Distribution of Blood Group Antigens and Isoagglutinins among Women of Varying Parity Levels in Owerri, Nigeria: Implications for Maternal and Child Health

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Abstract

Red blood cells and sera from 236 pregnant women (primigravid and multigravid) and 10 women with no history of pregnancy or blood transfusion (controls) were tested for blood group antigens [A, B, and Rh(D)] and isoagglutinins, respectively, using the tube agglutination technique. Information concerning age and pregnancy history – numbers of pregnancies, deliveries, miscarriages, stillbirths, premature deliveries, jaundice and/or death of neonate – were volunteered by the women. The mean age of the pregnant women was 28.4 ± 4.8, range, 18-47; and the mean number of pregnancies per woman (to the nearest whole number) was 3.0, range, 1-11. Blood group distribution among the 246 women including the controls was, O, 55.7%; A, 29.7%; B, 13.8%; AB, 0.8%; $Rh(D)^+$, 92.3%, and $Rh(D)^-$, 7.7%. Overall mean log_2 anti-A titer was 3.3 ± 1.5 with a range of 1-8; while anti-B was 3.8±1.2, range 1-7. Relatively, B antigen evoked higher antibody titers than A antigen. Higher anti-A and anti-B agglutinin titers were recorded among the control women than among the pregnant women. Rates of miscarriage were not significantly different in $Rh(D)^+$ (73.3%) and $Rh(D)^-$ women (68.4%). The probable effects of blood group incompatibility between mother and fetus are discussed.

Keywords: Blood group, ABO, Rh (D), antigens, parity status, isoagglutinin, abortions, pregnant women, Nigeria.

Introduction

Several blood antigens have been recognised but the most important ones, clinically, are the ABO and Rh(D) antigens. The A and B antigens are carbohydrate structures found not only on the surface of red blood cells but also on cells in various tissues and organs throughout the human body and secretions (1). They are recognised as major alloantigens and potent immunogens in individuals who lack them (2). Thus, their corresponding antibodies cause harmful immune reactions, thereby acting as histocompatibility barriers to successful red blood cell transfusion and tissue and organ transplantation (3).

Perhaps, the most serious problem associated with blood group antigens is the hemolytic disease of the newborn (HDN), which arises from incompatibility in blood group antigens between a pregnant woman and her fetus. Although the highest percentages of HDN cases may be due to ABO incompatibilities, most of them are very mild and subclinical. This is presumably so because the major group antibodies of A and B mothers are principally of the IgM class, which does not cross the placenta. However, the antibodies of the group O mothers include the IgG class, which cross the placenta. Thus, despite the fact that 23% of infants are ABO incompatible with their mothers, only about 0.67% of detectable HDN cases are due to antibodies of the ABO system (4). A large majority of the HDN cases and transfusion reactions that are not explicable on the basis of ABO incompatibility are due to reactions against Rh(D) antigens, i.e. due to IgG Rh antibodies. The anti-D antibodies do not fix or activate complement but bring about red cell destruction in the erythroblastotic fetus by preparing the cells for adherence to macrophages of the reticuloendothelial system, thereby rendering them osmotically fragile. The ensuing excessive lysis of the fetal red blood cells (rbcs) can lead to different clinical manifestations including abortions, stillbirths, jaundice of the newborn (*icterus gravis neonatorum*), deposition of bilirubin in the basal nuclei of the brain (*kernicterus*) and severe anemia.

Several cases of the above clinical conditions, particularly high abortion rates, stillbirths and neonatal jaundice are continually reported in Nigerian hospitals but there are no investigations to determine the causes. This work is a surveillance of the different blood group antigens among pregnant women in Owerri, Nigeria, with a retrospective survey of incidents of abortions, stillbirths and neonatal jaundice aimed at evaluating the probable risk of HDN among neonates in the sample population.

Materials and Methods

Sample Population: The sample population consisted of 236 pregnant women aged 18 – 47 years, who were attending the antenatal clinic at the Federal Medical Centre (FMC), Owerri. Only those booking that particular pregnancy for the first time were included. The personal data requested and

voluntarily supplied included age, marital status, number of pregnancies to date, outcome of the pregnancies (miscarried or completed and delivered), number of stillbirths or deaths shortly after birth, cases of neonatal jaundice and other clinical symptoms. Ten (10) controls were included and consisted of women aged 17 – 35 with no history of pregnancy or blood transfusion. These were mostly female students of tertiary educational institutions in Owerri.

