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Unilateral Temporo Occipital Cortex and Focal Leptomeningeal Involvement in MRI in a Case of Japanese Encephalitis

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Abstract **Case Report**

Acute encephalitis syndrome secondary to Japanese encephalitis is common in India and south east Asia. Neuroimaging is important in the diagnosis of JE along with Cerebrospinal fluid, IGM antibody against JE. Bilateral thalamus, hippocampus, caudate nucleus, substantia nigra, cortex and cerebellum are usually involved in JE. Unilateral involvement of the above-mentioned areas without involvement of thalamus and diffuse leptomeningeal enhancement has been reported in literature. Unilateral focal cortical involvement with focal leptomeningeal enhancement in JE has never been reported in literature till now. We are reporting a case of Japanese encephalitis with positive CSF IGM antibody against JE and MRI showing unilateral focal occipital cortex involvement with adjacent focal leptomeningeal enhancement without involvement of bilateral thalamus, caudate nucleus, putamen, substantia nigra and hippocampus.

Keywords: Focal Cortical, Leptomeningeal Enhancement, Japanese Encephalitis, No Thalamic Involvement, Acute Encephalitis Syndrome.

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INTRODUCTION

Japanese encephalitis is a mosquito borne viral disease spread by Culex tritaeniorhynchus. It is highly prevalent in India and South east Asia. The first case was documented in Japan in 1871. JE has an incidence of 68000 cases per year with 13600 – 20400 annual deaths.[1]

Common in children less than 15 years of age, but can occur in any age group. Among symptomatic cases, the case fatality rate is 30%. It also causes irreversible neurological damage in 30-50% of patients.[1]

WHO defines a case of acute encephalitis syndrome as acute febrile illness and a change of mental status and /or new onset seizures, excluding simple febrile seizure. If these patients are IgM positive for JE and if other causes of encephalitis/ meningitis/ meningoencephalitis are excluded, a diagnosis of JE can be made.[2] MRI plays a significant role in diagnosing Japanese encephalitis with characteristic involvement of bilateral thalamus, putamen, caudate nucleus, globus

pallidus, hippocampus and substantia nigra.[3] Cortical involvement and lepto- meningeal enhancement are rare in JE.

Unilateral cortical involvement of temporal and occipital lobe with focal lepto-meningeal enhancement without involvement of thalamus, basal ganglia, hippocampus in case of JE has notbeen reported in literature. We are reporting such a case here.

Case Report

An elderly female presented to our out-patient department with history of seizures at home, which was preceded by intermittent fever and chills, intermittent confusion and visual hallucinations in the form of formed images of Indian map in front of her and at times some demons sitting in front of her for 1 week.

Neurological examination revealed defective attention span and poor recall. Her cranial nerve examination, spinomotor and cerebellar examination were normal without any signs of meningeal irritation.

She satisfied the WHO criteria for acute encephalitis syndrome and investigations pertaining to the same were done in serum and CSF. EEG and MRI Brain with contrast were also done.

Her CSF showed lymphocytic pleocytosis with raised protein and normal glucose. Viral encephalitis panel revealed positivity for IgM JE and negative for other viruses, which included Nipah, West nile and Herpes simplex viruses. Cartridge based nucleic acid amplification test for tuberculosis was negative. Auto immune encephalitis panel which included antibodies against NMDA, AMPA, GABA and VGKC, which included LG1 and CASPR2 were negative. Her thyroid profile and Anti microsomal antibodies were also negative.

Her MRI brain revealed right temporo occipital T1 isointensity, T2 weighted hyperintensity involving the brain parenchyma as well as the gyri which was not suppressed by FLAIR. These areas showed restriction on DWI with slightly low ADC values and contrast showed patchy enhancement of the leptomeninges of the corresponding areas. There was no involvement of thalamus, basal ganglia and hippocampus.

She was treated with intravenous acyclovir, antipyretics and anti-epileptics. She showed remarkable improvement in two days. She became afebrile, her seizure were controlled, her cognition also showed significant improvement. She was discharged on day seven.



