



A Case of Myelin Oligodendrocyte Glycoprotein Antibody Disease Associated With Post-Dengue Transverse Presenting With Paraplegia and Urinary Retention

Rao Erbaz Hassan^{1*}, Syed Aamer Hussain², Shafiq Tanveer³, Qazi Fawad Ahmad¹, Arif Ullah Khan¹

¹Trainee Medical Office, Department of Surgery, Ayub Teaching Hospital, Abbottabad

²Consultant General Surgery, Department of Surgery, Ayub Teaching Hospital, Abbottabad

³Trainee Medical Office, Department of Ophthalmology, Khyber Teaching Hospital, Peshawar



Received date: 23-08-2023

Publication date: 12-10-2023

Abstract

Transverse myelitis is the inflammation of the spinal cord, which causes the is demyelination in a horizontal plane. Dengue virus is known to be associated with transverse myelitis para or post infectiously. Autoantibodies such as Aquaporin-4 autoantibodies and Myelin oligodendrocyte glycoprotein autoantibodies have been associated with transverse myelitis. However, the exact causal relationship is yet to be determined. We report a case of post-dengue fever transverse myelitis associated with myelin oligodendrocyte glycoprotein antibody disease. A 26-year-old previously healthy young man presented with acute urinary retention and paraplegia. He had a recent history of contracting dengue virus infection. He had a positive serology for antibodies against the dengue virus and imaging showed the inflammation of the spinal cord consistent with transverse myelitis. Further workup demonstrated the presence of Myelin oligodendrocyte glycoprotein autoantibodies. A diagnosis of Myelin oligodendrocyte glycoprotein antibody disease associated with post-dengue transverse myelitis was made. He was treated with high-dose methylprednisolone for 7 days followed by an oral tapering dose over 6 months. Follow-up examination and imaging showed subsequent improvement with complete recovery. Myelin oligodendrocyte glycoprotein antibody disease is a very less understood and rare complication of dengue virus infection. Further studies are required to determine the exact mechanism. Moreover, prompt diagnosis. treatment can result in early recovery and complete remission of the disease.

Keywords Myelin Oligodendrocyte, Post-Dengue, Glycoprotein, Myelitis, Paraplegia

1. Introduction

Dengue fever is a prevalent viral disease in humans and remains a significant cause of illness and mortality, particularly in tropical and subtropical regions (1). The literature has documented various neurological complications associated with Dengue virus (DENV) infection. Among these, Transverse Myelitis (TM) is a less frequently reported para- or post-infectious complication of DENV (2). TM can arise from autoimmune, vascular, metabolic, or para-infectious processes. In the context of post-infectious TM, two common antibodies, Aquaporin-4 immunoglobulin G (AQP-4 IgG) and Myelin oligodendrocyte glycoprotein immunoglobulin G (MOG-Ig G), have been identified (3).

In this case report, we present the clinical scenario of a young adult who developed paraplegia and urinary retention following the resolution of dengue fever. The diagnosis of TM secondary to DENV infection was established based on the detection of serum antibodies against DENV and findings from spinal imaging. Subsequent identification of autoantibodies against Myelin oligodendrocyte glycoprotein (MOG) confirmed the diagnosis of MOG antibody Disease (MOGAD). This case highlights the association between Dengue fever and TM, shedding light on the involvement of MOG antibodies in this autoimmune disorder.

Corresponding author at: Rao Erbaz Hassan
Email address: brawo1100@gmail.com

<https://doi.org/10.56600/jwmdc.v2i2.70>



2. Case Presentation

A previously healthy 26-year-old man presented to the Emergency room with acute urinary retention lasting for 3 hours. The patient underwent catheterization and was subsequently discharged by the ER staff. However, the following day, the patient returned with bilateral

lower limbs demonstrated 3/5 power and absent knee reflexes bilaterally. Babinski's sign was positive, but there were no signs of meningism. Although the patient complained of tingling sensations in the medial aspect of the upper limbs, neurological examination of the upper limbs revealed no abnormalities. Vital signs were stable, and the rest of the systemic examination was

