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Research Article

IMAGING IN JAPANESE ENCEPHALITIS

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ABSTRACT

We documented 5 cases of Japanese encephalitis. MRI and CT Brain were carried out on Philips 1.5T and Toshiba 16 slice machines respectively. Findings were noted in bilateral thalami, frontal white matter, corona radiata, posterior limb of internal capsule, posterior temporal lobe, substantia nigra, bilateral cerebellar hemisphere and vermis. Small hemorrhagic foci noted in bilateral thalami in 3 cases. Bilateral thalamic involvement, especially with hemorrhagic foci within it, are considered characteristic of Japanese encephalitis, especially in endemic areas.

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INTRODUCTION

Japanese encephalitis (JE) is the commonest endemic encephalitis in India and rest of south-east Asia [1]. The diagnosis is commonly based on demonstrating a rising titre of antibodies against JE virus in acute and convalescent sera [2, 3]. There is a delay of at least 7 days before the diagnosis can be confirmed. Common clinical features of JE include fever, headache, body ache, and occasional vomiting and altered sensorium. In an endemic area, it is important to differentiate JE from herpes simplex encephalitis for rational therapy with acyclovir. Commonest radiological findings in JE are thalamic lesions [4].

MATERIAL AND METHODS

5 clinically suspected cases of meningoencephalitis were referred to department of radiology for CT Brain. MRI Brain was performed for further evaluation. Scans were done using Philips 1.5T(MRI) and Toshiba 16 slice(CT) machines.

Objective

To evaluate CT and MRI findings in Japanese encephalitis.

RESULTS

Out of 5 cases, 3 cases were in age group 25-35years and 2 cases were noted in children less than 10 years. Out of 5 cases 2 were females and 3 were males.

CT Brain was performed as initial assessment in all cases followed by MRI Brain in 4 cases for further evaluation.

All patients presented with fever, headache and loss of consciousness and clinical suspicion of meningoencephalitis. Out of 5 cases, 4 cases showed normal CT Brain while in one case, CT Brain showed symmetrically enlarged and hypodense bilateral thalami with small hemorrhagic foci within it. Brainstem, B/L cerebellar and white matter edema and mass effect in form of compression of fourth ventricle and obstructive hydrocephalus was noted. Rest of 4 cases were followed up with MRI Brain. In MRI of 3 cases, bilateral thalami were symmetrically swollen which showed T1 hypointense and T2/FLAIR hyperintense signal with diffusion restriction and no significant enhancement. Small foci of blooming noted on gradient images in thalami s/o hemorrhagic foci. In 1 case T1 hypointense, T2/FLAIR hyperintense areas were noted in bilateral frontal white matter, corona radiata, posterior limb of internal capsule, posterior temporal lobe, substantia nigra, b/l cerebellar hemispheres and vermis in addition to B/L thalamic lesions. Lesions showed diffusion restriction with no significant enhancement. A provisional diagnosis of Japanese encephalitis was given, which was confirmed by CSF analysis (IgM capture ELISA) which showed IGM antibodies against JE virus.

