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CASE REPORT

Vertical Transmission of Scrub Typhus in Newborn: A Case Report

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Introduction

Scrub typhus is an acute febrile mite-born rickettsial infection caused by *Orientia tsutsugamushi* (formerly called *Rickettsia tsutsugamushi*). This infection is very uncommon in neonates. We report a case of 8-day-old newborn presenting with clinical features mimicking severe sepsis but were subsequently diagnosed with primary scrub typhus infection. The timely treatment resulted in dramatic response and complete recovery.

India being in the part of famous Tsutsugamushi Triangle, entire India is endemic for scrub typhus. Scrub typhus is the most prevalent rickettsial infection in India which is followed by Indian tick typhus followed by murine typhus.

Case Report

An 8-day-old female infant was admitted in Neonatal Intensive Care Unit of our hospital with history of high grade fever, abdominal distension and difficulty in breathing since last two days and decreased oral acceptance and vomiting for one day. She was delivered at CUSMC medical college & hospital in Surendranagar through normal vaginal delivery with a birth weight of 3 kg. The antenatal and intrapartum periods were uneventful. She was kept mother side and discharged on second day of life and was exclusively breast fed before the illness.

At admission, the baby was febrile and irritable, had pallor with few ecchymotic rashes present over abdomen and neck (Figure 1 and Figure 2). Her weight was 2.9 kg at admission. The axillary temperature was 38.9 °C, heart rate was 186/min, respiratory rate was 66/min and capillary refill time was less than three seconds.

A provisional diagnosis of late onset sepsis with meningitis was made and she was started on intravenous antibiotics and supportive measures to control fever and maintain adequate perfusion.

Investigations revealed anaemia (Hb 10.5 gm/dL), leukocytosis (40,800/cmm with 65% neutrophils) and thrombocyptopenia (platelet count- 44000/cmm). C-reactive protein (98.78 mg/dL, normal value less than 5 mg/dL) were highly positive. Kidney function

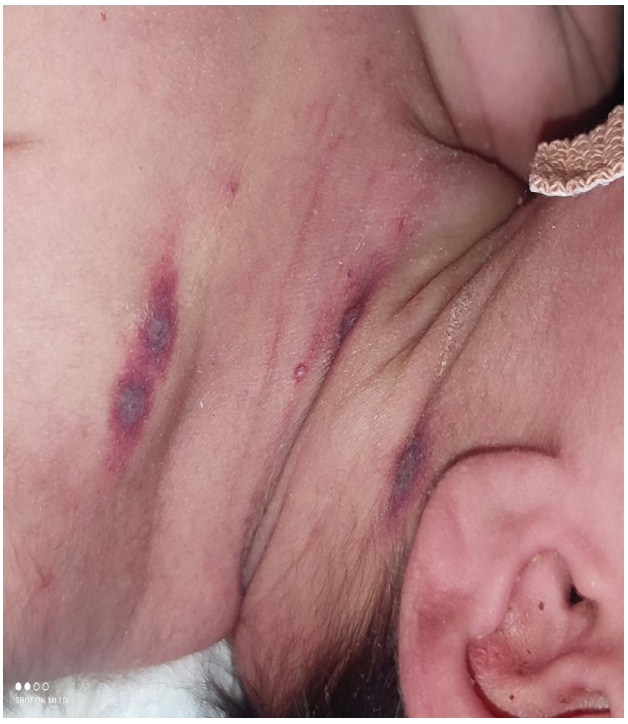

Figure 1: Infiltrative erythema with a black eschar in the center on day of admission.

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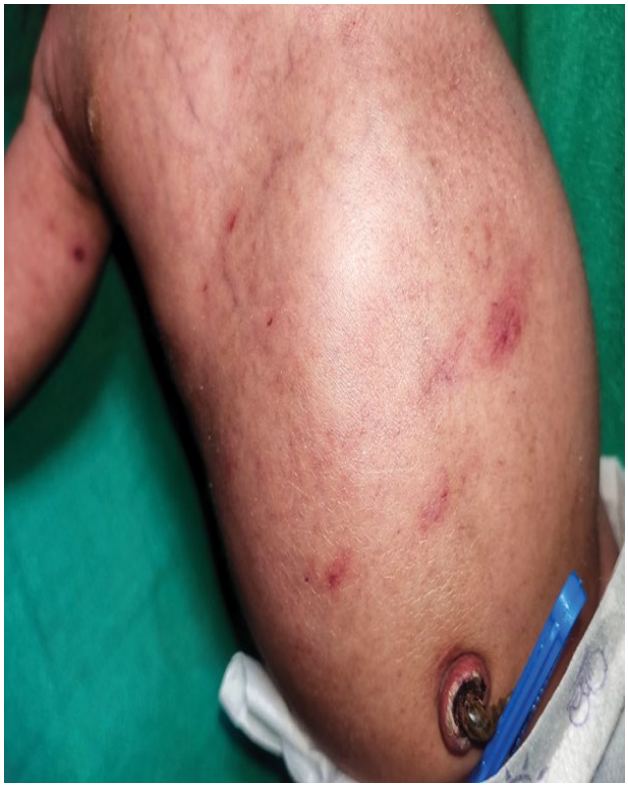