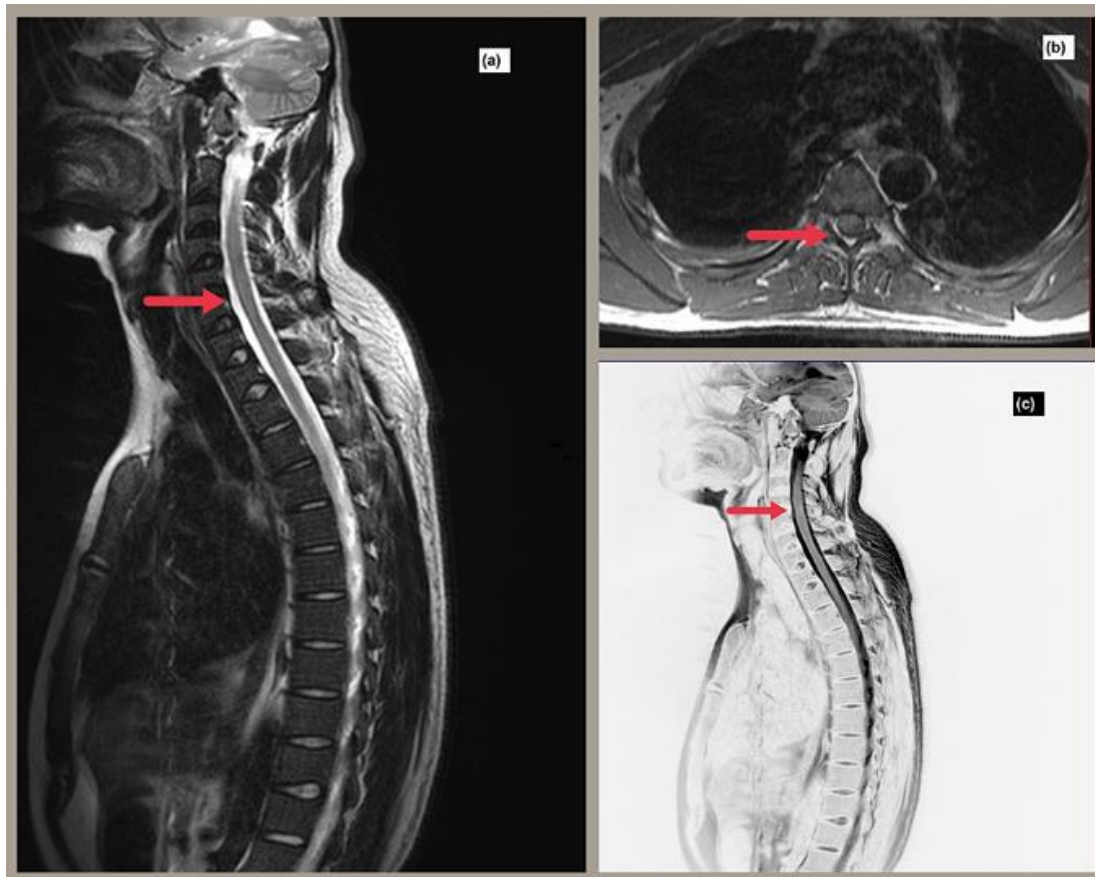


Figure 1: Multifocal T2 hyperintense signals involving the spinal cord. There is continuous involvement of C4-C6. The red arrows indicate T2 hyperintense signals in the spinal cord at C4 level in both sagittal (a) and axial planes (b) and also in Negative (c)

lower limb weakness. The patient resided in an area affected by the dengue virus outbreak. Approximately 12 days prior, he experienced high-grade fevers accompanied by chills, myalgias, backache, and headache. A serological test, specifically the non-structural protein 1 (NS1) antigen test, was performed and revealed dengue fever. Low platelet counts were also documented in serial complete blood counts. The patient managed the condition conservatively at home with the help of fluids and paracetamol. For the past two days, the patient had been afebrile before this new presentation.

Upon examination, the patient was awake, alert, and oriented to time, place, and person. Evaluation of the

unremarkable. Baseline investigations, including complete blood count, liver function tests, renal function tests, random blood sugar, and serum electrolytes, were all within normal limits.

A contrast-enhanced magnetic resonance imaging (MRI) of the spine was performed, revealing expansion of the cervical cord between the C4-C6 levels. The MRI showed symmetrical high signal changes involving both sides of the cord medially, accompanied by faint ill-defined enhancement in this segment of the cord. These findings were consistent with transverse myelitis. Subsequently, a lumbar puncture was performed, and cerebrospinal fluid analysis revealed oligoclonal bands. The blood test for Myelin oligodendrocyte glycoprotein

immunoglobulin G (MOG-IgG) antibodies was positive, while the test for Aquaporin-4 immunoglobulin G (AQP-4 IgG) antibodies was negative. Visual evoked potentials (VEP) showed no signs of optic neuritis.

