Dengue Encephalitis with Unilateral 3rd Nerve Palsy

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DOI: 10.29322/IJSRP.9.06.2019.p9008
http://dx.doi.org/10.29322/IJSRP.9.06.2019.p9008

Abstract- Dengue encephalitis with cranial mononeuropathy is a rare entity. Imaging being non-specific, diagnosis of the condition relies on detection of NS1 antigen and IgM antibodies in CSF. Treatment relies on maintenance of airways, adequate hydration and nutrition along with monitoring of vitals without any need of antivirals or antibiotics. Prompt recognition of the condition is necessary firstly due to the potential mortality associated with the condition and secondly to avoid unnecessary administration of drugs. Here we report a case of 25 year old female who presented with fever, vomiting and altered sensorium, had an isolated left 3rd nerve palsy, was investigated to have dengue encephalitis accounting for the same, was managed conservatively, discharged with stable vitals and resolution of the symptoms and normalisation of lab parameters.

Index Terms- Dengue encephalitis; Mononeuropathy; Neurotropism

I. INTRODUCTION

Neurological manifestations in dengue infection, caused by Dengue virus, a single-stranded RNA virus of the Flaviviridae family are relatively uncommon. It includes encephalitis, encephalopathy, neuromuscular disorders and neuro-ocular disorders with cranial mononeuropathy being a rare manifestation.[1]Dengue encephalitis is a rare entity which occurs due to direct neuronal infiltration by the dengue virus.[2,3]Here we report a case of dengue fever with encephalitis with unilateral 3rd nerve palsy.

II. CASE REPORT

A 25 year old female presented with the complaints of fever along with chills since last 5 days,multiple episodes of vomiting since last 2 days and decreased responsiveness since last 1 day. On examination patient was drowsy,Pulse-88/min,BP-100/60mm Hg, blanchable rashes were present, Chest, CVS, P/A examinations revealed no abnormalities, CNS examination revealed absent meningeal signs, pupils unequal in size with left sided pupil fully dilated not reactive of light(Fig 1),bilateral plantar being non-responsive, Fundus examination was normal. Investigations revealed Hb-7.7g/dl,Hct-23.7%,MCV-110 fl,MCH-36.4pg,Platelet-18,000/µl,TLC-4500/ µl(N-25%,L-60%,M-15%),peripheral smear didn't show any hemoparasites, RBCs showed moderate anisocytosis with macrocytes, normocytes and elliptocytes,Na/K-135/4.2 meq/L,Urea/Creatinine-35/0.4 mg/dl,Total Bilirubin-o.4 mg/dl,AST/ALT/ALP-451/114/182 U/L,INR-0.99,ABG,Chest X-ray,ECG , NCCT head revealed no abnormalities. CSF study revealed 110 cells with 98% lymphocytes,2% neutrophils,Protein-109mg/dl,Sugar-47mg/dl.USG abdomen revealed Gall bladder wall edema.She was started on IV acyclovir and artesunate along with crystalloids.Her fever subsided after 3 days,sensorium improved but there was persistent headache,nausea and absent vision in the left eye.She now had developed ptosis of the left eye with fully dilated pupil suggestive of 3rd nerve palsy(Fig 2).A repeat fundoscopic examination revealed Grade 1 papilledema.Meanwhile her serum for Dengue IgM came out to be positive,Malaria serology,Scrub typhus serology and Typhidot IgM were negative.An MRI brain was done which revealed altered signal intensity in left cerebral peduncle,bilateral medial temporal lobes,bilateral hippocampi,corpus callosum splenium and left parasagittal occipital lobe,iso-hypointense on T1,hyperintense on T2/FLAIR(Fig 3,4).The MRI findings were suggestive of encephalitis,likely of viral etiology.To look for the etiology,CSF for HSV IgM,JE serology,CBNAAT were sent which were negative, but was positive for Dengue IgM. Thus a diagnosis of Dengue encephalitis with left 3rd nerve palsy was established.IV Acyclovir was discontinued,repeat fundus examination revealed no papilledema,headache subsided consequently.Her vision improved,ptosis subsided and the left pupil was sluggishly reactive to light after ten days of treatment, LFT showed resolution of the derangement(AST/ALT/ALP-56/25/134). Patient was discharged with stable vitals,resolution of symptoms after two weeks and follow up after one month revealed no abnormalities.

