Neuro-Chikungunya: Acute Transverse Myelopathy Associated with Chikungunya Virus Infection

Rahul Kumar1, Prabal Rajvanshi2, Harshit Khosla3, Sahil Arora3

Abstract
Chikungunya is an arboviral infection caused by Chikungunya virus, an RNA virus from Togaviridae family.1 The disease manifests as fever, rash and characteristically, with arthralgia.2 Chikungunya is strongly believed to have neurotropism but has not been well studied like other neurotropic arboviruses.2 Encephalitis appears to represent the most common clinical manifestation1 and occurs either simultaneously or within few days of onset of systemic symptoms, during the period of viremia. A delay of more than two weeks has been reported with other complications like myelitis, Guillain Barre syndrome and optic neuritis. This case describes the clinical, serological, neuroimaging and CSF findings of Chikungunya induced acute transverse myelitis in a 13 years old male patient who responded to steroid treatment. It is a relatively unknown and very rare complication of Chikungunya virus infection during outbreak of Chikungunya infection in September 2016.

Introduction
Chikungunya virus is an insect-borne (Aedes mosquito) virus of the genus Alphavirus and the family Togaviridae.1 The word Chikungunya means, “become twisted” in the Kimakonde language, of ethnic group in southeastern Tanzania and Northern Mozambique. After a average incubation period of 2 to 4 days (2 to 12 days) the disease manifests, without prodromata, with typical features of fever, rash and arthralgia.3,4 Children are among the high risk group for severe manifestations of the disease and some clinical features in this group are distinct from those seen in adults. Chikungunya virus is considered primarily a non-neurotropic virus but neurological complication like meningoo-encephalitis, myeloneuropathy, Guillain Barre syndrome, optic neuritis6 et al. have been reported. A history of fever, polyarthralgia, and/ or rash in patients with neurologic symptoms may be the first clues to neuro-Chikungunya. Clinical findings or neuroimaging suggestive of demyelination process may lead to proper selection of appropriate serodiagnostic tests.

Case Report
A 13 year old school going male from Aligarh, Uttar Pradesh, presented with chief complaint of inability to walk since 5 days. There was history of high grade fever which lasted for 2 days, associated with arthralgia and myalgia, 10 days before the development of paraparesis. He had associated history of urinary retention, constipation, decreased sensation for pain, touch and temperature below T8 spinal segment level. There was no history of recent vaccination, trauma to spine/limbs, contact with tuberculosis patient or anti-tubercular therapy in past. Similarly there was no history of rash, bleed from any site, malena, joint swelling, respiratory problem or pedal edema. He had no complaint of blurred vision, diplopia, facial nerve palsy features, nasal regurgitation or features suggestive of any other cranial nerve involvement. There was no history suggestive of nystagmus, slurred speech, altered sensorium, seizures, ataxia, headache, vomiting.

On clinical evaluation, patient was conscious and alert. Further physical examination revealed hypertonia and brisk deep tendon reflexes in lower limbs. Power was grade [0/5] in bilateral lower extremities, in both proximal and distal segment. Plantar response was extensor bilaterally, absent abdominal reflex and ankle clonus were present in both lower limbs. Tone, power and all deep tendon reflex were normal in upper limbs. Respiratory, Cardiovascular and Abdominal examination were unre Markary

Laboratory results revealed, Hb 11.7gm/dl, ESR-20mm, TLC-4520/ cumm, Platelet count- 92000/cumm, B. Urea- 39mg/dl, S. Creatinine- 0.4mg/ dl, Serum Na+/K+-138/4.3 mEq/L, S.Bilirubin- 0.8mg/dl, SGOT/SGPT-60/50u/l, ALP- 145u/l, S.Albumin-3.3g/dl, S.Globulin- 2.7g/dl, Serum Calcium/Phosphate- 8.3/3.5mg/dl. HIV(I and II), Hepatitis B and Hepatitis C serology was negative. Dengue serology (IgG and IgM), NS1 Antigen, Malaria serology (and antigen), Peripheral smear for malaria parasite, and Widal test were discovered to be negative. IgM chikungunya Serology by ELISA was positive and chikungunya PCR was negative in serum sample. Autoimmune marker Antinuclear antibody, Rheumatoid factor, Anticardiolipin antibody and anti dsDNA were negative. Serum NMO Antibody against water channel protein aquaporin-4 was negative. Optic neuritis was ruled out on basis of normal visual evoked potential and absent relative afferent papillary defect. Fundus examination was normal. MRI Brain showed nonspecific demyelinating foci in bilateral fronto-parietal subcortical white matter with no restricted diffusion or post contrast enhancement, largest in the left parasagittal parietal white matter. MRI Spine showed long segment cross sectional altered intramedullary signal in cord from C5-C6 disc to D8 body (Figure 1). Another intramedullary hyperintense lesion was found opposite C5 vertebrae and C2-C3 vertebrae in posterior

---

1Senior Resident, 2Professor and Consultant, 3PG Resident, Department of Medicine, Vardhman Mahavir Medical College and Safdarjung Hospital, New Delhi

Received: 02.02.2017; Accepted: 04.04.2019
Arbovirus which mostly causes self-limiting febrile illness, had its recent outbreak in September 2016 in Delhi and various other states of India. Being associated with morbidity in the form of rheumatic symptoms (joint pains, swelling and stiffness) and rash, chikungunya can infrequently cause neurological sequelae which can be more or less life threatening.

Since 2005, small mutation in the E1 protein of the viral envelop have been considered as a major explanation of disease having varied complications and its expansion in South-east Asian countries. The exact pathophysiology of neurological involvement in Chikungunya infection has not been established. There are reports of association of neurological involvement in animal experimental studies, strongly points toward possible neurotropism of this virus.3

Although spectrum of neurological manifestations ranging from encephalitis, Guillain Barre syndrome, external ophthalmoplegia, sensorineural deafness6 etc. have been reported but Acute transverse myelitis is a rare complication of chikungunya. The presence of Chikungunya virus in the CSF establishes chikungunya as the cause of acute transverse myelitis on this case.

Our patient was confirmed as having Chikungunya infection on the basis of detection of CHIK IgM in serum and positive real time-PCR in CSF sample.

The negative result of CSF reverse transcriptase PCR (RT-PCR) assay for CHIKV RNA was not surprising because of late presentation of the patient and lumber puncture for CSF result was done after 20 days of symptoms onset. The time lapse between acute Chikungunya infection and the onset of myelopathic sequelae, and response to steroid, suggests an immune mediated phenomenon rather than direct activity of the virus itself. We chose to rely on RT-PCR detection of the virus to diagnose CHIKV infection rather than testing for IgM antibodies, which may persist for several months after infection and could reflect coincidental infection rather than an acute infection. In summary, during CHIKV outbreaks, clinicians should consider that CHIKV may be a likely cause of CNS infections among children.

This case emphasizes the fact that studies are scarce that report transverse myelitis confirmed with Chikungunya PCR. There is further paucity of literature on such patients showing response with steroids. Thus patients with chikungunya infection should be followed up for possible neurological complications.

Chikungunya is expanding its territory and is posing a threat to non-immune population in many countries. Therefore, physicians are expected to see more cases of Chikungunya and thus Neuro-chikungunya in the future.

References