CASE REPORT

Clinical Significance of Rare Maternal Anti Jk^a Antibody

Kshitija Mittal¹ · Tanvi Sood¹ · Naveen Bansal¹ · Ravneet Kaur Bedi¹ ·
Paramjit Kaur¹ · Gagandeep Kaur¹

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Abstract We hereby report a rare case of HDFN because of antibody to Kidd (Jk) blood group system—anti Jk^a. An EDTA sample of a baby along with mother's sample was received in the Department for Direct Antiglobulin Test (DAT) alongwith blood requisition for double volume exchange transfusion. On blood grouping, baby's and mother's blood group was found to be B Rh D positive. DAT with polyspecific anti human globulin (AHG) was positive. Screening of mother's serum for irregular antibodies showed anti-Jk^a antibody. AHG phase titers using tube technique were 1:64. Mother was found to be Jk^a antigen negative; father and neonate were found to be Jk^a antigenpositive. Antibody was observed to be of IgG type on Dithiothreitol treatment. Baby had total serum bilirubin of 20.5 mg/dl on day 3 of life and phototherapy was started. Exchange transfusion was not required in the baby. The present case emphasises the significance of minor blood group antigens other than Rh blood group system as a cause of HDFN. Although HDFN due to Jk antibodies is

rare, however, the clinician must be aware of the occurrence of these antibodies as they can lead to severe HDFN and persistent anemia in the infant.

Keywords Hemolytic disease of fetus and newborn · Kidd antibody · Total serum bilirubin · Exchange transfusion

Introduction

Hemolytic disease of the fetus and newborn (HDFN) is a condition in which the lifespan of the infants' red blood cells(RBC) is shortened as a result of maternally-derived alloantibodies that are transferred via the placenta and directed against paternally inherited antigens [1]. Though anti D is the most common cause but HDFN due to other antibodies have also been reported. We hereby report a rare case of HDFN because of antibody to Kidd (Jk) blood group system—anti Jk^a.

Case History

An EDTA sample of a 3 day old male baby along with mother's sample was received in the Department of Transfusion Medicine for Direct Antiglobulin Test (DAT)alongwith blood requisition for double volume exchange transfusion. On blood grouping, baby's and mother's blood group was found to be B Rh D positive. DAT with polyspecific (anti IgG and C3d) anti human globulin (AHG) was positive (4 +) using microcolumn gel technique (BioRad, Switzerland). Screening of mother's serum for irregular antibodies using a commercial three cell antibody screening panel (ID-Diacell I-II-III, BioRad, Switzerland) and LISS Coombs gel cards (BioRad,

✉ Ravneet Kaur Bedi
rkbedi15@yahoo.com

Kshitija Mittal
drkmittal@gmail.com

Tanvi Sood
drtanvisood@yahoo.in

Naveen Bansal
lovelybansal099@gmail.com

Paramjit Kaur
paramjit.gp71@yahoo.com

Gagandeep Kaur
docvpsingh@yahoo.co.in

¹ Department of Transfusion Medicine, Government Medical College and Hospital, Chandigarh, India

Switzerland) suggested the probability of anti c, E, K, Fy^b, Jk^a, Le^a, M and anti S. On further testing with 11 cell identification panel (ID-DiaPanel-P, BioRad, Switzerland) and enzyme treatment with bromelin, antibody was confirmed to be anti-Jk^a antibody (Table 1). Autocontrol was negative. AHG phase titers using tube technique were 1:64. Anti Jk^a antibody was identified in eluate obtained from neonatal red cells by heat elution method. On red cell phenotyping, mother was found to be Jk^a antigen negative; father and neonate were found to be Jk^a antigen positive. Antibody was observed to be of IgG type on Dithiothreitol (DTT) treatment. No history of blood transfusion was elicited in the mother. Jk^a antigen negative blood units were crossmatched with mother's and neonate's sample using microcolumn gel technique and found to be compatible.

Term baby delivered through Caesarian section with total serum bilirubin (TSB) 20.5 mg/dl on day 3 of life and phototherapy was started. TSB decreased to 14.5 mg/dl on day 4 and 13.7 mg/dl on day 5 of life. Phototherapy was stopped and exchange transfusion was not required. TSB was again repeated the next day which further decreased to 10 mg/dl and baby was discharged on 6th day of life. Mother was second gravida with one live issue and no history of abortion. There was no history of neonatal jaundice in the elder female sibling. The parents were counselled to get the elder sibling for extended phenotyping, however, they refused.

