

A REVIEW OF ABO ISOIMMUNISATION IN PREGNANCY

by

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SUMMARY

Incidence of ABO isoimmunisation was found in 5 out of 40 heterospecific cases (12.5%). High incidence of ABO isoimmunisation were associated in A group infants (83.33%). Only one out of five cases of ABO haemolytic disease of newborn required exchange transfusion.

Introduction

The haemolytic disease of the newborn due to ABO isoimmunisation can only occur when the pregnancy is heterospecific viz—if the mother's group is O and the foetus group A or group B. It shows that the foetal red cells of A or B group cross the placenta into the circulation of O group mother from about 24th week of gestation, stimulating the existing anti-A or anti-B to high titres. The immune anti-A then crosses the placenta into the foetal circulation, where it combines with foetal red cells, leading to their destruction (Bryant, 1982).

The diagnosis of ABO isoimmunisation in pregnancy which causes haemolytic disease of the newborn remains far from easy despite the major breakthroughs have been made in the study of Rh haemolytic disease. There is no definitive test available to diagnose ABO haemolytic disease. This is mainly because the ABO antibodies

are present in normal individuals and direct coombs test on the infant is negative (Gupta and Bhatia 1973). In this communication we present our experience with various hamatological and serological tests carried out for studying the ABO isoimmunisation.

Material and Method

The present study consists of 540 consecutive cases attending antenatal clinic of this hospital during the year 1982-83. All the cases were screened at random for ABO blood group and Rh typing. Of these 200 were found to be blood group O. Out of these only 40 were taken up as test cases where mothers gave birth to babies of other than group O. Saline Anti-A and anti-B titer of the mother was estimated by master dilution method at 38 weeks gestation period and during post-partum period. There was one pair of twins so that forty one babies comprised for the present study.

The cord blood of the baby was collected for the study of ABO and Rh(D) grouping, haemoglobin estimation, serum bilirubin estimation and direct coombs test.

All the newborn babies were carefully watched for signs of jaundice. The manage-

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ments of the affected babies were done with the help of neonatologists.

Observations

In the present study, 5 cases (12.5%) were found sensitised out of 40 hysterospesific mothers (out of total 540 cases). Clinicopathological profiles of 5 cases are shown in Table I. Two cases (40%) were primiparae, 1 case (20%) from para 2 to 4 and 2 cases (40%) from para 5 and above.

In the present series, out of 40 hetero-specific deliveries (one twin delivery), 22 (53.65%) babies were male and 19 (46.35%) were female. In immunised group, out of 6 immunised cases (one twin delivery), 5 male were affected and 1 female reflecting the male to female ratio 5:1. 5 cases (83.33%) are group A, and 1 case (16.67%) is group B.

There was increase of isoagglutinin titre in immediate postpartum period. Titre in antenatal period could not be done in one case as the case was not booked. In 4 sensitized mothers anti-A titre was more than that of anti-B. Of the 6 immunised babies, 4 babies (66.6%) were in between 2.0 to 3.0 kg and 2 (33.4%) were in between 1.0 to 2.0 kg at birth.

The cord serum bilirubin level in all immunised cases was above 6.5 mg%. In the first case, serum bilirubin level was 23.0 mg% on the third day of birth. Baby required two exchange transfusion to keep the level of bilirubin less than 20 mg%. In the second case it was 8.0 mg%, where exchange transfusion could not be performed due to nonavailability of compatible blood in time. In third and fourth case it was 7.5 mg% and 6.5 mg% respectively, while in the 5th case (twins), it was 12.5 mg% and 12.0 mg% respectively.

In all immunised cases, the haemoglobin

level was below 14.2 gms%. Direct coombs test was negative in all cases.

Discussion

The incidence of ABO isoimmunisation given by different workers varied from 5.0 to 17.6% (Rosenfield 1955, 11.0%; McElfresh 1960, 13.0%; Robinson *et al* 1960, 5.0%; Oski and Naiman 1972, 10.0%; Raha *et al* 1977, 17.6%. Desjardins 1979, 5.0%; Jain *et al* 1983, 8.7%). In the present study the incidence of ABO isoimmunisation was 12.5% (5 cases out of 40).

In ABO sensitization, firstborn infants are affected in about 50% of the cases (Mollison, 1972). In the present series, similarly, 50% (3 out of 6 babies) of the affected babies were firstborn. Since ABO antibodies are naturally occurring, ABO isoimmunisation occurs in first incompatible pregnancy (Levine and Rosenfield, 1961).

Nearly all the affected babies due to ABO isoimmunisation are group A, but blood group B babies can also be affected (Mollison 1972; Levine and Rosenfield 1961). In the present study 5 cases (83.33%) are group A, and 1 case (16.67%) is group B.

