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Case Report
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A Broken Smile in Dengue Fever: Isolated Bell's Palsy as an Uncommon Neurological Complication

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ABSTRACT

Background: Dengue is caused by Flavivirus and its infection is mostly asymptomatic but multi-organ complications can be fatal in some cases. Among those varied uncommon neurological manifestations, cranial neuritis involving facial nerve palsy is rarely reported. Here we describe a case of an isolated Bell's palsy as a complication secondary to dengue infection.

Case Presentation: A 15-year-old boy got admitted with fever, headache, & myalgia for 2 days and was diagnosed as dengue based on NS1 antigen positivity. He suddenly developed right sided LMN facial nerve palsy three days post-admission. The boy was treated supportively along with a short duration of oral steroids. His palsy recovered significantly later on follow-up.

Conclusion: This case shows that infrequent neurological manifestations such as isolated Bell's palsy can be a part of the dengue illness spectrum. Early recognition of such atypical clinical features enables early management and may lead to better recovery. Continued reporting of such cases is essential to understand the mechanisms behind these rare associations.

Keywords: Dengue, Dengue fever, Facial nerve palsy, Bell's palsy

INTRODUCTION

Dengue is one of the most prevalent arthropods borne viral diseases endemic globally, caused by genus Flavivirus, transmitted by Aedes mosquitoes and includes four serotypes DENV 1-4.[1] As per the 2009 clinical case classification system by WHO, three categories of dengue infection include- Dengue (without warning signs), Dengue with warning signs and Severe Dengue. Most DENV infections are subclinical and self-limited dengue fever is the usual clinical result of infection.[1] In recent years an increasing number of uncommon presentations and complications involving multiple organ systems have also been observed.[2] Neurological involvement was first reported in 1976 and incident rates vary from 0.5 to 20%.[3-4] All 4 serotypes dengue (DENV 1-4) have been related to neurological manifestations.[5] CNS features comprise 95% of neurological complications.[6-7] Cranial neuritis as a complication of dengue infection is rare with very few cases reports.[3,7]

Here we present a case report of an isolated Bell's palsy as a complication of Dengue fever.

CASE REPORT

A 15-year-old boy studying in class 8 from a remote village of Western Odisha, presented to the casualty with complaints of fever, frontal headache, severe myalgia and

arthralgia for two days. The fever was abrupt onset, high grade, continuous and partially subsided with acetaminophen. Fever was not associated with cough, chest pain, coryza, runny nose, earache or any discharge, pain abdomen, diarrhoea, burning micturition, urgency, frequency, seizure, neck rigidity, or altered sensorium. He neither had a history of any chronic illness, nor was hospitalised for similar complaints in the past. The patient had no history of any type of addiction or any high risk behaviour. On examination he was conscious, alert, febrile, mild pallor, no icterus, edema, or lymphadenopathy, a macular rash over trunks, arms, and neck, mild hepatomegaly, and was hemodynamically stable. The rest of the systemic examination was normal. The source of acute febrile illness couldn't be localized.

The boy was admitted to the infectious diseases ward (with suspicion of a communicable disease) and a complete panel of blood tests was advised as per the institutional protocol. It was confirmed as Dengue infection based on NS1 antigen positivity as well as IgM antibody positivity on the reports. Complete blood count showed WBC count of 8000 with relative lymphocytosis in differential count, hemoglobin of 10.4, normal hematocrit (47%), and severe thrombocytopenia of 60,000 but no signs of bleeding from any external site. ESR, liver transaminases (AST & ALT) were also elevated but bilirubin levels were in limits. His RFT, electrolyte levels, blood sugar levels, urine routine and microscopy test were normal. ECG, chest x-ray and USG of abdomen & pelvis was also normal with no signs of fluid accumulation. The lowest level of platelets observed was 40,000 during the total stay at hospital.

While in ward, the patient developed right sided LMN facial weakness (Fig 1) on day 3 of admission, as evidenced by -

- facial asymmetry
- deviation of angle of mouth towards left side
- loss of nasolabial fold on right
- absence of wrinkles on right half of forehead
- Inability to close the right eye completely
- bell's phenomenon positive.