Discussion

Majority of HDFN reported in literature are due to ABO or RhD incompatibility. Although Jk^a antigens are well developed on RBCs of neonates and have been detected on fetal RBCs as early as 11 weeks but are only rarely responsible for severe HDFN possibly because of poor immunogenicity [2–4]. Though anti Jk^a was identified by Allen et al. in 1951 in the serum of a Mrs. Kidd, whose infant had HDFN [5], but very few cases of anti Jk^a related HDFN have been reported in literature till date. Moreover, case reports from Asia are even rarer due to the low Jk^a allelic frequency [6]. HDFN associated with anti Jk^a is usually mild. However, Matson et al. reported a case of severe HDFN associated with anti Jk^a where the baby developed kernicterus and received exchange transfusion. The antibody appeared to be stimulated by pregnancy [7]. Similarly in our case, mother had high antibody titers of 1:64 and newborn had severe HDFN with TSB increased up to 20 mg/dl. Phototherapy was started. However, exchange transfusion was not required in our case.

Jk antibodies are usually of IgG type thus can cross placenta, bind complement and can cause either rapid intravascular and/or extravascular hemolysis in the infant

Table 1 Results of antibody screen and identification panel on the mother's serum with ID Panel lot no. 06217.67.x, expiry: 23.11.2015

Rh-hr	Kell						Duffy			Kidd		Lewis		P	MNS			Luth		Xg	Results		Enzyme treatment	Auto-control							
	D	C	E	c	e	C ^w	K	k	Kp ^a	Kp ^b	Js ^a	Js ^b	Fy ^a		Fy ^b	Jk ^a	Jk ^b	Le ^a	Le ^b		P ₁	M			N	S	s	Lu ^a	Lu ^b	IS	AHG
SC I	+	+	0	0	+	+	0	+	0	+	nt	nt	+	0	0	+	+	0	+	0	+	+	+	Neg	Neg	Neg					
SC II	+	0	+	+	0	0	0	+	0	+	nt	nt	+	0	0	+	+	0	+	0	+	0	+	Neg	4+	Neg					
SC III	0	0	0	+	+	0	+	+	0	+	nt	nt	0	+	+	0	+	0	+	0	+	+	+	Neg	4+	Neg					
ID 1	+	+	0	0	+	+	0	+	0	+	nt	nt	+	0	0	+	+	0	+	0	+	+	+	Neg	Neg	Neg					
ID 2	+	+	0	0	+	0	+	+	0	+	nt	nt	0	+	0	+	+	0	+	0	+	+	+	Neg	4+	Neg					
ID 3	+	0	+	+	0	0	0	+	0	+	nt	nt	0	+	+	0	+	0	+	0	+	+	+	Neg	4+	Neg					
ID 4	0	+	0	+	+	0	0	+	0	+	nt	nt	0	+	0	+	+	0	+	0	+	+	+	Neg	Neg	Neg					
ID 5	0	0	+	+	+	0	0	+	0	+	nt	nt	+	0	0	+	+	0	+	0	+	+	+	Neg	4+	Neg					
ID 6	0	0	0	+	+	0	+	+	0	+	nt	nt	+	+	0	+	+	0	+	0	+	+	+	Neg	Neg	Neg					
ID 7	0	0	0	+	+	0	0	+	0	+	nt	nt	+	0	0	+	0	+	0	+	0	+	+	Neg	4+	Neg					
ID 8	+	0	0	+	+	0	0	+	0	+	nt	nt	0	0	0	+	+	0	+	0	+	+	+	Neg	Neg	Neg					
ID 9	0	0	0	+	+	0	0	+	+	+	nt	nt	0	+	0	+	+	0	+	0	+	+	+	Neg	4+	Neg					
ID 10	0	0	0	+	+	0	0	+	0	+	nt	nt	0	+	0	+	0	+	0	+	+	+	+	Neg	4+	Neg					
ID 11	0	0	0	+	+	0	0	+	0	+	nt	nt	+	0	0	+	+	0	+	0	+	+	+	Neg	Neg	Neg					

SC screening cell, ID identification, IS immediate spin, AHG anti human globulin

[3]. Studies in the past have reported relatively low hemoglobin levels in infants due to the persistence of the antibody coating the red cells even up to 7 weeks of life [3, 8]. Thus, a close monitoring of the infant's hemoglobin levels for first few months of life is warranted. However, in our case, the patient was lost to follow up.

Kidd (Jk) antibodies are notorious and have been associated with delayed hemolytic transfusion reactions because of their tendency to become undetectable between transfusions following sensitisation. However, a strong anamnestic response is observed on subsequent re-exposure through blood transfusion or pregnancy. About 52 % of Jk antibodies disappear within months in comparison with 27 % of Rh antibodies [9]. Thus, the alloimmunised individuals should be provided special immunohematology antibody card so that the individual can receive lifelong antigen negative blood.

Conclusion

This case illustrates the importance of DAT in all the newborns irrespective of mother's Rh D phenotype and work up in case of DAT positivity. Moreover, it further emphasises the significance of minor blood group antigens other than Rh blood group system as a cause of HDFN. Although HDFN due to Jk antibodies is rare, however, the clinician must be aware of the occurrence of these antibodies as they can lead to severe HDFN and persistent anemia in the infant.

Compliance with Ethical Standards

Conflict of interest None.

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