newborn due to anti c antibodies

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Sri Lanka Journal of Child Health, 2012; **41**(1): 44-45

(Key words: Haemolytic disease of the newborn; HDN; Rh anti c antibodies)

Introduction

The Rh antibody is still the commonest cause of severe haemolytic disease of the newborn (HDN)[1]. Out of the Rh antigens D is the most potent antigen causing HDN. Other common Rh antigens C, c, E and e are weak and do not commonly cause HDN.

Though the maternal alloimmunisation to Rh anti D antibodies is low due to better detection and effective preventive options, other Rh red cell antigens remain a significant but uncommon cause of haemolytic disease of the newborn. Prophylactic immunoglobulins are not yet available to prevent the formation of these antibodies.

Case Report

A 33 year old mother of one living child gave birth to a baby girl at term by elective lower segment caesarean section (LSCS) due to bad obstetric history and past LSCS. There was no consanguinity in the parents. The only child the lady had was a healthy 6 year old girl from the first pregnancy. The second pregnancy was a macerated still birth at 35 weeks of gestation; the third was an intrauterine death (IUD) due to placental abruption, which has necessitated the mother to undergo a blood transfusion. Three units of group specific cross matched blood have been given. Current is her fourth pregnancy.

The antenatal period was uncomplicated and she had regular clinic follow up. The anomaly scan performed at 20 weeks and the repeat abdominal ultrasound scan at 33 weeks, were normal. She had had her rubella vaccination at 18 years of age. The baby had a birth weight of 2300 grams. She did not have any perinatal complications but was found to have icterus by 2 hours after birth and was admitted to the special care baby unit (SCBU).

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(Received on 20 March 2011: Accepted on 30 March 2011)

On admission to the SCBU the baby was found to be in mild respiratory distress. She appeared very pale and had mild jaundice. Abdominal examination revealed hepatosplenomegaly. There was no visible rash or oedema. The baby had persistently low oxygen saturation and bradycardia with mild respiratory distress. Urgent 2D echocardiogram showed severe pulmonary hypertension (PHT) with a large PDA and ostium secundum ASD with bidirectional shunts. She was started on ventilation due to hypoxaemia and severe PHT.

Investigations at 2 hrs of age showed the following:

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Haemoglobin (haematocrit):	61 g/L (20.8%)
Reticulocyte count:	45%
Blood picture:	Severe haemolysis
Serum bilirubin:	9.25 mg/dl

Both the mother and the baby had A Rh D positive blood group. Coombs test was negative. Baby was given an urgent blood transfusion after taking blood for investigations. Once the vital functions were stabilized, she had a double volume exchange transfusion at 26 hours of age. Phototherapy was continued for 10 days afterwards.

The baby was also investigated for congenital infections which was reported negative and for TSH level which was within the normal range. The motherôs and babyôs blood samples were investigated for Rh and minor blood group antibodies. The motherôs blood was positive for anti c antibodies.

Blood grouping and Rh phenotyping was carried out in the family members (Table 1):

Table 1: ***Blood grouping and Rh phenotyping***

	ABO status	Rh status	Rh phenotype
Mother	A	Rh D positive	DCe/DCe (R_1R_1)
Father	O	Rh D positive	DcE/dce (R_2r)
Elder sibling	O	Rh D positive	DCe/dce (R_1r)
Baby	A	Rh D positive	

most problems are due to D, C, c, E and e antigens. Unlike AB antibodies which are mostly IgM, Rh antibodies are mostly IgG thus causing HDN readily. There have been only a few cases reported in the literature of HDN due to anti c antibodies[2,3]. Our patient had clinical and laboratory evidence of severe haemolytic disease of the new born due to anti c antibodies.

To explain the inheritance of Rh system there are two theories. Fisher-Race theory describes the presence of genes D, C, E and their alleles c, d and absence of D and that they are inherited as a group of 3. Weiner describes 8 alleles R^0, R^1, R^2, R^Z, r, rô, rôô, r^y. Now it has been shown that two separate genes (RHD and RHCE) located on the short arm of chromosome 1, encode Rh proteins[4] (Table 2).

Table 2: ***Rh gene complexes, antigens, possible combinations and their percentage in whites***[4]

Haplotypes	Genes present	Antigens present	Phenotype	Percentage
R^1	RHD RHCe	D,C,e	R_1	42%
r	RHce	dce	r	37%
R^2	RHD RHcE	DcE	R_2	14%
R^o	RHD RHce	Dce	R_o	4%
r'	RHCe	dCe	r'	2%
r''	RHcE	dcE	r''	1%
R^z	RHD RHCE	DCE	R_z	<1%
r^y	RHCE	dCE	r^y	<1%

The presence of cde/cde phenotype in the father and the DCe/DCe phenotype in the mother and the previous two losses of intrauterine pregnancies indicate that the likely cause for the HDN was the exposure to c antigen from intrauterine transfusion from the baby. The presence of two C antigens in the mother is likely to have made the response higher thus explaining the severe HDN (dosage effect). Close follow up of the next pregnancy is needed due to high risk of recurrence of severe HDN.

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