Figure 2: Infiltrative erythema with a black eschar in the center on day of admission.

tests serum creatinine (1.83 mg/dL, normal biological reference interval 0.04-0.33) serum Urea (211.23 mg/dL). Cerebrospinal Fluid (CSF) examination showed aseptic meningitis (total cell count of 18 cells, with 97% lymphocytes, protein of 69 mg/L and sugar of 77 mg/L with sterile culture). The blood (both BACTEC and fungal) cultures showed no growth. Dengue and malaria were ruled out by NS1 (Non-Structural Protein 1) antigen and rapid malarial antigen test and peripheral smear examination. IgM antibody for TORCH (Toxoplasmosis, Other agents, Rubella, Cytomegalovirus and Herpes Simplex) group of infections was negative in both the mother and the neonate.

2D echo was done suggestive of ACHD: Two small ostium bsecundum ASD with left to right shunt. Normal biventricular function. We started Oral Furosemide (1 mg/kg/day).

On third day of admission, due to deteriorating b clinical status and development of respiratory distress and shock, the infant was mechanically ventilated by HFNC (High flow nasal cannula) and given inotropic support. Platelet concentrate were administered. The antibiotics were upgraded. The chest radiograph revealed presence of bilateral pleural effusions and ultrasound of abdomen revealed ascites. The infant, however, continued to have high grade fever, anasarca and persistent thrombocytopenia (Figure 3 and Figure 4).

On 8th day of admission, we reevaluated the history provided by the family and it was revealed that mother

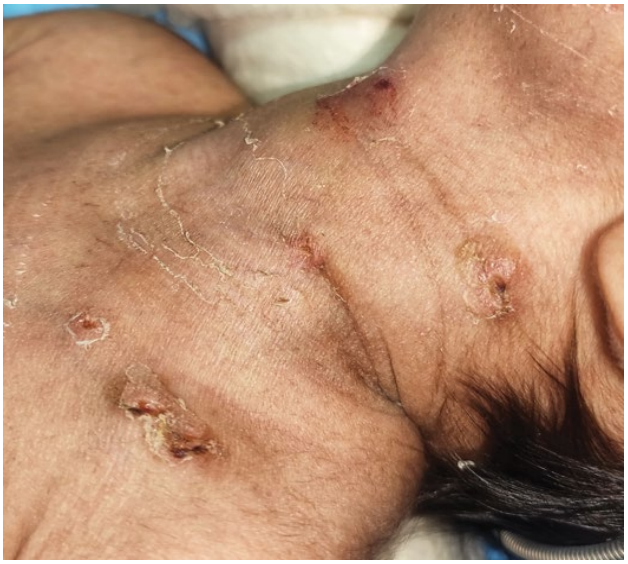

Figure 3: Skin lesions on day 5th with black Eschar formation at sites of chigger bites.