Based on the correlation of laboratory and radiological findings with the patient's signs and symptoms, a

NS1 antigen, along with the presence of MOG-IgG antibodies and demyelination of the spinal cord, it is highly likely that the post-infectious autoimmune complication of dengue virus infection led to the development of MOGAD-associated transverse myelitis.

The patient showed a favorable response to

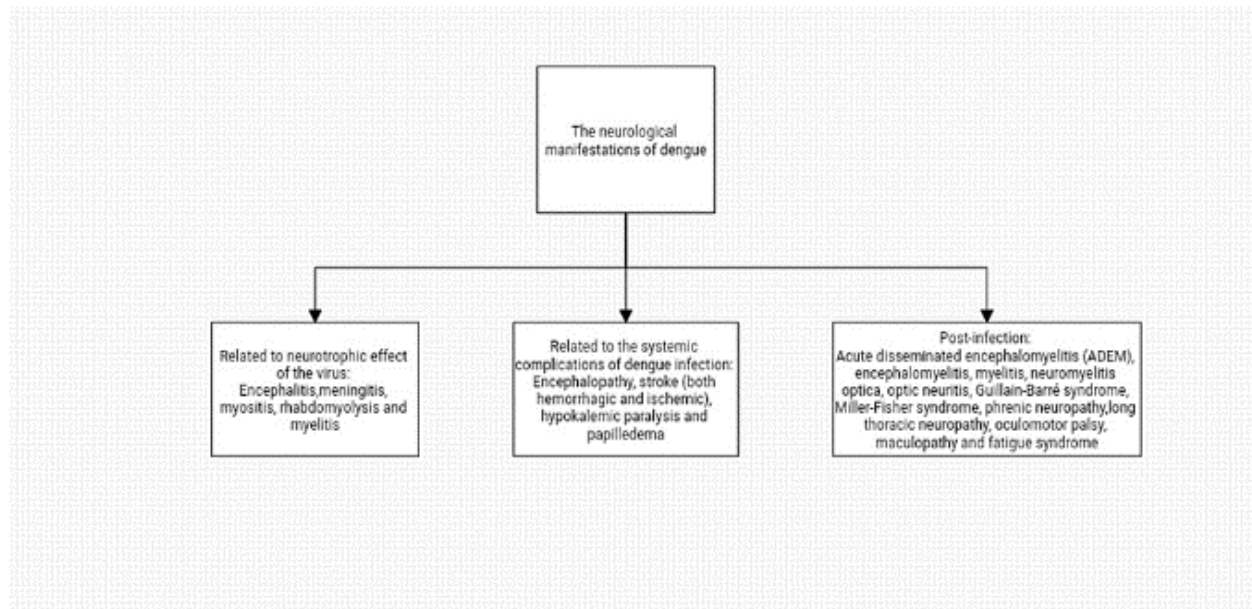


Figure 2: The neurological features of dengue by Murthy

diagnosis of MOGAD-associated transverse myelitis was made. Treatment was promptly initiated with methylprednisolone at a dose of 1 gm/day for 5 days, followed by a switch to oral prednisolone. Initially, prednisolone was administered at a dosage of 60 mg/day in two divided doses, which were gradually tapered over the next 3 months. The patient experienced improvement in weakness following the administration of the first dose of methylprednisolone, and urinary retention resolved on the third day of admission. After a 7- day hospital stay, the patient was discharged with an extensive rehabilitation exercise plan.

During the 4-week follow-up, the patient regained the ability to walk without support and perform most daily tasks independently. Adequate voiding was also achieved, with a post-void urine volume of 70 ml. The patient reported mild fatigue with exertion, which was reassured, and subsequent follow-ups showed an improvement in physical exertion capacity. A follow-up MRI of the spine after 3 months demonstrated complete resolution of demyelination.

Considering the patient's recent history of dengue fever with high-grade fever, body aches, and positive dengue

corticosteroid therapy, and no residual neurological deficits were observed during subsequent follow-ups.

3. Discussion

The World Health Organization (WHO) has classified the clinical presentation of dengue based on severity into two categories: Dengue fever with or without warning signs, and severe dengue fever. In 2009, the WHO introduced this classification system. Neurological manifestations and central nervous system (CNS) involvement in dengue fever are considered to be part of severe dengue (4).

