Chapter

Neurotropism of Chikunguniya Virus Depicted by Intramedulary Spinal Cord Hyperintensities and Diffuse Restrictions in Cerebral White Matter

Khatri Harsh[1], Shah Heli[2], Roy Dhara[3] and Kaushalendra Mani Tripathi[4]*

Department of Medicine, V.S. Hospital, Smt.N.H.L. Municipal Medical College, India

***Corresponding Author:** Kaushalendra Mani Tripathi, Department of Medicine, V.S. Hospital, Smt.N.H.L. Municipal Medical College, Ahmedabad, India

First Published **July 05, 2019**

This Book Chapter is an excerpt from an article published by Kaushalendra Mani Tripathi, et al. at Case Reports in Infectious Diseases in July 2018. (Harsh Khatri, Heli Shah, Dhara Roy, and Kaushalendra Mani Tripathi, "A Case Report on Chikungunya Virus-Associated Encephalomyelitis," Case Reports in Infectious Diseases, vol. 2018, Article ID 8904753, 4 pages, 2018. https://doi.org/10.1155/2018/8904753.)

Conflict of Interest: The author(s) declare(s) that there is no conflict of interest regarding the publication of this paper

Abstract

Chikungunya is a re-rising alphavirus infection that has resulted from enhanced vector competence. Chikungunya is a dengue like illness characterized by acute febrile polyarthralgia, arthritis, malaise, body ache, rash, headache and nausea.The Neurological menifestations are uncommon and incorporate meningoencephalitis, myelitis, Guillain-Barre syndrome, cranial nerve palsies, myelopathy and neuropathy,MRI abnormalities in patients with encephalopathy from india have been reported in the form of multiple punctuate white matter lesions that are more prominent on diffusion weighted MRI than on T2 or T1.Here we present an intriguing case of chikungunya encephalomyelitis who presented to our tertiary care hospital with quadriparesis and urinary retention.He was treated with 5 doses of intravenous Immunoglobulin along with supportive care with which he showed partial recovery.

Keywords

Chikuguniya; Encephalomyelitis

Introduction

Chikungunya is a re-emerging alphavirus infection that has resulted from enhanced vector competence. Alphaviruses having single-stranded RNA genome belong to Togaviridae family of viruses and are divided into arthritogenic viruses(old world) and encephalitogenic viruses (new world) including equine encephalitis viruses. It is transmitted by the mosquitoes of Aedes genus amongst which Aedes aegypti being the principal vector in earlier times now viral host range includes A. albopictus as well [1]. From first isolation of virus in 1952-53 from Tanzania [2] it has spread in tropical countries to such an extent that 2006 outbreak in India reported 1.5 Million cases with A. Aegypti implicated as the vector. Chikungunya virus was first

isolated in Calcutta, India, in 1963, with several reported outbreaks in India since then.In year 2016 clinically suspected cases of chikungunya in India were 64057[3].

Neurological complications of Chikungunya virus(CHIKV) infection are infrequent but it can include a spectrum of different neurological manifestations because of neurotropism demonstarted by this virus. Herein we describe an adult case of CHIKV associated central nervous system (CNS) disease.

Case Report

A 64 year old male patient ,who is a known case of Diabetes mellitus II, Hypothyroidism and Ocular Myasthenia gravis since 3 years presented to our tertiary care center with 8 days history of acute fever, malaise, generalized rash and multiple joint pains. He complained of acute onset of sensorimotor Quadriparesis and urinary retention since 7 days which was followed 1 day later by H/o altered sensorium. He had no history of headache, vomiting, seizure, dimness of vision, double vision, dysphagia ,change in voice, neck/back pain. Patient had already received Inj. Methyl Prednisolone pulse therapy 1gm each for 5 days before presenting to our institute to which he responded partially in form of improved level of consciousness.

