CASE REPORT

CONGENITAL MALARIA

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Neonates with congenital malaria may present with non-specific signs and symptoms which may be mistaken for neonatal sepsis and inborn error of metabolism resulting in delay of diagnosis and significant mortality and morbidity. Here we present a unique case of 25 days old premature female baby who was diagnosed to have mixed malarial infection. Despite standard treatment the patient was not responding well and was also diagnosed to have congenital adrenal hyperplasia.

Keywords: Congenital malaria, splenomegaly, vivax, chloroquine, premature
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INTRODUCTION

There is no precise definition of congenital malaria but the presence of asexual plasmodium species in the placenta at the time of delivery or in neonatal peripheral blood smear in the early neonatal period irrespective of clinical symptoms is termed as congenital malaria.[1] The prevalence of congenital malaria in endemic areas varies from 0.03% to about 40%.[2] There is a greater chance for a non-immune mother to transfer infection to the foetus than immune mother. Some infants may be parasitemic at birth but still will never become ill. This may be due to trans-placental passage of maternal antibodies which then clear malarial parasites and this clearance rate may approach 100%. In contrast some neonates may have no detectable parasitemia at birth but will become symptomatic later on.[3]

The mode of transmission for congenital malaria is transplacental route. Congenital malaria may negatively affect pregnancy outcome and may result in low birth weight, anaemia and prematurity.[4]

CASE REPORT

A 25 days old female baby was brought to Military Hospital (MH) Rawalpindi with history of failure to gain weight since birth, fever, off feed and lethargy for 1 day. The patient was born at home by spontaneous vaginal delivery at 35 weeks of gestation. The baby was well till 8 days of life when she was brought for the first time to MH with history of fever and jaundice for 3 days. The baby remained admitted in MH for five days and was treated as neonatal sepsis and neonatal jaundice. She also underwent exchange transfusion for indirect hyperbilirubenimia. Common causes of jaundice were ruled out at that time like: Rh and ABO incompatibility, G6PD deficiency and breast milk jaundice as the baby was on top feed.

The mother had history of fever 4 days before birth of child for which she took medication from local doctor and got well. But again 3 days after birth of child she became febrile and was brought to MH in an unconscious state. She was diagnosed to have mixed malaria. She also remained on mechanical ventilator for 3 days and was discharged once stable.

On examination we found an emaciated and lethargic neonate with heart rate of 170/min, respiratory rate of 65/min and temperature of 101 °F. Her length, weight and head circumference were well below 0.4 centile for age and sex. Her abdomen was soft and not distended. Liver was palpable 4 cm and spleen 5 cm below right and left costal margin respectively. She had hyper-pigmented female genitalia and clitoral enlargement. Rest of the examination was unremarkable.

Investigation revealed haemoglobin 8g/dl, platelets 30×10/l and C Reactive protein (CRP) was 24mg/l, peripheral blood film showed rings of *Plasmodium falciparum*. 17-hydroxyprogesterone (17-OHP) was 37.5nmol/l. ultrasound abdomen showed hepatosplenomegaly. Rest of investigations were within normal limits.

The patient remained admitted in neonatal intensive care unit of MH for more than a month. The patient was treated with broad spectrum antibiotics and anti-malarial drugs and with supportive care. On admission the patient had falciparum ring positive and CRP 24mg/l. So the patient was advised inj. Artesunate and antibiotics. On 4th day of admission blood film showed both falciparum and vivax parasites with malaria parasite index (MPI) more than 1% and CRP rose to 48mg/l. Patient's antibiotics were revised and syrup chloroquine also started. On 16th day of admission vivax cleared from the slid but falciparum still persisted with MPI <1%. The patient was started with injection quinine in standard doses. On 29th day of admission falciparum also disappeared from slide, CRP became negative but 17-OHP was markedly raised. The patient was started with tablet hydrocortisone and fludrocortisone. On 31st day of admission the patient was discharged with medication for congenital adrenal hyperplasia (CAH) and was advised follow up.

During her stay in hospital the patient had respiratory distress and remained on incubator oxygen for 4 days, head box oxygen for 9 days, continuous positive airway pressure for 3 days and on mechanical ventilator for 1 day, she had hypoglycaemic episodes which were treated with 10% dextrose boluses. She also

had recurrent fall in haemoglobin and platelets for which she was transfused with red cells concentrates thrice and platelets twice. On her follow up 1 month after discharge the patient had improved a lot. Her length was on 0.4 centile while head circumference and weight were between 0.4 and 2nd centile. She was also reviewed in retinopathy of prematurity clinic and was found normal. Her repeat blood complete picture was normal with no malarial parasite seen.

DISCUSSION

About three hundred cases of congenital malaria have been reported in world literature so far[5] and only four of them were premature. There are few cases of congenital malaria with mixed infections. In our case the patient was premature and suffered from both vivax and falciparum infections. In most of the cases the mother has history of exposure to malaria and our patient mother also suffered from severe malaria and also remained on mechanical ventilator for 3 days. Congenital malaria clinically present between 10–30 days post-partum. Our patient presented for the first time on 8 days of life but was not diagnosed at that time and was diagnosed when she was brought for second time on 25th day of life.

Fever is the most common symptom of congenital malaria. Other symptoms may include paleness, jaundice, loose stool, poor feeding and drowsiness.[6] Our patient was having fever and jaundice when presenting for the first time and the jaundice was so severe that she underwent exchange transfusion. The jaundice at that time was most probably due to malaria infection because at that time all the common causes of indirect hyperbilirubenemia were ruled out. And for the second time again she had complaints of off feed, lethargy and fever. The most common signs of congenital malaria are anaemia and splenomegaly. Other signs may include cyanosis, jaundice and hepatomegaly.[7] In a review from China spleen and liver were enlarged more than 2 cm below left and right costal margin respectively. In our patient spleen was palpable 5 cm and liver 4 cm below left and right costal margin respectively.[6] Our patient had most of the signs and symptoms of severe malaria like lethargy, respiratory distress and apnoeic spells for she remained on oxygen therapy for many days including mechanical ventilator for one day. For severe pallor she was transfused with red cells concentrate thrice.

Malarial diagnoses can be made by thick and thin blood film, rapid diagnostic tests and polymerase chain reactions. Each of these investigations has its own merits and demerits.[8] Our patient was diagnosed by using microscopy and was followed with blood thick and thin films and malaria parasite index.

With the exception of tetracycline, pyrimethamine-sulphdoxin and primaquine rest of the anti-malarial drugs can be used in the neonates with dose adjustment as per body weight.[9] Our case was very unique in the sense that she did not respond to 5 days course of parenteral artesunate and so it was given for another 5 days but still the patient was positive for malarial parasite. She was then given chloroquine with which vivax was cleared but falciparum still persisted. And at last the patient responded to 14 days course of parenteral quinine. We could not establish the resistance on molecular basis as this facility is not available with us. But in literature we also failed to find a case with such complex constellations of mixed congenital malaria, prematurity, low birth weight, and severe clinical presentation and with congenital adrenal hyperplasia.

Detailed history taking especially antenatal history is very important for congenital malaria diagnosis in neonates who are febrile, lethargic and have hepatosplenomegaly. Prompt treatment should be initiated to avoid malaria associated mortality and morbidity in neonates.

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