal meningitis accompanied with worsening of the patient's condition and repeated episodes of coma directed clinical diagnosis to the new etiologic agent of meningoencephalitis. In conclusion, the unusual clinical course of bacterial meningitis sometimes could indicate a concurrent viral infection.

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