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# Congenital malaria – a case report from a non-endemic area

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**Abstract.** Eighteen day old neonate presented with features of early neonatal sepsis. History of mother revealed a travel from non-endemic area of malaria to endemic area, and on the 7th gestational age mother detected as having malaria. She was treated with quinine and cured. Baby was also evaluated for congenital malaria in first few neonatal days and discharged. Now the baby on evaluation shows anemia, hepatosplenomegaly and diagnosed with a *Plasmodium vivax* infection on peripheral smear. The quinine failed to prevent transplacental transmission. Prolonged interval between birth and onset of symptoms may be explained by transmission late in pregnancy or during delivery or by presence of transplacentally acquired maternal antibody (IgG). Mother acquired malarial infection after travel to an endemic area and transmitted to the baby. A high level of suspicion is warranted in babies of malaria infected mothers even when the neonate peripheral smear shows no evidence of infection.

## INTRODUCTION

Congenital malaria due to *Plasmodium falciparum* is not uncommon in endemic areas (Fischer, 2003). However, the role of *Plasmodium vivax* in causation of congenital malaria is uncertain (Valecha *et al*., 2007). Recent postulation is that placental barrier and malaria antibody protect the fetuses in endemic areas thus limiting the incidence (Desai *et al*., 2007). It is well known that pregnant women constitute an important risk group for malaria infection, particularly in hyperendemic and holoendemic situations. However, more recent reports from both malaria-endemic and non-endemic areas show higher prevalence of congenital malaria ranging from 8% to 33 % (Akindele *et al*., 1993). On the whole, malaria infection during pregnancy could account for around 6% of the Infant Mortality Rate (Steketee *et al*., 1993). We present a case of an 18 day old baby who was brought to our hospital with symptoms of early neonatal sepsis and diagnosed congenital malaria. We believe this case report will be useful for clinicians when evaluating and managing patients with congenital malaria.

## CASE HISTORY

An 18 day old neonate presented to our pediatric emergency department with complaints of fever of five days duration and cough and breathlessness of last three days duration. He had fever exacerbated in the night with breathlessness, tachypnoea and grunting respirations. The child was sucking well at the breast, but became less active and playful.

The mother was native to Kerala which is non-endemic for malaria and after marriage (non-consanguineous) she went to Orissa which is endemic area for malaria. She conceived one year after marriage. In the 7th month of pregnancy

she had fever and rigor, which on evaluation was diagnosed as malaria. She had undergone treatment with Quinine and was declared cured. Antenatal ultrasound evaluation showed Intra Uterine Growth Retardation (IUGR) at 32 weeks of gestation. She delivered normally at term, a male baby weighing 1.8 kg. Because of maternal history baby was evaluated for malaria, peripheral smear on the 3rd neonatal day was negative for malarial parasites with no evidence of any neonatal jaundice. On evaluation there were no evidence of malaria, baby and the mother was discharged on the 3rd day itself. Baby was on exclusive breast feeding after discharge from the hospital

On physical examination baby had pallor, facial dysmorphism with depressed nasal bridge and a broad nose. Abdomen was distended and sacral dimple was present. He weighed only 1.825 kg against an expected weight of 2.04 kg. Liver was palpable clinically with a span of 6 cm, spleen tip was also palpable without any evidence of ascitis (Figure 1). Heart rate was 150 per minute, respiratory rate 50 per minute, temperature 38°C and BP of 64/40 mm Hg.

We admitted the child and started him on an empirical antibiotic treatment. Laboratory evaluation revealed hemoglobin of 9.2 gm, total count of 3000/mm3, differential count was P37% L63%, PCV of 31% and O-positive blood group. Random blood sugar was 58 mg, blood urea 34 mg, serum creatinine 0.45mg, SGPT 10 IU/L, SGOT 14 IU/L, blood and urine culture and sensitivity results were sterile. BUN and serum electrolytes were within normal limits. Peripheral smear examination showed *Plasmodium vivax* malarial parasites with high parasitemia. Baby was also evaluated for other congenital infections and was negative for *Toxoplasma*, Rubella, Cytomegalovirus (CMV), HSV-I and HSV-II. The neonate was also assessed for evidence of any hemolytic conditions and was negative. Imaging studies were normal except for mild hepatosplenomegaly.

The baby was treated with syrup chloroquine 10mg/kg immediately followed by 5 mg/kg after 6 hours and then followed by 5 mg/kg once a day for next two days. Hemoglobin status of the child dropped to 7 mg% on the second day of admission and was corrected by blood transfusion.

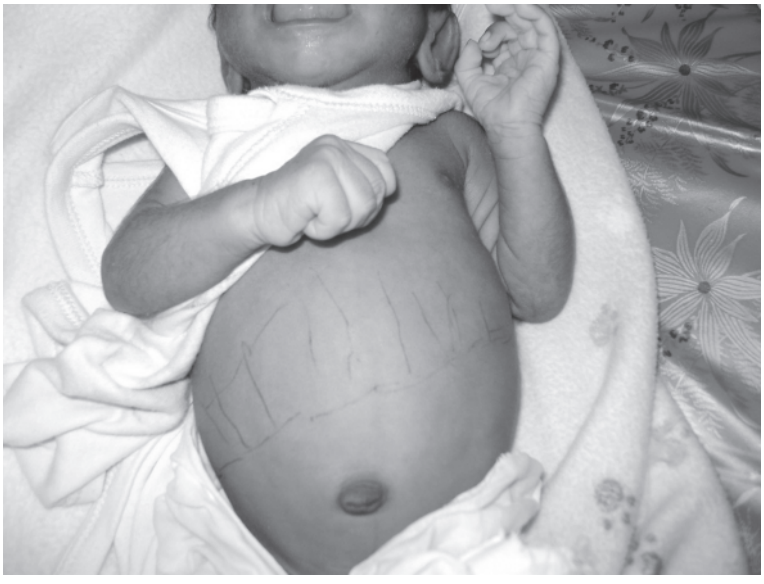

Figure 1. Picture of the abdomen showing enlarged liver without evidence of ascitis

Peripheral smear was repeated after therapy and was negative for malarial parasites on two consecutive days. The baby was discharged and followed up in outpatient department.

