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Case Report

A rare case of vertical Chikungunya transmission presenting with neurological depression and postnatal hyperpigmentation

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ABSTRACT

Chikungunya (CHIKV) is a virus that spreads through mosquito bites. Neonatal chikungunya is rare, and congenital transmission whether occurring when the virus crosses the placenta or is transmitted perinatally is even more uncommon. Due to limited awareness and overlapping symptoms with other neonatal infections or metabolic conditions, congenital chikungunya may be underdiagnosed. The increased risk of vertical transmission has been linked to maternal viremia near delivery. About 50% of neonates with CHIKV symptoms develop neurodevelopmental delay. We report a rare case of vertically transmitted chikungunya in a neonate presenting with repeated apnoea's and generalised hypotonia.

Keywords: Chikungunya, Neonates, Vertical transmission, Mother to child

INTRODUCTION

Chikungunya virus an RNA virus belonging to genus Alphavirus and family Togaviridae. It is an RNA virus. Chikungunya is transmitted primarily by bite of mosquitoes of the Aedes genus and rarely through vertical transmission.

First case of mother-to-child chikungunya virus transmission (vertical transmission) was first reported during a large epidemic on La Reunion island in June 2005.[1,2] When maternal infection coincides with the intrapartum period, the likelihood of vertical transmission rises significantly, reaching about 48.7%.[3]

Neonatal Chikungunya is rare but may present with severe or atypical manifestations such as irritability, fever, poor feeding, bleeding, apnea, diffuse limb edema, various skin manifestations, and encephalopathy.[4] The clinical features often resemble those of early-onset neonatal sepsis, making accurate diagnosis difficult.[5]

CASE REPORT

Preterm (34+1 wks.) male baby was born via C section in view of premature labour pains. The baby cried immediately at birth and was admitted to NICU in view of prematurity. The baby was placed on continuous positive airway pressure (CPAP) and started on intravenous antibiotics- Piperacillin-tazobactam, Amikacin, Meropenem for suspected neonatal sepsis. There were repeated episodes of Apnoea after which the baby was referred to our hospital at Day of Life 5. When we received the baby in NICU on Day 5 of life (DOL-5), he was lethargic, hypotonic and had an episode of apnoea. There were bullous lesions and oedema over extremities. The baby was subsequently placed on CPAP (PEEP-6, FiO2- 35%), and a loading dose of caffeine was administered, followed by maintenance therapy. Baby was initially started on I/V fluids. As septic markers were elevated (CRP-16.9mg/dL, PCT–2.79ng/ml) Intravenous antibiotics - Vancomycin, Meropenem, and Colistin were started after blood cultures were drawn.

As the baby experienced recurrent episodes of apnoea even while on CPAP support, the mode of ventilation was escalated to non-invasive mechanical ventilation (NIMV), with settings of PEEP-6, PIP- 15, FiO2- 21%, RR- 40/min). Following initiation of NIMV, the baby had no further episodes of apnoea and was subsequently weaned to CPAP on day 3 of admission. On day 3 of admission, enteral feeds were initiated and gradually advanced.

After 5 days, with blood culture showing no growth, Inj Colistin was stopped. A trial off CPAP was performed on day 6 of admission, which was well tolerated, after which the baby was maintained on room air. In view of persistent lethargy and hypotonia, a brain MRI was performed.

MRI findings: MRI of the brain revealed symmetrical areas of restricted diffusion involving the bilateral periventricular white matter, centrum semiovale, internal capsules, corpus callosum, and brainstem along the bilateral corticospinal tracts.? Metabolic/infective encephalopathy (Figure 1).

