Temporal lobe involvement: A diagnostic challenge in Japanese encephalitis

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Abstract

Japanese encephalitis (JE) and herpes encephalitis are the most common endemic and sporadic encephalitis, respectively. A 10-year-old boy, presented with features suggestive of viral meningoencephalitis, had neuroimaging suggestive of herpes encephalitis, but cerebrospinal fluid serology was positive of JE. Temporal lobe involvement in JE may cause problems in differentiating it from herpes encephalitis, which highlights the importance of both neuroimaging and serology for a complete diagnosis.

Key words: Japanese B encephalitis, Temporal lobe, Serology, Viral meningoencephalitis

apanese encephalitis (JE) is widely recognized as the most common vaccine-preventable encephalitis. It is a common disease in the Indian subcontinent, particularly affecting the northeastern part of the country [1]. It is a mosquito-borne, flaviviral infection similar to dengue, yellow fever, and West Nile fever. The first case of JE was reported from Japan in 1871. Worldwide, approximately 68,000 cases are reported annually [2]. The virus is transmitted to humans by *Culex* mosquitoes that breed in flooded paddy fields. About 50% of the patients recover with no residual deficit and the remaining patients tend to have a wide variety of neurological deficits [3]. The case fatality in JE ranges from 10% to 60% [3]. JE primarily affects the children and adults are immune following a childhood infection in endemic areas.

The evaluation involves neuroimaging and confirmation is by serology. Neuroimaging is useful in differentiating other forms of encephalitis such as herpes simplex virus (HSV) encephalitis, which is a potentially treatable condition. We are reporting a case of atypical involvement of the brain in a case of JE.

CASE REPORT

A 10-year-old boy child with no previous medical history presented to us with the complaints of fever of 3 days duration followed by one episode of generalized tonic-clonic convulsion which lasted for 15 min following which child lapsed into altered sensorium. There was no associated rash. No previous history of seizures was there and the child was normally developing for his age.

On examination, he had a Glasgow come scale of 9/15, 2 mm reactive pupils, and bilateral hyperemic disc on fundus

examination with positive signs of meningeal irritation. There was no cranial nerve abnormality with generalized increase in tone, brisk deep tendon reflexes, and extensor plantar reflexes without any abnormal movements. Other systemic examination was unremarkable. A provisional diagnosis of acute meningoencephalitis was made and the child was evaluated for the same.

Computerized tomography (CT) scan was done which showed hypodensities in the left temporoparietal lobe with effacement of sulci Figure 1 a & b. Cerebrospinal fluid (CSF) analysis was suggestive of viral meningitis (cell count - 30 cells, all lymphocytes, glucose - 85 g/dl, and proteins - 41 g/dl). CSF smear did not reveal any organism and culture was sterile. In view of the CT scan findings, he was initially managed as herpes encephalitis with intravenous acyclovir and mechanical ventilation and supportive measures. As the child was critically ill and unstable, magnetic resonance imaging was differed and electroencephalography could not be done. CSF was sent for both JE and herpes serology and immunoglobulin M enzymelinked immunosorbent assay for JE was positive; hence, a diagnosis of JE was made.

DISCUSSION

JE is the most common cause of endemic encephalitis in Asia. An estimated 3 billion people are at risk and to date there is no known effective antiviral therapy against this disease. Vaccination programs, improved living standards, and mechanization of agriculture have contributed to decrease in the disease incidence in countries such as Japan and South Korea; however, it is on an increasing trend in the Asian countries.



Figure 1: CT scan head showing hypodensities in the left temporoparietal lobe with effacement of sulci.

Most of the JE infections are mild with fever and headache only or no apparent symptoms at all. However, 1 in 250 infections tend to be severe characterized by fever, headache, and neurological symptoms. The case fatality rate is as high as 10-30% and half of the survivors having neurological sequelae [2]. Investigating a case involves serological confirmation on blood or preferably CSF. In neuroimaging though quick, the characteristic areas described in cases of JE could be misleading.

Neuroimaging in JE shows the involvement of bilateral thalamus, basal ganglia, substantia nigra, brain stem, cerebellum, and cerebral cortical and white matter. Bilateral T2 hyperintense and T1 hypointense to isointense thalamic lesions, especially hemorrhagic in an appropriate clinical setting is described as characteristic [4,5]. Whereas neuroimaging in HSV encephalitis, characteristically shows bilateral or unilateral T2 hyperintense lesions in the temporal lobes with or without hemorrhage and contrast enhancement [6]. The involvement of temporal lobes in JE has been described in up to 17% of the cases in various case series [7], causing a diagnostic dilemma where the serological diagnosis is not available.

Our patient's clinical profile was that of a neuroinfection without any specific clinical indicators such as focal convulsions for herpes or abnormal movements at a presentation for JE. Neuroimaging was suggestive of herpes encephalitis, but CSF serology changed the diagnosis. We would like to report this as an atypical manifestation of JE, and thus stressing the importance of both serology and neuroimaging for a complete diagnosis.

CONCLUSION

In a child with encephalitis, having temporal lobe involvement, JE should also be kept in differentials. However, before a confirmatory diagnosis can be made, it may be prudent to start antiviral therapy against potentially treatable herpes infection. We would like to recommend that whenever feasible, serological diagnosis should be attempted for confirmatory diagnosis for the better outcome of these sick children.

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