

Study on Detection of Rhesus-D Antibody and Titre of Chittagong Division

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A total of 400 cases of Rhesus D negative mother who attended in Transfusion Medicine Department of Chittagong Medical College Hospital were studied to detect the percentage of Rhesus-D antibody and its titre. It was also studied to find out the incidence of different blood groups of ABO system among Rhesus-D negative mothers. It is observed from the study that out of 400 pregnant women 142 (35.50%) were primigravida who have not produced anti-D antibody during their pregnancies. Remaining 258 (64.50%) were multigravida, 185(71.70%) of them had taken injection of anti-D prophylaxis after delivery. The rest 73 (28.29%) multigravida women had not taken any anti-D immune-prophylaxis, 7 (9.58%) had developed anti-D antibody. Out of 7 cases 2 women's antibody titre reached a level which causes haemolytic disease of new-born, treated with phototherapy and exchange transfusion. It is also observed from our study that Blood group B is the commonest among the Rhesus negative mothers.

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Introduction

Blood groups are genetically determined which cannot be changed.¹ All blood group systems are not equally important from clinical point of view. The clinical importance of a blood group antigen depends on the frequency of occurrence of corresponding antibody and their ability to haemolyse the red cells in vivo.² On the basis of the criteria among 23 blood group systems, ABO and Rhesus blood group system are of major clinical importance because they produce haemolytic disease of the new born (HDN) and haemolytic transfusion reaction (HTR).³ In Rhesus blood group system there are 50 blood group antigens of which D is the most immunogenic.⁴ Rhesus antigen is present only at RBC membrane.

Passage of Rh-D positive blood to the Rh-D negative mothers either by trans-placental haemorrhage or transfusion, sensitise the women to produce the anti-D antibody. All negative mothers don't produce antibody necessarily after trans-placental haemorrhage. Rhesus-D antibody production depends upon some factors like amount of D positive blood transfused, Rh phenotype and sex of the fetus. Another most important factor for antibody production is 'responder' or 'non-responder' status of the Rh-D negative mothers. 'Non-responder' means repeated stimulation by Rh-D positive blood fails to produce antibody development. The purpose of our study is to find out the incidence of Rh-D antibody production and its titre.

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Methods

A total of 400 mothers were studied in Transfusion Medicine Department of Chittagong Medical College Hospital from August 1992 to June 1994 for detection of Rh-D antibody and titre. Among them 142 (35.50%) were primigravida and 258 (64.50%) were multigravida. They were of 16 years to 44 years of age. All the mothers had their blood investigated three times during their pregnancy period; first at 12-16 weeks, 2nd at 28-32 weeks and finally at 36-37 weeks of pregnancy. The mothers who transfused Rh-D positive blood were excluded from our study. With all aseptic precautions 5cc of venous blood were collected in a clean and dried test-tube. Sera were taken for antibody detection and estimation of titre. Fresh, citrated R1r/R1R2 '0' -Rh-D positive red cells were taken for in vitro sensitization. The results were observed both macroscopically and microscopically. ABO and Rhesus blood groups were done in standard agglutination method.

Results

The result showed among the 400 Rh-D negative mothers. 142 (35.50%) were primi and 258 (64.50%) were multigravida.

Out of 142 primi, none had developed Rh-D antibody upto the end of delivery. Among 258 multigravida, 185 (71.70%) had taken injection anti-D immunoglobulin within 6 hours to 72 hours after delivery. Remaining 73 (28.29%) multigravida did not have anti-D immunoglobulin prophylaxis and 7 (9.58%) of them developed anti-D antibody (Table I).

Anti-D titre raised at peak during the end of pregnancy (Table I). Out of 7 detected cases who developed Anti-D antibody, did not have prophylaxis and 2 developed haemolytic disease of new-born.