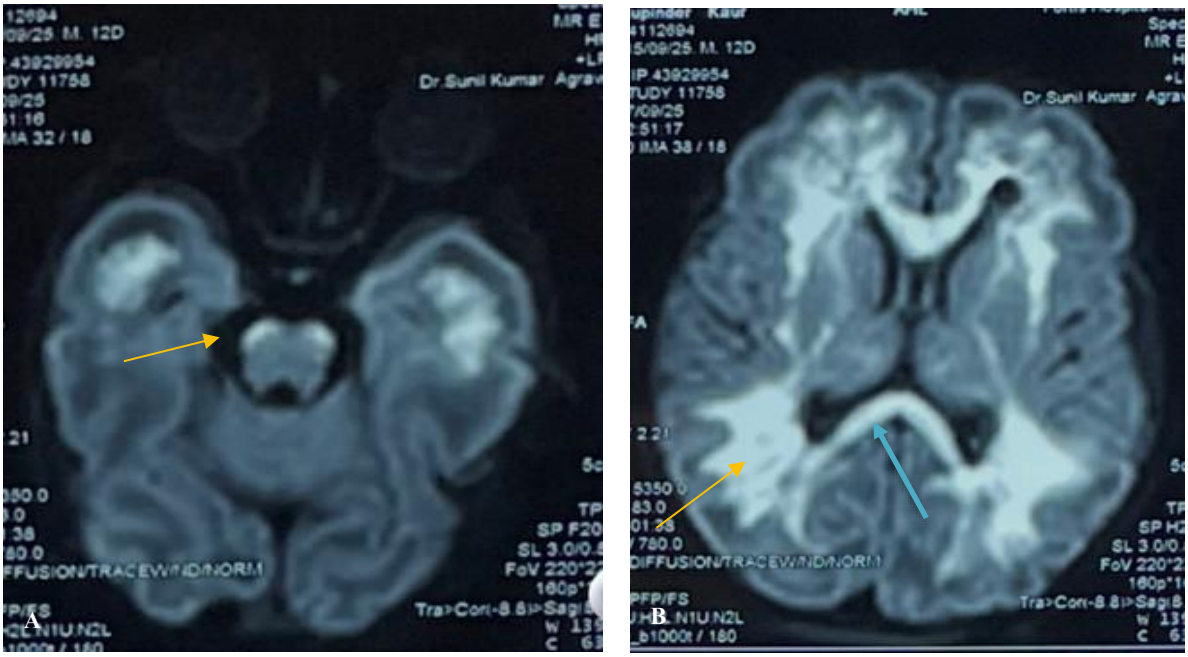


Figure 1: DWI Showing Symmetrical areas of restricted diffusion seen in ventral brain stem along bilateral corticospinal tracts (arrow in 1A), bilateral periventricular white matter (yellow arrow in 1B), centrum oval, internal capsule, Corpus callosum (blue arrow in 1B).

On the same day, cerebrospinal fluid (CSF) examination was normal, and the blood culture sent at admission (day 5 of life) showed no growth. Following these results, intravenous antibiotics were stopped.

Following the MRI findings, a metabolic workup was performed, including thyroid function tests, creatine kinase levels, newborn screening, and assessment of ACTH and cortisol levels. Considering persistence of hypotonia, genetic evaluation for myotonic dystrophy and Prader–Willi syndrome was undertaken. All results were within normal limits.

On day 6 of admission, the baby developed generalized hyperpigmentation. An endocrinology consultation was sought, and serum cortisol and ACTH levels were assessed, both of which were within normal limits. Over the next 2–3 days, the hyperpigmentation intensified, becoming more prominent periorally and over the tip of the nose a characteristic “chick sign” (Figure 2 and 3).



Figure 2: Hyperpigmentation periorally and over the tip of the nose, a characteristic “Chick Sign”.

The baby also had persistent thrombocytopenia since admission. In view of this combination of findings, a blood sample was sent for Chikungunya IgM antibodies, which returned positive.

Following this result, the mother was re-interviewed regarding her antenatal history, at which point she recalled having experienced fever and joint pain two days prior to delivery. Maternal testing for Chikungunya IgM antibodies was subsequently performed and also returned positive.

Table 1: Serial investigations.

INV	20/09/25	23/09/25	01/10/25	08/10/25	13/10/25
HB (g/dl)	12.9	11.1	12.2	12.1	7.5
TLC	11500	18130	8700	15220	15200
DLC %(N/l)	93/4	76/16	14/66	56/26	52/33
Platelets	114000	55000	75000	147000	325000
CRP (mg/l)	16.9	136	31	17.7	1.7
BUN/CR (mg/dl)	16/0.7	16/0.60	14/0.2	15/0.2	
Na/K/Cl (mmol/l)	140/5.78/100	133/4.35/98	143/4.18/108	139/4.3/102	
Ca/ Mg/ PO4	7.5/2.6/8.2		9.6/-/7.4		
Total protein	3.0		4.2		
Albumin	2.3		2.6		
TSB/DSB	7.11/ 0.98		1.08/0.5		
SGOT	109		74		
SGPT	9		75		
Alkaline phosphate	133		205		
LDH	1517		1662		
ACTH			<1.5		
Cortisol			11.47		
Procalcitonin			5.07	1.19	
T3/T4		2.06/1.34			
TSH		10.43			
Creatine kinase		2169			
Procalcitonin	2.79	1.520			
Blood culture	No growth			No growth	
Myotonic dystrophy type 1-not detected					
Prader Willi Syndrome MS PCR-not detected					
Newborn screening test - normal					

Table 2: Cerebrospinal fluid examination.

	CSF		
Date	24/09/25	26/09/25	09/10/25
Appearance	Hazy	Clear	Clear
Colour	Pale Yellow	Pale Yellow	Colourless
Glucose	31	42	41
Protein	148	148.9	90.8
TLC	40	60	40
Lymphocytes	20	60	20
Mono nuclear cells	15	40	80
Gram Stain	No microorganisms detected	No microorganisms detected	No microorganisms detected
Culture	No growth	No growth	No growth

Based on these findings, a diagnosis of congenital chikungunya infection was established, and the neonate was managed with supportive care.

By day 13 of admission, the baby was gradually started on spoon feeds, which had previously been deferred due to persistent hypotonia, lethargy and poor feeding efforts. The feeds were slowly advanced as tolerance improved,

and the neonate eventually accepted spoon feeds well. The baby was discharged after 26 days of hospital stay.



