

## CASE REPORTS

### CONGENITAL MALARIA DUE TO PLASMODIUM FALCIPARUM

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#### INTRODUCTION

Congenital malaria is defined as the presence of malaria parasites in the newborn within seven days of birth, or later if there is no possibility of postpartum infection by either mosquito bite or blood transfusion. Congenital malaria can be acquired by transmission of parasites from mother to child during pregnancy or perinatally during labour [1]. Congenital malaria has been documented for many years but it was previously thought to be uncommon. Normally symptoms occur 10 to 30 days postpartum and the most common clinical features are fever, irritability, feeding problems, hepato splenomegaly, anaemia and jaundice [2]. Due to non-specific clinical presentation of this disease, practitioners often fail to consider malaria in their initial differential diagnoses in neonates. One such case is reported.

#### CASE REPORT

A 16 days old male neonate was hospitalized with 2-days history of intermittent fever and breathing difficulty. The infant had been born at full term in an uncomplicated vaginal delivery at home by a 23 year old primipara. On examination, he was pale febrile and tachypnoeic. Blood counts indicated haemoglobin 6.2 g/dL, white blood cell count  $8.6 \times 10^9/L$  and platelets  $86 \times 10^{12}/L$ . Biochemical profile were normal. However, his peripheral blood smear revealed *P. falciparum* malarial parasites (parasitemia 4% of red blood cells). He was given oral quinine (10 mg/Kg body weight  $\times$  TDS) for one week. He had negative smears for malarial parasites on 4th day. Infant was transfused with 50 mL (15 mL/kg of patient body weight) of packed red blood cells before

discharge. On follow-up 10 days after discharge, the infant had no symptoms or signs of illness. A directed history revealed that the mother was having intermittent fever during last week of pregnancy and got some treatment. On examination, she was having hepatosplenomegaly. Her peripheral blood film also revealed *P. falciparum* (Malarial index 3%) and she was also treated for malaria during her stay in the hospital.

#### DISCUSSION

Congenital malaria is a major problem in tropical and subtropical countries and can be transmitted vertically from placenta of a pregnant woman to her fetus or perinatally during labour [3]. Studies have shown that the incidence of congenital malaria ranges from 0.3 to 33% in both endemic and non endemic areas [4]. A study conducted in infants at Karachi showed the prevalence of congenital malaria 4.45% and acquired malaria 14%. *P. falciparum* and *P. vivax* were the most prevalent species in this study [5]. All four species can cause congenital malaria but the majority of studies from Africa have focused on *P. falciparum* malaria. Congenital malaria by *P. vivax* has been described from Asia including Pakistan [6, 7]. The prevalence of congenital malaria was nearly three times higher among babies born preterm as compared to those born at term.

A hallmark of malaria during pregnancy is the sequestration of malaria-infected red blood cells containing late developmental stages in the intervillous spaces of the placenta. This is usually accompanied by the infiltration of maternal leukocytes, especially monocytes, in the intervillous spaces and haemozoin deposition. The sequestration of

infected red blood cells in the placenta is thought to be mediated in large part by the cytoadherence of infected red blood cells to placental receptors expressed in the

sulfate A (CSA) is the principal placental infected RBC receptor. Parasite-encoded surface ligands expressed on the membrane of infected RBCs are thought to facilitate this adherence. To date, the only well-studied cytoadherence parasite protein is the *P. falciparum* erythrocyte membrane protein-1 (PfEMP1) encoded by the highly polymorphic members of the var gene family. The most well characterized PfEMP1 variant identified to mediate infected RBC binding to the placenta is VAR2CSA [8]. Because of placental sequestration, peripheral blood film microscopy grossly underestimates the prevalence of placental malaria. Majority of neonates with congenital malaria were born to primiparous mothers as was our case and probably this could be explained by the increase with each pregnancy in levels of antibodies to variant surface antigen/chondroitin sulfate A, which inhibit the adherence of the parasite to placenta, thus decreasing its transplacental transmission with successive pregnancies [9].

A classic presentation of malaria may not occur in the newborn and absence of febrile episodes has been described. This has been attributed to transplacentally acquired antibodies (IgG), which confer transient protection to infant and thus manifestations are mild. Although IgG and IgM antimalarial antibodies can be detected in maternal blood, only IgG is normally found in the infant's blood. Onset of disease may be as early as 14 hours to as late as eight weeks of age but on an average it is between 10 to 28 days of life.

intervillous spaces and on the syncytiotrophoblast. Currently, it is believed that the glycosaminoglycan chondroitin

Fever, anaemia, splenomegaly occur in 80% cases. Reticulocytosis occurs in 50% cases and jaundice in 33% cases. Other features include hepatomegaly, poor feeding, loose motions and failure to thrive. Our case had respiratory distress which is not a common finding but has been well documented [10].

This congenital malaria should be considered in the differential diagnosis of ill neonates and young infants in Pakistan if symptoms points towards that.

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