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Figures and Tables

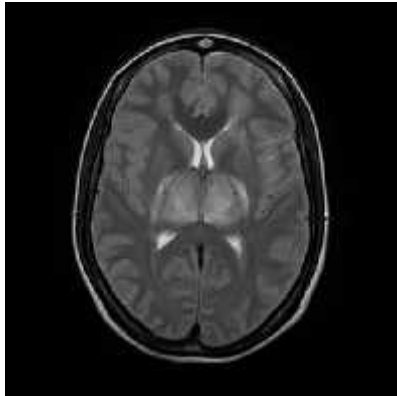


Fig.1

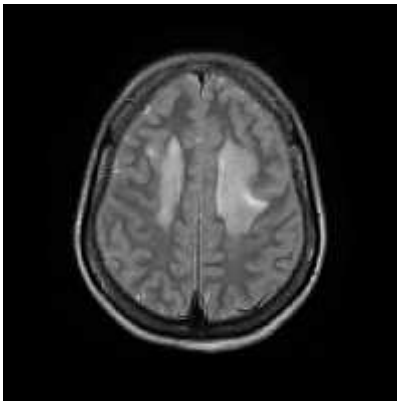


Fig.2

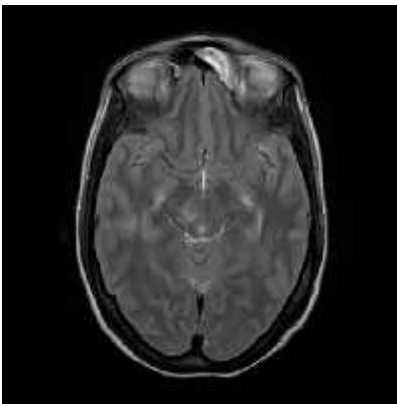


Fig.4



Fig.3

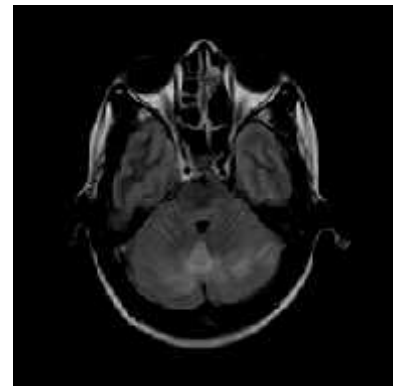


Fig.5

MRI Brain of a 29years old female patient showing T2/FLAIR hyperintensities noted in bilateral thalami(Fig.1), corona radiata(Fig.2), right substantia nigra (Fig.3),bilateral cerebellar hemispheres(Fig.4)and vermis (Fig.5)

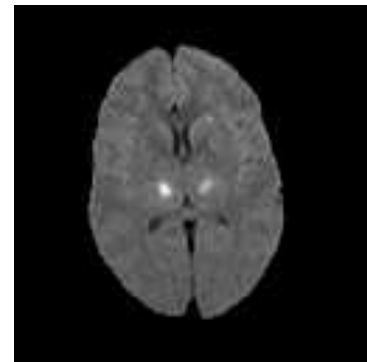


Fig.6



Fig.7

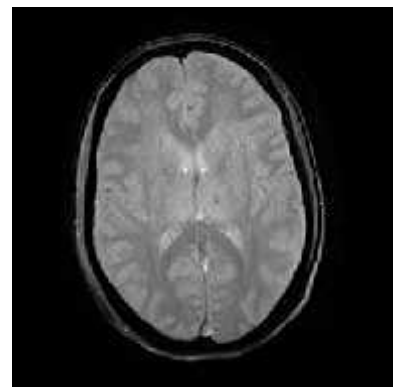


Fig.8

MRI Brain of a 32 years male patient showing Diffusion restriction in bilateral thalamus with blooming (Fig.6-8)

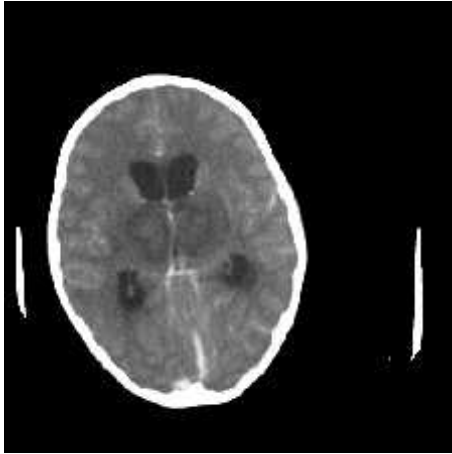


Fig.9

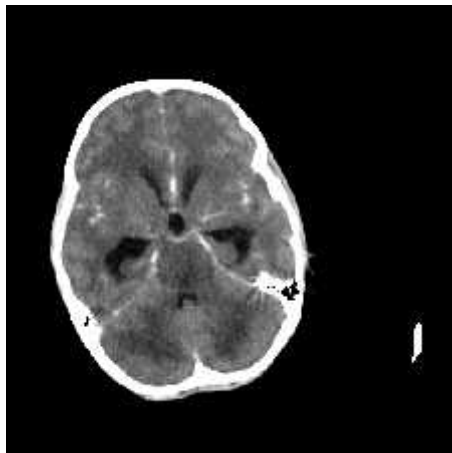


Fig.10

CT Brain of a 9 year old male child showing symmetrically enlarged and hypodense bilateral thalami with small hemorrhagic foci (Fig.9) with brainstem, B/L cerebellar and white matter edema and mass effect in form of compression of fourth ventricle and obstructive hydrocephalus.