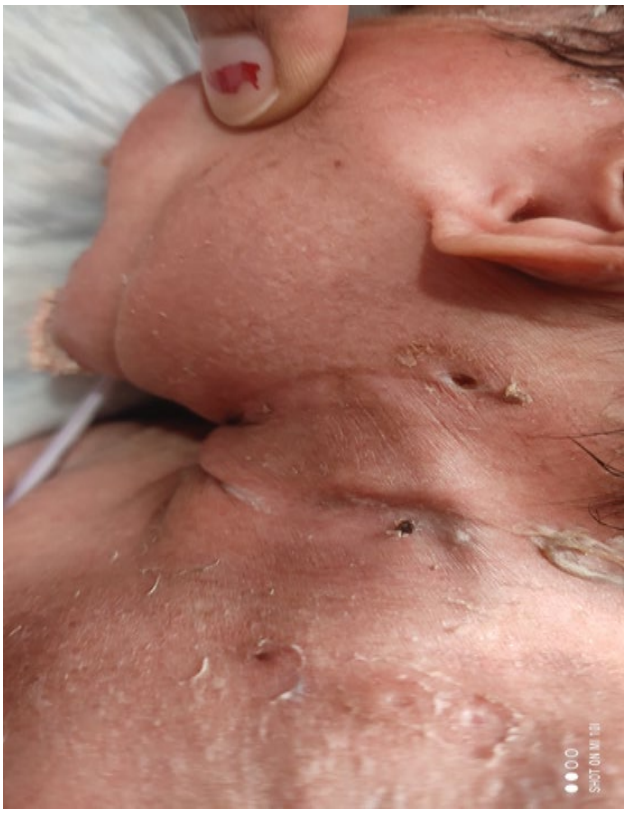

Figure 4: Skin lesions on day 7th with black Eschar formation at sites of chigger bites.

had complain of fever 10 days before delivery of baby living in village with skin lesions a week before the onset of illness in the neonate. We sent the rickettsial serology by card test (IgM ELISA) and it came positive for scrub typhus. The Weil Felix Test (Latex agglutination) (Table 1 and Table 2).

Oral Azithromycin (10 mg/kg/day in single dose) was initiated in the patient for 5 days. There was defervescence of fever with clinical improvement within 48 hours. HFNC and inotropic support was tapered and

Table 1: Weil felix test of baby.

Proteus Antigen	Reaction	Titre (in dilution)
OX 19	Non-Reactive	
OX 2	Non-Reactive	
OX K	Reactive	**1:40**

Table 2: Weil felix test of mother.

Proteos Antigen	Reaction	Titre (in dilution)
OX 19	Reactive	**1:160**
OX 2	Reactive	**1:8**
OX K	Reactive	**1:8**

withdrawn over next two days. The body cavity effusions started resolving. The platelet counts also normalised over one week. The antimicrobial was given for a total duration of 10 days. At the time of discharge, the infant had recovered fully and was on exclusive breast feeds.

Discussion

Scrub typhus is most prevalent human rickettsial infection caused by *Orientia tsutsugamushi* transmitted by the bite of an infected mite chigger. It has been observed to be an important emerging infection in India.

Orienta tsutsugamushi survives in the wild involving trombiculid mites (principal vectors) and other vertebrates (small mammals and birds), humans being the accidental hosts [1]. The infection is usually seen in people who come in contact with vector chiggers due to their occupational activities and in those who live in wide range of vegetation type from scrubs (terrain between woods and clearings), primary forests to gardens and beaches [2]. Scrub typhus infection has been rarely reported in the neonatal period [3-5].

The organism enters the human body and targets endothelial and reticulo-endothelial cells, leading to diffuse vasculitis and perivasculitis. At onset of fever, an eschar often develops at the site of the chigger bite. The typical lesion of scrub typhus begins as a red, indurated lesion about 1 cm in diameter; in eventually vesiculates, ruptures, and becomes covered with a black scab. Vasculitis has been implicated to be the basic pathogenetic mechanism responsible for skin rash, microvascular leakage, oedema, tissue hypoperfusion and end-organ ischemic injury. Widespread tissue hypoxic-ischemic insult can result in multi-organ dysfunction leading to high incidence of morbidity and mortality in untreated cases.

The incubation period of *Orienta tsutsugamushi* is 1-2 weeks.

The clinical manifestations may vary from mild undifferentiated fever to a severely potentially fatal disease leading to Multi Organ Dysfunction Syndrome (MODS).

The presentation of scrub typhus in children is non-specific and hence requires high index of suspicion for timely diagnosis. Due to wide variation in clinical presentation, the diagnosis of scrub typhus is often missed or diagnosed late.

The clinical manifestations of scrub typhus in neonates remain unexplored, perhaps due to low clinical suspicion and overlapping signs and symptoms in this age group.

Our case was an 8 day-old neonate who presented with features suggestive of severe sepsis. She did not respond to standard management protocol for sepsis and all the investigations including blood and CSF cultures were sterile.

This prompted us to investigate her for unusual infections at this age. After ruling out vector - borne diseases like dengue and malaria and congenital infections (TORCH), we sent the serology (IgM ELISA) for typhus which came out to be positive with vertically transmission of infection from mother to baby.

Conclusion

Our case highlights that a neonate presenting with high grade fever, respiratory distress, skin lesions, edema, thrombocytopenia and shock and showing no evidence of sepsis or congenital infections should always be investigated for scrub typhus especially in endemic regions of the country.

The diagnosis is essential as the infection is treatable with antibiotics and if untreated can lead to MODS and mortality.

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