

Post dengue neurological complication

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Abstract

Dengue infection is highly endemic in many tropical countries including Malaysia. However, neurological complications arising from dengue infection is not common; Gullain–Barre syndrome (GBS) is one of these infrequent complications. In this paper, we have reported a case in which a 39-year-old woman presented with a neurological complication of dengue infection without typical symptoms and signs of dengue fever. She had a history of acute gastroenteritis (AGE) followed by an upper respiratory tract infection (URTI) weeks prior to her presentation rendering GBS secondary to the post viral URTI and AGE as the most likely diagnosis. Presence of thrombocytopenia was the only clue for dengue in this case.

Introduction

Dengue fever is a mosquito-transmitted arboviral infection mainly found in subtropical and tropical countries.¹⁻³ It can present with neurological deficits and the incidence of this presentation ranges from 1% to 5%.^{3,4} Neurological manifestations of dengue infection include encephalopathy, encephalitis, meningitis, GBS, myelitis, acute disseminated encephalomyelitis, polyneuropathy, mononeuropathy and cerebromeningeal haemorrhage.² GBS accounts for 30% of these manifestations; however, it can be underestimated as the preceding dengue infection may be oligosymptomatic.^{2,5}

Case summary

A 39-year-old woman who had no underlying illness presented to the emergency department with a sudden onset of numbness of both feet. The numbness had rapidly progressed up to her hips within a few hours. It was associated with throbbing pain in the upper part of her lower limbs causing difficulty in standing and walking. There was absence of urinary or bowel incontinence. This acute presentation was preceded by a 4-day history of lethargy and myalgia.

A week before the onset of the lower limb numbness, she had a low-grade fever, rhinorrhea and minimal cough. However, the symptoms resolved after 3 days. In addition, she had a history of AGE for 2 days a month prior to this presentation.

On examination, she was alert and conscious,

but appeared weak. Her hydration status was normal. She was afebrile. Her pulse rate was 76 beats/min and her blood pressure was 120/80 mmHg with no evidence of postural hypotension. Her respiratory rate was 16/min with oxygen saturation of 100% at room air. Her higher mental functions were intact and cranial nerve examinations were unremarkable. She was able to stand from a sitting position but unable to walk due to 'heaviness' of the legs. However, examination of the both upper and lower limbs revealed normal power and reflexes. She had reduced sensation to light touch and pinprick from both feet to below the knee level.

Based on her clinical presentation, the provisional diagnosis was early GBS secondary to post viral URTI and/or AGE. Another differential diagnosis considered was post viral myositis. Thus, full blood count (FBC) was performed and it showed a haemoglobin level of 12.2 g/dL, white blood count of $5.5 \times 10^9/L$ and platelet count of $104 \times 10^9/L$. Her baseline renal profiles showed serum creatinine level of 72 mol/L, serum sodium level of 136 mmol/L and serum potassium level of 3.7 mmol/L. Levels of liver enzymes and serum creatine kinase were within normal range. Urinalysis was normal.

Her inability to walk and the rapid onset of the symptoms had warranted hospital admission and monitoring of her clinical progress. On day two of admission, the numbness started to involve both of her hands. However, the symptoms plateaued subsequently. Repeated FBC showed a drop

in platelet level to $87 \times 10^9/L$. Therefore, serum anti-dengue IgM antibody was taken, and subsequently reported as positive for dengue infection. Serial daily platelet levels showed an increasing trend and it returned to a normal level after 8 days.

She was treated conservatively in the ward and her symptoms improved after 4 days. On day eight of admission, her neurological symptoms resolved and she was able to walk unaided. A nerve conduction test (NCT) done on day four of admission was reported normal.

Discussion

The sudden onset and ascending presentation of neurological deficits in this patient suggested that the patient might have GBS. History of prior AGE and URTI supported this diagnosis. However, the symptoms were mainly associated with the lower limbs with predominant sensory impairment. Furthermore, her reflexes and NCT were normal. Cerebrospinal fluid (CSF) was unavailable to confirm the diagnosis. As she showed a remarkable improvement after 4 days, lumbar puncture was deemed unnecessary. Nevertheless, a diagnosis for GBS was still possible as a minority of patients with this syndrome could have symptoms confined to the legs or normal NCT.⁶

GBS has a wide clinical spectrum that ranges from mild self-limiting disease to acute fulminant disease with severe pandysautonomia.^{7,8} Clinical course of GBS secondary to dengue infection is similar to GBS caused by other infections whereby the neurological manifestation occurs after the infection subsided.⁹ It usually presents during the recovery phase of dengue fever, i.e. from 3 days to 6 weeks after dengue infection.⁹ Most of the patients have little or no clinical symptoms of dengue during its acute phase, often making dengue as an unconsidered differential diagnosis.¹⁰ In this patient, the presumed URTI occurred a week prior to her neurological symptoms was likely to be an acute dengue infection. Absence of typical symptoms of dengue fever such as high grade fever, headache, retro-orbital pain, arthralgia and rash had made the infection go unnoticed. Furthermore, the initial investigation only showed mild thrombocytopenia hence, dengue infection was initially regarded as a less likely diagnosis. Since the oligosymptomatic presentation of dengue infection is common in dengue related