DISCUSSION

Japanese encephalitis (JE) is a serious vector-borne viral encephalitis disease found worldwide, especially in Asian, the Western Pacific countries, and in northern Australia[5]. Over 3 billion individuals live in JE epidemic and/or endemic countries. It is estimated that approximately 67,900 JE cases have occurred annually in 24 countries, with only 10,426 cases reported in 2011[6,7]. The fatality rate in JE cases ranges from 20%–30%, with neurologic or psychiatric sequelae observed in 30%–50% of survivors[8].

JE is induced by infection with Japanese encephalitis virus (JEV), which belongs to the JEV serogroup in the genus *Flavivirus*, family *Flaviviridae*[7]. JEV has a single-stranded, positive-sense RNA genome of ~11 kb in length. The JEV virion contains seven non-structural proteins (NS1, NS2A, NS2B, NS3, NS4A, NS4B, and NS5) and three structural proteins: nucleocapsid or core protein (C), non-glycosylated membrane protein (M), and glycosylated envelope protein (E)[9]. The main JEV transmission vector is the *Cx. mosquito*

especially *Cx. tritaeniorhynchus*, and the main vertebrate amplifying hosts are pigs and wading birds[10].

Acute encephalitis with CT and MR imaging findings showing bilateral thalamic, medial temporal lobe, and hippocampal lesions (like JE), besides lesions elsewhere in the brain, has been reported from Korea[15].

The most characteristic MR imaging finding of Japanese encephalitis is T2 hyperintensity, typically with bilateral involvement of the posteromedial thalamus. Intralesional hemorrhages and restricted diffusion have also been described [13,14]. Other sites of involvement include the basal ganglia, substantia nigra, red nucleus, pons, hippocampi, cerebral cortex, and cerebellum. Japanese encephalitis and Murray Valley encephalitis more often involve the thalamus [12], whereas West Nile fever typically demonstrates bilateral involvement of the thalamus and the caudate and lentiform nuclei [11].

No antiviral agent is effective against Japanese encephalitis virus. Care is supportive, including management of intracranial pressure, if needed, airway protection, and seizure control.

CONCLUSION

Japanese encephalitis still remains significant public health problem in South East Asian countries including India. The MRI and CT are important modalities for establishing the diagnosis of Japanese encephalitis. Though MRI is considered more sensitive and specific than CT for diagnosis, in places where MRI is not available or contraindicated, CT can also be helpful in few cases. MRI findings of Japanese encephalitis help to differentiate from other viral encephalitis, encephalopathy and acute disseminated encephalomyelitis. Bilateral thalamic involvement with hemorrhage within is characteristic for JE[4].

References

1. Umenai T, Krzysko R, Bektimorov TA, Assaad FA (1985) Japanese encephalitis: current worldwide status. Bull WHO 63: 625–631
2. Mathur A, Arora KL, Rawat S, Chaturvedi UC (1986) Persistence, latency and reactivation of Japanese encephalitis virus infection in mice. J Gen Virol 67: 381–385
3. Bharucha NE, Bharucha EP (1991) Neurology in India. In: Bradley WG, Daroff RB, Fenichel GM, Marsden CD (eds) Neurology in clinical practice. Butterworth-Heinemann, Boston, pp1925–1941
4. Misra UK, Kalita J, Jain SK, Mathur A (1994) Radiological and neurophysiological changes in Japanese encephalitis. J Neurol Neurosurg Psychiatry 57: 1484–1487
5. Erlanger TE, Weiss S, Keiser J, Utzinger J, Wiedenmayer K. Past, present, and future of Japanese encephalitis. Emerg Infect Dis. 2009;15(1):1–7.
6. Japanese encephalitis surveillance and immunization – Asia and the Western pacific, 2012. MMWR Morb Mortal Wkly Rep. 2013;62(33):658–662.
7. Campbell GL, Hills SL, Fischer M, et al. Estimated global incidence of Japanese encephalitis: a systematic review. Bull World Health Organ. 2011;89(10):766–774. 774A.

8. Solomon T. Flavivirus encephalitis. *N Engl J Med.* 2004;351(4):370–378.
9. Chambers TJ, Hahn CS, Galler R, Rice CM. Flavivirus genome organization, expression, and replication. *Annu Rev Microbiol.* 1990;44:649–688.
10. Vaughn DW, Hoke CJ. The epidemiology of Japanese encephalitis: prospects for prevention. *Epidemiol Rev.* 1992;14:197–221.

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