Collection and processing of blood samples: Approximately 5.0 ml amount of venous blood was drawn with a syringe and needle. A 2.5 ml volume was discharged into a sequesterene bottle while the remaining 2.5 ml was put in a sterile plain specimen bottle. The sequesterene bottle was prepared by allowing 0.04 ml of 10% (w/v) EDTA in a sterile clean bottle, covered with a thin muslin cloth, to evaporate to dryness at room temperature.

Immediately after discharging the blood sample into the sequesterene bottle the contents were briskly and gently mixed to prevent clotting. The uncoagulated blood was transferred to a 10-ml centrifuge tube and the rbcs washed three successive times by alternate centrifugation and re-suspension of cells in isotonic saline. The packed (washed) rbcs were re-suspended and diluted to a working concentration of 2 % in isotonic saline and kept chilled at +4° C.

The 2.5 ml volume of blood in the sterile plain bottle was allowed to clot for about 2hr before centrifugation at 3000 rpm for 5 min to separate the serum. The serum was stored at –20° C in a clean sterile bottle. All blood samples were assayed within 24 hours of collection.

Determination of blood group of sample population: The antigenic group of each blood sample was assayed by the tube agglutination method described by Baker and Silverton (5).

ABO grouping: Two sets of experiments were carried out. One was to group the patients' sera using, as test reagents, standard A, B and O rbcs obtained from appropriately grouped donors. The other was to determine the blood group of each patient's rbcs using standard anti-A and anti-B sera and high tittered group O serum containing anti-A and anti-B antibodies (Wellcome).

Three rows of precipitin tubes (each of dimension, 50mm x 7mm) were arranged in a metal rack and labeled A, B and O, respectively. Using a Finnpipette and changing pipette tip after each addition, 50µl of the 2l% suspension of appropriate standard rbcs, A, B or O, was put into each tube. Then, 50µl of each patient's serum was added to duplicate tubes in a row. In the second experiment, three rows of precipitin tubes marked anti-A, anti-B and anti-AB (group O serum), respectively, were arranged. To duplicate tubes in a row were added 50µl of corresponding standard antiserum (anti-A, anti-B or group O serum, respectively). Subsequently, equivalent volume (50µl) of 2 % washed (patient's) rbc was added. Finally, an auto-control experiment consisting of 50 µl of each patient's serum added to 50µl of another patient's rbcs, in different combinations, was set up. The contents of the tubes were mixed by gently tapping the metal racks and the tubes incubated at room temperature (≅27° C) for 90 min before examining the tubes for agglutination, starting with the controls. Contents of tubes showing no agglutination were transferred to a cavity slide and examined microscopically using a x10 or x40 objective.

Rh (D) grouping: To each precipitin tube in a row was added 50 µL of standard group O serum (Wellcome). Then, 50µL of each patient's rbcs was added to duplicate tubes. Positive and negative controls consisting of known O-Rh(D)$^+$ and AB-Rh(D)$^-$, respectively, were included in each batch of experiments. Tube contents were mixed and incubated as described above but for 60 min and examined for agglutination. To tubes showing no agglutination, 100 µL of 30% bovine serum albumin (Sigma) was gently run down the side without disturbing the settled cells. Each was re-incubated at 37° C in a water bath for 60 min. Thereafter, the cells were resuspended and re-examined both macroscopically and microscopically for agglutination. For those tubes that still showed no agglutination, the cells were re-washed 4 times and supernatant completely drained. Then 50µl of anti-human globulin was added followed by centrifugation at 1000 rpm for 1 min, before macroscopic and microscopic examination for agglutination.

Determination of antibody (anti-A and anti-B) titers of patients' sera: Anti-A and anti-B titers were determined for 246 serum samples using the method described by Dacie and Lewis (6). Two-fold serial dilutions of each patient's serum were made in precipitin tubes with isotonic saline as diluent. Each row consisted of 11 tubes; the first tube in each row contained undiluted (neat) serum sample while the eleventh, containing saline and no serum, was the cell control. After dilution, 50µL of 2% A or B red cell suspension was added to all the tubes, including the cell controls. All experiments were in duplicates. Contents of the tubes (serum or saline and red cells) were mixed by tapping the tube-rack gently before incubation at 37° C in a water bath for 90 min. The antibody titer was recorded and expressed as $\log_2$ of the reciprocal of the last serum dilution showing agglutination.

Results

The sample population consisted of a total of 246 women of childbearing age (range 17-48 years).