As the number of reported neurological manifestations of dengue fever continues to increase each year, it is evident that they cannot be overlooked as rare occurrences. The overall incidence of CNS-related complications in dengue fever is approximately 6% (5). Dr. Murthy has classified the neurological complications of dengue fever into three categories (6). Transverse Myelitis (TM) refers to the inflammation of one or more segments of the spinal cord without compression. It is a well-studied complication that can occur during the para-infectious or post-infectious

period following dengue fever. Para-infectious TM results from the direct invasion of the virus, while post-infectious TM is believed to be immune-mediated and typically occurs 1-2 weeks after the initial symptoms of dengue fever (7-9).

The primary auto-antibodies associated with immune-mediated TM are Aquaporin-4 (AQP4) antibodies and Myelin Oligodendrocyte Glycoprotein (MOG) antibodies. MOG Antibody Disease (MOGAD) represents a distinct entity with its characteristic pathophysiological features. MOG-IgG-associated myelitis is usually a monophasic disease, with rare instances of relapses (10).

The treatment approach for MOGAD involves initiating high-dose glucocorticoids for 3-5 days, followed by a gradual tapering of steroids over an extended period to reduce the risk of flare-ups and relapses. In cases where patients do not respond to steroids, intravenous immunoglobulin (IVIG) infusion, mycophenolate mofetil, and rituximab can be considered as alternative options (10).

4. Conclusions

Myelin Oligodendrocyte Glycoprotein (MOG) IgG-associated Transverse Myelitis is a condition that has been frequently reported in medical literature. However, its occurrence in the context of Dengue fever has never been documented before. This case report brings forth new opportunities for researchers to explore the pathophysiology, management, and evaluation of treatment options in this particular scenario. It opens up new horizons for further investigation in this field.

Additional Information

Disclosures

Human subjects: Consent was obtained or waived by all participants in this study. Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following:

Payment/services info: All authors have declared that no financial support was received from any organization for the submitted work. Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. Other

relationships: All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

Conflict of Interest The authors declared that they have no competing or conflict of interest

Acknowledgments We are grateful to all the participants who willingly participated in the study.

References

1. Bhatt S, Gething PW, Brady OJ, Messina JP, Farlow AW, Moyes CL, et al. The global distribution and burden of dengue. *Nature* . 2013;496(7446):504–7. Available from: <http://dx.doi.org/10.1038/nature12060>
2. Mota MT de O, Estofolete CF, Zini N, Terzian ACB, Gongora DVN, Maia IL, et al. Transverse myelitis as an unusual complication of dengue fever. *Am J Trop Med Hyg* . 2017;96(2):380–1. Available from: <http://dx.doi.org/10.4269/ajtmh.16-0284>
3. Comtois J, Camara-Lemarroy CR, Mah JK, Kuhn S, Curtis C, Braun MH, et al. Longitudinally extensive transverse myelitis with positive aquaporin-4 IgG associated with dengue infection: a case report and systematic review of cases. *Mult Scler Relat Disord*. 2021;55(103206):103206. Available from: <http://dx.doi.org/10.1016/j.msard.2021.103206>
4. World Health Organization: Dengue: Guidelines for Diagnosis, Treatment, Prevention, and Control. New edition. World Health Organization, Geneva; 2009.
5. Hendarto SK, Hadinegoro R. Dengue encephalopathy. *Pediatr Int*. 1992;34(3):350–7. Available from: <http://dx.doi.org/10.1111/j.1442-200x.1992.tb00971.x>
6. Murthy JMK. Neurological complication of dengue infection. *Neurol India* . 2010;58(4):581. Available from: <http://dx.doi.org/10.4103/0028-3886.68654>
7. Lim PAC. Transverse Myelitis. In: *Essentials of Physical Medicine and Rehabilitation*. Elsevier; 2020. p. 952–9. Available from: <http://dx.doi.org/10.1016/B978-0-323-54947-9.00162-0>
8. Leão RNQ, Oikawa T, Rosa EST, Yamaki JT, Rodrigues SG, Vasconcelos HB, et al. Isolation of

- dengue 2 virus from a patient with central nervous system involvement (transverse myelitis). *Rev Soc Bras Med Trop.* 2002;35(4):401–4. Available from: <http://dx.doi.org/10.1590/s0037-86822002000400018>
9. Seet RCS, Lim ECH, Wilder-Smith EPV. Acute transverse myelitis following dengue virus infection. *J Clin Virol.* 2006;35(3):310–2. Available from: <http://dx.doi.org/10.1016/j.jcv.2005.08.006>
 10. Perez Giraldo GS, Ortiz Garcia JG. Immune-mediated disorders affecting the spinal cord and the spine. *Curr Neurol Neurosci Rep.* 2021;21(1). Available from: <http://dx.doi.org/10.1007/s11910-020-01088-6>