Hyper-intense thalamic lesion in Magnetic Resonance Imaging: Two common causes

Manjari Basu[1], **Subrata Chakraborty**[2]

Sri Lanka Journal of Child Health, 2012: **41**(2): 100-101

(Key words: Thalamic hyper-intensity; ADEM; Japanese B encephalitis)

Thalamus may be hyper-intense in many diseases such as Japanese encephalitis (JE)[1], other viral encephalitis[1], acute disseminated encephalomyelitis (ADEM)[2], systemic lupus erythematosus (SLE)[2], Wernicke encephalopathy[2] and Wilson disease[2]. The most common causes with acute presentations are JE and ADEM.

Case-1

A 5 year old boy with an average build and normal developmental milestones had low grade fever persisting for 7 days, 17 days prior to admission. Three days prior to admission child developed blurring of vision with intense headache and vomiting. On examination, the higher functions were normal. There was no menace reflex, no perception of light (PL) in both eyes but both pupils reacted to light. There were no other focal neurological signs. On ophthalmoscopy papilloedema was present.

The complete blood count (CBC) was normal, the Mantoux test was negative and the chest x-ray was normal. A study of the cerebrospinal fluid (CSF) showed 30 cells /cu mm, all lymphocytes, protein 102 mg/dl, normal sugar and adenosine deaminase activity (ADA) 9 U/L. Magnetic resonance imaging (MRI) scan of the brain showed bilateral thalamic hyper intensity in both T_1 and T_2 images and lesion enhanced with contrast. No changes in other part of MRI-brain. Serology for Japanese encephalitis (JE) was negative. We considered the case as probable ADEM and gave 5 day methyl prednisolone followed by oral prednisolone and child dramatically improved in 2 days with complete recovery of vision.

Case-2

A 3 year old girl with average build and normal developmental milestones presented with low grade fever for 2 weeks. After 1 week child developed repeated convulsions and altered sensorium which persisted for 5 days; thereafter sensorium gradually improved but child developed dystonic posture of limbs and loss of speech.

The complete blood count (CBC) was normal. The CSF showed 100 cells/cu mm with protein and sugar normal. Mantoux test was negative and the chest x-ray was normal, MRI scan of the brain showed right thalamic involvement with T_2 hyper-intensity. JE IgM was elevated. We diagnosed the case as Japanese encephalitis. We continued conservative management and the child gradually improved.

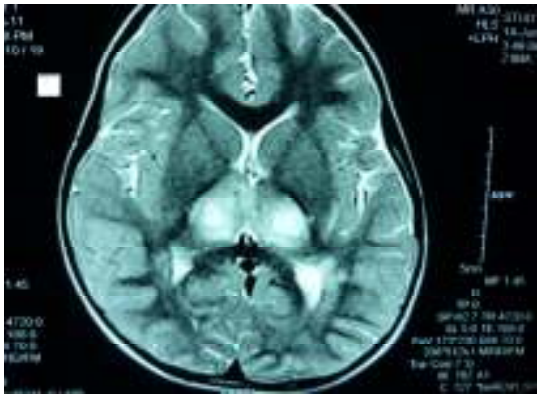

Figure 1: *MRI brain of case 1 (ADEM)*

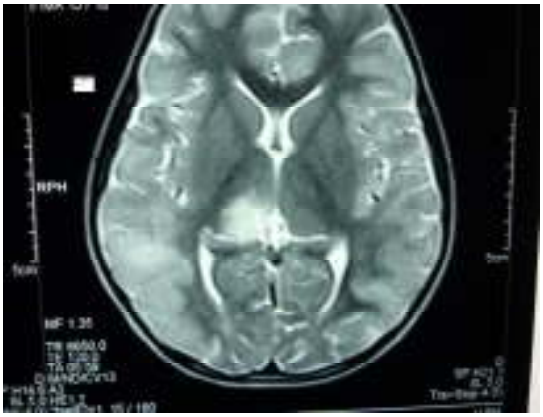

Figure 2: *MRI brain of case 2(JE)*

Discussion

In MRI scan of the brain hyper-intensity of thalamus in T2 image may be found in JE[1], other flavivirus

[1]*Senior Resident,* [2]*Professor, Department of Paediatrics, Institute of Child Health, Kolkata, India*

(Received on 13 July 2011: Accepted after revision on 10 August 2011)

encephalitis[1], ADEM[2], SLE[2], tuberculosis and glioma[2]. Metabolic causes of T2 thalamic hyper-intensity are Wilson disease[2] and Wernicke encephalopathy[2]. Among these ADEM and JE commonly present with feature of acute encephalopathy. In ADEM white matter is involved. Lesions in thalamus, basal ganglia and cortical gray-white junction strongly favour ADEM[3]. The MRI lesions have no mass effect and enhances with contrast. This lesion is due to demyelination. In JE thalamic lesions never enhance with contrast in MRI but may be haemorrhagic. These lesions are due to thalamic oedema[3]. Differentiation between these 2 diseases is important as ADEM has definitive treatment with steroid and JE has only conservative management.

References

1. Kumar S, Misra UK, Kalita J, Salwani V, Gupta RK, Gujral R. MRI in Japanese encephalitis. *Neuroradiology* 1997; **39**:180-4. http://dx.doi.org/10.1007/s002340050388

2. York DH Jr. Magnetic resonance imaging of CNS disease, a teaching file. 2nd ed. St Louis: Mosby Inc; 2002:403.

3. Krishna Murthy SN, Faden HS, Cohen ME, Bakshi R. Acute Disseminated Encephalomyelitis in Children. *Pediatrics* 2002; **110** (2): e21 http://dx.doi.org/10.1542/peds.110.2.e21