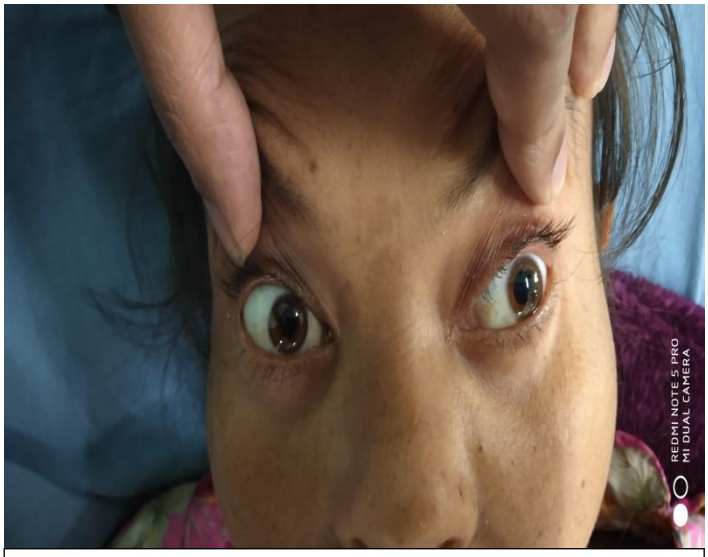

Fig 1 showing dilated left pupil

Fig 2 showing left sided ptosis

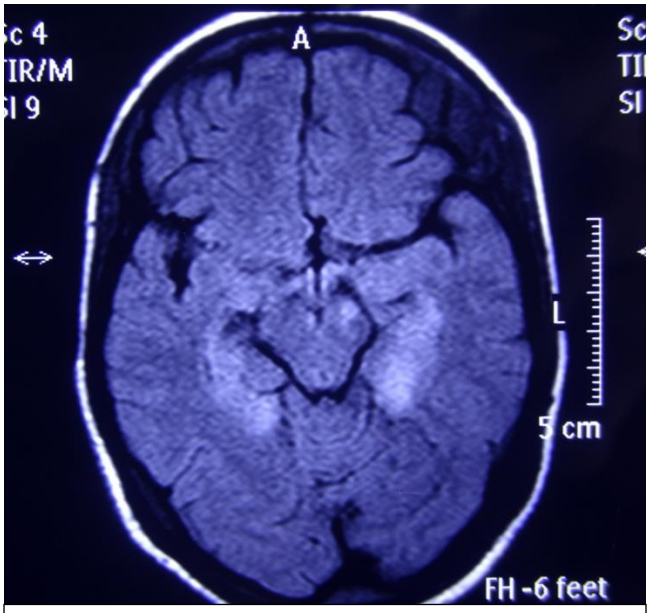

Fig 3 showing T2 FLAIR MRI image with hyperintensity noted in bilateral medial temporal lobes.

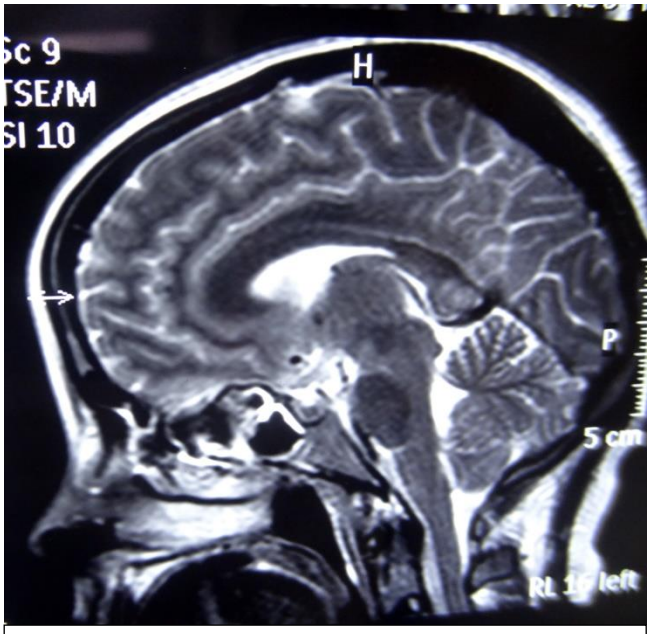

Fig 4 showing T2W MRI image with hyperintensity noted in corpus callosum

III. DISCUSSION

The incidence of dengue encephalopathy ranges from 0.5 to 6.2 %.[4] The proposed possible mechanisms responsible for the same being liver failure (hepatic encephalopathy), cerebral hypoperfusion (shock), cerebral edema (vascular leak), deranged electrolytes, and intracranial bleeding due to thrombocytopenia or coagulopathy, which is secondary to hepatic failure.[5] In a subset of patients the cause for neurological injury could not be elicited even after excluding the above-mentioned indirect mechanisms which raises the possibility of direct neuronal injury due to the dengue virus.[3] Neurotropism and CNS invasion in Dengue virus(DENV) infection has been demonstrated in recent years and detection of viral antigen in brain tissue and DENV RNA amplification in the CSF support the invasive neurotropism of DENV.[5] DENV-2 and DENV-3 serotype infections and young age have been found to be the risk factors for dengue encephalitis.[2] The mortality associated with the condition may be high and the prognosis depends on the causal factors,associated comorbidities and early supportive treatment.[6] Dengue encephalitis is manifested by a reduced level of consciousness, headache, fever, nausea and vomiting, seizures, focal neurological deficits, and behavorial symptoms.[7] There aren't any specific neuroimaging findings suggestive of dengue encephalitis and brain MRI may be normal or show some focal parenchymal abnormalities.[4] These include symmetric gyral edema,altered signal intensity involving bilateral temporal perisylvian regions, hippocampi, and cingulated gyri;[8] thalamus, pons, and bilateral cerebellum cortex can be involved as well.[9]Meningeal enhancement has also been seen on postcontrast MRI.[10] To differentiate between dengue encephalitis from encephalopathy, CSF analysis has been helpful. A lymphocytic pleocytosis with mild increase in CSF protein can be found in

the CSF of patients with dengue encephalitis although a normal CSF cellularity does not exclude the diagnosis.[4] Dengue encephalitis is confirmed by the presence of dengue NS1 antigen and DENV-specific IgM antibodies in the CSF.[9] The management of dengue encephalitis is primarily supportive with maintenance of airway,adequate hydration and nutrition and monitoring of consciousness level without any role of antivirals.[5] Though the ophthalmic complications associated with dengue infection were once thought to be rare,the incidence has increased in the time being with more reported cases with maculopathy being the most common complication whereas retina vasculopathy, optic neuropathy and cranial nerve palsy,the lesser common ones.[12] Although the exact mechanism is incompletely understood,the pathogenesis in dengue-related neuro-ophthalmic complication is believed to be immune mediated and the delayed onset of visual symptoms of up to 1-week following dengue infection favours the same.[11] The overall prognosis for dengue-related ocular complications has been found to be good, and complete recovery coincided with improved platelet counts.[11,12]

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