Post Chikungunya Brain Stem Encephalitis

LA Gauri*, BL Ranwa**, K Nagar**, A Vyas***, Q Fatima***

Abstract
Chikungunya is an arthropod born acute febrile arbo viral illness characterized by acute severe polyarthralgia. During last few years there has been scattered out breaks with associated neurological complications in India. Here we report a case of post chikungunya reversible demyelinating encephalitis who presented with vertigo, dysarthria and ataxia. There was complete clinical as well as radiological improvement with steroids.

Introduction
Chikungunya virus was first isolated in Calcutta, India, in 1963, with several reported outbreaks in India since then. The first isolation of the disease 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 isolation of both dengue and chikungunya from sera of patients has been reported. Chikungunya virus, an Old World alphavirus, 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. There was a recent outbreak of chikungunya in December 2010 in Bikaner, North-west Rajasthan. We present the clinical, neuroimaging, and CSF findings of Chikungunya brainstem encephalitis, a relatively unknown and rare complication of the infection.

Case Report
We here by present a case report of 55 years old male who admitted to our department with complaints of remittent fever for 4-5 days, 15 days back followed by progressive vertigo and dizziness, disturbed speech along with progressive difficulty in walking and meaningful use of hands for 5 days. On examination patient was afebrile, drowsy, responding to verbal commands (GCS 13/15), inattentive, therefore mental state examination could not be performed accurately, however, sometimes he was oriented to time, place, person with normal intelligence, emotion, behavior, memory and persistent bulbar dysarthria. Cranial nerve examination revealed prominent nystagmus without directional preponderance, bulbar weakness involving IX, X, XI and XII nerves.

Caloric stimulation test and Dix Hallpike manoeuvre were normal. Abnormal cerebellar signs were present which included in coordination involving both upper limbs i.e. abnormal finger nose test, finger-finger-nose test, past pointing, rebound phenomenon, dysdiadochokinesis, nystagmus, truncal ataxia with falling towards right side and lower limbs i.e. drunken gait, abnormal heel knee test, foot pat test, pendular jerks.

Motor system examination revealed no motor deficit apart from pendular knee jerks. Sensory nerve examination was normal and no signs of meningeal irritation and autonomic dysfunction were elicited. Blood, CSF examination, MRI, EMG and NCV were carried out immediately with accredited laboratory. Blood examination revealed all the parameters within normal limits. Serology (IgM) for chikungunya by NIV Pune Kit was positive. Blood RT-PCR for chikungunya was negative.

Physical CSF examinations were within normal limit. In CSF chemistry total protein was 45.50mg%, globulin – normal, glucose 90mg%, chloride 127mg%. In CSF cytology total cell count was two, all lymphocytes, gram stain, auramine screen and ZNS was negative. CSF IgM for chikungunya was positive (ELISA kit). IgG positive (titre 87.0). CSF RT-PCR was negative. NCV and EMG studies were normal. MRI brain revealed T2 and flair hyper intensity in B/L middle cerebellar peduncles (Figure 1a), medulla and visualized part of cervical spinal cord, parietal and parieto-occipital white matter and right capsule ganglionic area - possibility of demyelination ? (Figure 1b).

Patient is still being followed regularly for persistent neurological sequelae.

Discussion
Arbo viruses are known to cause viral encephalitis in many epidemics in the past. In 15 cases (out of 2424 seropositive), meningoencephalitis has been reported in chikungunya out break in Reunion island from March 2005-January 2006. In a study of 20 cases Rampal et al2 had shown the affection of CNS at various levels in the form of encephalitis (in 15 cases), encephalomyelitis (in 03 cases) and optic neuritis (in 02 cases).

As compared to other published case reports and articles it is still unclear whether neurological manifestation in chikungunya are due to infection or autoimmune demyelination.

Our case report points towards demyelination due to clear

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latent period between fever and neurological deficit, negative CSF DNA PCR and almost complete resolution of clinical and neuro-radiological deficits with steroids.

Ganesan et al\(^4\) reported two cases of post chikungunya encephalo-myelorediculitis with predominant clinical features of cortical involvement with altered consciousness and derangement of mental functions. MR findings of bilateral frontoparietal white matter lesions with restricted diffusion, which is described as an early sign of viral encephalitis.

Chandak et al\(^5\) reported 300 cases of CHIKV infection cases during the period of June 2006 to December 2006 was 300. 49 out of 300 patients enrolled for the study had neurological complications. These patients were grouped under encephalitis n=27, myelopathy n=7, neuropathy n=7, myeloneuropathy n=7 and myopathy n=1. They concluded that recent CHIKV infection was associated with neurological complications. Their house-developed ELISA system based on CHIKV antigen detection is more sensitive than IgG and IgM and equivalent to RT-PCR assay.

Musthafa et al\(^6\) reported a case of ADEM following Chikungunya Fever. Their patient developed a rapidly progressive quadriplegia after few days of subsiding of the fever. His MRI showed multiple white matter lesions and he improved remarkably with methyl prednisolone. His clinical picture, MRI findings and response to treatment are consistent with ADEM.

Our case is rare in that demyelination involved brain stem predominantly with almost complete radiological and clinical improvement after treatment.

**References**