Table I: Antibody titre at different weeks in 7 cases of multipara

At 12-16 weeks	At 28-32 weeks	At 36-38 weeks
(n=2) 1:4	(n=3) 1:2	(n=1) 1:4
	(n=2) 1:4	(n=2) 1:16
	(n=2) 1:16	(n=2) 1:32
		(n=2) 1:64

n=number of mothers

Out of 400 Rh-D negative women, 166 (41.50%) were Group B, 112 (28%) were Group a, 91 (22.75%) were Group A and 31 (7.75%) were AB (Table II).

Table I: Incidence of ABO groups in Rh-D negative mothers

Blood Group	Numbers of Women	Percentage (%)
B	166	41.50
O	112	28.00
A	91	22.75
AB	31	07.75
Total	400	100

Discussion

Rhesus-D antibody production to Rh-D negative mother are either due to transfusion of Rh-D positive blood or by transplacental haemorrhage. It is first shown by Chown in 1954. Transplacental haemorrhage occurs during normal delivery, Caesarean section or manual removal of placenta during delivery. Other causes of transplacental haemorrhage are maternal trauma. Menstrual Regulation (MR), abortion, miscarriage, procedure which damage the foetus or placenta like amniocentesis, external version, chorionic villus sampling or placental chorioangioma.

Although foetal and maternal circulation are separated but during delivery or above conditions, transplacental haemorrhage occurs.⁵ The passage of foetal red cells possessing an antigen lacking in mother's blood which has inherited from father, cross the placenta into mother's circulation. This

sensitizes the mother and provokes the immune-competent cells (ICC) to produce antibody. This type of antibody is of IgG class which crosses the placenta, coats the foetal red cells and produce haemolytic disease of the new-born.

The minimum dose for primary immunization is 0.5 ml of R1R2 or R1r positive blood.⁶ It is found that a single dose of 1 ml Rh-D positive red cells produce anti-Rh antibody less than 50% of cases. Usually large dose of antigens elicit the best response. During normal delivery, a trace amount of foetal blood enters into mother's circulation. Bredenoen in 1953 found less than 1% antibody production in primi. But in our study, it is found that an antibody is developed even at the end of full term pregnancy. Sometimes it would appear that about 33% of Rh-D negative mothers are incapable of producing anti-D antibody by repeated passage of Rh-D positive cells.⁷

Blood group B has the highest incidence in Bangladeshi population. It is observed that there are more B negative mothers than other groups. In our study, it is revealed that one dose anti-D immunoglobulin reduce much Rhesus D immunization to negative mothers. It is concluded that proper antenatal check up and by both obstetricians and blood transfusion specialist would prevent immunization of Rhesus D negative mother and also prevent haemolytic disease of the new-born baby.

References

1. Marcela contraries et al. Blood Group serology, in AV Hoffbrand and SM Lewis (editors). Post Graduate Haematology, 3rd edition Delhi, CBS, 1991:208.
2. Dacie Sir John V & Lewis SM, Practical Haematology, 8th editions ELBS with Churchill Livingstone. 1994:445.
3. Frank firkin, Colin Chesterman et al. de Grauchy's Clinical Haematology in

- Medical practice. 5th edition, Delhi, Oxford University Press, 1989-479.
4. Branch R. Donald Ph. D. Dzik H Walter MD. AABB Technical Manual 11th edition. 1993: 23 & 651-53.
5. Rahman M. Guide to Blood Transfusion, 1st edition, 1978:72.
6. Mollision PL, Engelfried CP and Contretrees M Blood Transfused in Clinical Medicine, 8th edition, London: Blackweil, Scientific Publication, 1988:351-356,669.
7. Boorman Kathleen E, Dodd Barbara E. et al Blood Group Serology, 6th edition London Churchill Living Stone Publication. 1988: 28-30.
8. Hollborn AM & Prior DM, Clinical Haematology The UK National External Quality Assessment scheme Blood Group Serology, 1983;8.
9. Sturgeon, P & Jennings, E.ZR. Transfusion Anti & Globulins in women treated with anti-Rh & globin: 1968; 8, 343.