RELAPSE OF PLASMODIUM VIVAX LEADING TO CONGENITAL MALARIA: TREATMENT COULD NOT PREVENT TRANSMISSION FROM MOTHER TO CHILD

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ABSTRACT

Congenital malaria is an established though infrequent disease entity. So far, 300 cases are reported in literature which are mostly caused by transplacental transfer of parasitized erythrocyte during pregnancy or at the time of delivery from an undiagnosed or untreated mother. We report a case where mother inspite of being adequately treated for Plasmodium vivax during her antenatal period, couldn’t prevent transplacental transmission to the neonate. The probable mechanism was relapse from hypnozoites which were not eliminated due to restrictions on use of Primaquine during pregnancy. The case brings in forth the importance of early neonatal screening in such situations of Plasmodium vivax malaria in mother irrespective of treatment or recovery. The case also emphasizes the need to evaluate the role of chemoprophylaxis in pregnant mothers of endemic regions. Presence of parasite in the microscopical section of the placenta, parasite illustrated within 7 day of life in newborn’s peripheral blood smear and that of maternal blood smear after delivery and IgG anti malarial antibodies in infant’s blood are indicators of congenital malaria. The onset, spectrum of clinical presentation, and prognosis are however diverse.

Key words: Congenital malaria, plasmodium vivax, relapse

INTRODUCTION

Congenital malaria is an infrequent disease mostly caused by transplacental transfer of parasitized erythrocyte during pregnancy or at the time of delivery. The disease may present with diverse clinical presentations. Presence of parasite in the microscopical section of the placenta, or newborn’s peripheral blood smear, IgG anti malarial antibodies in infant’s blood and similar findings in mother indicates congenital malaria. A 21 days old male infant presented with progressive pallor, fever, abdominal distension, poor feeding & lethargy. His mother had Plasmodium vivax infection one
Relapse of Plasmodium vivax causing congenital malaria

and a half month before delivery for which she received 3 day course of Chloroquine and became asymptomatic. However the infant’s as well as mother’s blood smear revealed trophozoites of Plasmodium vivax and Anti-malaria IgG. Infant was treated with Chloroquine and mother by Chloroquine and Primaquine. Both were completely cured and doing well in follow up.

CASE REPORT

A 21 days old male infant weighing 3 kg was admitted to pediatric ward for the evaluation of progressive pallor for last 10 days and fever, abdominal distension, poor feeding, lethargy for last 7 days. There was no history of bleeding from any sites, jaundice or blood transfusion; no respiratory distress, diarrhea, vomiting, convulsion or any other significant complains. He was a full term child born to a non-consanguineous marriage with birth weight 2.5 kg, undergone normal vaginal delivery at hospital and cried after birth. He was exclusively breast fed since birth and was absolutely normal before this illness.

Her mother was 26-year old, primigravida of urban residence. She had taken 2 doses of tetanus toxoid and regular iron-folic acid supplements as per recommendations. Throughout the antenatal period she had no exanthematous disease or other illness and her routine investigations were within normal limits. One and half month prior to her delivery she developed high grade fever with chill & rigor. She was detected to have Plasmodium vivax malaria and completed a course of Chloroquine for 3 days. She was thereafter afebrile and had no further ante-partum or post-partum complaints. Peripheral blood examination of mother after treatment failed to reveal any parasitemia.

There was no significant family history of any disease or blood transfusion. On physical examination there was severe pallor, mild icterus. The child had body temperature 38.5 degree centigrade, was conscious alert, normal cry, reflex, activity; capillary refill time 2 sec, pulse rate of 130/ min, respiratory rate 42/ min, blood pressure of 80/50 mmHg and a head circumference of 36 cm. Hepato-splenomegaly was present (liver 5 cm below costal margin, spleen was 8 cm below costal margin). There was no ascites. Other systems were normal on examination.

Laboratory tests revealed - Hb 5.2 gm%, erythrocyte-1.87 million/cu mm, WBC count 19,900/ cu mm, neutrophil-52%, lymphocyte-44% and platelet-1 lakh/cu mm, few toxic granule, 5% band cell. PCV 17.5%, MCV-93.6 fl, MCH-27.8 pg, MCHC-29.7 g/dl, RDW(CV)-20.8%. Corrected reticulocyte count was 3.5%. C reactive protien (CRP) was 56.2 mg/dl (normal <5 mg/dl). Serum bilirubin 4.5 mg/dl (indirect fraction 3.8 mg/dl), SGPT-20 U/L, SGOT-35 U/L, ALP-558 U/L; total protien 5.6 g/dl, albumin 3.7 g/dl, urea-15 mg/dl, creatinine-0.8 ng/ml. The remaining biochemistry parameters and metabolic values were within normal limits. Blood culture, urine RE/ME and Culture as well as chest X-ray were normal. TORCH screening was also negative.

HPLC of both parents was normal. Thick and thin blood film microscopy of the infant revealed P.vivax trophozoites(Figure 1) with parasitaemia 2%. The mother’s peripheral blood smear was also positive for P.vivax. Both mother and baby had positive antimalarial IgG antibody in blood.

The infant was administered oral chloroquine 10 mg/kg on the first and the second day followed by 5 mg/kg on the third day along with PPC transfusion. Mother was also
Relapse of Plasmodium vivax causing congenital malaria

treated with Chloroquine along with primaquine after G-6-PD assay. Four days after treatment peripheral parasitaemia was completely cleared from the child’s as well as mother’s blood and the CRP came down to 10.5 mg/dl. The infant was discharged on day five with normal red and white blood cell counts, haemoglobin value, platelets count, biochemical profile, and normal inflammatory index (CRP < 5 mg/dl). Both are doing well in follow up.