On Presentation, his CNS Examination: Patient was conscious oriented following verbal command, no neck rigidity. Cranial nerves: Left eye ptosis+;No facial/neck flexor weakness; Mixed dysarthria+. On motor examination: Nutrition- no undue wasting or hypertrophy; Tone: Spastic both Upper limbs(UL) with flaccid both lower limbs; Power(acc to MRC scale) : 4/5 in both UL and 1/5 in both LL ; with B/L hand grip weakness and B/L dorsiflexor weakness ;Deep tendon reflexes(DTR) were +3 in both UL with B/L pectoralis reflex and jaw jerk+;DTR were absent in both lower limbs(LL) well as absent planters. Sensory Examination: Impaired joint, position & vibration sensations upto metatarsophalangeal joints in both Lower limbs and upto metacarpophalangeal joints in both upper limbs with normal cerebellar examination.

Investigations

His routine Investigations Including Complete blood count, Random blood sugar, Renal & liver function tests , serum electrolytes were within normal limits except for slightly raised WBC to 14500/µL , CRP 16 and CPK Total 84. Serum Chikungunya RT-PCR was positive.Electroencephalogram(EEG) showed generalised bilaterally symmetrical diffuse theta-delta slowing suggestive of diffuse encephalopathy. His electrophysiology study showed distal symmetrical sensory motor axonal polyneuropathy more in lower limbs than upper limbs.CSF routine micro : Protein- 96mg/dL; Glucose -60mg/dL; Total cells- 140 with lymphocytic pleocytosis with Negative Gram and ZN stain. CSF Viral Panel was negative for neurotropic viruses. ANA blot was negative. His Aquaporin-4 Ab (NMO Ab) was negative.

MRI Brain(P+C) with whole spine screen: Multiple tiny altered signal intensity foci with restricted diffusion seen in subcortical and periventricular white matter of bilateral(B/L) cerebral hemispheres, corpus callosum and b/L corona radiata with normal MR Angiogram. Patchy areas of T2W hyperintensity in the cervico-dorsal cord predominantly from C7 to D9 level without significant post contrast enhancement .These findings were consistent with features of encephalomyelitis.

Management

Our patient was managed with IV immunoglobulin (IgG) in a total dose of 140 grams as well as supportive treatment for his co morbidities. He was discharged in stable hemodynamic condition with power 5/5 in both UL and 3/5 in both LL.Patient was advised supportive treatment and neuro-rehabilitation in form of active physiotherapy. The Patient responded well in a follow up after one and a half months his power improved; He is able to walk with one stick support with a normal bladder function with current Modified rankin scale (mRS) of 4.

Discussion

The Neurological menifestations associated with chikungunya are uncommon and variable. CHIKV has got predilection for Central and peripheral nervous system. Various manifestations include meningoencephalitis, myelitis, Guillain-Barre syndrome, cranial nerve palsies, encephalomyeloradiculitis and neuropathy1,2.

In this unique case of Chickungunya Encephalomyelitis, diagnosis was based on positive concordance between clinical presentation, serology, CSF and neuroimaging features. Other differential diagnosis were ruled out by thorough investigations.

MRI brain showed bilateral multiple tiny punctuate white matter lesions with increased signals on DWI(diffusion weighted sequence) which is quite characteristic of early Chickungunya encephalitis as demonstrated by case reports in brazil by Licia Pacheco Pereira et al [4] In India by by Ganesan et al [5]. and in southern Thailand by Sarunyou Chusri et al [6]. Restricted diffusion precedes signal abnormalities seen on fluid-attenuated inversion recovery (FLAIR) images and is also known to resolve earlier than the FLAIR signal abnormalities during the recovery period [4]. MRI spine showed T2W intramedullary hyperintensities in cervico- dorsal region with long segment transverse myelitis. These changes of Encephalomyelitis were similar to the case report by D Taraphdar et al [7] in west Bengal.

The Pathophysiological changes leading to such characteristic neuroimaging findings include bilateral white matter lesions with restricted diffusion which is implicatd to cytotoxic edema secondary to plasma leakage from capillaries and venules. Such changes can also occur in vasculitis or acute demyelination induced by sensitization to viral antigen and microglial activation [4].

