Indian Journal of Basic and Applied Medical Research; September 2018: Vol.-7, Issue- 4, P . 329 - 332

Original article

Role of IVIG in Preventing 2nd Exchange Tranfusions in Newborn Rh Hemolytic Disease

Dr. (Col.) D.Y. Shrikhande, *Dr. Jimmy Chaudhari, Dr. Shantanu Verma, Dr. Priyanka Nayak

Department of Pediatrics, Rural Medical College, PIMS (DU) , Loni
Corresponding author *

Abstract

Hemolytic disease of the newborn falls into three basic categories based on cause and serological diagnosis, ie, Rh incompatibility, ABO blood group incompatibility, and alloantibody reactions. Rh incompatibility is managed by maternal administration of Rh(D) immune globulin which binds to and destroys fetal cells circulating in maternal blood before a full maternal immune response can be initiated(1). Blood group incompatibility occurs in 15%–25% of pregnancies(2–4). From this case series we may conclude , We think that delaying exchange transfusion by 8 hours, until the results of IVIG treatment are known, may at least partially reduce the need for 2nd transfusions. As IVIG therapy can be administered quickly, this may gain some valuable time for the patient, as it take may take hours to prepare for an exchange transfusion.

Introduction

Hemolytic disease of the newborn falls into three basic categories based on cause and serological diagnosis, ie, Rh incompatibility, ABO blood group incompatibility, and alloantibody reactions. Rh incompatibility is managed by maternal administration of Rh(D) immune globulin which binds to and destroys fetal cells circulating in maternal blood before a full maternal immune response can be initiated(1). Blood group incompatibility occurs in 15%–25% of pregnancies(2–4). Mothers with the blood group O develop anti-A and anti-B antibodies. If a fetus is A or B blood type, an incompatibility exists and maternal anti-A or anti-B antibodies attach to the fetal blood cell, leading to destruction and the development ABO blood group incompatibility. However, only 1 in 150 infants develop mild hemolysis and even fewer, 1 in 3,000, develop severe disease(3). Rare alloantibodies, including anti-D, anti-C, anti-E, anti-Kell, anti-Kidd, and anti-Duffy, can also lead to hemolytic anemia in the newborn(5). Of these, anti-D remains the most common, affecting 1 to 1,200 pregnancies(6–8). Prenatal maternal testing can identify these antibodies, so health care providers can provide close monitoring and possible prenatal interventions(5)

Neonatal use of IVIG to treat hemolytic anemia was first reported in 1987 by Hara et al as being successful in the treatment of late anemia due to rh incompatibility(9). Intravenous immunoglobulin (IVIG) treatment has been reported to decrease requirements for exchange transfusion, phototherapy, and to shorten hospitalization time for newborns with Rh-ve mother(10-12). It has been shown that IVIG is also effective in prevention of repeated exchange transfusions when used after the first exchange transfusion(13). Here we report four cases of Rh-ve mother with DCT+ve newborn at birth which 2nd exchange transfusion was indicated, but the patients were treated with IVIG followed by blood transfusion.

www.ijbamr.com P ISSN: 2250-284X , E ISSN : 2250-2858

Material and method

All the case described developed early icterus due to maternal Rh incompatibility. After that 1st early exchange transfusion was done. This was followed by IVIG after 24 hours of completion of exchange transfusion followed by blood transfusion. All the cases were admitted to neonatology department of pravara rural hospital.

Case studies

Case 1 was a term female male infant. At birth, the infant showed marked icterus with splenomegaly. The cord blood hemoglobin (Hb) level was 9.3 g/dL, and the total bilirubin level was 5.4 mg/dL. Early Exchange was done. After 24 hours of life IVIG was administered followed by blood transfusion. Eight hours later, the patient's Hb and total bilirubin levels were 16.2 g/dL and 11.3 mg/dL, respectively.

Case 2 was a term male. On admission to our hospital at 26 hours after birth, the patient was icteric and his spleen was palpable. The serum Hb was11 g/dL, the total bilirubin level was 23 mg/dL. Exchange transfusion was done. He was given IVIG 24 hours after followed by blood transfusion. Phototherapy was started. Eight hours later, the baby's Hb level was 17 g/dL and his total bilirubin had dropped to 18 g/dL.

Case 3 was a term male. On admission to our hospital 10 hours after birth, the infant was icteric and spleen was enlarged. The serum Hb level was 10 g/dL, total bilirubin was 20.5 mg/dL. Exchange transfusion was done. The baby was given IVIG 24 hours after followed by blood transfusion and phototherapy was initiated. Eight hours later, his Hb level was 14.5 g/dL and her total bilirubin had fallen to 19 mg/dL.

Case 4 was term female. Physical examination at birth revealed splenomegaly along with icterus. Cord blood Hb was 9 g/dL, and total bilirubin was 8 mg/dL. Early Exchange was done. After 24 hours of life IVIG was given followed by blood transfusion Eight hours later, her Hb was 12 g/dL, and her total bilirubin level was 16 mg/dL.

