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Case Report

Dengue Encephalitis Presenting as Complex Partial Status Epilepticus

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Abstract
Dengue is a potentially fatal acute febrile illness caused by infection with any of the four dengue viruses, DENV 1,2,3,4. Spectrum of disease may vary from asymptomatic infection to more fatal dengue haemorrhagic fever (DHF) and dengue stroke syndrome (DSS). Neurological manifestations of dengue can be seizures, encephalitis, meningitis, encephalopathy, stroke and Guillain barre syndrome. We report a case of 24 year old male who presented to us with fever and complex partial status epilepticus and was diagnosed as Dengue encephalitis.
Keywords: *encephalopathy, stroke, seizures, neurological, DHF, DSS.*

Introduction

Dengue fever, an arboviral infection is recognised as one of the most important mosquito born human infection caused by dengue virus transmitted in humans through Aedes mosquito. Four different serotypes of DENV 1,2,3,4 causes fever with various infectious outcome i.e; asymptomatic to severe haemorrhagic fever. WHO classified dengue infection into 3 categories[1]: Dengue with no warning signs, Disease with warning signs, Severe dengue with CNS involvement. Neurological complications in dengue can be encephalitis, encephalopathy, meningitis, stroke, cerebellar syndrome[(6)], Transverse myelitis ,Acute disseminated encephalomyelitis.[2-10] Other uncommon neurologic complications are Guillain barre syndrome, myositis, hypokalaemic paralysis, neuritis , lateral rectus palsy and peripheral facial palsy.[11-16] Ophthalmic complications are maculopathy and retinal vasculopathy.[17]

Case Report

A 24 year old male presented to us with abnormal bizarre behaviour, fever and diplopia since one day, as per the history given by the relative the patient was alright one day back, then had fever which was high grade associated with headache,

myalgia, neck and back pain. There was no history of chills, cough, expectoration, burning micturition, oliguria, diarrhoea, vomiting. Twelve hours after the fever the patient suddenly had bizarre behaviour in form of excessive talkativeness with intermittent mutism and subtle abnormal movements in his face and upper limb with complex movements like lip smacking ,intermittent picking at clothes and his shirt buttons and not responding to verbal commands .This episode lasted for 15 minutes and patient became normal. Patient was shown to general practitioner where antipyretics were prescribed and referred to higher canter.

On examination in this hospital, patient was febrile, pulse 110 /min regular, blood pressure was 110/70mm Hg. Other general examination was normal .There were no rash or petechial spots present on the body. CVS, RS and Per abdomen examination were normal.

CNS examination; patient was alert but was not responding to verbal commands ,abnormal automatisms was present in the form of lips smacking and picking movements involving the left hand. He was moving all fourlimbs, cranial nerve examination revealed bilateral 6^{th} nerve and right 3^{rd} nerve palsy (figure 1-4). Bilateralplantars were mute. Neck stiffness was present. Kernig's sign was positive. A presumptive diagnosis of meningoencephalitis was kept. The patient was treated with IV fluids, antipyretics, inj lorazepam was given 2mg iv stat, seizures subsided and empirical antibiotics were started.

Investigations revealed, Hb:11.5 gm%, MCV: 101fl, Absolute platelet counts 94,000/mm^3, TLC: 5200 mm 3, DLC showed lymphocytic pleocytosis. PS was negative for malarial parasite. Paracheck was negative. Dengue NS 1/ Ig M were positive.

CSF examination: TLC: 60cells/HPF, 90% lymphocytes, sugar 148mgdl, protein : 96 mg/dl. Dengue Ig M in CSF was positive. PCR for HSV was negative. KFT, LFT was normal. Serum sodium was 132 mEq/L. Other electrolytes were normal.

CT report showed right temporoparietal cortical enhancement (Figure 5).EEG was suggestive of Complex partial seizures.

Patients medication was initiated with tab carbamazepine 100mg bd, tab Levetiracetam 500mg, tab. folic acid 5mg and plenty of fluids. Patient started to respond well , complex partial seizures subsided, cranial nerve palsy recovered (Figure 6) and over a course of 1week patient recovered and was discharged when he appeared to be symptomatically normal.