Figure 3: Generalised hyperpigmentation, more prominent at the tip of the nose and periorally.

At the time of discharge, hypotonia, hyperpigmentation, and mild feeding difficulties were still present, though the overall clinical condition had improved. The baby continues to be followed up in the outpatient department for ongoing monitoring and developmental assessment.

DISCUSSION

Chikungunya fever (CHIKF) is endemic in India and has caused multiple outbreaks since its re-emergence in 2005. The most recent major outbreak occurred in 2016, resulting in 64,057 confirmed cases. These recurring outbreaks are largely attributed to the reintroduction of the virus from outside the country along with the circulation of newly emerging viral strains within India.[6] Chikungunya remains 3rd most common vector-borne disease in India. Chikungunya is a viral disease transmitted by the bite of infected Aedes mosquitoes. In adults, the illness can be debilitating, presenting with symptoms such as fever, headache, fatigue, nausea, vomiting, muscle pain, rash, and severe joint pain.[7]

Maternal-to-child transmission of Chikungunya is rare and was first reported during the 2005–2006 outbreak on La Réunion Island.[2] Intrapartum period is the critical time for transmission to neonate.[2,8] The placental barrier is generally effective in preventing trans placental CHIKV transmission. However, vertical transmission can occur near term, when high maternal viremia coincides with breaches in the placental barrier caused by uterine contractions during labor.

The rate of vertical transmission is approximately 48.7% among neonates exposed during labor.[2] Maternal infection is considered intrapartum when symptoms appear within the window of two days before to two days after delivery.[3] Importantly, transmission occurs regardless of the mode of delivery and is almost always associated with maternal viremia in the peripartum period.[1]

Our case highlights the key clinical features observed in a neonate with vertically transmitted chikungunya infection. Vertical transmission was confirmed by the presence of serum IgM antibodies in both the mother and the new-born. Notably, IgM antibodies can appear as early as two days after infection and may persist for more than three months, supporting their diagnostic utility in early neonatal presentations.[9]

The neonate exhibited multiple clinical manifestations, including unexplained apnoea, hypotonia, hypoactivity, refusal to feed, vesicobullous lesions with oedema in limbs, and extensive generalized hyperpigmentation. Among these, the presence of diffuse hyperpigmentation, particularly the characteristic “CHIK sign” (nasal hyperpigmentation) and perioral darkening that developed on day 11 of life, served as important clues directing us toward the diagnosis of chikungunya.

Ferreira et al previously described these striking pigmentation patterns (CHICK Sign) as hallmark features of neonatal chikungunya.[1] Without such distinctive findings, the clinical picture could easily have been mistaken for early-onset neonatal sepsis or an underlying metabolic disorder. Hyper melanosis in chikungunya is believed to represent a form of post-inflammatory hyperpigmentation mediated by increased intraepidermal melanin retention following viral injury.[10] Typically, these pigmentary changes become evident by the end of the second week of life.[11]

Neurological involvement in our patient manifesting as hypotonia, hypoactivity, feeding difficulty, and recurrent apnoea was supported by MRI findings suggestive of encephalopathy. Chikungunya virus can disseminate to the central nervous system through areas with incomplete blood–brain barrier protection.

Proposed mechanisms of neuronal injury include viral invasion of the choroid plexus and leptomeninges leading to disrupted neuronal migration, as well as microglial activation causing inflammatory damage.[12] Persistent thrombocytopenia was another significant laboratory finding in this case. Gerardin et al have reported an association between the severity of thrombocytopenia and severe neonatal chikungunya, highlighting the importance of vigilant hematologic monitoring.[2]

Currently, there is no specific antiviral therapy for chikungunya; thus, management remains entirely supportive. Optimal care involves regular monitoring of vital signs, assessment of skin and neurological status, ensuring adequate hydration and nutrition, and meticulous fluid management.[1,3] Early recognition of these characteristic clinical features is essential to guide appropriate supportive care and reduce the risk of complications.

CONCLUSION

This case underscores the importance of considering chikungunya as a differential diagnosis in neonates presenting with fever, characteristic hyperpigmentation, and encephalopathy, particularly in regions where the virus is endemic. Early recognition is essential, as neonatal chikungunya can closely mimic other conditions such as sepsis or metabolic disorders.

A thorough maternal history is crucial in identifying possible perinatal infection; in dengue-endemic areas, chikungunya should be suspected in cases of early-onset neonatal sepsis that do not respond to conventional antibiotics, especially when mothers report flu-like symptoms around the time of delivery.

Although there is no specific antiviral treatment and management remains primarily supportive, timely diagnosis allows for appropriate monitoring of vital signs, hydration status, skin manifestations, and overall systemic condition, thereby reducing the risk of complications. Long-term follow-up is vital due to potential sequelae, including neurocognitive impairment, microcephaly, and cerebral palsy. This case highlights the need for heightened clinical awareness to improve outcomes in affected neonates.

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