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A CASE OF LONGITUDINALLY EXTENDING TRANSVERSE MYELITIS POST DENGUE INFECTION

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ABSTRACT Association of dengue fever with longitudinally extensive transverse myelitis is a rare neurological feature of dengue infection. We describe a 53 year old male who had a history of dengue fever (serologically confirmed) 1 month ago and in whom symptoms of longitudinally extending transverse myelitis developed 4 weeks after fever (post-infectious stage). Magnetic resonance imaging confirmed the diagnosis of longitudinally extensive transverse myelitis involving dorso-lumbar cord upto the conus medullaris. Our patient recovered with minimal residual neurological deficit in the form of minimal gait imbalance after a six weeks course of corticosteroids and supportive management including physiotherapy.

KEYWORDS : Dengue, Longitudinally extending transverse myelitis, Neurological manifestations of Dengue

INTRODUCTION

Dengue fever and dengue hemorrhagic fever is a very important vector borne illness in India. As per data from NVBDCP, There have been 101192 reported cases of dengue last year in India, of which there were 172 reported mortalities. Various neurologic complications of dengue viral infection are rare but have been reported, including central and peripheral nervous system involvement. Encephalopathy, encephalitis, seizures, mononeuropathy, polyneuropathy, and Guillain–Barré or Miller-Fisher syndromes have been associated. However, spinal cord involvement associated with dengue viral infection has been rarely reported. There have been very few previously reported cases of longitudinally extending transverse myelitis in association with dengue infection.

CASE REPORT

A 53 year male presented to the emergency department of a tertiary care hospital with complaints of twitching (flexor spasms and parasthesias) in both lower limbs along with imbalance of gait and difficulty getting up from a supine and a sitting position since the past 15 days. The patient also gave a history of slipping of footwear and involvement of bowel and bladder in the form of decreased sensation and incontinence. There was no history of slurred speech/vision abnormalities/deviation of angle of mouth/seizure/memory impairment.

The patient was hemodynamically stable on admission with CNS examination suggestive of spastic weakness in both lower limbs (4 out of 5 according to MRC grading of power.) Superficial reflexes were normal, deep tendon reflexes were exaggerated in both lower limbs. Sensory examination was suggestive of impaired pain and temperature sensation and impaired sensation of fine touch upto the hip in both lower limbs. Joint position sense was also impaired in both lower limbs.

Magnetic resonance imaging of the whole spine was suggestive of a diffuse ill defined long segment intramedullary hyperintense signal on T2W involving dorsal spinal cord extending from level of inferior end plate of D7 vertebral body to the conus medullaris. It involved more than 2/3rd circumference to near complete involvement of the spinal cord in a few places. The signal was isointense on T1 images.

The patient gave a history of fever with chills and rigors 1 month ago from which he recovered with supportive management. This episode of fever was serologically confirmed as dengue fever via a positive IgM ELISA study and with a positive NS1 antigen capture assay from 1 month ago.

Workup for other common causes(CSF oligoclonal bands, Serum IgG, Serum Neuromyelitis optica antigen and CSF neuromyelitis optica antibody) for LETM came out to be negative. ANA by IIF was negative.

The patient was treated with supportive management and intravenous pulse therapy with methylprednisolone at a dosage of 1 g/day for 3 days and then was switched to oral prednisolone. The oral prednisolone dosage was started at 60 mg/day and then was tapered gradually over 4 weeks. The patient's bladder was catheterized for the first week, and then the catheter was removed after intermittent clamping and bladder training. Rehabilitation physical therapy exercises were done simultaneously with medical treatment.

Over the course of 2 weeks of hospital stay and 4 weeks of regular follow up the patient improved significantly with return of normal power in lower limbs, normal bowel and bladder functions and return to activities of daily living with only minimal imbalance of gait while walking.

DISCUSSION

Neurologic manifestations associated with dengue fever were considered very rare initially, but in the last 20 years there has been increasing recognition of the spectrum the neurologic manifestations. After a thorough search of the literature, we found very few reports of dengue illness associated with spinal cord involvement in the form of longitudinally extending transverse myelitis.

Our patient had typical history of dengue fever with a clinical history, rash, thrombocytopenia, leukopenia, and positive NS1 antigen and immunoglobulin M serologic findings for dengue virus. With all these features on examination and imaging, the lack of any other important physical findings and negative autoimmune workup, LETM in this patient was probably post dengue infection. The patient responded very well to corticosteroid treatment, and the neurologic deficit recovered almost completely by 6 weeks.

In our patient, myelitis developed in the acute postinfectious phase of dengue illness, similar to earlier case reports. It was also postulated that acute parainfectious dengue infection presents with Spastic paralysis, whereas late-stage (postinfectious) dengue infection presents with spastic weakness, and our case had similar findings of flaccid paralysis. Usually, LETM is associated with a poor prognosis; however, our patient showed a dramatic improvement in neurologic deficit after treatment with corticosteroids and supportive physiotherapy.

We were unable to determine the strain of dengue in our patient. However, it would be interesting to know the epidemiology for future such cases.

CONCLUSION

Even though transverse myelitis is a rare neurologic manifestation of dengue infection, manifesting mostly during parainfectious phases and sometimes after the infection has resolved, it is important that clinicians are aware of this entity. It is vital that clinicians look for dengue virus as a cause of transverse myelitis or other inflammatory neurologic manifestations in patients presenting with typical dengue fever symptoms and living in or visiting dengue-endemic areas.

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