



NEUROLOGICAL MANIFESTATIONS OF CHIKUNGUNYA: CASE SERIES

Pathology

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KEYWORDS

INTRODUCTION :

Chikungunya fever (CHIK) is a viral disease transmitted by the bite of *Aedes aegypti* and *Aedes albopictus*. The causative agent is chikungunya virus (CHIKV), a RNA alpha virus belonging to the *Togaviridae* family.[1] Outbreaks of this virus have occurred in India since 2005, and Delhi recently witnessed the outbreaks of CHIK and dengue fever, presenting with high-grade fever, rash, arthralgia, and thrombocytopenia.[2] However, atypical presentation and severe complications such as myocarditis, hepatitis, Guillain-Barre syndrome (GBS), and meningoencephalitis can happen rarely.[3] Here, we have reported two cases of CHIKV with not so common neurological manifestation. First patient had atypical GBS (sensory motor poly neuropathy affecting lower limbs more than upper limb) and the second patient had acute motor sensory axonal neuropathy (AMSAN variant of GBS) as neurological manifestations.

Case reports :

Case 1 :

A 49 year old male patient known case of diabetes mellitus presented with complaint of fever with chills with joint pain and abdominal pain for since 23/09/2018. Patient was admitted at private hospital on 24/09/2018 with these complaints where during hospital stay patient had developed AKI for which he was treated with intra venous fluids and antibiotics. Patient was investigated further which revealed peripheral smear for malaria parasite, leptospira IgM antibody, Dengue profile and Chikungunya Ig M antibody tests were negative. Total WBC count, ESR, PCT and CRP level were elevated but platelet count was normal. Patient with this history and reports was referred to DHIRAJ hospital on 18/10/2018 where he was examined and further investigation was done. On clinical examination patient was conscious oriented had weakness in bilateral lower limb was more in left lower limb as compare to right lower limb there was no weakness in both upper limb. Patient had increased tone with decreased power with hypo reflexia and extensor planter in left side lower limb for that patient underwent neurology consultation and advised all four limb NCV. NCV revealed sensory motor poly neuropathy affecting lower limbs more than upper limb and diagnosed as a GBS. On investigation patient was positive for Chikungunya PCR. Patient was treated with immunoglobulin therapy along with supportive therapy in form of antibiotics and iv fluids and given mechanical ventilation for acute respiratory failure during hospital stay but eventually patient passed away on 25/10/2018 at 5:55 a.m.

Case 2:

48 year old male presented with complains of high grade fever with chills with multiple joints pain and generalized weakness since 27/10/2018 to private hospital where diagnosed as chicken gunia fever with viral encephalitis Chikungunya PCR was positive and MRI brain revealed multiple demyelinating lesions in bilateral subcortical and periventricular white matter with normal MR Angiography of brain which was suggestive of Chikungunya encephalitis. For further management patient was referred to DHIRAJ hospital on 1/11/2018. Where on examination there was hypotonia with hyporeflexia of all four limbs NCV was done all four limbs revealed severe sensory motor axonal neuropathy of all four limbs and diagnosed as acute motor sensory axonal neuropathy (AMSAN). For that patient was given 3

cycles of plasmapheresis and treated with antibiotics. Patient was put on mechanical ventilation for acute respiratory failure but eventually patient passed away on 8/11/2018 at 10:15 A.M.

Discussion:

Neurologic manifestations of the disease are significantly less common, but incidence appears to be rising over the last decade, particularly among more severe cases. A review of 405 cases of suspected CHIKV fever in India in 2006 found neurologic symptoms in 9% of confirmed CHIKV cases and 3.14% of suspected cases [4]. Another study in the same region found that 16.3% of CHIKV suspected cases had neurologic disease, with patients above 60 years of age showing a higher proportion of neurologic disease [5]. Indeed, CHIKV appears to have increased neurovirulence among the severely ill, elderly, and neonates. Neurologic manifestations of CHIKV most commonly include encephalitis, encephalopathy, and peripheral neuropathy (including Guillain-Barre syndrome).

Guillain-Barre syndrome (GBS) can occur after a number of infections, and has been described in association with other arboviruses, including Zika [6]. Increases in the number of GBS cases were seen during the outbreak of CHIKV in French Polynesia in 2013–2014, suggesting an association with the virus. Case series from 2006 outbreak on La Reunion Island also demonstrated elevated anti-CHIKV IgM in patients with GBS and an absence of other etiologies, strengthening the evidence for a clinical link between viral infection and subsequent development of GBS.

Conclusion:

Though neurologic symptoms associated with CHIKV remain relatively uncommon, their frequency appears to be increasing with poor outcomes. Clinicians treating patients with neurologic symptoms from CHIKV endemic areas should be aware of the growing association between CHIKV and neurologic sequelae to help guide diagnostics.

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