

NEONATAL SCRUB TYPHUS: CASE REPORT

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Article Received on
27 April 2018,

Revised on 17 May 2018,
Accepted on 07 June 2018

DOI: 10.20959/wjpr201812-12662

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INTRODUCTION

Also known as tsutsugamushi disease.^[1] this acute febrile disease is caused by orientia tsutsugamushi [Japanese word tsutsuga (“dangerous”), mushi (“bug”)^[1], which is a small gram negative, obligate intracellular organism whose polysaccharides bear an antigenic relationship to proteus OX-K, which is thus used in serologic tests to confirm scrub typhus.^[1] O. tsutsugamushi is transmitted to humans by the bite of the larva of trombiculid mites (chiggers) which are almost microscopic, often brilliantly colored (red). Infected chiggers are found particularly in areas of heavy scrub vegetation during wet season, (therefore this disease has also been called river/flood fever^[1]). when mites lay eggs.^[2] usually June through November.^[3] The word “scrub” was applied because of the type of vegetation that maintains the chigger-mammal relationship even

though other regions also support rodents and mites.^[1]

Scrub typhus is endemic to a part of the world known as the “tsutsugamushi triangle”. In India the presence of scrub typhus has been known for several years. The disease is widely spread all over the country and was reported in several states. Though scrub typhus in pregnancy is uncommon. Transmission via bite of the larval stage (chigger) of trombiculid mite (lepto trombidium) serves as both vector and reservoir. Transmission is most commonly trans ovarian and regurgitation of infected saliva during feeding.

Case

7 days post dated female baby weighing 3.55 kg with normal Apgar score born to a primi mother 22 years was admitted to NICU with history of vasambo application over the

abdomen with oral grape water on 23rd day of life for cold 2 days abdominal distension 1 day breathing difficulty 1 day with reduced activity.

Investigations

23-10-2015- haemoglobin 12.5 gm/dl, TC 14500, RBC 4.2, platelet 1.81 lacs, CRP positive 96 mg, peripheral smear normal

28-10-2015 - blood culture no growth

30-10-2015 – ET Tube culture gram positive cocci and gram negative bacilli. E coli scanty growth. haemoglobin 817 gm/dl, TC 11400, RBC 2.9, platelet 1.2 lacs, peripheral smear Normocytic normochromic cytic normal chromic anemia with thermocyto penia

1-11-2015- calcium 8.3 mg

4-11-2015- CRP 48 mg, echo pattern foreman ovale 2.5 m with left risen, outside source scrub thypus IgM+ve, USG abdomen- ascites, x ray chest and abdomen- bilateral full efficient with epitomegaly.

Course in the hospital

Baby was kept NPO and oxygen by hood injection piptaz and injection amikacin given for 5 days. baby was put on ventilator therapy and ABG, then was normal in view of shock, NS bolus 10 ml per kg Infusion with dopamine. In view thermocyte apenia abnormal PT and APTT and suspected DIC, platelet transfusion and FFP given. In view of E coli growth antibiotics changed to injection meropenum and Vancomycin given. In view of ascites Hepatosplenomegaly with bad child rearing practises, neonatal scrub typhus was suspected and Doxycycline started and given for 10 days. Abdominal distension, liver span reduced. maintaining saturation. Baby was extubated and kept on CPAP and weaned from oxygen and started on breast feeds and discharged after 16 days of admission with a weight 3.2 kg.

DISCUSSION

Tsutsugamushi disease is caused by orientia tsutsugamushi. Organisms enter human body, multiply locally then enter the bloodstream and reticulo-endothelial system. Vasculitis is the basic mechanism responsible for skin rash, micro vascular leakage, edema and tissue hypo perfusion and end-organ ischemic injury.^[3,4] There is formation of thrombi leading to tissue infraction and hemorrhagic necrosis. Inflammation and vascular leakage cause interstitial pneumonitis, pulmonary edema, cerebral edema and meningoencephalitis and serositis. Infection of endothelial cells leads to procoagulant activity and coagulation factor consumption, platelet adhesion and leukocyte emigration producing disseminated

intravascular coagulation as seen in our case. Incubation period of the disease ranges between 1-30 days.^[4] There are 3 possible route of infection in neonate's transplacental infection, perinatal blood born transmission and postnatal infection. It has been well documented that an elevation of IgM. Antibodies during the neonatal stage indicates an intrauterine infection.^[5] Most newborn present with respiratory distress, fever, decreased oral intake, abdominal distension, hepatosplenomegaly, seizure and lethargy mimicking neonatal septicemia as seen in our case and also in cases reported. However eschar formation was not reported in that neonatal cases Scrub typhus, a maculopapular rash is present less than 30%. It affects almost all systems neonates may develop complications such as shocks, seizures, encephalopathy, pleural effusion, pneumonitis and respiratory failures as seen in our case. On hemogram, total leukocyte and platelet count mostly normal although thrombocytopenia in one quarter to one third patients^[4] thrombocytopenia was noted in our case and cases reported.^[6] Hyponatremia, hypoalbuminemia, elevated hepatic transaminases and elevated blood urea are biochemical findings. Blood cultures are generally found with *E coli* growth in our case.

Diagnosis mostly based on history, clinical feature and serological marker. Antibodies mediated test like microimmunofluorescence, immunoperoxidase assay, latex agglutination and indirect hemagglutination, ELISA, dot blot immunoassay and Weil-felix test. Other investigation includes Immunohistochemistry, isolation of organism, PCR : detects rickettsial DNA in whole blood, buffy coat fraction or tissue specimen. Laboratory findings such as WBC counts $>10,000/\text{mm}$ and serum albumin level $\leq 3.0 \text{ g/dl}$.^[7]

Measles, dengue fever, malaria, meningococemia, typhoid, leptospirosis, infectious mononucleosis, collagen vascular Kawasaki disease are important differentials. Treatment includes antibiotics like tetracyclines, chloramphenicol, macrolides and fluroquinolones. Doxycyclines is the drug of choice. Long course of tetracycline to newborn and young children leads teeth related problems. Use of quinolones during neonatal period may cause problems related to cartilage and bone. however short term use has not demonstrated such problems. Azithromycin is a safer alternative. As per DHR-ICMR guidelines for diagnosis and management of rickettsial disease in india recommendation include (a) Doxycycline in the dose of 4.5 mg/kg body weight/day in two divided doses for children below 45 Kg or (b) Azithromycin in the dose of 10 mg/kg body weight for five days. Good supportive therapy is

very important. Nested PCR TEST is known to be 100 times more sensitive than performing single PCR.^[8,9,10]

Prognosis

Case fatality rate for untreated classic cases is 7%. Poor prognostic factors include requiring care in intensive care unit or high APACHE-II scores and absence of eschar (making diagnosis more difficult).

Prevention

It is based on avoidance of chiggers that transmit *O. Tsutsugamushi*, accomplished by insect repellents and by the use of protective clothing impregnated with benzyl benzoate. Vaccines were developed and tested. However no single antigen has been identified that induces protection against all of the antigenically diverse strains of *O. tsutsugamushi*.^[1]

Scrub typhus in pregnancy

Doxycycline, is associated with fetal risk and hence contraindicated. Azithromycin seems to be an effective agent against scrub typhus because it efficiently penetrates polymorphonuclear leukocytes and macrophages, which are target cells for *O. Tsutsugamushi*.^[11]

CONCLUSION

Scrub typhus should be kept in differentials in newborns with fever and skin papules/eschar.

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