Table 1: Age Distribution of Number of Pregnancy among the 236 Pregnant Women Sampled

Age Range	Number screened	Number of Pregnancies (%) 1	2	3	4	5	6	≥7	χ± SD	Range
≤ 20	10 (4.2)	8 (13.8)	1 (2.1)	0 (0.0)	1 (3.5)	0 (0.0)	0 (0.0)	0 (0.0)	1.3 ± 0.9	1 - 4
21- 30	158 (66.9)	41 (70.7)	36 (76.6)	42 (89.4)	14 (48.3)	8 (53.3)	9 (47.4)	8 (40.0)	2.8 ± 1.8	1 - 9
31- 40	67 (28.4)	9 (15.5)	10 (21.3)	35 (10.6)	15 (51.7)	7 (46.7)	10 (52.6)	11 (55.0)	4.3 ± 2.4	1-10
41-50	1 (0.4)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (5.0)	11.0± 0.0	11
Total	**236 (100)**	**58 (100)**	**47 (100)**	**47 (100)**	**30 (100)**	**15 (100)**	**19 (100)**	**20 (100)**	**3.2 ±2.2**	**1 - 11**

Note: Average age of child bearing women = 28.4± 4.8; Age range = 18-47; Total number of pregnancies recorded = 762

Table 2: Age Distribution of Pregnancies, Deliveries and Abortions among the 236 Pregnant Women Sampled

Age Range	Number screened (%)	Pregnancies Number (%)	(χ± SD)	Deliveries Number (%)	(χ± SD)	Number (%) Abortions
≤ 20	10 (4.2)	14	(1.4 ± 0.9)	3 (21.4)	0.3 ± 0.6	11 (78.6)
21- 30	158 (66.9)	445	(2.8 ± 1.8)	271 (60.9)	1.7 ± 1.7	174 (39.1)
31- 40	67 (28.4)	289	(4.3 ± 2.4)	208 (71.97)	3.1 ± 2.2	81 (28.0)
41-50	1 (0.4)	11	(11.0 ± 0.0)	10 (90.9)	10.0 ± 0.0	1 (9.1)
Total	**236 (100.0)**	**759**	**(3.2 ±2.2)**	**492 (64.8)**	**2.1 ± 1.2**	**267 (35.2)**

Table 3: Age distribution of Blood–group Antigens among women of childbearing age

Age Range	Number[a] (%) screened	Frequency occurrence of Blood-group Antigen (%) A	B	AB	O	Rh (D)$^+$	Rh (D)$^-$
≤20	12 (4.9)	7 (58.3)	0 (0.0)	2 (16.7)	3 (25.0)	10 (83.3)	2 (6.7)
21 - 30	162 (65.9)	49 (30.2)	24 (14.8)	0 (0.0)	89 (54.9)	154 (95.1)	8 (4.9)
31 - 40	71 (28.9)	17 (23.9)	10 (14.1)	0 (0.0)	44 (62.0)	62 (87.3)	9 (12.7)
41 - 50	1 (0.4)	0 (0.0)	0 (0.0)	0 (0.0)	1 (100.0)	1 (50.0)	1 (50.0)
Total	**246 (100.0)**	**73 (29.7)**	**34 (13.8)[b]**	**2 (0.8)**	**137 (55.7)**	**227 (92.3)**	**19 (7.7)**

[a] This includes the 10 control women who had never been pregnant or transfused with blood or blood-products. [b] Blood group Index (I) = A + AB/B + AB = 29.7 + 0.8/13.8 + 0.8 = 30.5/14.6 = 2.09 (Note: The European Caucasians index is ≥2.5)

Of this number, 236 were pregnant women registering and attending antenatal clinic first time for the current pregnancy and 10 (control) were women who had never been pregnant and had never been transfused with whole blood or blood product. The number of pregnancies among the 236 pregnant women varied from 1 to 11 with an average of 3.2±2.2 pregnancies per woman (Table 1). The pregnant women could be categorised into four age groups, ≤20, 21-30, 31-40 and >40 years, respectively. The average age of the pregnant women was 28.4±4.8 years. The mean number of pregnancies per woman varied with age from 1.3±0.9 among the ≤20 year olds to 11.0±0.0 for a 49 year old woman, the single case >40 years of age. Table 2 shows the distribution of pregnancies, deliveries and miscarriages among the different age groups. The highest number of pregnancies was among women aged 21-30 years with a mean of 2.8±1.8 (i.e. ≅3.0) followed by the 31-40 year age group, which had a significantly higher mean of 4.3±2.4 (≅4.0; $P<0.05$). The highest proportion of miscarriages (78.6%) occurred among the ≤20 age group followed by the 21-30 age group (39.1%).

