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Case Report

A Doughnut in the Brain

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ABSTRACT

Dengue fever, a mosquito borne viral illness, is endemic in many parts of the world, particularly in tropical and subtropical regions. It is caused by four distinct serotypes (DENV 1–4) of single stranded RNA viruses from the Flaviviridae family. Although it is generally considered non-neurotropic, certain strains, especially DENV-2 and DENV-3, have been associated with neurological involvement. Neurological complications can include encephalopathy, encephalitis, meningitis, acute disseminated encephalomyelitis (ADEM), stroke, and Guillain-Barré syndrome. Dengue encephalitis, a relatively uncommon but serious manifestation, arises from direct viral invasion of neural tissue. This case report presents a patient with altered mental status and characteristic MRI findings symmetrical involvement of the thalami and cerebellum, known as the "double doughnut sign" indicative of dengue encephalitis. Cerebrospinal fluid analysis confirmed the diagnosis. Early recognition, neuroimaging, and supportive management with immunomodulation played a crucial role in the patient's recovery. Clinicians should maintain a high index of suspicion for dengue encephalitis in endemic areas when encountering unexplained encephalopathy.

KEYWORDS

• Dengue fever • Infectious encephalitis • MRI findings • Expanded dengue syndrome & complications

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INTRODUCTION

Dengue encephalopathy is usually secondary to multisystem derangement like shock, hepatitis, coagulopathy, and concurrent bacterial infection.[4] Dengue encephalitis is a different entity altogether, which occurs due to direct neuronal infiltration by the virus. The CNS manifestations can be attributed to three factors (a) neurotropic effect, (b) secondary to systemic manifestation, and (c) postinfectious sequelae including immune-mediated reactions.[1] It is postulated to occur mainly because of direct neuronal injury involving basal ganglia and thalamus complex bilaterally, causing cerebral oedema with haemorrhage secondary to vascular leak.[2] Resulting in a characteristic 'Double doughnut' appearance on MRI brain.

Case Report

A 51-year-old female presented with history of fever in the past 2 days along with generalized weakness. On Day 3 of illness in the morning, she had multiple episodes of vomiting along with severe headache. Thereafter she became drowsy and unresponsive by same day afternoon she was brought to the emergency department in obtunded state. She had no previous medical & surgical history. Upon arrival, she had tachycardia, (118 beats/min), blood pressure of 100/60 mm hg, respiratory rate of 18 breathes/min and saturation of 94%. She had depressed consciousness level with Glasgow coma scale (GCS) of E1V1-2M3. Pupils were small and reactive. Eyeballs were turned down and inwards. Doll's eye was absent. Bilateral decorticate posturing with extensor plantar were noted. There was no neck rigidity.

In view of low GCS and threatened airway, she was intubated in the emergency department was put on ventilatory support. Urgent MRI Brain done, and blood was drawn sent for investigations (*Table 1*).

MRI brain Showed (*Figure 1-4*) "symmetrical hyperintensity involving bilateral Thalamus, Pons, Cerebellum with frontal subcortical Hyperintensity". Patient was subjected to CSF tapping and sent for analysis (*Table 2*).

Table 1: Blood investigations as follows:

Investigation	Day-1
Hb(g/dl)	13.6
WBC Count /mm3	10,900
Hematocrit %	39.1
Platelet count	68,000
Neutrophil %	92.4
S. creatinine (mg/dl)	0.64
S. Sodium (mmol/l)	136
S. potassium (mmol/l)	
SGOT (IU/L)	1428
SGPT (IU/L)	1270
S. procalcitonin	0.48

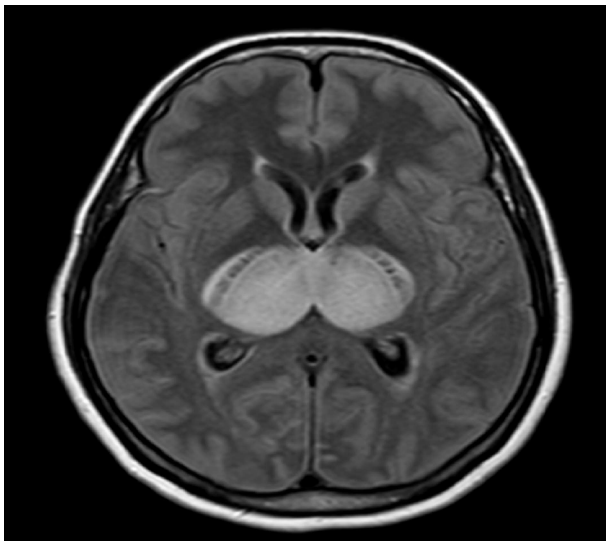

Figure 1: Axial FLAIR MRI showing bilateral thalamic hyperintensities part of the characteristic "doughnut in the brain" appearance

