

## Case Report

# Hemiplegia with dysarthria: an initial manifestation of Japanese encephalitis in a 4-year old child

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Japanese encephalitis (JE) was first recognised after an outbreak in Japan in 1924 that led to 6125 cases. Nearly, all the cases occur in children younger than 10 years, and more than 99% of infections are sub-clinical. After an incubation period of 4 to 14 days, the earliest symptoms are lethargy, nausea or abdominal pain, headache and fever. During a period of 2–3 days, lethargy increases and the child may exhibit uncharacteristic patterns of behaviour and motor abnormalities. Extra pyramidal signs usually develop in the second week of illness<sup>1</sup>. JE has a mortality rate of 10–20%, but may rise to 40–50% for comatose patients<sup>2</sup>. Here we describe a 4-year old child who presented with fever and left hemiplegia with left facial weakness and preserved consciousness throughout his illness but after fifth day onward developed extra pyramidal symptoms.

A right handed 4-year old boy presented with history of fever for one day without chills and rigors and headache, followed by seizure of the left half of body, without loss of consciousness. A day later he developed left sided hemiplegia and left facial weakness with well-preserved consciousness. Routine evaluation on Day 3 revealed a conscious, but less alert child with slurred speech and excessive crying. Fundi were normal. On Day 5, he developed some altered behaviour and rigidity more marked on the left side along with choreoathetoid movements in limbs. On

examination, meningeal signs were absent. All the deep tendon reflexes were brisk and plantar was bilaterally extensor. Till Day 10 his condition remained static, after that he progressively improved. Laboratory investigations revealed Hg 11 g/dl, total leukocyte count 11000/mm<sup>3</sup>, polymorphs 71%, lymphocyte 25%, eosinophil count 4%, ESR was 47 mm in the first hour which was normalised over a week. Serum electrolytes were found normal. CSF examination on Day 2 of illness revealed no abnormality, culture of blood and CSF was found sterile. Initial CT scan of head (Fig. 1) was performed before lumber



*Fig. 1: CT scan on Day 7*



*Fig. 2:* CT scan on Day 10 showing focal acute infarct in right frontal and parietal lobes

puncture and was within normal limit. A repeat CSF was done on Day 8 which revealed almost normal findings but IgM ELISA for JE virus revealed titers of 229 units in serum and 221 units in CSF strongly suggestive of Japanese encephalitis. ELISA test for antimycobacterial antibodies was negative. Electroencephalogram showed diffuse theta wave slowing. Repeat CT scan (Fig. 2) was done after 10 days of previous and showed focal acute infarct in right frontal and parietal lobes, with hypodensities in bilateral basal ganglia. Patient was managed symptomatically and discharged after improvement. During the follow-up over a period of 10 months he showed residual weakness with stiffness of the left half of body with choreoathetoid movements and slurred speech. Intellectual functions were grossly deteriorated.

Hemiplegia as an initial manifestation of JE is an unusual phenomenon and symptoms like tremors, rigidity, thick slurred speech may be initial features, but more frequently, choreoathetosis and other extra pyramidal signs become evident in the second week of illness<sup>1</sup>. Our patient from endemic area to JE and presented with a dense left hemiplegia, facial weakness and progressed to the encephalitic stage. There-

after he developed extra pyramidal manifestations within the first week of illness. A specific diagnosis can be confirmed by JE virus-specific IgM antibody in serum or CSF by enzyme-linked immunosorbent assay (ELISA)<sup>3</sup>. In the present case, the CSF IgM ELISA titer was diagnostic of JE. The CT scan in patients with JE show diffuse white matter edema and non-enhancing low-density areas, mainly in the thalamus, basal ganglia, and pons. Thalamic lesions frequently are associated with unilateral or bilateral haemorrhage<sup>4</sup>. A thalamic location of involvement is consistent with the electroencephalograms, pattern of slowing of background activity mainly in delta to theta range<sup>5</sup>. Bilateral symmetrical thalamic lesions are also seen in metabolic diseases such as Wilson's disease, anoxic encephalopathy and childhood hepatocerebral degeneration. However, the clinical and laboratory features of these diseases are distinctive compared to JE. The pathological lesions in JE are foci of neuronal degeneration with parenchymal and perivascular inflammatory responses are found principally in thalamus and brain stem, as well as in the hippocampus, temporal cortex, cerebellum, and spinal cord<sup>6</sup>. These changes based upon the anatomical locations describe the most of clinical manifestations of JE. In this case, it may be possible that the thalamus and the internal capsule may get involved initially as a result of involvement of right middle cerebral artery, prior to involvement of encephalon by the virus and resulting into the dense hemiplegia and facial nerve palsy. It can be explained on the basis of CT scan findings which show infarct in the territory of right middle cerebral artery.

In conclusion, the initial clinical symptoms like fever, headache and hemiplegia with dysarthria are unusual presentation in JE, especially in endemic areas. Hence, any child who is presenting like this should be evaluated in early stage of JE so that the mortality as well as morbidity can be prevented.

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