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General Medicine

"DENGUE ENCEPHALITIS – A RARE MANIFESTATION OF DENGUE FEVER."

Dr. Showkat Nazir Wani	MD Department Of General Medicine
Dr. Anish Garg Atul Kaushik	MBBS Department Of General Medicine
Dr. Atul Kaushik*	M.B.B.S., Post Graduate Resident, Dept. of General Medicine, Sharda Hospital, SMSR, Greater Noida, U.P., India. 201308.*Corresponding Author
Dr. Naveen Tariq	MBBS Department Of General Medicine

ABSTRACT We report a case of dengue fever with features of encephalitis from Northern India. A 22 years female presented with history of high-grade fever with chills for 2 days followed by altered sensorium and MRI brain, revealed characteristic changes consistent with Encephalitis with hemorrhage mainly involving the bilateral thalami. The diagnosis of dengue was confirmed by the presence of dengue antigen in the serum and the presence of dengue antibodies in the serum as well as cerebrospinal fluid. Dengue is not primarily a neurotropic virus and encephalopathy is an uncommon finding in dengue. This case explains the importance of considering dengue encephalitis as the differential diagnosis of fever with altered sensorium, especially in countries like India where dengue is rampant.

KEYWORDS : CNS, Dengue, Encephalitis.

INTRODUCTION

The Dengue virus is a single-stranded RNA virus of the Flaviviridae family causing dengue fever, dengue shock syndrome and dengue hemorrhagic fever. Unlike other arboviral infections, dengue virus does not usually cause neurological manifestations [1]. Dengue encephalopathy is usually secondary to multisystem derangement like shock, hepatitis, coagulopathy, and concurrent bacterial infections [2]. Dengue encephalitis is a different entity, which occurs due to direct neuronal infiltration by the dengue virus [3,4]. We report a case of dengue fever with encephalitis, with peculiar MRI brain findings.

Case History

A 22 years female patient presented with history of high-grade fever with chills since past 2 days, followed by altered sensorium after 1 day. There was no significant past medical history.

On admission patient was afebrile, pulse 98/minute, BP 100/70mmh of Hg. On pulse-oximetry, oxygen saturation was 97% at room air. There were no signs of respiratory distress. Pallor was present with no icterus, cyanosis, edema, skin rash or clubbing.

The neurological examination revealed GCS of E2V1M4 and signs of meningeal irritation were absent. Pupils were bilaterally small sized and not reacting to light, and both eyes were deviated medially and downward . There was generalized hypotonia, deep tendon reflexes were 2+ in all 4 limbs and babinski sign was present bilaterally. Rest of the systemic examination was within normal limits.. Patient was resuscitated with IV fluids and empirical therapy with 3rd generation cephalosporins, acyclovir and parenteral antimalarial was started.

Investigations revealed Hb-10.4 gm/dl, TLC-8080/mm^3, Neutrophils 49%, lymphocytes 40%, and monocytes 8% with a platelet count of 1.05 lac/mm^3. The liver enzymes were raised SGOT - 171.70 U/L, SGPT-109 U/L. Serum albumin was 3.5 gm/dl. Blood urea nitrogen was 24.30 mg/dl with creatinine of 0.4 mg/dl. The electrolytes were within normal range. X- ray chest, USG of abdomen and 2-D echocardiography were normal. Malarial antigen raid test was negative and PBF for malarial parasites was also negative. Serum Dengue serology for IgM and Dengue Ns1Ag were positive.

The urgent NCCT head revealed no evidence of infarction or intracerebral hemorrhage (ICH). MRI brain was performed in which T2/FLAIR images show ill-defined symmetric areas in bilateral frontal centrum semiovale, thalami, mid brain, posterior part of pons and cerebellar hemisphere. Areas of diffusion restriction are noted within the thalamus, Pons, bilateral Cerebellar hemispheres, Hippocampus and left subcortical white matter in parietal lobe. Blooming of SWI is seen in bilateral thalami, pins and cerebellar hemispheres suggestive of hemorrhage. Patchy post contrast enhancement is noted in bilateral thalami. (Fig.1)

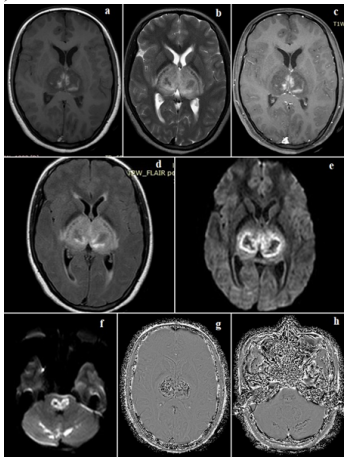

Fig. 1 –MRI Brain a.T1W-SE; b. T2W-TSE; c. T1W-SE Post contrast; d. T2W-FLAIR post contrast; e, f. DWI; g,h. SWI. (Description in text.)

