Laboratory findings in acute bacterial meningitis and acute viral encephalitis

Hasan Tahsin Gozdas, Ahmet Dogan

Dear sir,

We read the case report of Wright and Fox-Lewis[1] recently published in the *New Zealand Medical Journal* with great interest. They reported the first case of imported human rabies in Aotearoa New Zealand. Their patient presented with a 3-day history of fever, vomiting and inability to swallow food or fluids. We have a criticism about the empirical treatment of this patient.

Empirical treatment was started for this patient with ceftriaxone, clarithromycin and aciclovir, suspecting acute meningoencephalitis. Blood tests of this patient revealed leukocytosis with neutrophilia and normal C-reactive protein (CRP) level. Cerebrospinal fluid (CSF) analysis showed lymphocytic pleocytosis, normal protein and mildly elevated glucose. In CSF examination of acute bacterial meningitis cases, neutrophilic pleocytosis, low glucose and very high protein levels are expected. In addition, a high CRP level is almost always seen in acute bacterial central nervous system (CNS) infection cases. In CSF analysis of acute viral encephalitis cases, lymphocytic pleocytosis, normal glucose and normal or mildly elevated protein levels are expected.[2] Their patient's blood and CSF changes were compatible with acute viral encephalitis rather than acute bacterial meningitis. We wonder whether CSF gram staining was done to discriminate bacterial and viral aetiology. We are of the opinion that indication of antibacterial treatment with ceftriaxone and clarithromycin is not clear in this patient.

COMPETING INTERESTS

No conflict of interest was declared by the authors.

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Response to: Laboratory findings in acute bacterial meningitis and acute viral encephalitis

Hamish Wright, Andrew Fox-Lewis

Thank you for your interest in the paper. CSF gram staining was performed, and no organisms were seen. We agree that the patient's blood and CSF changes were not typical of acute bacterial meningitis. Differential diagnoses included acute viral encephalitis. In this case, empirical meningoencephalitis treatment was commenced on day 4 post-symptom onset when the patient was admitted to the local hospital closest to where his vessel was in port. Lumbar puncture and CSF analysis were undertaken on day 5 following transfer to the intensive care unit at the regional tertiary hospital. The CSF was obtained for analysis after approximately 24 hours of antibacterial treatment, polymerase chain reaction testing failed to detect any common viral causes of encephalitis, and the CSF glucose value was confounded by concurrent hyperglycaemia. Given the patient was critically unwell, the clinical decision was made to err on the side of caution and continue empirical treatment for both acute viral encephalitis and a broad range of bacterial causes of meningoencephalitis while awaiting investigations. CSF parameters cannot confidently exclude bacterial meningitis, especially if pre-treated, and 10% of cases of bacterial meningitis have a lymphocytic predominance.[1] The most likely indication for commencing a macrolide antibiotic was ground-glass pulmonary infiltrate that was seen on computed tomography chest imaging on day 4, and was later thought to be non-significant.

It is valuable to emphasise that empiric treatment of meningitis for the most common pathogens normally consists of ceftriaxone with dexamethasone. Vancomycin is added if risk factors are present for infection with strains of *Streptococcus pneumoniae* with reduced susceptibility to ceftriaxone. Benzylpenicillin or amoxicillin are added if risk factors are present for infection with *Listeria monocytogenes*. Acyclovir is often also added to empiric CNS treatment, to cover predominantly for HSV encephalitis.[1]

COMPETING INTERESTS
Nil.

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