

Chikungunya Myeloradiculopathy: A Rare Complication

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ABSTRACT

Chikungunya, an alpha virus belonging to the family of Togaviridae is transmitted to humans by the bite of *Aedes aegypti* mosquito and presents with fever, headache, rash, and severe arthralgia. Chikungunya virus is not known to be neurotropic, but cases of meningoencephalitis have been reported during outbreaks. The clinical, laboratory and neuroimaging findings of a 56-year-old man who initially developed Chikungunya fever with arthralgia and later on lead to Chikungunya myeloradiculopathy, a relatively unknown and rare complication of the infection has been presented.

Key words: *Aedes aegypti*, Chikungunya virus, Myeloradiculopathy

INTRODUCTION

Chikungunya is a viral fever caused by a single stranded RNA virus of the genus alpha virus in the family Togaviridae and transmitted to humans by the bite of *Aedes aegypti* mosquito. The name “Chikungunya” is derived from the Makonde word meanings “that which bends up” in reference to the stooped posture which develops as a result of arthritic symptoms of the disease. Chikungunya virus is of African origin and is maintained among non-human primates by *Aedes* mosquito of subgenera *stegomyia*. Disease is endemic in rural areas of Africa.^[1]

Chikungunya virus was first isolated in Calcutta, India, in 1963^[2] with several reported outbreaks in India since then.^[3,4] The first isolation of the virus worldwide was in 1952, following an outbreak on the Makonde Plateau. The symptoms include fever, headache, rash, and severe arthralgia. Many of these symptoms are indistinguishable from dengue fever, and simultaneous isolations of both dengue and Chikungunya from sera of patients have been reported.^[5] Chikungunya virus is related antigenically to O'nyongnyong virus and is not known to be neurotropic. However, meningoencephalitis has been reported in

outbreaks in India and the Reunion Islands. The clinical, laboratory and neuroimaging, findings of Chikungunya myeloradiculopathy, a relatively unknown and rare complication of the infection have been presented.

CASE REPORT

A 56-year-old male patient came with the complaints of inability to walk without support and past H/O fever 20 days back which lasted for 10 days and was associated with the multiple joint pain, swelling and restriction of movements. The joint pain persisted for 2 weeks along with the fever. He was a smoker and occasional alcoholic. There was no other positive history or symptoms pertaining to other system.

O/E wrist and ankle skin excoriation were present. CNS examination revealed loss of abdominal reflexes, decreased power (3/5) in upper and lower limbs, decreased pain and temperature sensations in bilateral deltoid, neck, and abdominal regions. Considerably, proximal muscle weakness was more than distal muscle weakness.

It was diagnosed as post viral arthritis with CNS involvement and hence further evaluated. Laboratory analyses for all parameters were normal except elevated ESR. Serological studies were positive for CRP and Chikungunya IgM ELISA (Done at KING INSTITUTE, Chennai) when all the other tests like RA Factor, ANA, DSDNA, Dengue IgM ELISA, leptospirosis IgM and JE IgM ELISA were

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negative. CSF analysis showed 95 lymphocytes, protein 83 g/dl, glucose 50 mg/dl and negative results for gram positive or negative organisms. MRI spine revealed findings related to demyelination at C2-C3 and T5-T7 levels. The patient was treated with steroids, NSAIDs and given supportive treatment. He improved clinically but, further follow-up was lost.

DISCUSSION

The precise reasons for the re-emergence of Chikungunya in the Indian subcontinent as well as the other small countries in the southern Indian Ocean are an enigma. Studies need to be conducted on virus isolates obtained during the current outbreak in order to understand if any mutation has occurred in the virus that has facilitated the large-scale spread of this virus in the region. Alternatively, one could take the simplistic view that the lack of herd immunity within the country probably lead to its rapid spread across several states.^[1] Chikungunya virus, with spectrum of diseases ranging from a self-limiting febrile illness to crippling acute and lingering arthritis and sometimes serious complication like encephalitis and death.^[6] There was no definite histological evidence of Chikungunya neurotropism.^[7] The neurovirulence and neuroinvasiveness of several other alpha viruses were well established, and chikungunya virus had been isolated from two children with clinical signs of encephalitis and meningitis.^[8]

Rampal *et al* also reported three cases of lower motor neuron type paraplegia, diminished deep tendon reflexes without any focal neurological deficit in seven cases and involuntary movements in upper limbs in four cases. There had also been cases of meningoencephalitis in neonates, Guillain Barré syndrome and other cases of CNS involvement.^[9] Ganesan *et al* also reported 2 cases of encephalomyeloradiculitis with similar findings after

Chikungunya infection and which was discussed with neuroimaging and autopsy reports.

CONCLUSION

This report highlights that Chikungunya virus which causes a self-limiting disease involving mainly the joints in the acute phase, may be presented with significant demyelinating sequel weeks afterward. So, the clinicians should treat the infected patients effectively and watch for important complications during the follow up.

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