On day 1 of admission, a lumbar puncture (LP) of CSF was performed and details mentioned in table 1. CSF analysis of day 5 are shown in table 2. It was positive for dengue IgM and IgG antibodies. Virus isolation was not possible. CSF for viral panel [HSV (Herpes Simplex Virus), EBV (Epstein Bar Virus) and JE (Japanese Encephalitis)] were negative.

Table 1: CSF analysis of Day 1 Lumbar Puncture.

Color	Colorless
Appearance	Clear
Total cell count	05
Differential	All lymphocytes
Gm stain	Negative
AFB stain	Negative
Culture	Negative
ADA	Normal

Table 2: CSF analysis of Day 5 Lumbar Puncture.

Color	Colorless
Appearance	Clear
Total cell count	120
Differential	N-20%, L-80%

Protein	110mg/dl
Glucose	57mg/dl
Gm stain	Negative
AFB stain	Negative
Culture	Negative
ADA	Normal
Dengue IgM	Positive
Dengue IgG	Positive

Patient was intubated on the 1st day after few hours of admission in view of low GCS. Patient developed generalized tonic-clonic seizures with decerebrate posturing of the body. Course got complicated by ventilator associated pneumonia, multiorgan dysfunction and shock. Empirical antibiotics and antimalarials were continued till reports of the causative agent were obtained, and then withdrawn. Acyclovir was stopped on the basis of CSF analysis and MRI brain findings.

In spite of all the treatment she continued to remain comatose, with hemodynamic compromise, and succumbed on the 45 day after admission.

DISCUSSION

Dengue virus infections are among the most common causes for hospital admissions in India. It is estimated that 50 to 100 million infections and 25,000 fatalities occur worldwide every year. The World Health Organization (WHO) surveillance shows that the global incidence is in rising trend [5]. Numerous neurological manifestations like transverse myelitis [3], myositis [6], and Guillain-Barre syndrome [7] have been reported. Dengue encephalopathy is a well-recognized and common entity, the incidence ranging from 0.5 to 6.2 % [6].

The possible mechanisms are 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 [8]. There are subsets of patients in whom the cause for neurological injury remains unclear even after excluding the above-mentioned indirect mechanisms. These raise the possibility of direct neuronal injury due to the dengue virus. Dengue is thought to be a non-neurotropic virus [4]. However, there are reports of the demonstration of dengue virus and IgM antigen in the cerebrospinal fluid (CSF) of patients with encephalopathy. In the study described by Misra *et al*. [6], 11 patients were seen with confirmed dengue infection, but no CSF study was reported. Solmon *et al.* [3], diagnosed dengue encephalitis in nine patients, but virus or antibody was found in the CSF of only two cases. Kankirawatana *et al*.[9]and Kularatne *et al.*[10], had a similar study in which they showed the association of dengue with encephalitis.

Positive MRI and CSF showed evidence of encephalitis. This showed the co-existence of encephalopathy and encephalitis. The MRI findings noted in our case are most characteristic of JE and not commonly seen with dengue fever [11]. The pattern of involvement found in MRI in this case is very uncommon with dengue. There is only one similar case report by Kamble *et al*., with similar MRI findings [11]. This case highlights the possible extensive involvement of the brain by dengue virus.

CONCLUSIONS

Dengue is not classically a neurotropic virus, although there is recent evidence of direct neuronal injury. Dengue encephalitis must be thought of as differentials of encephalopathy, in patients with dengue. In such cases, neuroimaging and CSF analysis should be done whenever possible. The virus or antibody can be isolated from the serum, but the CSF samples may be negative. The dengue encephalitis is thought to be benign, but can be fatal at times. The role of an antiviral in such cases needs to be further defined because of the extensive parenchymal involvement and possible unfavorable outcome.

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