Of the 246 women in the sample population, including the 10 controls, 73 (29.7%) had blood group sA antigen, 34 (13.8%) group B, 2 (0.8%), AB while 137 (55.7%) belonged to blood group O. There were 227 (92.3%) Rh(D)$^+$ and 19(7.7% RhD$^-$) in the whole sample population (Table 3).

The anti-A or anti-B agglutinin titers varied with parity status among the 236 pregnant women, as shown in Table 4. The range of anti-A titers was 2.81.8 in the women who had experienced 4 pregnancies to 3.6±1.7 among women with 6 and ≥7 pregnancies, respectively. No specific pattern of increase or decline of antibody titers with parity emerged; for example, while women with 4 pregnancies had mean $\log_2$ anti-A titer of 2.8±1.8, those with 1 or 2 had titers of 3.6±1.3 and 3.4±1.3, respectively. Similar pattern was observed for anti-B titers in groups of women of the various parity levels. The mean $\log_2$ anti-B titers ranged from 3.4±0.7 among women with history of 5 pregnancies to 4.3±1.2 among women with 6 pregnancies. Overall mean $\log_2$ anti-A titers was 3.3±1.5 with a range of 1-8 while mean anti-B titer was 3.8±1.2, range, 1-7. Summary of mean $\log_2$ antibody titers in different ABO blood groups shows that the B antigen stimulated higher antibody response both in individuals possessing O cells and individuals possessing A cells

Table 4: Distribution of Log_2 Titers of Antibody to A and B Blood Antigens according to Pregnancy Status and Blood Group

Pregnancy Status	Number (%) screened	Blood Group	Number (%)/Blood Group	Mean log_2 antibody titer ± SD Anti-A	log_2 titer range	Anti-B	log_2 titer range
1	58 (24.6)	O	31 (53.4)	3.7 ± 1.4	1 - 6	3.8 ± 0.9	1 - 5
		A	22 (37.9)	-	-	5.2 ± 1.9	1 - 6
		B	4 (6.9)	2.8 ± 0.4	2 - 3	-	-
		Sub-total	**57 (98.3)**	**3.6 ± 1.3**	**1 - 6**	**3.8 ± 1.2**	**1 – 6**
2	47 (19.9)	O	28 (59.6)	3.6 ± 1.3	1 - 7	3.9 ± 1.1	2 - 6
		A	9 (19.1)	-	-	3.4 ± 0.9	2 – 5
		B	10 (21.3)	3.4 ± 1.7	2 - 7	-	-
		Sub-total	**47 (100)**	**3.4 ± 1.3**	**1 - 7**	**3.8 ± 1.0**	**2 – 6**
3	47 (19.9)	O	24 (51.1)	3.7 ± 1.1	2 - 6	3.2 ± 1.4	4 - 6
		A	16 (34.0)	-	-	3.9 ± 0.7	3 – 5
		B	7 (14.9)	2.3 ± 0.6	1 - 3	-	-
		Sub-total	**47(100)**	**3.4 ± 1.7**	**1 - 6**	**3.5 ± 1.3**	**1 – 6**
4	31 (13.1)	O	13 (41.9)	3.9 ± 1.9	1 - 8	4.1 ± 1.6	1 - 7
		A	10 (32.3)	-	-	3.6 ± 1.2	2 - 5
		B	7 (22.6)	2.4 ± 0.9	1 - 3	-	-
		Sub-total	**30 (96.8)**	**2.8 ± 1.8**	**1 - 8**	**3.9 ± 1.5**	**1 – 7**
5	14 (5.9)	O	11 (78.6)	3.0 ± 1.0	2 - 5	3.4 ± 0.8	2 - 5
		A	3 (21.4)	-	-	3.3 ± 0.5	3 - 4
		B	0 (0.0)	0	0	-	-
		Sub-total	**14 (100)**	**3.0 ± 1.0**	**2 - 5**	**3.4 ± 0.7**	**2 – 5**
6	19 (8.1)	O	10 (52.6)	4.0 ± 1.7	2 - 6	4.6 ± 1.3	3 - 6
		A	6 (31.6)	-	-	3.8 ± 1.4	2 - 6
		B	3 (15.8)	2.3 ± 0.8	1 - 3	-	-
		Sub-total	**19 (100)**	**3.6 ± 1.7**	**1 - 6**	**4.3 ± 1.2**	**2 – 6**
≥7	20 (8.5)	O	15 (75.0)	3.7 ± 1.7	1 - 7	3.9 ± 1.6	2 - 7
		A	4 (20.0)	-	-	4.7 ± 1.7	3 - 7
		B	1 (5.0)	2.0 ± 0.0	2	-	-
		Sub-total	**20 (100)**	**3.6 ± 1.7**	**1 - 7**	**4.0 ± 1.6**	**2 – 7**
Grand-Total	**236 (100)**		**234**[a]	**3.3 ± 1.5**	**1 - 8**	**3.8 ± 1.2**	**1 - 7**

