Case Report

Hemispheric encephalitis secondary to HAV

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CASE PRESENTATION

A 2-year male child, presented to National Institute of Child Health (NICH), with acute onset high grade fever and focal left sided seizures for 1 day, followed by left hemiparesis and encephalopathy. Developmental and family history was unremarkable. On physical examination, patient's body temperature rose up to 38.7 °C. Though he had pallor along with hepatomegaly, there were no signs of jaundice or ascites. Central nervous system examination showed encephalopathy as well as positive neck stiffness. Motor system examination revealed generalized decrease in bulk of upper and lower limbs, but rest of the findings were localized to left side of the body, showing hypertonia, decreased power, brisk muscle stretch reflex, and positive ankle clonus and left Babinski sign.

Blood investigations showed anemia (hemoglobin 9.1g/dl), leukocytosis (white blood cells 22.1 cells/µL) and raised aspartate aminotransferase (AST) levels (826 IU/L). Total bilirubin and direct bilirubin levels were normal (0.3 mg/dL and 0.1 mg/dL, respectively). Serum ammonia (64 µg/dL) and lactic acid (1.6 µg/dL) levels were also within normal ranges. The cerebrospinal fluid (CSF) was also clear, with 0 leukocytes/µL, protein levels of 25 mg/dL, and normal glucose levels (82 mg/dL), no organism was seen on gram stain. Hepatitis A Ig M antibody came out to be reactive.

Brain computed tomography showed large hypo density along with effacement of sylvian fissure, sulci, gyri on right side involving frontal, parietal, and temporal and occipital lobe on ipsilateral side. On post contrast, there was remarkable meningeal enhancement on right side. MRI brain revealed cortical and subcortical large area of abnormal signal intensity seen in fronto-parietal and occipital cortex on the right side along with laminar necrosis.

Seizures were controlled by given intravenous injection phenytoin and leviteracetam in bolus and then maintenance doses. Intravenous acyclovir was started due to clinical suspicion of herpes encephalitis but was stopped after observing clinical improvement and identification of Ig M antibody of HAV. Patient conscious level improved after 2 weeks. AST levels also decreased to 20 IU/L and he was discharged with advice to follow up after 14 days.

DISCUSSION

Hepatitis A virus (HAV) is a major public health problem worldwide. It is a picornavirus, and is transmitted by fecal-to-oral route. The clinical presentation may range from mild flu like symptoms to acute fulminant hepatitis. Diagnosis may be impeded during the anicteric phase, due to atypical clinical findings. Usually HAV has a prodromal phase of flu like illness, which progresses to symptoms due to hepatic involvement and rapid elevations in liver enzymes. Transaminase levels follow static course in a week and then gradually decreases to normal levels. Serum IgM antibody of HAV can be detected to make the diagnosis.[1-3]

Neurological involvement is rarely associated with HAV, especially encephalitis, myelitis, peripheral neuropathy and Guillian-Barre' syndrome.[4, 5] Acute viral encephalitis however, can be caused by a variety of viruses, such as herpes virus, alpha virus, flavi virus, Chandipura virus, rabies virus, influenza A virus, measles and enterovirus.[6] In addition to these viruses, cases of encephalitis and meningoencephalitis associated with HAV have also been rarely reported.[7] Pathogenesis behind this is attributed to a disturbed function of the damaged liver and metabolic abnormalities, such as fluid electrolyte imbalances, or direct invasion of the central nervous system through encephalitis.[8]

First case report on HAV associated encephalitis was reported by Bromberg *et al* in 1982.[9] After reviewing a few case reports, it was observed that altered consciousness and seizures were the common presentations. CSF pleocytosis was also noted in most but not all cases. Positive HAV PCR results were reported in one case, of which CSF cells and protein were normal. Specific radiological or EEG findings did not appear in all the cases.[10]

Conflict of interest: The authors declared no conflict of interest exists.
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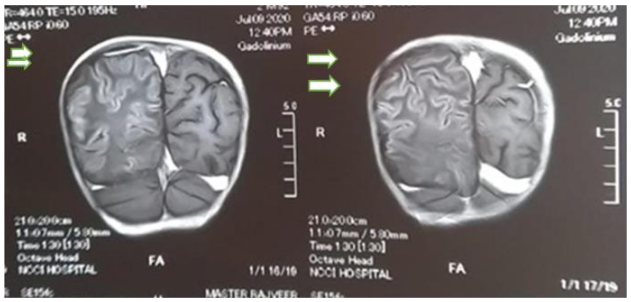

Figure 1. MRI brain coronal images revealed cortical and subcortical large area of abnormal signal intensity seen in fronto-parietal and occipital cortex on the right side along with laminar necrosis

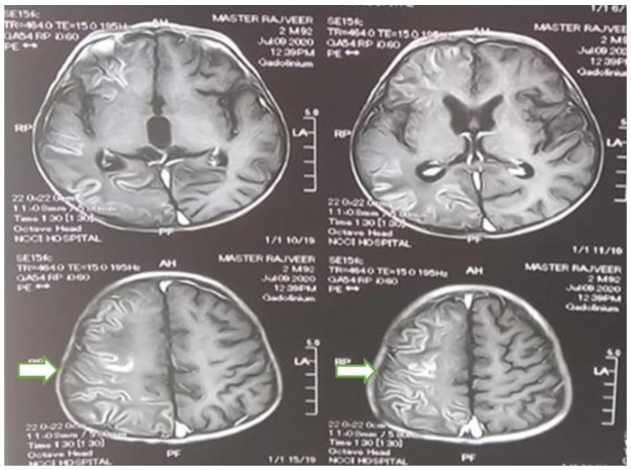

Figure 2. MRI brain axial imaged revealed cortical and subcortical large area of abnormal signal intensity seen in fronto-parietal and occipital cortex on the right side along with laminar necrosis

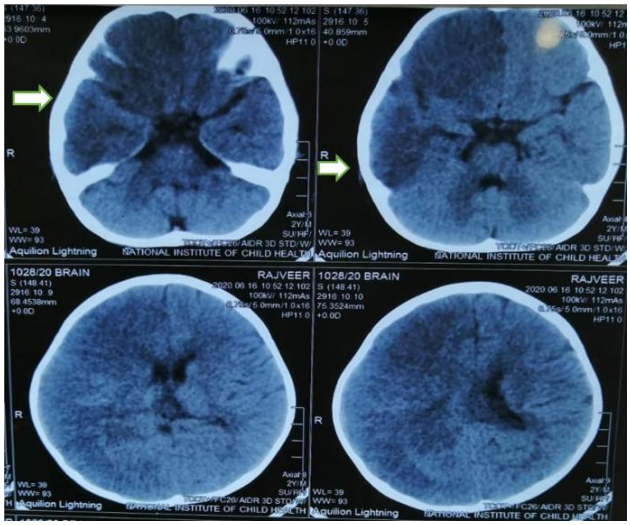

Figure 3. Brain computed tomography showed large hypo density along with effacement of sylvian fissure, sulci, gyri on right side involving frontal, parietal, and temporal and occipital lobe on ipsilateral side. On post contrast, there was remarkable meningeal enhancement on right side.

HAV associated encephalitis is a very rare disorder and an exclusive finding of our case was involvement of the unilateral hemisphere. In the anicteric phase, the diagnosis may be troublesome without a clinical suspicion. Possibility of HAV associated disease should be considered when liver transaminases are progressively increasing in patients with encephalitis.

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