

Double doughnut sign in dengue encephalitis

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HOW TO CITE THIS ARTICLE IN VANCOUVER STYLE?

Sharma S, Shrestha GS. Double doughnut sign in dengue encephalitis. *Journal of Nepalese Society of Critical Care Medicine*. 2023 Jan;1(1):26-27.

Submitted : 21 Nov 2022
Accepted : 22 Nov 2022
Published Online : 2 Dec 2022
Conflict of Interest : None
Source of Support : None

Key words: dengue encephalitis, double doughnut sign.



Figure A: Arrowheads indicating hyperdense lesion surrounded by hypodensity involving bilateral thalamus and basal ganglia regions (Double Doughnut sign) suggestive of Dengue encephalitis.

A 38 year old female with no known co-morbidities presented with the complaints of fever, abdominal pain and altered consciousness of 4 days duration. At presentation, her GCS was 7/15; eye opening to pain, flexor response to pain and incomprehensible sounds. Her vitals were stable with a SpO₂ of 92% with O₂ via nasal cannula at 2 L/min, blood pressure 110/70 mmHg, heart rate 100/min and temperature 99 degree Fahrenheit. Petechial rashes were present over multiple areas of the body. In the background of a low GCS score, the patient was intubated. Plain CT scan of head (Fig A) revealed hyperdense lesion surrounded by hypodensity involving bilateral thalamus and basal ganglia regions. Patient had leukopenia and thrombocytopenia (3,800/cu mm and 40,000/cu mm respectively) and elevated liver enzymes (ALT/AST- 120/86 IU/L). The patient tested positive for Dengue IgM antibody, Dengue IgG antibody and NS1 antigen. Evaluation for other tropical infections including Scrub typhus, Leptospirosis, Malaria, Brucellosis and Japanese encephalitis were negative. Lumbar puncture was performed which revealed total white cell

counts of 10 with 20% neutrophils and 80% lymphocytes, sugar of 50 mg/dl and protein of 90 mg/dl. A diagnosis of dengue encephalitis was made and supportive treatment was initiated. Viral isolation in CSF could not be performed because of unavailability of the test. The patient gradually deteriorated over the next three days and finally succumbed on the fourth day of ICU admission because of refractory septic shock.

Neurologic complications of dengue fever include encephalopathy, encephalitis, Guillain-Barre syndrome, transverse myelitis, and neuromuscular disorders.¹ In our patient, acute febrile illness with encephalopathy, positive dengue IgM serology, suggestive lumbar puncture findings and exclusion of differentials satisfied the diagnostic criteria of dengue encephalitis.² Various pathogenic mechanisms have been implicated for these complications which include direct viral invasion, systemic inflammatory response, and immune-mediated mechanisms. Dengue virus inflicts direct neuronal injury leading to cerebral edema and hemorrhage secondary to vascular leak, which usually involves bilateral basal ganglia

and thalamus complex and manifests neuro-radiologically as “double-doughnut” sign.³ This imaging finding is unique to dengue encephalitis and is rarely seen in other central nervous system infections.

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