[a] *Number excludes the 2 AB-positive women*

Table 5: Summary Distribution of log_2 Antibody Titer in different (blood) Antigenic groups (A, B and O)

Antigenic Group	Mean log_2 antibody titer ± SD Pregnant women (N = 236)		Non-pregnant women, Control (N = 10)	
	Anti-A	Anti-B	Anti-A	Anti-B
O	3.7 ± 1.5	3.8 ± 1.2	4.4 ± 2.2	4.8 ± 1.3
A	0.0	4.0 ± 1.2	0.0	5.7 ± 2.1
B	2.5 ± 1.1	0.0	5.3 ± 1.7	0.0

than A antigen did in individuals with O and B cells, respectively. Furthermore, anti-A and anti-B titers were higher in the control cases that had neither history of pregnancy nor of transfusion of blood cells or products (Table 5).

Table 6 shows the prevalence of abortions in pregnant women of different blood grouped – A, B, AB, O, $Rh(D)^+$ and $Rh(D)^-$. Subtracting primigravid data from the overall pregnancy rate leaves a difference of individuals with the likelihood of primary sensitization with $Rh(D)^+$ cells and likely development of anti-D antibodies. On the whole, 91.9% of the 236 pregnant women had Rh(D) antigens on their red cells whereas 8.1% had none.

Table 6: Prevalence of abortion among pregnant women of different ABO and Rh_o Blood Groups

ABO Groups	Number of pregnant women	No. D^+ (%)	Frequency of miscarriages among Rh Groups			No. D^- (%)			
			Miscarriages				Miscarriages		
			Overall (%)	Primigravidae (%)	Difference (%)		Overall (%)	Primigravidae (%)	Difference (%)
A	69	66 (95.7)	66 (95.7)	23 (33.3)	43 (62.3)	3 (4.3)	3 (4.3)	0 (0.0)	3 (4.3)
B	32	30 (93.8)	29 (90.6)	4 (12.5)	25 (78.1)	2 (6.3)	2 (6.3)	0 (0.0)	2 (6.3)
AB	2	1 (50.0)	1 (50.0)	0 (0.0)	1 (50.0)	1 (50.0)	1 (50.0)	1 (50.0)	0 (0.0)
O	133	120 (91.9)	116 (87.2)	26 (19.5)	90 (67.7)	13 (9.8)	13 (9.8)	5 (5.8)	8 (6.0)
TOTAL	**236**	**217 (91.9)**	**212 (89.8)**	**53 (22.5)**	**159 (67.4)**	**19 (8.1)**	**19 (8.1)**	**6 (2.5)**	**13 (5.5)**

[a] The rate of miscarriage among RhD^+ women with history of ≥2 pregnancies = 159/217 or 73.3% [b] The rate of miscarriage among RhD^- women with history of ≥2 pregnancies = 13/19 or 68.4%

Approximately, 73.3% $Rh(D)^+$ women with ≥2 pregnancies had abortions compared with 68.4% $Rh(D)^-$ individuals. The difference between the two Rh(D) groups was not statistically significant ($P < 0.05$).

Discussion and Conclusions

Among the 236 pregnant women studied, the mean number of pregnancies was ≅3.0 with the highest rate among persons aged 31-40 (mean parity, ≅4.0) followed by those aged 21-30 years. The fewer pregnancies among women aged ≤20 (mean = 1.3±0.9, range, 1-4) reflects a shift in the age of motherhood from 14-16 years in Igbo traditional society to 18-20 years. This shift is presumably due to higher rate at which women are being educated presently. At 14-16 years the girls are in the secondary schools and most of them would not consent to marriage, whereas at 18-20, a good number are in tertiary institutions and would consider marriage while studying. This explanation is consistent with the average age of 28.4 ± 4.8, range 18-47, among women of childbearing age sampled and with the average age of 25.2 ± 5.9, range 18-34, among the controls drawn randomly from young women undergoing nursing or university education in Owerri. Education, also raises the awareness of birth control measures among the latter group, thus preventing unwanted pregnancies.

