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*Case Report*

## **Japanese B Encephalitis and Uremic Encephalopathy in Same Patient with MRI Brain Imaging**

**Authors**

**Shyam Baboo**

Radiology Department, Darbhanga Medical College and Hospitals, India

**M A Hashmi**

EKO CT & MRI Scan Centre. At Medical College and Hospitals, Campus, Kolkata, India

**Gautam G.**

Neurology department, Medical College and Hospitals, Kolkata, India

**D Bandyopadhyaya, D Roy, P Sarkar, P Singhanian and S Singh**

Medicine department, Medical College and Hospitals, Kolkata, India

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## **Abstract**

Japanese B Encephalitis is a viral disease, which presents with multiple findings in brain. Uremic encephalopathy is also a known cause of multiple signal changes in brain. We present a case of a 30 yr male patient who was having Japanese encephalitis and was also having acute renal problems leading to uremic encephalopathy. The patient did not have any renal problem before. Diagnosis was confirmed by CSF examination for Japanese encephalitis virus (JEV) by RT-PCR, which was positive, and renal function test. To our knowledge, MR imaging findings of encephalopathy in same patient caused by Japanese B and hyperuremia have not been reported. Possibility of Renal failure may be a complication of sequelae of Japanese B encephalitis.

**Keywords:** Japanese B Encephalitis, Magnetic Resonance Imaging (MRI).

## **Introduction**

Japanese B Encephalitis is a viral disease transmitted by is transmitted through the bite of an infected female culex mosquito. Patients with the disease usually present within the first week or two as 'flu like illness with muscle pains and headache. These early clinical feature are than followed by gastrointestinal symptoms like diarrhea and vomiting. Mark Neurological signs are seen as the virus affects the patient's brain tissue. Typical MRI finding of JE is bilateral T2 thalamic hyperintensity with involvement of basal ganglias and upper brainstem.

Encephalopathy caused by uremia can also present as focal or diffuse hyperintensities in subcortical parieto occipital region but

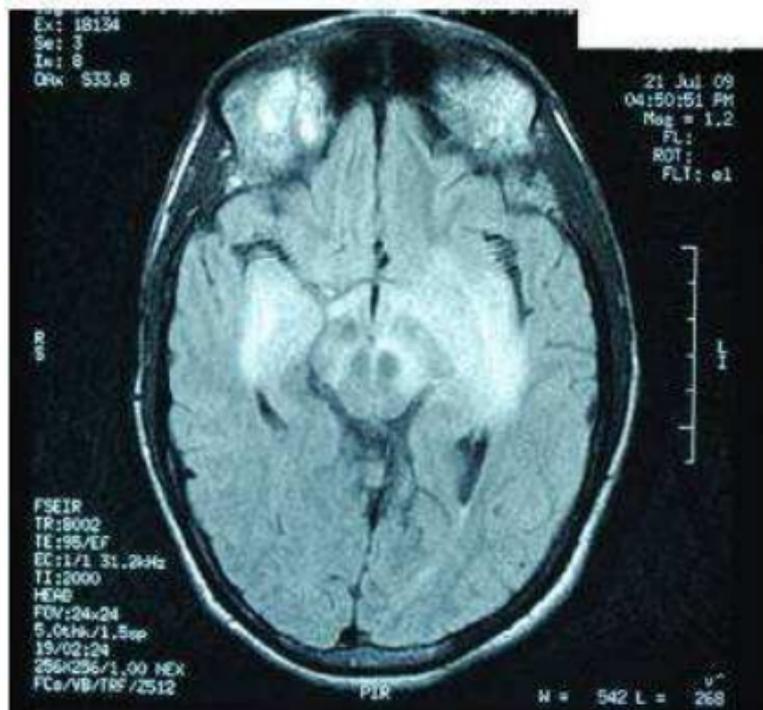
can also involve the deep brain parenchyma. They usually resolves as the uremic condition improves.

## **Case Report**

A 30 year-old male presented with convulsions for 20 days with diminish urine output. 3 days later he developed drowsiness and diminished speech. He has been unconscious for last 10 days and also had fever. There was no previous history of any disease like jaundice, altered behavior, fits or renal problems.

Patient was anemic hemoglobin 7.2 gm/dl. His liver function test was within normal limit. MRI revealed hyperintensities involving bilateral thalamus, basal ganglias and adjacent periventricular region with involvement of brainstem [Fig 1 and 2]. Japanese

encephalitis virus (JEV) RT-PCR was done from CSF, which was positive. His Ceruloplasmin level was normal and presentation was well before 20 days. His renal function test was abnormal. Urea level was 220mg/dl and creatinine was 12.8mg/dl, which after dialysis came to 87mg/dl and 9.7mg/dl.



**T2 Weighted Flair is Showing Hyperintense Areas Seen Involving Upper Brainstem and Adjacent Thalamic Areas**



**T2 Weighted Flair Imaging is Showing Hyperintense Areas Seen Involving Bilateral Thalamus and Basal Ganglias**

## **Discussion**

Japanese Encephalitis which is caused by infection with the JE virus belongs to the mosquito-borne flavivirus group. Culex mosquito is the main vector while Pigs and some birds like herons and sparrows are the natural hosts. Most of the infected patient has subclinical infections or flu like symptoms. When fulminant acute neurological involvement occurs. Seizures and paralysis are seen and the condition carries a high mortality rate of 10% to 40% as mentioned by Matsui M. In up to 80% of those who survive there may be residual neurological findings. The definitive serologic diagnosis of JE is based on antibody detection in serum and CSF by immunoglobulin M (IgM) capture enzyme-linked immunosorbent assay test.

Characteristic MRI finding of JE is bilateral T2 thalamic hyperintensity as said by Singh P and Ohtaki E as seen in Fig 2. Other common sites of involvement include substantia nigra, red nucleus and pons as seen in Fig 1. Hippocampus, cerebral cortex, and cerebellum are other common sites as mentioned in study by Ohtaki E , Abe T and Kumar S. Unilateral involvement has also been reported but is less common. Subcortical white matter involvement is also reported in some patients, but this has always been in combination with the more characteristic gray matter lesions. Some lesions, especially those in the thalamus, may be haemorrhagic. Enhancement is not usually observed, indicating only minor blood-brain barrier deficit. Abe et al discovered autoantibodies to myelin basic protein and neurofilaments in some patients with JE and raised the possibility of a superimposed immunologic component to this disease that

may selectively involve certain portions of the central nervous system.

Uremic encephalopathy also involves nervous system as small hyperintensities in subcortical parieto-occipital region and in deep brain parenchyma, which regresses as the patient condition improves as said in research by Schmidt M and Okada J. In above case the patient did not have any prior history of renal dysfunction. Coexistent of both the condition in same patient is rare with mark signal changes in MRI and possibility of autoimmune mechanism against renal tissue.

## **Conclusions**

JE leads to typical MRI findings in brain and it is very helpful as an additive tool in its diagnosis. Uremic encephalopathy also involves nervous system and its MRI findings are almost same. In such situation other pathological examinations result also has to be considered.

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