## DISCUSSION

Malaria during pregnancy, particularly close to term, may entail two opposite effects on child survival. It may protect the infant against malaria infection and severe disease via acquired maternal immunity (AMI) (Rasheed *et al*., 1995; Okoko *et al*., 2003). However it may also increase the risk of infant mortality, particularly neonatal mortality, by increasing the risk of low birth weight, premature labour, intra-uterine growth retardation, placental infection and stillbirth.

The important factor noticed in this case is that the mother hailed from a non-endemic area and acquired malarial infection after travel to an endemic area and thereafter malarial infection was transmitted to the child who got infected.

Case definition of congenital malaria is a dilemma because the presence of parasites in the placenta at delivery and clinical diagnosis of malaria in a newborn, yield different results. In a place outside the endemic area, where postnatal transmission can be reasonably excluded, the clinical manifestation of disease is usually delayed for several weeks. The prolonged interval between birth and onset of symptoms may be explained by transmission in late pregnancy or during delivery or by presence of transplacentally acquired maternal antibody (IgG). Transfer of protective immunity is one of the main factors that affects the age of symptom onset. Literature describes symptom onset of congenital malaria as typically occurring three to six weeks after birth, coinciding with the half-life of maternal IgG antibody in infants, but possibly as late as 15 months (Quinn *et al*., 1982).

Even though the mother was adequately treated with quinine, it failed to prevent transplacental transmission of infection or congenital malaria. Most clinical trials failed to consider a possible diminishing effect of antimalarial therapy on AMI and incidence of congenital malaria (Greenwood *et al*., 1992; Menendez *et al*., 1994). Thus a better modality of treatment should be adopted in malaria infected pregnant females with special emphasis on preventing congenital malaria and improvement in AMI.

In addition to this the baby was parasite free on peripheral smear at third neonatal day and subsequently showed parasites. Thus a high level of suspicion is warranted in babies of malaria infected mothers even when the neonate peripheral smear shows no evidence of infection. It has previously been suggested that infants should be presumptively treated if their mothers are identified as being parasitemic at delivery (Zucker & Campbell, 1993). Good cohort studies should be undertaken to test this suggestion. The newer tools used for the early diagnosis of congenital malaria was inadequate in cases of *P.vivax* infection (Singh *et al*., 2005). This case also brings to the limelight the importance of considering congenital malaria as a diagnosis even in non-endemic areas.

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## REFERENCES

Akindele, J.A., Sowunmi, A. & Abohweyere, A.E. (1993). Congenital malaria in a hyperendemic area: a preliminary study. *Annals of Tropical Paediatrics* **13**: 273–276.

Desai, M., ter Kuile, F.O., Nosten, F., McGready, R., Asamoa, K., Brabin, B. & Newman, R.D. (2007). Epidemiology and burden of malaria in pregnancy. *The Lancet Infectious Diseases* **7**(2): 93-104.

Fischer, P.R. (2003). Malaria and Newborn. *Journal of Tropical Pediatrics* **49:** 132-134.

Greenwood, A.M., Armstrong, J.R., Byass, P., Snow, R.W. & Greenwood, B.M. (1992). Malaria chemoprophylaxis, birth weight and child survival. *Transactions of the Royal Society of Tropical Medicine and Hygiene* **86**(5): 483–485.

Menendez, C., Todd, J., Alonso, P.L., Lulat, S., Francis, N. & Greenwood, B.M. (1994). Malaria chemoprophylaxis, infection of the placenta and birth weight in Gambian primigravidae. *The Journal of Tropical Medicine and Hygiene* **97**(4): 244–248.

Okoko, B.J., Enwere, G. & Ota, M.O. (2003). The epidemiology and consequences of maternal malaria: a review of immunological basis. *Acta Tropica* **87**(2): 193–205.

Quinn, T.C., Jacobs, R.F., Mertz, G.J., Hook 3rd, E.W. & Locklsey, R.M. (1982). Congenital malaria: a report of four cases and a review. *The Journal of Pediatrics* **101**(2): 229-232.

Rasheed, F.N., Bulmer, J.N., De Francisco, A., Jawla, M.F., Jakobsen, P.H., Jepson, A. & Greenwood, B.M. (1995). Relationships between maternal malaria and malarial immune responses in mothers and neonates. *Parasitology and Immunology* **17**(1): 1–10.

Singh, N., Saxena, A., Awadhia, S.B., Shrivastava, R. & Singh, M.P. (2005). Evaluation of a rapid diagnostic test for assessing the burden of malaria at delivery in India. *American Journal of Tropical Medicine and Hygiene* **73**: 855-858.

Steketee, R.W., Nahlen, B.L., Parise, M.E. & Menendez, C. (1993). The burden of malaria in pregnancy in malaria-endemic areas. *American Journal of Tropical Medicine and Hygiene* **64**(Suppl 1–2): 28–35.

Valecha, N., Bhatia, S., Mehta, S., Biswas, S. & Dash, A.P. (2007). Congenital malaria with atypical presentation: A case report from low transmission area in India. *Malaria Journal* **6:** 43.

Zucker, J.R. & Campbell, C.C. (1993). Malaria: principles of prevention and treatment. *Infectious Disease Clinics of North America* **7**(3): 547-567.