Figure 1-4: showing bilateral lateral rectus palsy and right superior rectus palsy

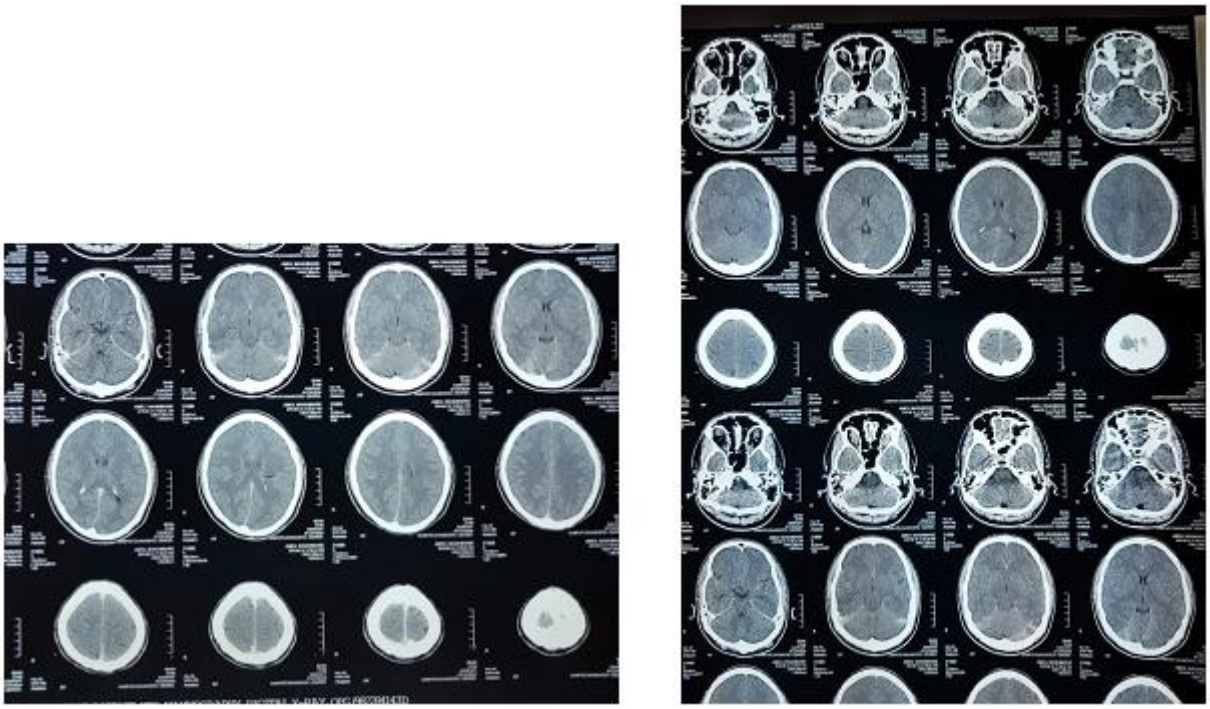

Figure 5: CT Brain showing; right temporoparietal cortical enhancement.

Figure 6 showing recovery of 6th nerve palsy

Discussion

Dengue meningoencephalitis is usually rare[18] The neurological signs of dengue were reported in 1976, though it was still considered to be an atypical manifestation [19] In the recent years the incidences of neurological manifestations varies from 1-20 %[20] .

Though dengue virus is not typically neurotropic but studies now have proven that it does invades central nervous system. The virus has been detected in CSF [21].It has been also demonstrated to invade the blood brain barrier[22]. There is no paucity of data that now clearly declares dengue to be neurotropic.[23]

The neurological complications of dengue has been classified into 3 categories 1)metabolic encephalitis 2)meningitis, encephalitis, meningo-encepalitis, myositis ,transverse myelitis and, 3) auto immune reactions like ADEM, neuromyelitis optica and Guillain barre syndrome [24]

Dengue meningits is rare occurrence meningoencephalitis is rarer still.[25] The usual symptoms are fever, headache and neck stiffness and the CSF picture typically reveals lymphocytic pleocytosis increased protein levels and normal sugar levels, diagnosis can be confirmed by positive IgM in CSF. Encephalitis is one of the severest of neurological manifestations in dengue

endemic regions. Our patient had meningoencephalitis because he typically showed signs of meningeal irritation with CSF confirming it. The abnormal behaviour and complex partial seizures, with hyperintensities in the temporoparietal area confirmed encephalitis in our case.

Definite criteria for diagnosing dengue encephalitis has been laid down[25,26]

It is;

1) Presence of fever
2) Acute signs of cerebral involvement such as altered consciousness or personality and/or seizure and/or focal neurological signs
3) Reactive IgM dengue antibodies ,NSI antigen or positive dengue PCR on serum and /or CSF .the choice of one of these laboratory methods should be performed according to time of infection onset
4) Exclusion of other causes of viral encephalitis and encephalopathy

Still to date there is no classical feature to neuro imaging to suggest definite dengue encephalitis.[27] Encephalopathy in dengue also presents as cognitive impairment acute mania, depression, psychosis and seizures. Our patient was unique because he developed complex partial seizures which is rarer. The causes of encephalopathy in dengue are due to cerebral oedema, hypoxia, haemorrhage and hyponatremia.

As far as management of dengue encephalitis is concerned no specific anti viral is available till date and the treatment is usually supportive in form of haematological monitoring, fluid resuscitation ,treatment of thrombocytopenia ,blood transfusions, anticonvulsant therapy were needed.

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