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Case Report

Late onset severe anemia due to rhesus isoimmunization

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ABSTRACT

Rh isoimmunization usually presents with severe neonatal jaundice associated with anemia needing exchange transfusion and phototherapy. Sometimes babies who have mild or no symptoms at birth may present later with severe hemolytic anemia. In this case report we have described a newborn infant who had mild jaundice initially and presented with severe anemia in the fourth week of life. This case signifies the importance of regular follow up and close monitoring of Rh isoimmunized infants for the first two months for delayed onset anemia.

Keywords: Anemia, Blood transfusion, Direct coombs test, HDN, Jaundice, Newborn, Phototherapy, Rh isoimmunization

INTRODUCTION

Hemolytic disease of the newborn (HDN) is caused by the trans-placental passage of maternal antibody active against paternal RBC antigens of the infant and is characterized by an increased rate of RBC destruction.[1] Haemolysis after Rh isoimmunization primarily causes anemia in the intrauterine period but leads to jaundice and anemia of variable intensity after birth.[2] Late anemia is a recognized complication of HDN regardless of the initial disease severity.[3] Now-a-days HDN is becoming less common, because of introduction of anti D antibodies, early intervention and specialty care centers. In this case report, we present a 24 day old infant born to an Rh negative mother with initial mild jaundice followed by late onset severe anemia.

CASE REPORT

A 24 day old male baby born of a non-consanguineous marriage, to a 26 year old mother was admitted to our unit with severe anemia. The blood group of the mother was AB negative and that of father was B positive. The mother had conceived twice before, the first one was an abortion and the second a term male baby (B positive) born by normal vaginal delivery. On both occasions mother had received anti D immunoglobulin prophylaxis within 24 hours of delivery. The present pregnancy was uneventful. The baby was born by normal vaginal delivery and weighed 2.5 kg at birth. He was apparently normal for the first 48 hours. The baby developed jaundice on day 3, (TSB - 17.22 mg/dl, DSB - 1.38 mg/dl, Hb - 15.2 g/dl) which was treated with phototherapy for 4 days (TSB - 10 mg/dl, DSB - 1.26mg/dl). A rebound hyperbilirubinemia on day 9 (TSB - 22.76 mg/dl, DSB - 1.72 mg/dl) necessitated repeat phototherapy for 48 hours (TSB - 11.5 mg/dl). Baby recovered and was discharged on day 15 when the TSB was 9.95 mg/dl.

The baby was next seen on day 24 of life with a viral upper respiratory infection associated with marked pallor. He weighed 3.6 kg and on examination had severe pallor with hepatosplenomegaly. His Hb was 4.5 gm/dl, retic count -3%, MCV - 90.7 fl , MCH - 34.9 pg, Peripheral smear showed microcytic and macrocytic RBCs with 5%

nucleated RBCs, marked anisopoikilocytosis, ovalocytes and tear drop cells . Baby's blood group was AB positive; DCT was strongly positive; total serum bilirubin - 4.04 mg/dl; direct bilirubin - 0.85 mg/dl; Indirect coombs test done on mothers blood was positive. The baby's blood could not be crossmatched with AB positive blood. Baby was transfused with O negative packed RBCs following which the hemoglobin increased to 9.8 g/dl. During follow-up the hemoglobin dropped to 8 g/dl on day 7 and 7.6 g/dl on day 21 post-discharge.

DISCUSSION

The incidence of Rh haemolytic disease in newborns has decreased dramatically after the introduction of anti D immunoglobulin prophylaxis.[1,2] In majority of mothers, sensitization occurs during labour. This provides the rationale for administration of anti D immunoglobulin within 72 hours of delivery. However, feto maternal transfusion leading to sensitization is known to occur in some mothers after 28th week of gestation. Therefore, a single dose of anti D during 28th week or two doses at 28th and 34th week may further reduce the incidence of Rh isoimmunisation3. In our case the baby developed Rh haemolytic disease despite anti D prophylaxis given post-delivery during previous two pregnancies.

Rh isoimmunisation can present occasionally with mild initial symptoms and severe late onset anaemia. The initial mild haemolysis may accelerate after the first week of life, probably due to improved reticuloendothelial function. A parallel maturation of liver function prevents a concomitant rise in serum bilirubin.[4] Late onset anaemia is especially common in babies who had not received an initial exchange transfusion.[5,6] More recent evidence has shown that haemolysis due to passively acquired antibodies may continue for six to 10 weeks in infants with HDN.[7] Another subgroup of babies with late onset anaemia is those receiving in utero transfusions. Here, reduced survival of transfused RBCs, continued immune destruction of neonatal RBCs and suppression of fetal erythropoeisis could be possible causes.[8,9]

In our case, the baby developed haemolytic disease despite mother receiving anti D prophylaxis during previous two pregnancies. Feto-maternal transfusion has been known to occur after 28th week and could be the possible cause for sensitization in our case. This emphasizes the need for anti D during pregnancy along with the postnatal dose. Our case had mild jaundice but no anaemia during the first week, but presented with anaemia on day 24 of life. The baby recovered well with O negative PRBC transfusion. Mitchell S et al reported 2 cases, in which babies were asymptomatic at birth and later developed severe anaemia on day 12 and day 18 respectively.[10] These babies were treated with double volume exchange transfusion.

This case was reported to highlight two issues. First, a single dose of anti D given within 72 hours of delivery may not prevent all cases of isoimmunisation. Second, close follow up is a must for all babies with Rh haemolytic disease irrespective of severity of initial disease.

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