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Research Article

ACUTE DISSEMINATED ENCEPHALOMYELITIS – A RARE COMPLICATION OF CHIKUNGUNYA FEVER

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ABSTRACT

Chikungunya fever is usually a self-limiting viral illness with fever, rash and polyarthralgia are commonly encountered manifestation[1]. Here we are reporting a case of acute disseminated encephalomyelitis (ADEM) following chikungunya fever.

INTRODUCTION

Chikungunya is a virus of *Togaviridae* family, endemic in Africa and Asia. . It is transmitted by female aedesaegypti mosquito. It commonly manifests as fever, rash and severe arthralgia. Persistence of arthralgia poses significant morbidity for the affected individuals. Though neurological complications are rare which includes encephalitis, myelopathy, peripheral neuropathy, myeloneuropathy and myopathy[2]. Acute disseminated encephalomyelitis (ADEM) is a very rare complication of Chikungunya fever.[3, 4]

Case report

A 35 year old female from Bharatpur (Rajasthan, India) presented with complaints of fever, rash and polyarthralgia from five days. The fever was associated with chills and rigors and do not relieved even on taking medication. She has difficulty in doing household activities due to severe joint pains mainly involving large joints. Just 2 day before the presentation to the hospital, she developed altered sensorium in the form of dizziness and unconsciousness and was rushed to the emergency department of our hospital by the family.

On physical examination, the patient was febrile, axillary temperature was 103.4° F. The heart rate was 110 beats per minute, the blood pressure 130/90 mm Hg in both arms, the respiratory rate 18 breaths per minute, and the oxygen saturation 100% while she was breathing ambient air. Erythematous rash were present all over the body.

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Her consciousness level on Glasgow Coma Scale was four [E2V1M1] i.e. she responds only to pain stimulus with eye opening. Bilaterally, pupils were round, regular and reactive to light. Fundus examination revealed mild disc pallor on both sides. There was clasp knife spasticity in all the four limbs along with brisk Deep Tendon Reflexes. Neck rigidity was present although kerning's sign could not be interpreted On Investigation, complete blood counts were normal (Hemoglobin 13.2 gm/dl, Total leucocyte count 6800/cmm, Platelet counts 1.48 lacs/cmm). Liver and Renal function tests were normal.Blood sugar level was 116 mg/dl at the time of admission. Serum electrolytes Na^+, K^+ and Cl^-were 138 mmol/l, 4.3 mmol/l and 98 mmol/l respectively. Her serum Creatinine phosphokinase (CPK) level was 110 U/L and Lactate Dehydrogenase (LDH) was290 U/L.Her Erythrophilic Sedimentation Rate (ESR) was 78 mm in 1st hour.

Screening her as a case of tropical fever syndrome, malaria, dengue and scrub typhus were negative. Chikungunya IgM antibody by ELISA method was positive. Chest x-ray and abdominal ultrasound were normal. Further evaluating for the altered sensorium, cerebrospinal fluid (CSF) cytology shows lymphocyte predominant pleocytosis (150/cu.mm,80% lymphocytes and 20% neutrophils) along with elevated protein – 137mg/dl, Glucose level was 39 mg/dl.PCR (polymerase chain reaction) testing for viral antigens in CSF were negative for common viruses (Herpes Simplex Virus & Ebstein Barr Virus). Human Immunodeficiency Virus (HIVI, HIV II) screening test by rapid ELISA method was negative.

After the examination as well as blood and CSF investigation, the differential diagnosis could be either meningitis, encephalitis or meningoencephalitis probably due to viral

etiology i.e Chikungunya. Further evaluating the case, radiological examination was done.

MRI Brain with contrast with diffusion weighted images showed diffuse involvement of posterior one third of the corpus callosum. Multiple focal hyperintense lesions in T2 and FLAIR sequence were seen in subcortical white matter of bilateral cerebral hemisphere and right thalami-capsular regions. No abnormal contrast enhancement of the meninges was seen. (Figure 1 and 2). All these findings are suggestive of acute disseminated encephalomyelitis (ADEM)[5]. MRI Cervical Spine with Screening of whole spine was normal.

