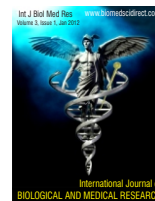


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Case Report

An Unusual Case Of Encephalitis In A Young Girl

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ABSTRACT

Japanese encephalitis (JE) is caused by Japanese encephalitis virus which belongs to the Flaviviridae family. It is transmitted by mosquitoes. Pigs and wild animals serve as reservoirs of the virus. Those infected by the virus can present with symptoms ranging from fever and headache to severe neurological deficits. This report documents a case of JE in a 16 year old girl who initially presented with symptoms of headache, fever, vomiting followed by altered sensorium and seizures. MRI brain showed (FLAIR) hyperintensity in bilateral thalami and basal ganglia. CSF Japanese encephalitis IgM antibody was positive. She showed improvement over a period of two months with some residual neurological deficits. The main aim of this case report is to highlight the fact that a high index of clinical suspicion is needed to diagnose JE when it occurs in a non-epidemic setting in urban areas.

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1. Introduction

Japanese encephalitis (JE) is a mosquito borne viral encephalitis. It is caused by Japanese encephalitis virus (JEV), a flavivirus. It is a zoonotic disease and the transmission cycle mainly involves mosquitoes, pigs and bats. The most important mosquito vector of this disease is *Culex tritaeniorhynchus*. Humans become infected when they are bitten by the mosquito and they are dead-end hosts. Pigs are the main contributors in the transmission cycle with respect to human infection.

It is estimated that around 30000 cases of Japanese encephalitis and 15000 deaths occur each year in southern and eastern Asia². Japanese encephalitis epidemics have been reported from the Indian state of Uttar Pradesh. JE is a disease of rural areas. Cases of Japanese encephalitis have been reported from Bellary district of our state (Karnataka). It is difficult to diagnose Japanese encephalitis when it occurs in a non-epidemic setting. We describe a case of Japanese encephalitis from an urban setting in a 16 year old female from Mangalore, Karnataka.

2. CASE REPORT

A 16 year old girl presented with history of headache since 3 days, fever since 2 days and altered sensorium since 1 day. On admission her temperature was 103°F, pulse- 100/min and BP-

120/80mmHg. Neurological examination revealed that she was disoriented and there was neck stiffness. Her blood counts, electrolytes, liver and renal function tests revealed no abnormalities. CECT brain revealed cerebral edema. Malarial test was negative. CSF analysis showed cell count - 85 (per µl), DC - Neutrophils-29%, Lymphocytes-71%. CSF protein - 44.1mg/dl, Glucose - 77mg/dl. Gram stain, Ziehl Nielsen stain and culture of CSF were negative. On the basis of clinical and lab findings empirical antibiotics were started along with acyclovir, mannitol and antiepileptics.

On day 3 of admission she developed focal seizures with paucity of movements in the left half of body. On clinical examination there was extensor plantar reflex on left side. MRI brain showed FLAIR hyperintensities in right thalamus and right frontal cortex (Figure 1).

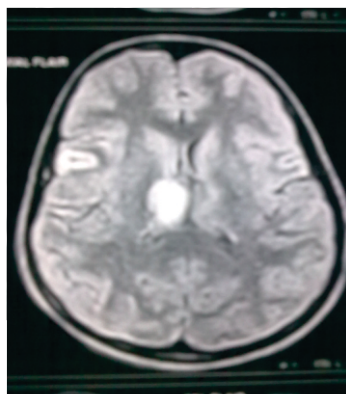
On day 4 she showed worsening of sensorium (GCS-3) with bilateral extensor plantar. Patient developed status epilepticus despite being on 4 antiepileptic drugs. She was mechanically ventilated and propofol infusion was started and inotropes were started as the patient developed hypotension. Repeat MRI showed FLAIR hyperintensity in bilateral thalami and basal ganglia (R>L) (Figure 2). CSF Japanese encephalitis IgM antibody was positive.

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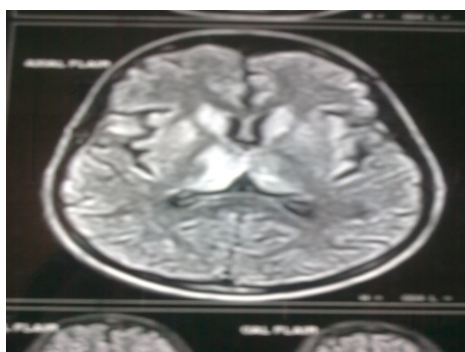
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MRI brain showing(FLAIR) hyperintensities in right thalamus and right frontal cortex (Figure 1).



Repeat MRI showing(FLAIR) hyper intensity in bilateral thalami and basal ganglia (R>L) (Figure 2).



Patient was on mechanical ventilation for a total of 20 days. During her stay in the ICU she developed rigidity of left upper and lower limbs with dystonia. She showed gradual improvement in sensorium and resolution of left hemiparesis at the end of 2 months. Residual deficits such as dystonia in left upper limb persisted even after symptomatic improvement. Patient was discharged after 80 days of hospital stay.

3. DISCUSSION:

JE is a disease of children and young adults. Infection with JEV may be asymptomatic or may present as meningitis, encephalitis or myelitis.⁴ The course of the disease can be divided into three stages: prodromal stage, an encephalitic stage and a late stage characterized by recovery or persistence of signs of CNS dysfunction.⁵ Severe encephalitis is associated with a higher frequency of seizures.¹ Our patient had fever, altered sensorium, convulsions and later had residual neurological deficit. Convulsions were present in 98.7% of the cases and was the first neurological manifestation of illness, followed by altered mentation.³ Apart from the classical presentation other atypical presentations of JE like acute transverse myelitis has also been documented.⁵ About 30% of JE cases are fatal and 50% result in permanent neuropsychiatric sequelae.⁶

Diagnosis of JE is established by the detection of antibodies, viral antigens or by detection of viral genome in serum or cerebrospinal fluid. World Health Organization has laid down criteria that needs to be fulfilled to diagnose a case as JE.⁷ CSF Japanese encephalitis IgM antibody was positive in our patient. MRI of brain can also assist in making a diagnosis of JE. On MRI, thalamus, basal ganglia and brainstem involvement are common.⁸ MRI brain can be normal in some patients.⁹ MRI showed FLAIR hyperintensity in bilateral thalami and basal ganglia in our patient. Imaging studies may be useful in distinguishing Japanese encephalitis from herpes simplex encephalitis. In Herpes changes are mainly in frontotemporal regions.

There is no cure for JE and treatment is mainly supportive.¹⁰ Malaria, dengue, herpes and tuberculosis are common causes of fever with altered sensorium in our state. It is difficult to identify Japanese encephalitis during initial stages as it is mainly characterized by non-specific symptoms. A high index of clinical suspicion is needed to diagnose JE when it occurs in a non-epidemic setting in urban areas.

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