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Case Report

A Rare Case of Dengue Fever Presenting with Oculomotor Nerve Palsy Followed by GBS in a 36 Year Old Female

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Abstract:

Dengue is a mosquito-borne, RNA virus of the family Flaviviridae. The vector is *Aedes aegypti* mosquito. Clinical manifestations range from an asymptomatic state to haemorrhagic fever. Guillain-Barré syndrome (GBS), myositis, myelitis, and encephalitis are among the neurological symptoms that have been previously described. These symptoms are mostly linked to infections with DENV-2 and DENV-3. These serotypes have been identified in myelitis, meningitis, and encephalitis cases.

Rarely, as a result of the humoral immune reaction, it may appear as neuritis. Cranial neuropathies which have been encountered earlier in dengue fever are optic neuritis, oculomotor nerve palsy, abducens nerve palsy, lower motor neuron motor facial nerve palsy.

Although the precise cause of dengue-associated cranial neuropathy is uncertain, immune-mediated processes are believed to be involved. A neurotropic virus, dengue can proliferate in the central nervous system after passing through the blood-brain barrier. Following direct infection of endothelium cells, dendritic cells, and monocytes, it would cause an excess of cytokines, which would lead to immune-mediated endothelial dysfunction.

Guillain–Barré syndrome (GBS) is an infrequent neurological complication of dengue viral infection. GBS is an acute immune mediated polyradiculoneuropathy characterised by rapidly progressing ascending paralysis and hypo or areflexia. The most common antecedent pathogen is Campylobacter jejuni, which is followed by Mycoplasma pneumoniae, Epstein-Barr Virus, and Cytomegalovirus (CMV).

Previous studies have shown AMAN and AMSAN variant to be most frequently associated GBS types following dengue in Asian countries.

We herein report a case of a 36 year old female who presented to us with dengue fever and left oculomotor nerve palsy and developed GBS after the onset of dengue fever.

Introduction

A 36-year-old female, who was a known case of hypothyroidism presented to us in emergency with chief complaint of High-grade fever for 4 days, thunderclap headache 1 day back This was followed by drooping of the left eye lid which was acute in onset, non-progressive and associated with diplopia which disappeared on closing either of the eyes. No diurnal variation was present. No history of altered sensorium, seizure, neck pain or any other neurological deficit was there.

On examination the patient was conscious, oriented to time, place and person. Vitals were normal.

There were few petechiae on the extremities. There was no evidence of arthritis.

CNS examination showed no signs of meningeal irritability or evidence raised intracranial pressure. Left eye ptosis was present, anisocoria was present, left pupil dilated and absent pupillary reflexes. All movements Apart from abduction and intorsion impaired in left eye.

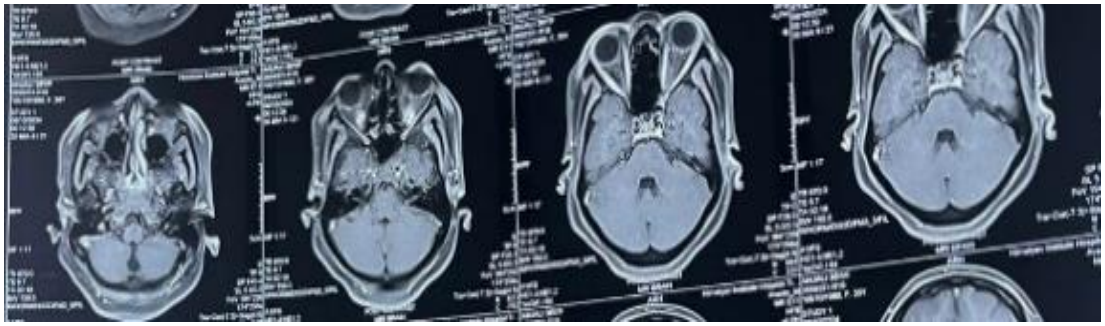
All other systemic examinations were with in normal limit.

Investigations

Complete blood counts were suggestive of thrombocytopenia (50,000) and a raised ESR (34).

Dengue NS1 antigen – positive

MRI Brain -Normal



CSF Examination was normal

A diagnosis of dengue fever with extra-axial 3rd cranial nerve palsy was made.

Preliminary treatment- She was given supportive treatment in the form of iv fluids, short course of oral steroids and antibiotics which resulted in partial resolution of ptosis and diplopia.