The order of prevalence of the ABO blood groups, namely, O>A>B>AB, has generally been observed in several human populations (1, 7). The prevalence rate of these groups in this study, O (55.7%), A (29%), B (14%) and AB (0.8%), varies from those of the American Caucasians – 43-47%, 38-41%, 9-13% and 3-5%, respectively (1) and from those of the Kenyan blacks – 47%, 26%, 22% and 4%, respectively (8). It even differs to some degree from that obtained among the Yorubas, another ethnic group in Nigeria – 50%, 21%, 23% and 4%, respectively (9). All these show that variations exist in blood group antigen distribution, not only between races but also between ethnic groups of the same race. However, any prospects of using such variations in anthropological deductions seem likely to be confounded by the effects of mixed marriages, except in relatively isolated communities.

As has been noted earlier, A and B antigens are the major polymorphic alloantigen systems and potent immunogens. Antibodies against these antigens have been reported to be responsible for fatal haemolytic transfusion reactions, rejection of organ transplants and delay in engraftment of transplanted erythropoietic marrow (2). However, blood group antigen substances A, B and H have been found in species other than humans including bacteria and plants (1). Thus, the sensitization resulting in high levels of antibodies against A and B group antigens even in the control group could be explained by possible contact with these other species since the controls had never received transfused blood or blood products. The higher mean $\log_2$ titers of anti-A (5.3 ± 1.7) and anti-B (5.7 ± 2.1) antibodies among the 10 non-pregnant control women in contrast to the 2.5 ± 1.1 and 4.0 ± 1.2, respectively, obtained for the 236 pregnant sample women (a log difference of approximately 1-3) is not easily understood. However, the state of pregnancy is generally associated with lowered immune responses. It is not known whether this could account, at least in part, for the lower antibody titers observed among the pregnant women. Besides, there is a physiological fluid expansion with resultant hemo-dilution in pregnancy, which is unlikely to occur in the non-pregnant state.

Out of the 759 pregnancies in the history of the 236 pregnant women sampled, 492 (64.8%) were delivered at full term while 267 (35.2%) were miscarried. The causes of the abortions or miscarriages were not indicated. The highest delivery rate was among women aged 31-40 (71.97%) followed by the 21-30 year old (60.9%). The highest abortion rate (78.6%) was apparently among women aged ≤20 years. The causes of high abortion rate among the primigravid women, in particular, need to be investigated. Attention may be paid to the possible effects of contraception on reproductive health of this group of women.

The 7.7% prevalence of Rh(D)$^-$ women in this study contrasts with the 15% among the Caucasians whites, the 5.15% among the Bantu Negroes (1) and 4.8% among the Palestinian women (10). This may imply a lower risk of hemolytic disease of the newborn in the study population than in the Caucasian race, at least. However, high Rh(D)$^+$ prevalence in the population presumably increases the chances of the fewer Rh(D)$^-$ women marrying Rh(D)$^+$ men and carrying Rh(D)$^+$ fetuses in their pregnancies with resultant maternal red cell alloimmunization. Anti-D remains the most common clinically important antibody in pregnancy and accounts for the greatest fetal and neonatal morbidity and mortality (11). The prevalence of abortions among the multiparous mothers was 73.3% for the Rh(D)$^+$ and 68.4% for the Rh(D)$^-$. Thus, there appears to be a confounding effect of other causes of abortions, which may not permit clear determination of the contributions of blood group incompatibility in these clinical cases. HDN cases due to ABO-incompatibility are expected to be low but cannot be distinguished in both Rh(D)$^+$ and Rh(D)$^-$ mothers. The actual HDN-induced abortions may not be discernible unless the cord blood and fetal red cells are studied as well. Even for the low rate of HDN expected in the population studied a preventive measure involving antenatal administration of anti-D immunoglobulin may be introduced, particularly to forestall sensitization in primigravid individuals among the 7.7% young women in the population that may be Rh(D)$^-$. Anti-D immunoglobulin administration has been reported to significantly reduce the levels of sensitization in primigravid women (12). It has been estimated that about 60% of untreated infants who show positive reactions to direct Coombs test for maternal anti-D immunoglobulin die of cardiac failure within a few hours of birth or rapidly develop jaundice and kernicterus (4). Therefore, careful diagnostic surveillance of Rh(D) status of both mother and the newborn and timely treatment remain important life-saving measures.

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