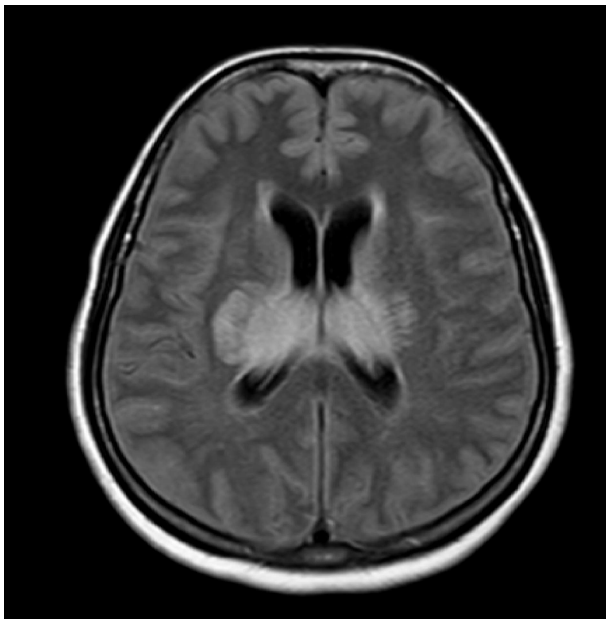

Figure 2: Axial T2-weighted MRI demonstrating symmetrical thalamic involvement, consistent with the "doughnut sign" in dengue encephalitis

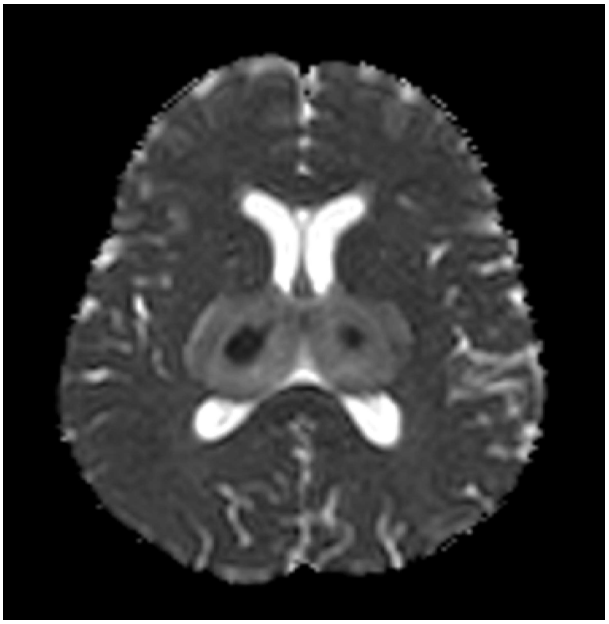

Figure 3: Axial DWI image showing diffusion restriction in bilateral thalami, contributing to the "double doughnut" appearance

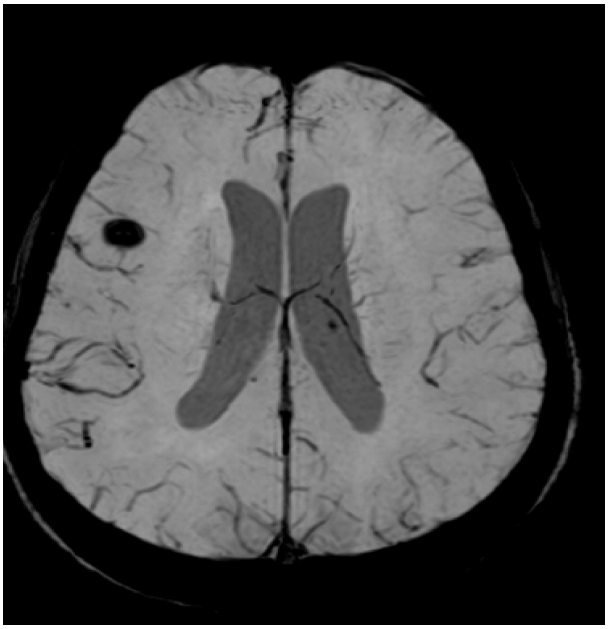

Figure 4: Axial GRE image showing blooming in the bilateral thalami, indicating hemorrhagic foci completing the "doughnut in the brain" pattern.

