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Interesting Cases

Anti-c Antibodies Causing Neonatal Hyperbilirubinaemia - A Case Report

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A B S T R A C T

Haemolytic disease of a new born is a heterogenous disease that ranges from jaundice to erythroblastosis foetalis and kernicterus. We present a case of a neonate who developed jaundice on day 3 of life which was progressive despite the use of intensive double surface phototherapy and finally required double volume exchange transfusion for management. The neonate was found to have minor blood group incompatibility. Anti-c antibodies were detected in the neonate's blood.

Keywords: Minor Blood Group Incompatibility, Anti-c, Neonatal Hyperbilirubinaemia, Exchange Transfusion

Introduction

Haemolytic disease of a new born is a heterogenous disease that ranges from jaundice to erythroblastosis foetalis and kernicterus. The exact mechanism of the disease was described in 1940 by Levine et al. who demonstrated that HDN occurs when a mother who is herself Rh D negative and is carrying a foetus who is Rh D positive, develops antibodies against Rh D positive red blood cells and the transplacental migration of these anti-Rh D positive antibodies cause destruction of foetal RBCs.[1] The most common causes of haemolytic jaundice in newborns are ABO incompatibility, minor blood group incompatibility, Rh incompatibility, and G-6-PD deficiency.[2]

Case

We present a case of a newborn baby, with a primi mother of 26 years of age. The baby was born of a non-consanguineous marriage by lower segment caesarean section in view of non-progress of labour at 39 weeks and 2 days period of gestation. The neonate was born with a birth weight of 3.6 kg, cried immediately after birth and required no resuscitation. The antenatal period was un-eventful. The baby was started on exclusive breastfeeding and was feeding well with adequate urine and stool output. On day 3, the baby was found to be icteric till legs. Serum bilirubin was 15.7 mg/dl, and haemoglobin was 14.6 mg/dl. The mother's blood group was AB positive and the baby's blood group was B positive. Direct Comb Test (DCT) was negative. The baby was started on phototherapy. Despite double surface phototherapy, serum bilirubin was 19.7 mg/dl after 12 hours. The baby was continued on DSPT and samples were repeated after 12 hours which increased to 21.4 mg/dl. Other investigations were repeated: haemoglobin was found to be 12.6 mg/dl, peripheral blood smear revealed

no features of haemolysis, thyroid profile was normal, G6PD was normal, and repeat DCT was 4+. The baby was clinically normal with a good cry, tone and activity. Sucking reflex and Moro's reflex were normal and the baby was feeding well. On day 5, serum bilirubin increased to 23.1 despite continued DSPT.

At this stage, a presumptive diagnosis of ABO incompatibility or minor blood group incompatibility was made, and in view of rising serum bilirubin despite continuous DSPT, the baby was given IVIg @ 1 gm/kg over 6 hours. Serum samples were collected prior to IVIg therapy and were sent for minor blood group incompatibility testing.

The baby, however, showed no response to IVIg therapy, and serum bilirubin after 8 hours of therapy further increased to 24.1 mg/dl. The baby was clinically normal with no features suggestive of bilirubin-induced neurological damage. Minor blood group incompatibility testing revealed Indirect Comb's Test (ICT) positive (2+) in the mother with identification of anti-c alloantibody. Rest C, E, e, and K antigens were negative. Double volume exchange transfusion was done with "c antigen" negative and E and K negative cross-matched compatible red cell units. Post transfusion serum bilirubin was reduced to 11.7 mg/dl. The baby was observed for 2 more days and remained well with no further rise in serum bilirubin values. The baby was on exclusive breastfeeds with normal neurological examination and was discharged on day 10 of life. On follow-up after a week, the baby was comfortable, feeding well on exclusive breastfeeds and repeat serum bilirubin was 10.8 mg/dl.

Discussion

Minor blood group incompatibility has been reported to cause haemolytic jaundice in nearly 3-5% of cases.[3,4] The highly common reasons leading to an incompatibility between a baby and the mother are C, c, Kell, E, e, Duffy, MNS Diego, and Kidd.[3,4,5] Anti-c is generally considered to be the cause of the most severe form of HDN after anti-D.[3,5] Haemolytic disease caused by minor blood group antigen can occur in the form of a wide range of clinical disorders ranging from Hydrops foetalis and recurrent pregnancy losses to haemolytic jaundice requiring phototherapy to exchange transfusions in the neonatal period.[3,5] Positive Coombs test has been reported in cases of HDN, however, it does not correlate with the severity of HDN. Coombs test can be negative in many cases due to weak antigen-antibody interaction or intravascular haemolysis.[5] Treatment options consist of using phototherapy, IVIg therapy, and exchange transfusion. A few reports have been presented regarding the usage of IVIg in minor blood group incompatibility, however, Cochrane review does not recommend the use of IVIg in routine cases.[6] Some neonates may also require blood transfusions later for anaemia.[5]

Conclusion

Minor blood group incompatibility is a significant cause of jaundice in neonates. Once the usual causes of neonatal jaundice have been ruled out, extended panel for minor blood group testing should be sent. Treatment can range from phototherapy to the requirement of exchange transfusion. An antibody panel is a must for correct diagnosis and ordering blood for exchange transfusion.

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