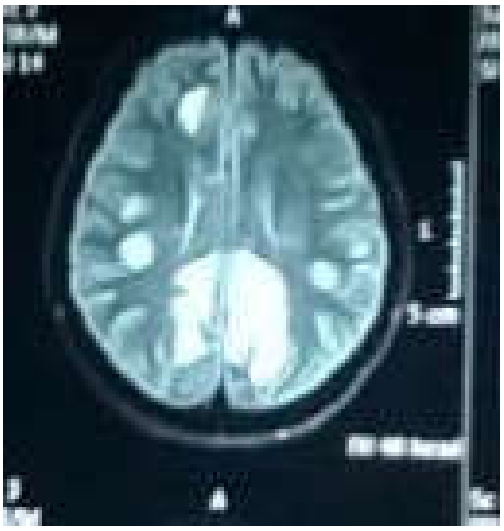

Figure 1

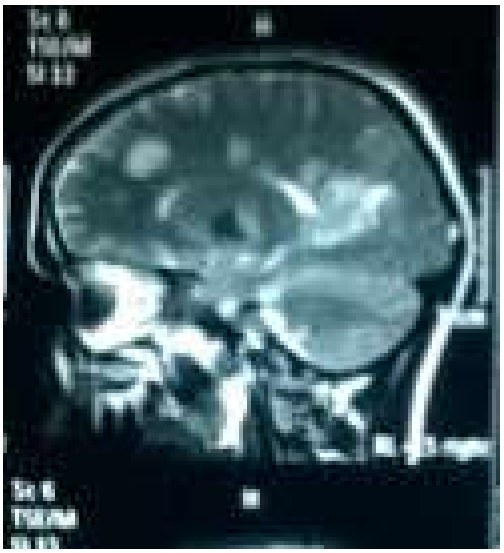

Figure 2

She was treated with intravenous Methylprednisolone 1 gram for 5 days. Patient partially responded to the treatment. Oral prednisolone 1 mg/kg body weight was started. After 5 days of oral treatment her consciousness level improved and GCS (Glasgow Coma Scale) score was 15. After regaining consciousness, she had difficulty in moving both upper and lower limbs, power graded was 0/5, along with blurring of vision.After continuous treatment with oral steroids, patient's vision came to normal. Power in both upper and lower limbs increased to 3/5. She was discharged in stable condition on oral prednisolone 1 mg/kg body weight for 7 days to review in follow-up. Hence blood investigations, CSF, and MRI findings concludes the diagnosis as acute disseminated encephalomyelitis (ADEM) due to Chikungunya Fever.

DISCUSSION

Acute disseminated encephalomyelitis (ADEM) is a monophasic disease that can be triggered by systemic viral infections or more rarely by vaccinations. The incidence of ADEM is reported to be 0.4–0.8 per 100,000 and it commonly affects children and young adults. The higher incidence in this age group is due to high frequency of exanthemata's and other infections and vaccinations. It can be classified either on the basis of areas of CNS involvement as well as its course of illness. [6 7 8 9]. Pathophysiology of the disease is thought to be auto immune injury to the central nervous system. ADEM doesn't have any specific biological markers, it is difficult to differentiate it from some microbial infections (herpes simplex encephalitis, listeria meningoencephalitis), vasculitis and demyelinating disorders. Multiple Sclerosis closely resembles ADEM.[10]

Chikungunya fever clinical spectrum is wide i.e. from a self-limiting febrile disorder to development of acute as well as chronic arthritis. It is associated with many neurological complications, which suggests that the virus has neurotropic nature too although the outcome of the complications is good. Regarding the prevalence of neurological complications, the most common is encephalitis followed by myelopathy, peripheral neuropathy, myeloneuropathy and myopathy in decreasing order.[2] Accounting for 25% of atypical cases and up to 60% of severe atypical cases, these neurological complications are one of the major cause of admission to the intensive care as well as death due to Chikungunya fever[11 12]

Treatment of ADEM is mainly supportive care. Specific treatment includes high-dose intravenous methyl prednisolone, intravenous immunoglobulin (IVIg), and plasmapheresis. Intra venous methylprednisolone 10-30 mg/kg for three to five days is used as first line therapy.If this therapy fails then immunomodulation can be tried by intravenous immunoglobins (IVIg) or plasmapheresis. Choice of second line therapy is individualised according to patient's clinical status, course of illness and affordability of the treatment. Many patients with ADEM has significant residual weakness. Physical Rehabilitation has a major role.[13]

CONCLUSION

An ADEM like life-threatening presentation is quite uncommon following Chikungunya virus infection, though the outbreak is not infrequent in India.[14] So in conclusion, our case of ADEM following Chikungunya fever is a rare and interesting association

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