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P. Vivax Malaria: A Rare Cause of Cholestatic Jaundice in a Neonate

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Abstract: Congenital malaria is thought to be rare in neonates even in malaria endemic regions. Recent reports from Africa suggest that the incidence of congenital / neonatal malaria is rising, possibly due to the increased resistance and virulence of the parasite resulting from altered antigenic determinants as well as increased reporting. The clinical features of congenital malaria are non-specific and overlap with those of sepsis. The typical malaria paroxysm is absent, with the infant instead having fever, refusal to suck, excessive crying and irritability, and anaemia. In contrast to unconjugated hyperbilirubinemia, which can be physiologic, cholestasis (conjugated bilirubin elevation of any degree) in the neonate is always pathologic and prompt differentiation is imperative the clinical features of any form of cholestasis are similar. In an affected neonate, the diagnosis of certain entities, such as galactosemia, sepsis, or hypothyroidism, is relatively simple. In most cases, the cause of cholestasis is more obscure. Differentiation among biliary atresia, idiopathic neonatal hepatitis, and intrahepatic cholestasis is particularly difficult. Congenital P.vivax malaria is generally not considered while evaluating a case of Neonatal cholestasis; however, the presenting signs and symptoms should also alert the clinician to consider congenital malaria in the appropriate clinical setting. Since many of the classic features of malaria such as chills and sweating may not be observed in these patients, the diagnosis of congenital malaria must be suspected and daily examination of thick and thin smears / malaria antigen rapid kit test should be done for evidence of intracellular parasites.
Keywords: Congenital malaria, Neonatal cholestasis.

INTRODUCTION

Malaria presenting in neonatal period is a rare disease even in endemic areas, [1].Congenital malaria has been documented for many years, but it was previously thought to be uncommon especially in indigenous populations[2]. Congenital malaria is acquired from the mother prenatally or perinatally and is a serious problem in tropical areas. It occurs in 0.1% of immune and 10% of non-immune mothers in endemic areas, [3].

Neonatal cholestasis is defined as prolonged elevation of serum levels of conjugated bilirubin beyond the 1st 14 days of life. Jaundice that appears after 2 wks of age, progresses after this time, or does not resolve at this time should be evaluated and a conjugated bilirubin level determined. Cholestasis in a newborn can be due to infectious, genetic, metabolic, or undefined abnormalities[5].

The 1st sign or symptom of congenital malaria most commonly occurs between 10 and 30 days of age (range 14 hrs to several months of age). Signs and symptoms include fever, restlessness, drowsiness, pallor, jaundice, poor feeding, vomiting, diarrhea, cyanosis, and hepatosplenomegaly, [4]. Jaundice is commonly seen with falciparum infection, [6].We is reporting a rare and interesting case of cholestatic jaundice caused by congenital P. vivax malaria infection.

CASE REPORT

A term infant born with average birth weight, out of a non-consanguineous marriage, presented at 1month of age because of fever for five days, appearance of jaundice and clay colored stools for 3 days, feeding difficulty and lethargy. There is no history of blood transfusion, jaundice or any significant illness in mother with normal antenatal history. Mother's HBsAg was also negative.

On Physical Examination, Pallor Was Present (Fig :1). The Infant Had Weight Of 5 Kg and was icteric and had a palpable liver 2.5cm below right costal margin with span of 6.4 cm and spleen 3.8 cm below left costal margin, firm and non tender. The stools were clay colored and urine was dark yellow. The laboratory findings were as follows – hemoglobin 8 gm/dl with MCV -90, MCH 30, and MCHC 31and RDW 14.8,

platelet counts were 70,000, TLC – 13,000 with N32%, L67%. Reticulocyte counts were 0.2%.

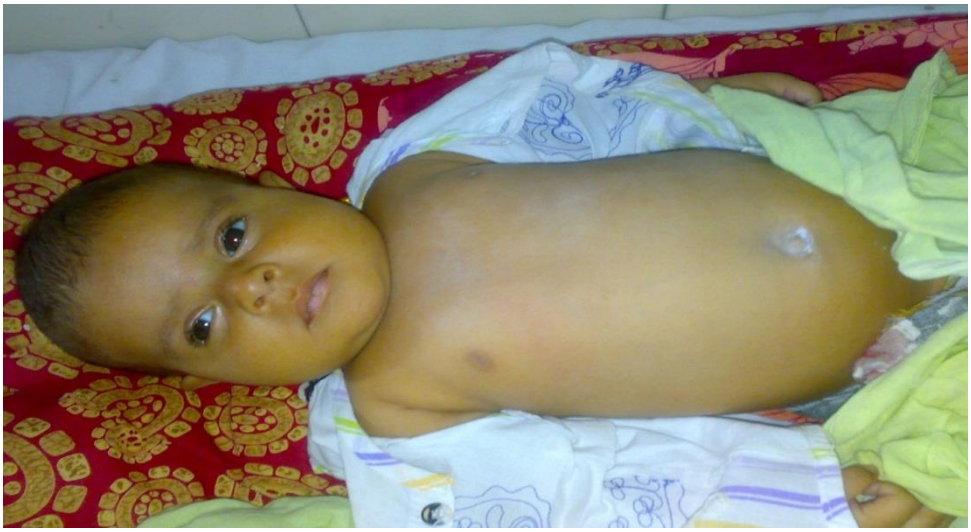

Fig-1: Cholestatic Jaundice

PBF report showed schizonts of P.vivax, Bilirubin level was 15.8 mg/dl with a conjugated fraction of 6.8mg/dl. Alkaline phosphatase was 432 IU/L, SGPT 1678 and SGOT 1998 U/L and GGT 125 U/L.