GBS, dengue serology should be performed in patients suspected with GBS who live in endemic dengue areas.^{10,2}

GBS is a post dengue neurological complication caused by autoimmune reaction.^{11,2} This cell-mediated immunological reaction is initiated by the dengue virus and targets myelin or axon.^{11,12} Other post dengue immune mediated neurological manifestations include acute transverse myelitis and acute disseminated encephalomyelitis (ADEM).¹³ Widespread neurological complications of dengue infection in recent years may be due to a change in the virological characteristics of dengue viruses.^{2,7} Presence of all four dengue viruses serotypes (DENV1 to DENV4) in endemic countries may complicate its presentation and co-infection of different serotypes may occur.^{1,13}

Most of the patients with post dengue GBS recover spontaneously without neurological deficits.^{2,7,12,14} Thus, in the ward, the patient was given supportive treatment only. Pulses of intravenous methylprednisolone have been recommended as a treatment; however, its efficacy is still unproven.¹³ In severe cases, high dose intravenous immunoglobulins may also be useful.^{1,15} Therefore, patients suspected with GBS should be admitted for monitoring of their clinical progress regardless of its severity. This is because the course of the disease may be unpredictable, although the prognosis of GBS caused by dengue infection is usually good.^{2,7,12,14} This allows timely provision of appropriate management including high dose intravenous immunoglobulins if the symptoms deteriorate.

Conclusion

GBS is a possible diagnosis of dengue infection in patients presented with sudden onset ascending neurological deficits following febrile episodes in endemic countries. Even without prior history of febrile illness, dengue infection should be suspected and investigated. Furthermore, clinicians should familiarise with the wide spectrum presentation of post dengue GBS to prevent misdiagnosis. This case report has highlighted the importance of early recognition of the neurological complications of dengue. Thus, a close monitoring of clinical progress can be done to ensure appropriate management being given in timely manner. Furthermore, this early recognition could avoid unnecessary invasive

investigation such as lumbar puncture because GBS caused by dengue infection is commonly mild and benign with good prognosis. However, some of the neurological complications are serious and life threatening, which require timely specific therapeutic interventions.

Conflict of interest

None.

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References

- Domingues RB, Kuster GW. Diagnosis and management neurologic manifestations associated with dengue virus infection. *J Neuroinfect Dis*. 2014;5:138.
- Puccioni-Sohler M, Orsini M, Soares CN. Dengue: A new challenge for neurology. *Neurol Int*. 2012;4(e15):65–70.
- Puccioni-Sohler M, Rosadas C, Cabral-Castro MJ. Neurological complications in dengue infection: A review for clinical practice. *Arq Neuropsiquiatr*. 2013;71(9B):667–71.
- Pancharoen C, Thisyakorn U. Neurological manifestation in dengue patients. *Southeast Asian J Trop Med Public Health*. 2001;32:341–5.
- Soares CN, Faria LC, Peralta JM, et al. Dengue infection: Neurological manifestations and cerebrospinal fluid (CSF) analysis. *J Neurol Sci*. 2006;249:19–24.
- Fokke C, van den Berg B, Drenthen J, et al. Diagnosis of Guillain–Barre syndrome and validation of Brighton criteria. *Brain*. 2014;137:33–43.
- Verma R, Sharma P, Garg RK, et al. Neurological complications of dengue fever. Experience from a tertiary center of North India. *Ann Indian Acad Neurol*. 2011;14(4):272–8.
- Kumar S, Subashini P. Guillain–Barre syndrome occurring in the course of dengue fever. *Neurol India*. 2005;53:250–1.
- Yuki N, Hartung HP. Guillain–Barre syndrome. *N Eng J Med*. 2012;366:2294–304.
- Soares CN, Cabral-Castro MJ, Oliveira C, et al. Oligosymptomatic dengue infection: A potential cause of Gullain–Barre syndrome. *Arq Neuropsiquiatr*. 2008;66:234–7.
- Warke RV, Becerra A, Zawadzka A, et al. Efficient dengue virus (DENV) infection of human muscle satellite cells up regulates type I interferon response genes and differentially modulates MHC I expression on bystander and DENV-infected cells. *J Gen Virol*. 2008;89:1605–15.
- Murthy JM. Neurological complications of dengue infection. *Neurol India*. 2010;58:581–4.
- Carod-Artal FJ, Wichmann O, Farrar J, et al. Neurological complications of dengue virus infection. *Lancet Neurol*. 2013;12:906–19.
- Qureshi NK, Begum A, Saha PR, et al. Guillain–Barre syndrome following dengue fever in adult patient. *J Medicine*. 2012;13:246–9.
- Jayashree K, Manasa GC, Pallavi P, et al. Evaluation of platelets as predictive parameters in dengue fever. *Indian J Hematol Blood Transfus*. 2011;27(3):127–30.