All four patients in this report developed hemolytic disease due to Rh-incompatibility (DCT +ve). 2nd Exchange transfusion was indicated but was withheld, and treatment with 1 g/kg IVIG significantly reduced the rate of hemolysis in all cases.

TABLE I- Characteristics and Laboratory Findings.

	Case 1	Case 2	Case 3	Case 4
Birth weight (g) 2690	3250	3580	2670	
Gestational week at delivery	37	39	40	38
Cord blood Hb (g/dL)	9.3	ND	12.8	9.0
Tbil (mg/dL)	5.4	ND	4.5	8.0
Direct Coombs'	+	+	+	+
Before+/after* IVIG Hb (g/dL)	10.3/13.2	12/13	11/11.5	10/12
Tbil (mg/dL)	5.4/11.3	19/14	10.5/9	18/16
No. of Transfusions	2	1	1	1

+24 hours after exchange transfusion

*8 hours after IVIG treatment.

ND: Not determined; Hb: Hemoglobin, Tbil: Total Bilirubin

www.ijbamr.com P ISSN: 2250-284X , E ISSN : 2250-2858

Results and Discussion

It has been hypothesized that the anti-D sensitized neonatal erythrocytes are destroyed by antibody dependent cellular cytotoxic effects mediated by the Fc receptor on the cells of the reticuloendothelial system. IVIG would occupy the Fc receptor sites, thus competing with the anti-D sensitized neonatal erythrocytes and preventing hemolysis(14). This mechanism explained the abrupt block in hemolysis after 1st exchang and arrest in rising bilirubin levels with adjuvant phototherapy and blood transfusion in all four cases, but this observation has to be validated by other studies as well. IVIG administration has been endorsed and included as a standard of practice in the American Academy of Pediatrics 2004 Management of hyperbilirubinemia in the Newborn Infant guidelines (15).

Conclusion

We think that delaying exchange transfusion by 8 hours, until the results of IVIG treatment are known, may at least partially reduce the need for 2nd transfusions. As IVIG therapy can be administered quickly, this may gain some valuable time for the patient, as it take may take hours to prepare for an exchange transfusion.

REFERENCES

1. Mundy CA. Intravenous immunoglobulin in the management of hemolytic disease of the newborn. Neonatal Netw. 2005;24(6):17–24.

2. Mollison PL, Engelfreit CP, Contreras M. Blood Transfusions in Clinical Medicine. 9th ed. Oxford, UK: Blackwell Scientific Publications; 1993.

3. Ziprin JH, Payne E, Hamidi L, Roberts I, Regan F. ABO incompatibility due to immunoglobulin G anti-B antibodies presenting with severe fetal anaemia. Transfus Med. 2005;15(1):57–60.

4. Maisel JM. Neonatal jaundice. Pediatr Rev. 2006;27(12):443–454.

5. Murray NA, Roberts IA. Haemolytic disease of the newborn. Arch Dis Child Fetal Neonatal Ed. 2007;92(2):F83–F88.

6. Howard H, Martlew V, McFadyen I, et al. Consequences for fetus and neonate of maternal red cell alloimmunisation. Arch Dis Child Fetal Neonatal Ed. 1998;78(1):F62–F66.

7. Stockman JA, de Alarcon PA. Overview of the state of the art of Rh disease: history, current clinical management and recent progress. J Pediatr Hematol Oncol. 2001;23(8):385–393.

8. Thompson J. Haemolytic disease of the newborn: the new NICE guidelines. J Fam Health Care. 2002;12(5):133–136.

9. Hara T, Mizuno Y, Kawano M, Ueki Y, Ueda K. Treatment of immune hemolytic anaemia with gammaglobulin. J Pediatr. 1987;110(5): 817–818

10. Rübo J, Albrecht K, Lasch P, Laüfkötter E, Leititis J, Marsan D, et al. High-dose intravenous immune globulin therapy for hyperbilirubinemia caused by Rh hemolytic disease. J Pediatr 1992; 121: 93-97.

11. Dagoglu T, Ovali F, Samanci N, Bengisu E. High-dose intravenous immunoglobulin therapy for rhesus hemolytic diseases. J Int Med Res 1995; 23: 264-271.

12. Mukhopadhyay K, Murki S, Narang A, Dutta S. Intravenous Immunoglobulins in RhesusHemolytic Disease. Indian J Pediatr 2003; 70: 697-699.

13. Aggarwal R, Seth R, Paul VK, Deorari AK. High dose Intravenous Immunoglobulin therapy in the treatment of Rhesus HemolyticDisease. J Tropical Ped 2002; 48: 116-117.

14. Urbaniak SJ. ADCC (K cell) lysis of human erythrocytes sensitised with Rhesus alloantibodies. II. Investigation into mechanism of lysis. Br J Haematol 1979; 42: 315-328.
15. American Academy of Pediatrics. Management of hyperbilirubinemia in the newborn infant 35 or more weeks of gestation. Pediatrics. 2004;114(1):297–316.