1 week later, during the course of stay in the hospital, she developed weakness in the b/l lower limbs characterised by difficulty in standing from squatting position and slipping of slippers from the b/l feet. weakness progressed to involve the b/l upper limbs in the next two days which was characterised lifting of her hand's overhead b/l and breaking chapatis and holding glass in hand. There was no H/O of paresthesia or numbness.

On Examination

Power - 3/5 over both flexor and extensor surface of and shoulder and elbow, 2/5 over wrist, hand grisp was weak b/l. Power was 3/5 in b/l proximal and distal extremity of lower limbs. Generalized areflexia was present. Plantars were bilaterally flexor

NCS: Showed reduced CMAP and normal SNAP (s/o AMAN variant of GBS)

Treatment

After confirming the diagnosis, the patient was planned for 5 cycles of plasmapheresis but could only undergo 2 cycles due to development of septicemia, which in turn was managed by IV Antibiotics. Once Sepsis was resolved the patient was discharged after a total duration of 22 days in the hospital. At the time of discharge the power was 4/5 in all four limbs and the patient was able to walk back home.

The patient's follow up examination one month later showed complete resolution of ptosis and diplopia. She also regained full power in all her extremities.

Discussion

8Dengue-associated cranial neuropathy is believed to be immune-mediated, while the precise pathophysiology is uncertain. The neurotropic dengue virus has the ability to multiply in the central nervous system and penetrate the blood-brain barrier. After then, it would directly infect monocytes, dendritic cells, and endothelial cells, causing an excess of cytokines and immune-mediated vascular dysfunction [4]. The management of Dengue related cranial neuropathy is usually supportive and has been known to respond to immunoglobulin and corticosteroids previously [5]. Our patient also showed partial resolution of diplopia and ptosis at the time of discharge.

GBS is an uncommon neurological sequela of dengue fever.

The clinical symptoms of GBS are believed to be caused by cell-mediated immune responses to non-self-antigens that misdirect to host nerve tissue, a process known as molecular mimicry [6].

Plasma exchange and intravenous immunoglobulin therapy are two typical therapeutic options that are equally effective if initiated within two weeks of symptoms.

In 1992, a randomized controlled trial demonstrated that IvIG is just as effective as plasma exchange [7].

Conclusion

Cranial mononeuropathy can be one of the clinical features of dengue fever, as seen in our patient but other causes of cranial nerve palsy need to be ruled out as it is a rare presentation. A short course of steroid therapy may enhance recovery avoiding longer stays in hospital.

Dengue fever can result in GBS. The characteristics of dengue fever-associated GBS differ from those of conventional GBS, including greater bifacial involvement, greater axonal involvement, and mild CSF pleocytosis.

References

1. Trivedi S, Chakravarty A. Neurological Complications of Dengue Fever. Curr Neurol Neurosci Rep. 2022 Aug;22(8):515-529. doi: 10.1007/s11910-022-01213-7. Epub 2022 Jun 21. PMID: 35727463; PMCID: PMC9210046.
2. Chaturvedi UC, Dhawan R, Khanna M, Mathur A. Breakdown of the blood-brain barrier during dengue virus infection of mice. J Gen Virol.

1991 Apr;72 (Pt 4):859-66. doi: 10.1099/0022-1317-72-4-859. PMID: 1707947.

3. Carod-Artal FJ, Wichmann O, Farrar J, Gascón J. Neurological complications of dengue virus infection. Lancet Neurol. 2013 Sep;12(9):906-919. doi: 10.1016/S1474-4422(13)70150-9. PMID: 23948177.
4. Chaturvedi UC, Dhawan R, Khanna M, Mathur A. Breakdown of the blood-brain barrier during dengue virus infection of mice. J Gen Virol. 1991 Apr;72 (Pt 4):859-66. doi: 10.1099/0022-1317-72-4-859. PMID: 1707947.
5. Biswas NM, Pal S. Oculomotor nerve palsy in dengue encephalitis--a rare presentation. Indian J Med Res. 2014 Dec;140(6):793-4. PMID: 25758582; PMCID: PMC4365359.
6. Hughes RA, Hadden RD, Gregson NA, Smith KJ. Pathogenesis of Guillain-Barré syndrome. J Neuroimmunol. 1999 Dec;100(1-2):74-97. doi: 10.1016/s0165-5728(99)00195-2. PMID: 10695718.
7. van der Meché FG, Schmitz PI. A randomized trial comparing intravenous immune globulin and plasma exchange in Guillain-Barré syndrome. Dutch Guillain-Barré Study Group. N Engl J Med. 1992 Apr 23;326(17):1123-9. doi: 10.1056/NEJM199204233261705. PMID: 1552913.