Figure_1A Details T1W



Figure_1B Details T2W

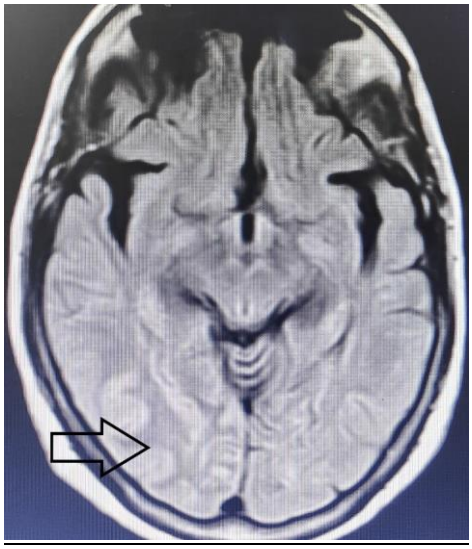


Figure_1C Details FLAIR

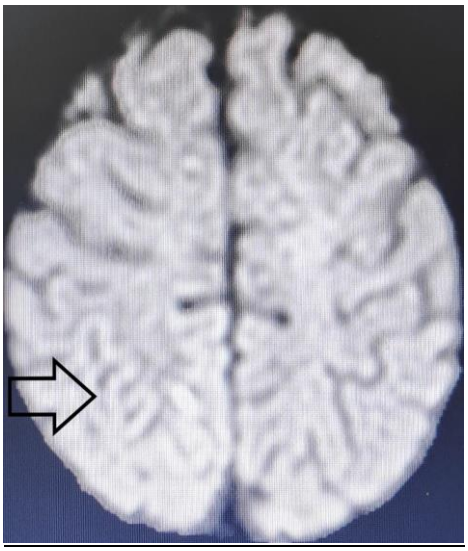


Figure_1D Details DWI

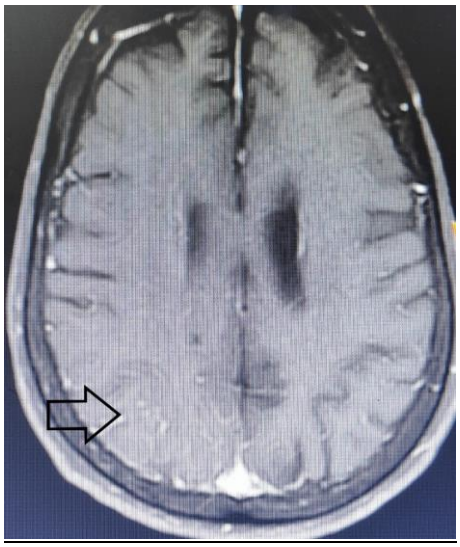


Figure_1E Details CONTRAST

DISCUSSION

MRI features of Japanese encephalitis in the descending order of frequencies are T2 hyperintensities involving bilateral thalamus, caudate nucleus, hippocampus, substantia nigra, cerebellum and cerebral cortex. Unilateral involvement of above said areas are uncommon but a few cases have been reported.[4] Lobar involvement especially temporal and frontal lobe involvement are more common with herpes simplex encephalitis. Diffuse leptomeningeal enhancement along with bilateral frontal, parietal, insular, caudate, putamen and hippocampal involvement without involvement of thalamus has been reported in Japanese encephalitis [5].

Other differential diagnosis based on MRI imaging considered were reversible posterior leucoencephalopathy and Variant CJD but were ruled out as our patient was normotensive and our patient's temporal profile, rapid clinical improvement and absence of EEG changes ruled out CJD. Autoimmune encephalitis was considered but was ruled out due to the short clinical duration of the illness, improvement without steroid or immunoglobulins and the antibodies for Autoimmune encephalitis were negative.

In our case T2 hyperintensities involving the right temporal and occipital cortex were noted which was not suppressed by FLAIR along with diffusion restriction with low ADC values in MRI was noted. Along with this focal leptomeningeal enhancement was seen in the meninges involving the occipital cortex.

The changes in MRI in our patient could be due to the cytotoxic edema as evidenced by diffusion restriction and low ADC values. These changes are attributed to the glial nodularity and necrosis caused by JE virus which spreads hematogenously to involve these areas [3,4,5].

Leptomeningeal enhancement in contrast MRI in JE could be attributed to breakage of blood brain barrier subsequent to inflammation caused by infected blood cells [5].

Unilateral temporo-occipital and focal leptomeningeal involvement without involving thalamus, caudate nucleus and hippocampus in JE in MRI seen in our case is very rare and unreported till now.

CONCLUSION

In patients from an endemic area of JE presenting with history of fever and encephalopathy it's important to do CSF analysis for IgM antibody for JE. Classic MRI features of JE may not be present in all cases as in our patient and this makes history, clinical profile and CSF analysis for appropriate antibodies along with neuro imaging for a final diagnosis.

Author's Contribution

Suresh Thirunavukarasu
Contribution: Concepts, Definition of intellectual content, Manuscript preparation, Manuscript editing and review

Devi Varadharaj
Contribution: Concepts, Definition of intellectual content, Literature search, Manuscript preparation

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The authors certify that they have obtained all appropriate patient consent.

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Use of Artificial Intelligence (AI)-Assisted Technology for manuscript preparation

The author(s) confirms that there was no use of Artificial Intelligence (AI)-Assisted Technology for assisting in the writing or editing of the manuscript and no images were manipulated using the AI.

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