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A Case Report of Delayed Presentation of Congenital Malaria

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Abstract
We present here a case report of a congenital malaria admitted in the tertiary care hospital presenting in the late neonatal period, with no symptoms of acute febrile illnesses. The mother developed febrile illness in the late second trimester trimester and was diagnosed plasmodium vivax malaria and received treatment. Baby delivered was asymptomatic later on presented with icterus and hepatospleenomegaly and was diagnosed as plasmodium vivax malaria after detection of trophozoites in the thick and thin films.

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I. Introduction

Congenital malaria is defined as the presence of malarial parasites in the cord blood or presence of asexual parasites in the neonatal peripheral blood in the first week of life[1][2]. Similar presentation may result rarely due to perinatal or intrapartum transmission as well in infants of asymptomatic mother[3]. It has also been noted that raised resistance and virulence as an important factor behind raised incidence of congenital malaria [4].The consequences vary with transmission intensity. When the transmission is high, maternal anaemia is common, and infant low birth weight due to foetal growth restriction and/or premature delivery is frequent. In low transmission areas, when non-immune pregnant women become infected, malaria infection may become severe and life-threatening, requiring emergency treatment [5]. Delay in the appearance of the symptoms may be upto 3-6 weeks after birth due to the transplacentally acquired maternal antibody. Differentiation from vector borne malaria in infants can be made by taking past history and making thorough investigation of the mother. In congenital malaria due to absence of exoerythrocytic phase, radical treatment with potentially deleterious antimalarial is not required.

II. Case Presentation :

A 21-day male neonate was admitted for marked yellowish discoloration of skin and poor feeding. Baby was born by uneventful spontaneous normal delivery at 38 weeks of gestation.The birth weight of the newborn was 2,900 g. The infant was on exclusive breast feeding, and jaundice was detected. On admission, physical examination revealed afebrile, pallor, poor feeding and hepatosplenomegaly. Mild tachypnea and tachycardia were also appreciated. Complete blood count showed severe anaemia with haemoglobin (Hb) 6.0 g/dl, hematocrit 18.8 %, MCV 89.1 fl, MCH 28.4 pg, MCHC 31.9 g/dl, red blood cell count $2.12 \times 10^6/\mu l$, total leucocyte count $3.5 \times 10^3/\mu l$ and platelet count 76,000/cu mm (thrombocytopenia). No ABO or Rh incompatibility was detected. Other laboratory findings included hyperbilirubinemia with total bilirubin 5.1 mg/dl (conjugated bilirubin 4.0 mg/dl, unconjugated bilirubin 1.1 mg/dl), aspartate aminotransferase 23 U/l, alanine aminotransferase 15 U/l, and an elevated alkaline phosphatase (2,050 U/l). Mildly elevated CRP (34mg/l) with normal serum electrolytes noted.Blood, urine & stool cultures were sterile. Ultrasound of abdomen was normal. finally, thick and thin blood smears were performed and *P. vivax* trophozoites with 0.1% parasitemia were detected. The diagnosis was confirmed by malaria rapid diagnostic test Malaria HRP2/pLDH [Pf/Pv] and antimalarial therapy with intravenous artemisinin was initiated. Three days after treatment peripheral parasitemia was completely cleared, the white blood cell count increased up to $4.2 \times 10^3/\mu l$, Hb was 6.5 gm/dl and platelet count $1.3 \times 10^3/\mu l$. The neonate was also transfused with packed red cells (10 ml/kg body weight) for 3 h to raise the Hb content. The history of the mother was re-evaluated. She was a primigravida and history of overt fever or other clinical symptoms of malaria during pregnancy or related treatment history could not be elicited. Mildly elevated CRP (34mg/l) with normal serum electrolytes noted. Peripheral blood smears were suggestive of no malarial parasite. However rapid malaria test for pan malaria pLDH was positive, implying low parasitemia not recognized on peripheral smears. Full course of antimalarial treatment with choloroquine was given to her.

Fig 1 : Table of Investigation report

	D 1	D2	D3	D4
Hb (gm%)	6.5	8.0	8.9	10
TLC (/micro. Lt)	3.2 x 10^3	3.8 x 10 ^3	4.0 x 10^3	4.2 x 10^3
PLC (/mm^3)	37000	60,000	80,000	1,00,000
TB	5.1	4.8	2.6	1.1
CB	4.0	4.0	2.0	1
UB	1.1	0.8	0.6	0.1
Trophozoite	3,800	3,000	120	0

III. Discussion :

Congenital malaria was first described in 1876 [6]. Transmission of the malarial parasites can be acquired by the child from mother during pregnancy and perinatally during labour. Malarial infection of placenta is characterized by syncytiotrophoblast and villous disruption, syncytial knot formation and fibrin-type fibrinoid deposition. Syncytial destruction may result in low birth weight and congenital infections [7]. Though placental sequestration is not a major feature of *P. vivax* infection, but the infection is common in pregnancy and has been associated with adverse pregnancy outcomes including decreased birth weight and maternal anaemia [8,9]. However, a recent study found relatively low infection prevalence and modest associations with morbidity in five countries including India [10]. Malaria in pregnancy is by far more common in the first pregnancy of the mothers belonging to the high transmission areas, but it has been noted that in subsequent pregnancies prevalence and densities of parasitemia decreases. By contrast in low transmission areas, all pregnancies are equally at risk for *P. falciparum*, and probably *P. vivax* infection [11]. Microscopy of stained blood smears remains widely used to monitor the prevalence of malaria. For point-of-care testing, rapid diagnostic tests (RDT) are very effective for the diagnosis of symptomatic malaria infection, which tends to be accompanied by high parasitaemia. These are less effective as screening tools, being unable to reliably detect

low-density infections which are common [12]. Histological examination of placental tissue at delivery is a sensitive tool for detection of active or past malaria infection. Past infection is detected as the malaria pigment, haemozoin, most commonly in fibrin deposits. Active infection can be accompanied by leucocytes infiltrates, principally monocytes, termed intervillositis, particularly in first-time mothers with little pregnancy-associated malaria immunity. In this group, it is strongly associated with low birth weight and maternal anaemia [13]. The newborn was not treated with primaquine like her mother; in fact, *P.* vivax congenital malaria is found to have been transmitted by trophozoites but not by sporozoites and due to the absence of exorythrocytic phase it necessarily excludes the need for eradicating the latent hepatic forms [14,15]. The delayed presentation of symptoms in this case may be attributed to the fact that, though some authors describe the occurrence of congenital malaria in newborns aged less than 7 days, the interval between birth and onset of symptoms may be prolonged. Transmission of infection in late pregnancy or during delivery or due to transplacental acquisition of maternal antibody can be the well suited explaination.The main factor determining the age of onset of symptoms is the transfer of the protective immunity.Available literature usually describes the onset of symptoms of congenital malaria typically between 3 and 12 weeks after birth, coinciding with the half-life of maternal IgG antibody in infants. However, cases have been seen where onset of congenital malaria occurred as late as 15 months after the birth of the child [16,17].

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