Serum isoagglutinin titre (Anti-A and anti-B) was appreciably increased in immediate postpartum period. Titre above 1/64 was taken arbitrarily as high titre (Roha *et al* 1977). In the present series anti-A titre is more in 4 cases and anti-B titre is more in one case only.

Gupte *et al* (1975) observed that the incidence of ABO incompatibility in low birth weight (LBW) babies was not significantly higher than among full term babies. In our study LBW of 2 immunised babies weighing between 1 to 2 kg can be attributed to their twin birth.

TABLE I
Clinico-pathological Profile of 5 Cases of ABO Isommunisation

Case No.	Age in years	Gravida	Mother		Blood Group			Mother titer			Mode of delivery
			No. of affected baby	Mother	Father	Baby	Anti-A	Anti-B	Anti-A	Anti-B	
Time of onset of jaundice in hours	Sex	Weight in kg.	Serum Bilirubin mg%	Cord Blood mg%	Maximal conc. in mg%	Hb in gms%	Direct Coombs' test	Number of Exchange transfusion	Foetal outcome		
1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
1.	30	5	3	O+	A+	A+	—	—	1:128	1:32	FTND
2.	24	5	—	O+	A+	A+	1:128	1:64	1:356	1:32	-do-
3.	19	1	—	O+	A+	A+	1:64	1:32	1:128	1:32	-do-
4.	29	2	—	O+	B+	B+	1:32	1:64	1:32	1:128	-do-
5.	20	1	—	O+	A+	A+	1:128	1:64	1:256	1:128	-do-
13.	14.	15.	16.	17.	18.	19.	20.	21.			
1. Within 24 hrs.	M	2.200	—	26.2	12.2	—	2	Well			
2. -do-	F	2.600	8.0	28.5	13.5	Negative	—	Well			
3.	32	M	2.400	7.5	10.0	14.2	-do-	Well			
4.	30	M	3.000	6.5	15.5	13.0	-do-	Well			
5.	24	M	1.750	12.5	—	11.5	-do-	Well			
	24	M	2.000	12.0	—	13.6	-do-	Well			

Robinson (1960) thought that a cord serum bilirubin value of 3.0 mg% was highly suggestive of ABO haemolytic disease. However, in the series of Haque (1978), 79.1% infants who had concentrations under 3.0 mg% required treatment by phototherapy. Hence he considers that the cord blood bilirubin level is unreliable for predicting hyperbilirubinaemia due to ABO incompatibility. In the present series, cord blood bilirubin level ranged from 6.5 mg% to 12.5 mg%.

In the series of Mannen *et al* (1963), 11 out of 22 cases of ABO haemolytic disease of newborns required exchange transfusion. In the present study, only one out of five cases required two exchange transfusions.

Because the manifestation of the ABO haemolytic disease are variable, the gynaecologist and neonatologist require acumen to detect the disease early as it may lead to kernicterus.

ABO incompatible couple (Woman O or Man A or B), attending genetic counselling clinic may be explained about possible risk of congenital haemolytic disease to offspring due to ABO incompatibility.

References

1. Bryant, N. J.: An introduction to immuno-haematology, 2nd Edition, W. B. Saunders Co., 1982.
2. Dasjardins, L.: *J. Paediat*, 95: 75, 1974.
3. Gupte, S. C., Kothari, J. and Bhatia, H. M.: *Ind. Ped.*, 12: 477, 1975.
4. Gupte, S. C. and Bhatia, H. M.: *Ind. J. Med. Res.*, 61: 1336, 1973.
5. Haque, K. N.: *Brit. Med. J.*, 2: 1604, 1978.
6. Jain, P. C., Bhala, A., Singh, S. H. and Rohatgi, P.: *J. Obstet. Gynec. India*, 33: 32, 1983.
7. Levine, H. and Rosenfield, R. E.: ABO incompatibility Progress in Medical Genetics. Edited by A. G. Steinberg. Grune and stratton Inc. 120 USA, 1961.
8. Mammen, K. C., Jadhao, M. and Webb, J. K.: *Ind. J. Child Health*, 12: 297, 1963.
9. McElfresh, A. E., Kurkuoglu, Vaughan: *J. Paediatrics*, 56: 39, 1960.
10. Mollison P. L.: Blood transfusion in clinical Medicine, Blackwell scientific Publication, Edinburgh, 1972.
11. Oski and Naiman: Haematological problems in the Newborn, Vol. IV, W. B. Saunders, 1972.
12. Raha, P. K., Mitra, A. K. and Ghosh, M.: *J. Obstet. Gynec. India*, 27: 899, 1977.
13. Rosenfield, R. E.: *Blood*, 10: 17, 1955.
14. Robinson, G. C., Dynn, H. G. and Wong, L. C.: *Acta. Ped.*, 49: Suppl. 120: 53, 1960.