Fig. 1: Right Facial Nerve Paralysis (LMN type)

This is not associated with any other cranial neuropathy or neurological deficit anywhere in the body nor parotid gland pathology. An interdepartmental call for neurological opinion was sought. A non-contrast computed tomography of the brain was done immediately which showed no signs of any acute ischemic or hemorrhagic stroke event nor any presence of space occupying lesions. MRI brain revealed no evidence of any altered signal intensity in brain parenchyma or in any part of the right facial nerve, any ischemic or hemorrhagic stroke signs nor any leptomeningeal enhancement. He was diagnosed with Bells' palsy as a complication secondary to dengue fever.

The patient was treated conservatively for his dengue fever and was prescribed oral prednisolone 60mg for 7 days along with artificial tear eye drops. He was advised for facial physiotherapy and was discharged on day 10 post-admission with partial improvement. Other deranged blood parameters including the platelets value were also normalized before discharge. When he visited for follow-up three weeks later, his facial palsy had resolved noticeably.

DISCUSSION

Dengue was classically described as "breakbone fever" by Benjamin Rush in 1780. Dengue is the most rapidly spreading mosquito borne viral infection of mankind and has been identified as one of the neglected tropical diseases by WHO.[8] Dengue is endemic in more than hundred countries worldwide. More than half of the world's population is at risk, although Asia bears 70% of the global burden.[9] Flaviviruses are mainly spread in tropical regions that lie between about 35° north and 35° south latitude — the same areas where Aedes aegypti, the primary mosquito carrier, is commonly found. Other mosquito species such as Aedes albopictus and Aedes polynesiensis can also transmit dengue viruses in these regions.[10]

In India, every state is endemic for dengue and there has been a dengue fever outbreak multiple times in the past. Dengue incidence is currently increasing for the past few years while the case fatality rate has significantly declined and is currently at 0.1% and stagnant since 2019.[11] The disease shows a seasonal pattern that is the cases peak in and after monsoon with a surge during July to November every year. Year-round construction activities, life-style changes and deficient water management, improper water storage, stagnation of rain water in containers lying outside houses and poor water storage practices leading to proliferation of vector breeding sites in urban, peri-urban and rural areas.[11]

Although most people infected with dengue virus will not have symptoms, those who do typically experience abrupt onset high fever, body aches, arthralgia, retro-orbital headache, rash, and nausea. If symptoms occur, they usually begin 4–10 days after infection and last for 2–7 days, starting with a febrile phase, after which most patients will recover.[12-13] Other patients, often with increased capillary permeability, will enter the critical phase around the time of defervescence on the 4th-5th day of illness, when warning signs become evident and when worsening of symptoms, organ dysfunction, and occasionally severe bleeding can occur; however, not all patients with severe dengue will have warning signs. Severe dengue can be fatal, and patients need in-hospital care for judicious intravenous fluid infusion and to manage other associated complications. Following the critical phase, patients typically enter the recovery phase with reabsorption of extravascular fluid. Most patients will recover in 1–2 weeks. [13-14] A biphasic ("saddleback") fever curve has been described in approximately 5 percent of cases; in such patients, acute febrile illness remits and then recurs approximately one to two days later; the second febrile phase lasts one to two days.[15] The severity of illness and clinical manifestations vary with age and DENV type. DENV-2 and DENV-4 may be more likely to cause inapparent infections in flavivirus-naïve persons.[16] Disease severity may be increased among infants and elderly people and patients with concomitant comorbidities.[17] There are no known specific antiviral treatments for dengue virus infections. Medications can be used to manage pain and fever.

In the last two-three decades, the virological characteristics of dengue viruses have been changing, and neurological features of dengue infection are reported more frequently.[18] Classically dengue virus was believed to have no CNS predilection.[19] However, latest reports on neurotropism or neuroinvasion of the dengue virus, causing a constellation of CNS manifestations has been documented. The neurological spectrum of dengue patients is not clarified because of less case reports, lack of imaging facilities and neurophysiological studies in many facilities.[2] Neurological manifestations occur more frequently in younger patients, during epidemics than in isolated cases and can arise in any spectrum of dengue fever.[6] The neurological complications in dengue infection can be categorized into three groups based on one of the pathogenic mechanisms: (1) neurotropism leading to encephalitis, meningitis, myositis, and myelitis; (2) systemic complications resulting in encephalopathy, stroke, and hypokalemic

