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Radiology

A CASE REPORT OF DENGUE ENCEPHALITIS

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ABSTRACT Dengue is classically considered a non-neurotropic virus, various case reports have shown there is increasing evidence for dengue viral neurotropism, suggesting that, there may be an element of direct viral encephalitis in patients with dengue. We present a case of dengue encephalitis, diagnosis of which was confirmed by blood and cerebrospinal fluid dengue serology and (NS1) antigen assay. The case showed extensive lesions involving the thalamus ,midbrain, and cerebellum on both sides of the brain in symmetrical fashion

KEYWORDS :

Introduction:

Dengue virus is a single stranded RNA virus of Flaviviridae family.The CNS manifestations can be attributed to three factors i.e. neurotropic effect, secondary to systemic manifestation and postinfectious sequelae including immune mediated reactions.[1,2]. Although the exact mechanism by which dengue virus crosses the blood-brain barrier is unclear, it has been proposed that the entry occurs through infected macrophages. [3] Neurological manifestation usually results from multisystem dysfunction secondary to vascular leak.MRI is modality of choice for evaluation of dengue encephaltits& findings often mimics Japanese encephalitis.

II. Case report: A 7 year old female patient presented at our hospital with Complain of Fever with chills, Weakness Headache and generalised bodyache since 2 days. On examination she was febrile, with a temperature of 101°F, pulse of 132 / minute, blood pressure of 90 / 40 mmHg (MAP-60 mmHg) and cold extremities. There were no signs of respiratory distress. She was pale, and had skin rashes. She had one episode of epistaxis on the day of admission. The neurological examination did not reveal any significant abnormality. There was no neck stiffness. The rest of the systemic examination was also within normal limits. There was no hepatosplenomegaly.Hemoglobin was 12.3gm/dl,Total leucocyte count on admission was 8800/cu.mm. Platelate counts on admission was 2.14lacs/cu.mm& After 12hrs of admission count decreased to 1.74lacs/cu.mm. Hematocrit increased over 12 hrs from 38.7% to 42.3%.Serum electrolyte panel and DIC panel were normal. Patient was suspected to have a dengue fever.Dengue virus NS-1 antigen test came out reactive. Malaria rapid antigen test was non-reactive. Patient was sent for USG abdomen and chest to look for signs of plasma leakage.usg showed pleural effusion with moderate ascites. Patient had a drop of Platelate counts over next 2 days and the lowest counts were 10,000/cu.mm.Patient was treated for dengue fever,blood pressure and urine output improved and gradually patient recovered symptomatically and Platelate counts increased over next 3-4days.Patient became afebrile on day 7 of admission and was fit for discharge, when she suddenly had an episode of generalised tonic-clonic seizures followed by loss of consciousness. Temperature increased over next 12hrs.Again Platelate counts dropped over next 24hrs.SGOT and SGPT increased markedly over next 24hrs. (472IU/L and 350IU/L respectively). Serum. Urea and creatinine and electrolyte panel were normal. Patient was suspected to have dengue encephalitis. An MRI brain scan was done as an emergency which showed symmetrical flair hyperintensity in b/l thalamus ,midbrain,pons & cerebellum which showed diffusion restriction and heterogenous contrasr enhancement.

Figure 1 abdominal usg shows extensive oedematous wall of gallbladder & moderate pleural effusion

Fig.1

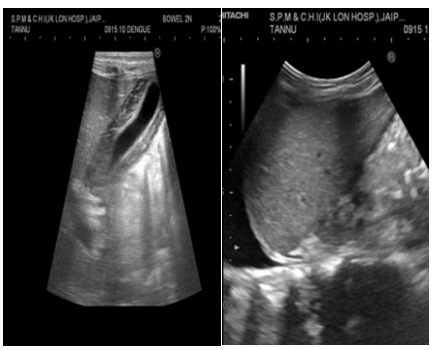


Figure 2 shows bilateral symmetrical FLAIR hyperintensities in thalami, , and mid brain, pons & cerebellar hemisphere .

Fig.2

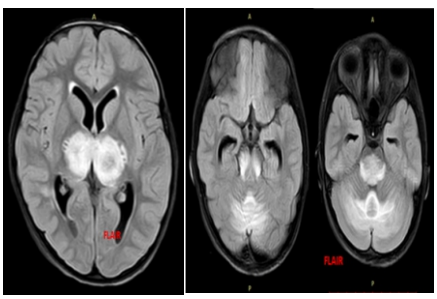


Fig.3 Thalamic & cerebellar Lesions are showing diffusion restriction

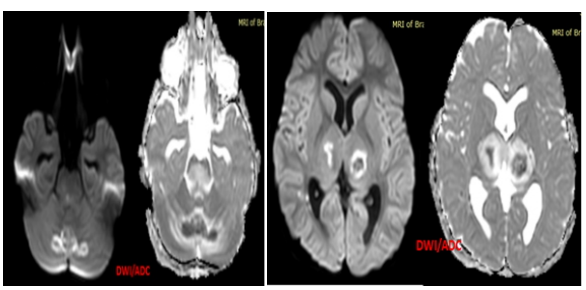
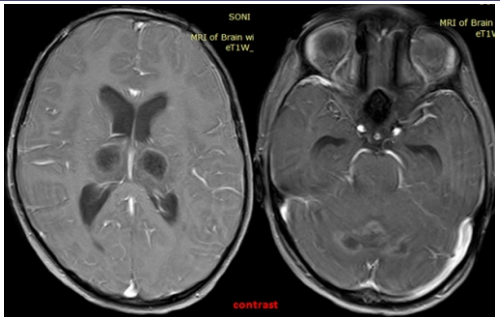


Fig.4 shows heterogenous contrast enhancement in bilateral thalamus & cerebellum



- **III. Discussion& Conclusion ;** Neurological manifestations of dengue usually result from multisystem dysfunction secondary to liver failure, cerebral hypoperfusion, electrolyte imbalance, shock, cerebral edema, and hemorrhage related to vascular leak which leads to encephalopathy. [4,5]. various neurological manifestations of dengue encephalitis includes seizure, encephalopathy, meningitis, myelitis, Guillain-Barré syndrome ,and myoclonus that are commonly reported[6].Our case showed bilateral symmetrical FLAIR and T2 hyperintensities in thalami,midbrain, pons, and cerebellum with heterogenous or peripheral enhancement on contrast administration, with few of these lesion showed diffusion restriction which was similar to cases described by Bhoi et al.,[7] &Souren et al.,[9]. Chikungunya encephalitis also presents with clinical presentations similar to dengue encephalitis.However, Chikungunya encephalitis shows T2 weighted hyperintense white matter lesions with restricted diffusion.[8] Bilateral thalamic involvement with positive IgG/IgM in CSF for dengue virus is diagnostic.Polymerase chain reaction (PCR) for confirming the viral RNA is required, as antibodies are not seen in all cases. Although dengue encephalitis is rare but in appropriate clinical settings diagnosis of the same must be considered.

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