Conclusion

Chikungunya related CNS complications should be searched for in patients from endemic areas with prominent neurological features on examination. Radio imaging along with Serology and CSF help to

confirm the diagnosis. This case report does highlight the importance of clinico-radiologic findings in patients with encephalomyelitis following Chikungunya infection.

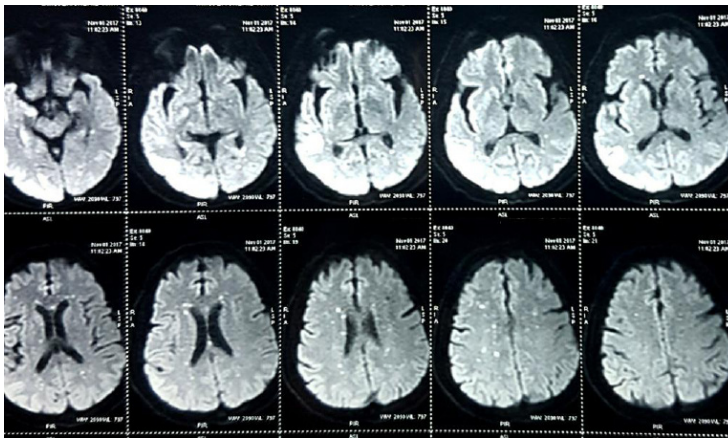

Figure 1: Multiple tiny areas with restricted diffusion in subcortical and periventricular white matter on axial DWI brain.

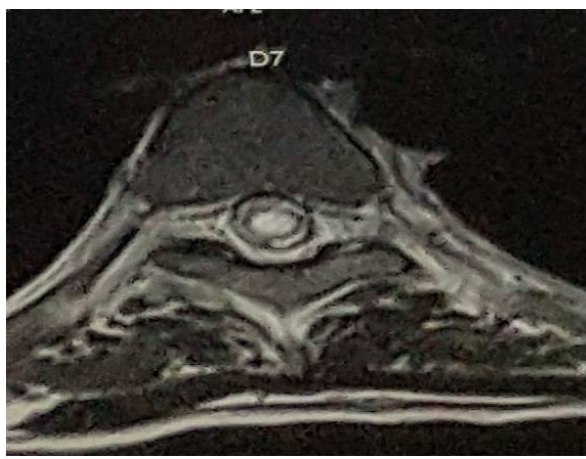

Figure 2: Axial T2W sagittal section showing D7 intramedullary hyperintensity.

Figure 3: T2W sagittal image showing intramedullary hyperintensity extending from C7–D9 segment (long-segment myelitis).

References

1. García-Moncó, Juan Carlos , tropical viral CNS infections. In: CNS infections clinical approach, 2014 edn. London: Springer. 2014.
2. Noshir Wadia, Satish Khadilkar. Acute viral encephalitis. In: Neurological practice, 2nd edn. India: Elsevier. 2014.
3. Rhythma Kaul. Chikungunya virus emerges as bigger threat to people in north India. Hindustan Times, India.
4. Licia Pereira, Rafaela -Bôas, Stephanie Scott, Paulo Nóbrega, Manoel -Neto, et. al. Encephalitis associated with the chikungunya epidemic outbreak in Brazil: report of 2 cases with neuroimaging findings. Revista Da Sociedade Brasileira De Medicina Tropical. 2017; 50: 413-416.
5. K Ganesan, A Diwan, SK Shankar, SB Desai, GS Sainani, et al. Chikungunya Encephalomyeloradiculitis: Report of 2 Cases with Neuroimaging and 1 Case with Autopsy Findings,American Journal of Neuroradiology. 2008; 29: 1636-1637.
6. Sarunyou Chusri, Pisud Siripaitoon, Siriporn Hirunpat, Khachornsakdi Silpapojakul. Case Reports of Neuro-Chikungunya in Southern Thailand. The American Journal of Tropical Medicine and Hygiene. 2011; 85: 386–389.
7. Taraphdar D, Roy BK, Chatterjee S. Chikungunya virus infection amongst the acute encephalitis syndrome cases in West Bengal, India. Indian Journal of Medical Microbiology. 2015; 33: 153-156.