paralysis; and (3) postinfectious immune-mediated acute disseminated encephalomyelitis, Guillain–Barre syndrome, and peripheral neuritis.[4,20] Among these features, encephalitis and encephalopathy are the most common neurological complications.[21-22] Both encephalopathy and encephalitis reflect severe disease and are associated with poorer recovery.[4,23] Dengue viral antigens have been isolated in human brain tissue via immunohistochemistry in cases of fatal dengue infection.[24] Neurotropic potential of dengue virus leading to encephalitis has also been suggested by the presence of DenV RNA in the CSF and cerebral tissue of patients.[25-27] Few case reports are available regarding dengue-associated neuritis, such as brachial neuritis, long thoracic nerve palsy, phrenic, oculomotor, abducens, and peripheral facial palsy.[3] The pathogenesis of neuritis is likely to be immune-mediated. They usually respond to supportive treatment or steroids.[4]

Our patient developed a rare neurological complication of isolated right sided Bell's palsy on the 5th day of dengue fever. Bell's palsy is a peripheral facial nerve palsy involving the lower motor neuron, causing weakness of all muscles of facial expression. Bell's palsy occurring with viral infections have been previously reported in association with Herpes simplex virus, Herpes zoster, Human Immunodeficiency Virus etc. [28] Reports describing flavivirus as a cause of seventh nerve palsy are scarce.

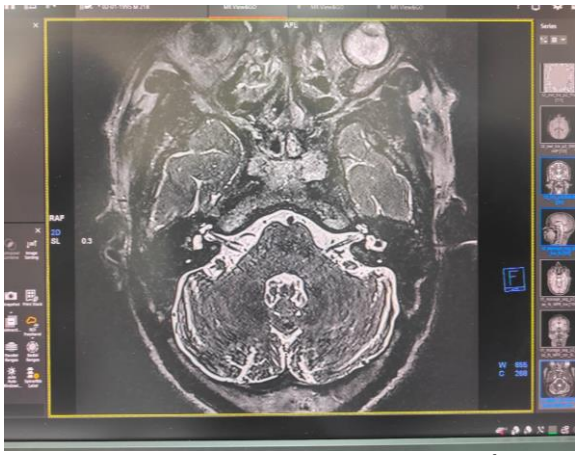

Fig. 2: MRI of brain at the level exit of 7th CN.

The diagnosis of bell's palsy in our case report was suggested by the patient's history and clinical examination along with imaging studies. We chose not to perform a lumbar puncture as it was unlikely to change the management course and posed an added risk in view of the patient's thrombocytopenia. Radiological imaging did not suggest any evidence of ADEM encephalitis or any other possible pathologies that could result in facial nerve paralysis (Figure 2). The close temporal association between symptom onset and a confirmed dengue infection makes dengue-associated etiology the most plausible explanation. The underlying mechanism behind this presentation is not clearly defined in current literature likely owing to the limited number of reported cases. As the patient symptom appeared early in the course of illness (day five), direct viral effect- through neural invasion or injury of the cranial nerves- can be hypothesized in our case report. Concurrently, the recovery following the use of steroids pointing towards an immune mediated mechanism cannot be ignored and is debatable.

CONCLUSION

Dengue has become an emerging public health alarm globally with its rising incidence. Continuous surveillance, adopting sustainable methods to reduce transmission and maintaining the environmental hygiene will reduce its outbreaks. Beyond its usual clinical picture, dengue infection can present with unusual manifestations, with neurological complications being increasingly recognized. These manifestations can affect nearly any part of the nervous system through different pathological mechanisms. However, with limited literature, the true relationship between dengue and many of these atypical presentations is still not fully understood. Recognizing and addressing neurological involvement early is essential to manage patients efficiently, limit disability, and reduce mortality. This case draws attention to the need for deeper research into the diverse neurological spectrum of dengue fever and

emphasizes clinicians to remain alert to these unusual yet clinically significant manifestations that may alter patient outcomes.

Declaration by Authors

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