Figure 1. Infant’s Peripheral blood smear showing Plasmodium vivax trophozoites.

DISCUSSION

Malaria is endemic in different parts of the world, specially in the tropics, causing substantial morbidity & mortality in all age groups. Pregnant women are more susceptible to malaria than non-pregnant women, especially in first and second pregnancy by virtue of the hormonal and immunological changes leading to immuno-suppression and loss of acquired immunity to malaria. Due to similar reasons parasitemia tends to be 10 times higher in pregnancy. Malaria infection is more frequent and serious during the first pregnancy, as is the occurrence of congenital malaria. Placenta is a new organ in the primigravida and allows the parasites to by-pass the existing host immunity. Development of placenta specific immunity may thus explain the decreased susceptibility of malaria in multigravida. Congenital malaria was first described in 1876. Congenital malaria, defined as the presence of malaria parasites in the erythrocytes of newborns aged less than 7 days, was considered rare in endemic areas until recent studies started reporting high prevalence rates. In most cases, the mother has been infected with the parasite during pregnancy.
Congenital transmission of malaria parasites occur due to maternal transfusion into the fetal circulation either at the time of delivery or during pregnancy, direct penetration through the chorionic villi, or penetration through premature separation of placenta. The transmission to the fetus is however lower than expected and possibly reflect the physical barrier of the placenta to infected red cells, the passive transfer of maternal antibodies, and the unfavourable environment offered by foetal erythrocytes for plasmodial replication due to their foetal haemoglobin composition and low free-oxygen tension. Between 1% and 4% of pregnant women with overt malaria attacks have babies with congenital malaria and in endemic countries these are mostly caused by P. falciparum. The majority of studies from endemic areas have focused on falciparum malaria. Although placental infections with P. falciparum have been described, the role of other species especially P. vivax, which exists in good proportion in some countries, including India, is not well studied. In a study conducted in Central India, although 2.2% (4/182) women were placenta positive for P. vivax or mixed infection, none of the infants developed parasitaemia up to 6 months of age. Congenital malaria in P. vivax has been however described from Sri Lanka, Italy, Singapore, Thailand. P. vivax is common in Asia and, unlike P. falciparum, does not cytoadhere in the placenta, yet, is associated with maternal anaemia and low birth weight.

At birth 29% of newborns suffering from congenital malaria have parasitemia. The prenatal and neonatal mortality may vary from 15 to 70%. Normally symptoms occur in the newborn 10 to 30 days postpartum. However, the disease can be seen in a day-old baby or be delayed for weeks or months. The clinical features of neonatal malaria include anemia (77%), fever (74%), liver and spleen enlargement (68%), poor feeding/lethargy/irritability and jaundice. Severe thrombocytopenia without bleeding, is also a frequently reported feature of congenital malaria. Parasites are detected as usual in peripheral blood of neonates. Anti malarial IgG passively transferred from mother to the fetus is often detected and may delay and modify the clinical presentation of congenital malaria in newborns. The detection of parasites in placenta remains an important tool for diagnosis although the presentations in most cases are too late for procurement of placenta. However it was not done in our case as the possibility of such a transmission of malaria from treated and cured mother was probably not considered by gynaecologists and they didn't also asked for opinion from Pediatric Department.

The different case reports across the world occurred in neonates whose mothers were either undetected to have malaria during pregnancy, untreated, or were having asymptomatic parasitemia during antenatal period. Our case was different in this aspect as the mother was adequately treated before delivery, yet could not prevent transmission to the fetus. Whether this was due to persistent low grade parasitemia as a result of Chloroquine resistance or a relapse by hypnozoites remains unsolved. Successful treatment of the neonate and mother in the present admission possibly rules out Chloroquine resistance and speaks more in favour of the second possibility. The hypnozoites of P. vivax couldn’t be terminated during pregnancy as Primaquine is contraindicated in pregnancy and this probably led to this serious consequence.

The incubation period for P. vivax is 14 days. It also takes few days time to develop severe pallor. The child developed symptoms on 11th day after birth. Hence it is unlikely to be a case of newly acquired malaria. Moreover the detection of Plasmodium vivax in peripheral blood of asymptomatic mother confirms it to be a case of congenital malaria.
Relapse of Plasmodium vivax causing congenital malaria

The treatment of the neonate with chloroquine only was adequate since there is no exoerythrocytic life cycle in congenitally acquired vivax infection. The report suggests that the diagnosis of congenital malaria should be considered as an important differential diagnosis in newborns presenting with unexplained pallor, lethargy, poor feeding or sepsis like presentation particularly in areas where transmission of malaria is high. Early neonatal screening in such situations of antenatal P. vivax infection in mothers should be seriously considered irrespective of treatment and recovery of the mother. There may be a delayed presentation of congenital malaria and hence long term supervision may be necessary in selected cases. Chemoprophylaxis of all pregnant mothers in endemic region is another preventive strategy that can be implemented. Although infrequent, this entity called Congenital Malaria must be given proper attention and formulation of well defined strategies are essential.

CONCLUSION

Congenital malaria is quite existent and an important differential in appropriate clinical setting. Early neonatal screening in such situations of antenatal P. vivax infection in mothers should be seriously considered irrespective of treatment and recovery of the mother. Well formulated strategies are essential for chemoprophylaxis of all pregnant mothers in endemic region.

REFERENCES

Relapse of Plasmodium vivax causing congenital malaria