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Case Report

# NEONATAL CHIKUNGUNYA: A CASE REPORT

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### ABSTRACT

Vertical transmission of Chikungunya has been described in neonates but postnatal Chikungunya has rarely been reported. We report a neonate from Bangalore, who presented with fever, rash and irritability followed by typical visceral and perioral skin hyperpigmentation. Chikungunya infection was suspected and confirmed by serology (IgM) which was positive in the baby and negative in mother. It is important to remember viral infections in the differential diagnosis of suspected sepsis.

Keywords: Chikungunya Virus, Fever, Neonate

### **INTRODUCTION**

Chikungunya is a relatively rare form of viral fever caused by an alphavirus that is spread by mosquito bites from the Aedesaegypti mosquito. The disease was first described by Marion Robinson and W.H.R. Lumsden in 1955, following an outbreak on the Makonde Plateau, along the border between Tanganyika and Mozambique, in 1952. Symptoms appear between 4 and 7 days after the patient has been bitten by the infected mosquito. A high fever and headache occur, with significant pains in the joints (ankles, wrists) and can persist for several weeks. Neonates present at 3-5 days of life with fever, excessive crying, dermatological manifestations like maculopapular rash, nasal blotchy erythema, freckle like pigmentation over centrofacial area, vesiculobullous lesions, apnea, shock, disseminated intravascular coagulation. The time of greatest risk of transmission from mother to fetus appears during birth if mother acquired the disease days before delivery. Viral chikungunya perinatally transmitted leads to encephalitis in newborn (Lahariya and Pradhan *et al.*, 2006).

## CASES

A 5-day old male baby presented to us with history of fever with rash since 1 day. No history of convulsion. The baby was born by normal vaginal delivery at to 24-year old, 2nd gravida mother at 36-weeks of gestation. There were no history of pregnancy induced hypertension, diabetes or antenatal problems or fever with rash or joint pain. The baby cried immediately after birth and was put on the breast feeds within one hour. On the 5th day of life, baby was brought to the hospital for high grade, intermittent type fever along with poor feeding and generalized rash. On admission baby was excessively irritable and continuously crying. Vitals were normal. Anterior fontanelle was not bulging. The fever continued and hedeveloped generalized hyperpigmentation. At admission he weighed 2.4 Kg, had deep generalized hyper pigmentation especially, over faceand nose and also over abdomen, extremities and knuckles. Capillary refilling time was < 3second. Generalized oedema was present especially as cites and pedal oedema. Hepatosplenomegaly present on perabdominal examination. Cardiovascular, respiratory and neurological examination was normal. In view of suspected sepsis Patient was started on piperacillin +tazobactem (300mg/ kg/day) and Amikacin (15mg/kg/day). Sepsis screen and blood culture was taken. In view of oxygen dependency baby was started on 2L of oxygen through nasal prongs. Initial diagnosis was thought to be sepsis/dengue or chikungunya infection.

Investigations revealed hemoglobin was 16.5 g/dL, total leukocyte count of 13,100/mm3 (4000–11000 cells/ mm3) with 67% granulocyte, 31% lymphocyte, 1% eosinophil, 1% monocytes and platelet count was 84000. Serum creatinine 0.5, blood urea 66, Sodium 137, Potassium6.5, Serum bilirubin 12.5mg/dl, s.albumin-2.4. Blood culture was sterile and CRP was negative. Chikungunya virus specific IgM antibodies by IgM capture ELISA test were positive. Dengue serology was negative and CSF analysis was normal. Mothers chikungunya IgM antibody was negative. Baby was conservatively managed with IV fluids and was symptomatically better.

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Figure 1: Showing Hyperpigmentation all over Figure 2: Peeled off Hyperpigmented Skin the Body



### Discussion

Chikungunya is an RNA virus belonging to the Alpha virus genus of the Togaviridae family. It is transmitted to humans by mosquitoes of either the Aedes or Culex genus. The word chikungunya has been derived from a Makonde word meaning "that which bends up". Repeated outbreak has been observed in west, central and southern Africa and many areas of Asia. Chikungunya was first described in Tanzania, Africa in 1952. The virus first appeared in India in 1963, when along with dengue, it caused very extensive epidemic in Calcutta, Madras, and other areas. Chikungunya outbreaks occurred at irregular interval along the east coast of India and in Maharashtra till 1973. Since then the virus has been quiescent (1-3). The incubation period can be 2-12 days, but is usually 3-7 days. It can cause a debilitating illness and symptoms mainly in adult include abrupt onset of fever, chills, headache, and severe joint pain with or without swelling (usually the smaller joints), low back pain, and rash. Our case presented with fever and irritability. Unlike dengue, hemorrhagic manifestations are relatively rare and as a rule shock is not observed in chikungunya virus infection. Neonates present at 3-5 days of life with fever, excessive crying, dermatological manifestations like maculopapular rash, nasal blotchy erythema, freckle like pigmentation over centrofacial area, vesiculobullous lesions, apnea, shock, DIC. Neurological complications such as meningoencephalitis have been reported in patients during the first Indian outbreak as well as the recent French Reunion islands outbreaks (Quatresous, 2006).

Mother to child transmission of chikungunya virus was reported during the recent epidemic in the Reunion Island off the coast of Africa. One hundred and sixty pregnant mothers were infected with chikungunya. Of the thirty three with viremia at the time of delivery, sixteen newborns were symptomatic in the neonatal period (Lenglet et al., 2006). The time of greatest risk of transmission from mother to fetus appears during birth if mother acquired the disease days before delivery. Viral chikungunya perinatally transmitted leads to encephalitis in newborn (Robillard et al., 2006), also reported that transplacental transmission of chikungunya can also occur before 16 weeks and suggest the virus played a direct role in fetal deaths. Diagnosis is made by CHIK IgM and PCR. Most often chikungunya is a selflimiting febrile illness and responded to conservative or supportive therapy (Gerrardin, 2014). The CHIMERE cohort study provides neurocognitive functions of infants infected by maternal-fetal transmission of CHIKV at birth. They found infected children exhibit poorer neurocognitive skills than uninfected peers, as evidenced by lower global developmental quotient scores and diminished specific neurocognitive skills. Thus, incidence of global neurodevelopmental delay (GND) in infected children is

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just over 50% but with a caveat: CHIKV encephalopathy gives the poorest neurocognitive outcome and prostration. Finally, Gérardin P concluded that the neurocognitive outcome of children exposed to perinatal mother-to-child CHIKV infection is poor. Severe CHIKV neonatal encephalopathy is associated with an even poorer outcome. The neurocognitive outcome of infected children is poor and must be monitored throughout childhood to anticipate the psychomotor, cognitive and behavioral therapies. The neurocognitive outcome of children exposed to perinatal mother-to-child CHIKV infection is poor. Severe CHIKV neonatal encephalopathy is associated with an even poorer outcome of children exposed to perinatal mother-to-child CHIKV infection is poor. Severe CHIKV neonatal encephalopathy is associated with an even poorer outcome. In conclusion, this case report shows that viral chikungunya can be transmitted postnatally and clinical presentation is similar to that of septicemia or meningitis.

Our case presented without any neurological symptoms/signs have been conservatively managed. The disease was transmitted postnatally, as most of the case reported are vertical transmission from mother to baby. So, this case report holds clinical significance which shows more scope for further learning.

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