Table 2: CSF analysis

CSF -Count	100
CSF-Glucose	110
CSF-Cell type	Lymphocytes
CSF-Protein	565.1
CSF-Chloride	139

CSF c/s was negative for other infective pathology, CSF PCR for Dengue came positive, and Negative for Japanese Encephalitis & Herpes simplex virus. Patient was treated with 3% hypertonic saline for next 5 days to treat cerebral oedema. Patient also received Pulse Steroids in the form of intravenous methylprednisolone 1000mg once a day for 5 days.

Day 5 of illness Possibility of immune mediated insult was considered as opposed to direct viral insult in view of albumin-cytologic dissociation even though CSF PCR was positive for Dengue. Inj. Acyclovir 500mg thrice a day continued for 7 days.

During the stay in hospital, patient sensorium improved by 4th Day of admission to a GCS of E3VTM3. Patient started opening eyes spontaneously, responding and obeying by day 5th, weaned off ventilator and tracheostomised and has recovered to grade 3+/5 power in limbs and on RT feeds, Patient shifted to ward and underwent Physiotherapy & mobilisation.

Patient was Decannulated and was breathing spontaneously by Day 12th, Patient is hemodynamically stable and discharged with stable vitals

Table 3: Blood investigations as follows

Investigation	Day-5	Day-7	Day-12
Hb(g/dl)	11.2	12.6	13.8
WBC Count /mm3	8,200	7,000	4,900
Hematocrit %	33.9	30.6	32.8
Platelet count	75,000	1,03,000	2,77,000
Neutrophil %	75.4	60.2	49.6
S. creatinine (mg/dl)	0.7		
S. Sodium (mmol/l)		138	143
S. potassium (mmol/l)			3.5
SGOT (IU/L)	266	106	49
SGPT (IU/L)	403	88	99

DISCUSSION

This case shows an encephalitis as an important neurological manifestation of dengue fever. Dengue fever produce neurological impairment via different pathways.

1. The blood-brain barrier's endothelial cells are impacted by immunological mediators released during active dengue infection. The barrier is breached and the virus directly enters into the brain's glial and neuronal cells.
2. Dengue encephalitis can also occur in the context of dengue shock syndrome and dengue haemorrhagic fever due to multisystemic dysfunction.
3. The dengue virus can cause encephalitis by ADEM, which is similar to a post-infectious immune response.[3]
4. For the Diagnosis of Dengue Encephalitis – A "Double Doughnut" is sine quo non and similar findings are seen in Japanese encephalitis and Herpes simplex virus infections which were ruled out by CSF PCR test.

"Double Doughnut sign" it is characteristic of Dengue encephalitis , It is postulated to occur because of direct neuronal injury causing cerebral oedema and haemorrhage secondary to vascular leak involving basal ganglia and thalamus complex bilaterally resulting "Double Doughnut" appearance on MRI brain.

If the case falls clinically into accepted criteria for Dengue encephalitis

"Double Doughnut sign" is considered a diagnostic feature in patients.[7] Other differentials to be considered radiologically are:

1. Japanese Encephalitis which shows Bilateral thalamic involvement with haemorrhagic foci
2. Herpetic encephalitis shows asymmetrical involvement, Basal ganglia and Thalamus is spared differences based on MRI to differentiate between JE and Dengue.

Our patient outcome was better probably because of early steroid intervention.

CONCLUSION

Dengue is still considered non -neurotropic virus. Dengue encephalitis must be thought of in differential in patients of encephalopathy. Neuroimaging & CSF analysis plays a predominant role. The virus can be isolated from serum but CSF may or may not show results. The role of antiviral is still unclear. To sum-up, physician should have a suspicion of Dengue encephalitis while treating patients with poor sensorium.

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