Ultrasound abdomen revealed distended gall bladder. The Australia antigen, IgM-anti HAV, and Anti-HEV were negative. Prothrombin time was 14.2 sec. with INR of 1.12 and serum total protein was 6.4 g/dl with albumin 3.3 g/dl. Urine examination was normal.

A diagnosis of "Cholestatic jaundice with congenital P. Vivax malaria" was madeand during 5 days of treatment with IV Artesunate the icterus and fever disappeared. On 5th day the serum bilirubin level decreased to 3.1 mg/dl with a direct fraction of 1.4mg/dl. The SGPT and SGOT were 20 and 60 U/L respectively. PBF showed absence of parasite.

DISCUSSION

Congenital infections such as cytomegalic inclusion disease, toxoplasmosis, rubella, and syphilis may be manifested as anemia, jaundice, hepatosplenomegaly, and thrombocytopenia, but the direct Coombs test result is negative and these conditions usually have other distinguishing clinical findings[5]. Anemia and jaundice may occur in infancy from hereditary spherocytosis and other red cell membrane defects, and, if untreated, can result in kernicterus. Congenital malaria is an uncommon cause of fever, splenomegaly, and anemia in infants.

Investigation of jaundice in an infant or older child must include determination of the accumulation of both unconjugated and conjugated bilirubin. The initial step is identification of cholestasis. The next step is to recognize conditions that cause cholestasis and for which specific therapy is available to prevent further damage and avoid long-term complications such as sepsis, an endocrinopathy (hypothyroidism, pan hypopituitarism), nutritional hepatotoxicity caused by a specific metabolic illness (galactosemia), or other metabolic diseases (tyrosinemia)[7]. In infants with biliary atresia, US findings may include small or absent gallbladder; nonvisualization of the common duct; and presence of the triangular cord sign, a triangular/tubular-shaped echogenic density in the bifurcation of the portal vein, representing fibrous remnants at the porta hepatis[5]. Since many of the classic features of malaria such as chills and sweating may not be observed in these patients, the diagnosis of congenital malaria must be suspected and daily examination of thick and thin smears / malaria antigen rapid kit test, should be done for evidence of intracellular parasites. In congenital malaria after treatment serum bilirubin level comes to normal level in 1-2 weeks after appropriate treatment, whereas in patients with acute viral hepatitis it takes 6-8 weeks, [8].

CONCLUSION:

In conclusion, Neonatal Cholestatic jaundice is a very rare and interesting consequence of congenital P.vivax malaria.The diagnosis can easily be missed if it is not considered and often results in the performance of unnecessary investigations, procedures, ineffective treatments, and potentially significant morbidity and expenses.The disease most commonly presents during the first 4 weeks of life but its onset can be delayed for few months.

REFERENCES

1. Logic DE, McGregor IA; Acute malaria in newborn infants. Br Med J. 1970;3(5719) 404.

2. Sotimehin SA, Runsewe-Abiodun TI, Oladapo OT, Njokanma OF, Olanrewaju DM; Possible risk factors for congenital malaria at a tertiary care hospital in Sagamu, Ogun state, southwest Nigeria. J Trop Pediatr. 2008; 54(5):313-20.
3. Elizabeth D Barnett; Malaria. In: Textbook of Pediatric Infectious Diseases. Philadelphia, Pennsylvania: Saunders. 5th edition, 2004; 2: 2714-2729.
4. John CC, Krause PJ; malaria (Plasmodium) Prototozoan diseases, infectious diseases. InNelson Textbook of Pediatrics. Nineteenth Edition. Editors: Robert M. Kliegman, Bonita F. Stanton, Joseph W. St. Geme III, Nina F. Schor, Richard E. Behrman, Elsevier Saunders Philadelphia.
5. Nelson; Textbook of Pediatrics.Eighteenth Edition. Editors: Robert M. Kliegman, Bonita F. Stanton, Joseph W. St. Geme III, Nina F. Schor, Richard E. Behrman, Elsevier saunders Philadelphia, 1662,1668.
6. Hazra BR, Chowdhury RS, Saha SK, Ghosh MB, Mazumder AK; Changing scenario of malaria: a study at Calcutta. Indian J Malariol 1998; 35(2): 111-6.
7. Moyer V, Freese DK, Whitington P, Olson A.D, Brewer F, Colletti R.B *et al.;* Cholestasis clinical practice guideline. North American Society for Pediatric Gastroenterology, Hepatology and Nutrition: Guideline for the evaluation of cholestatic jaundice in infants: Recommendations of the North American Society for Pediatric Gastroenterology, Hepatology and Nutrition. J Pediatr Gastroenterol Nutr 2004:39(2):115-728.
8. Patwari A, Aneja S, Berry AM, Ghosh S; Neonatal malaria a clinico haematological profile. Indian Pediatr 1978 : 15-10:847-9.