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Case Report

Neonatal chikungunya with facial pigmentation and encephalitis

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ABSTRACT

Neonatal Chikungunya infection in postnatal period is a rare entity. Clinical presentation varies from a simple febrile illness with or without centrofacial hyperpigmentation to symptoms like poor feeding, lethargy, apnea and seizures. Here, we report a case where chikungunya infection was suspected and confirmed by IgM serology, which was positive in the baby and negative in mother.

Keywords: Chikungynya, Hyperpigmentation

INTRODUCTION

Chikungunya is a vector borne arboviral disease, caused by chikungunya virus (CHIKV) of genus alpha virus and family Togaviridae transmitted by mosquitoes of the genus Aedes (mainly *A. aegypti* and *A. albopictus*).[1] It was first reported in Tanzania during 1952, however first outbreak in India was reported from Calcutta in 1963. The virus has re-emerged and there is a surge in chikungunya cases from northern part of India including Delhi.[2] These mosquitoes breed in artificial collections of water like water coolers, cans, pots, open tanks and discarded tires. It is highly susceptible to the virus, prefers to live close to people, seeks a blood meal during day time and bites several people in a short period for one meal.

Here, we report a case of chikungunya infection in a neonate which was confirmed by Mac-ELISA test and recovered completely with conservative management.

CASE REPORT

16 days, female neonate was admitted to our unit with complaints of fever and poor feeding for 3 days, marked nasal and perioral hyperpigmentation starting over the face and progressing to the rest of the body in 3 days. There was no history of inconsolable cry, abnormal movements, loose stools, vomiting and jaundice. Baby was born to a primigravida, at term vaginally with birth weight of 2.5 kg, cried immediately after birth and started on breast milk immediately after birth.

At admission, the baby had signs of dehydration and patchy hyper-pigmentation over face and whole body with poor activity, without any organomegaly and with normal cardiorespiratory system. Investigations showed hemoglobin of 16.3 g/dL, total leukocyte count of 12,100/cumm (26% polymorphs, 72% lymphocytes), platelets 2,58,000/cumm, C-reactive protein 15.1 mg/L, sodium 139 meq/L, potassium 4.3 meq/L. Cerebrospinal fluid (CSF) examination showed glucose (CSF:blood)-0.67, protein-37 mg/dL, leukocyte count 60-70/cumm with 95% polymorphs. Liver and renal functions were normal. Blood and CSF cultures were sterile. IgM for chikungunya, sent on 5th day of illness was positive in baby. Ultrasound cranium and Otoacoustic emissions (OAE) result done at one month after discharge was normal. She was treated conservatively and was discharged after 14 days. On follow up at 3 months, infant was gaining weight, achieving milestone appropriately. Hyperpigmentation of the skin had diminished.

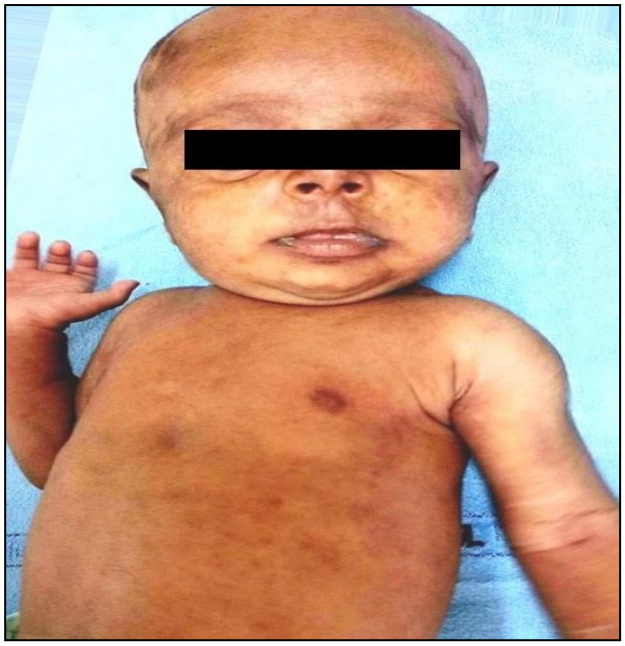

Figure 1: Facial hyperpigmentation.

DISCUSSION

Chikungunya fever is a vector borne viral disease caused by an RNA virus of genus Alphavirus of Togaviridae family; transmitted by the bite of infected Aedes mosquitoes. The incubation period is usually 3-7 days, but can vary from 2-12 days. The disease has differential presentation depending upon the age. In adults and children symptoms include fever, headache, fatigue, nausea, vomiting, rash, severe muscle and joint pain leading to inability to walk; however, in neonates presenting symptoms include fever, excessive crying, dermatological manifestations like maculopapular rash, nasal blotchy erythematic, freckle like pigmentation over centrofacial area, vesiculobullous lesions, apnea and shock.[3] CHIKV is not a 'true' neurotropic virus; however, encephalopathy appears to be the most common clinical manifestation among the newborns infected via mother-to-child transmission.[4]

Diagnosis is made by chikungunya virus real-time-polymerase chain reaction (RT-PCR) during viremia phase i.e. the 1st week of illness. Serum IgM levels are detectable as early as 2 days after infection and remain detectable beyond 3 months.

In our case, clinical features were short duration fever, patchy hyperpigmentation over face and extrimities and decreased oral acceptance. In our case, C-reactive protein was raised similar to the findings reported in a case series by Mangalgi et al.

Being a viral infection, CSF examination from patients with Chikungunya encephalitis has shown elevated protein and leukocyte count (often <100/µl), predominantly lymphocytes.[6,7] However, CSF cell count was normal in our case was predominantly polymorphonuclear and glucose and protein were normal. As reverse transcriptase-PCR is not available at our center, presence of CHIKV IgM in CSF from this case was taken as a feature suggestive of CNS invasion of the virus, which is consistent with previous reports of chikungunya encephalitis.[8] Mother when tested for IgM Chikungunya was negative suggesting that illness was a post-natal Chikungunya infection.

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