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The prevalence of ABO blood group antigens and antibodies in Lagos State, Nigeria

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Summary

A total of 1239 normal donors from the Lagos University Teaching Hospital (LUTH) and 111 staff of the National Institute for Medical Research (NIMR) Yaba were screened for ABO antibodies. Of the number from LUTH, 220 (17.8%) were found to be in group A, 282 (22.8%) in group B, 85 (6.9%) in group AB and 652 (52.6%) in group O. The number from NIMR consisted of 20 (18.0%) in group A, 25 (22.5%) in group B (7.2%) in group AB and 58 (52.3%) in group O.

The mean titre avidity time of sera from 789 (62.66%) potent LUTH donors was less than 35 seconds. Only 97 (6.91%) of this reacted within 10 seconds. On the other hand, only 11 (9.9%) of the NIMR sera reacted within 35 seconds and none reacted within 10 seconds. Group O individuals from LUTH and NIMR did not always have anti-A and anti-B components of their sera with equal avidity or potency. It was also observed that high avidity of antibody did not necessarily correspond with high potency. The commonest titre for group B (anti-A) sera was 256 and that for group A (anti-B) was 512. In general, anti-B titres tended to be consistently higher than anti-A. There was a bimodal peak at titres 32 and 256 in group B (anti-A) sera. This repeated itself in the anti-A component of group O sera (i.e., anti-A+B), but here the peaks occurred at 32 and 128.

We conclude that ABO antisera examined in Lagos State, Nigeria, exhibited distinct characteristics and that the socio-economic status of the donors influenced avidity and titre.

Résumé

Une somme totale de 1,239 donateurs normaux du Lagos University Teaching Hospital (LUTH) et 111 membres du personnel du National Institute for Medical Research (NIMR) à Yaba sont soumis à une

visite de dépistage pour des anticorps ABO. Du nombre de LUTH, 220 (17,8%) étaient du groupe A, 282 (22,8%) groupe B, 85 (6,9%) du groupe AB et 652 (52,62%) du groupe O. Le nombre du NIMR comprenait 20 (18,0%) du groupe A, 25 (22,5%) du groupe B, 8(7,2%) du groupe AB et 58 (52,3%) du groupe O.

La période moyenne d'avidité titre des sera de 789 (62,66%) donateurs puissants de LUTH était moins de 35 secondes. Seulement 87 (6,91%) de ce nombre ont réagi avant 10 secondes. De l'autre côté, seulement 11 (9,9%) des sera du NIMR ont réagi avant 35 secondes et aucun n'a réagi avant 10 secondes. Les individus du groupe O du LUTH et du NIMR n'ont pas toujours eu des composants anti-A et anti-B de leurs sera avec d'avidité ou puissance égales. Il a été observé aussi que l'avidité élevée d'anticorps n'était pas nécessairement en conformité avec une puissance élevée. Le titre le plus commun pour les sera du groupe B(anti - A) était 256 et celui du groupe A (anti-B) était 512. En général, les titres anti-B avaient la tendance à être uniformément plus élevés que ceux d'anti-A. Il avait une pointe bimodale aux titres 32 et 256 dans les sera du groupe B(anti-A), Ceci s'est répété dans le composant anti-A des sera du groupe O (c.a.d., anti-A+B), mais dans ce cas, les pointes se sont présentées aux titres 32 et 256.

En conclusion, nous constatons que les antisera ABO examinés à l'Etat de Lagos au Nigeria, ont montre des caractéristiques distinctes et que le statut socio-économique des donateurs ont influé sur l'avidité et le titre.

Introduction

In the human population, the ABO system could be divided into 4 blood groups (A, B, AB and O) according to the presence or absence of antigenic determinants located on the red blood cells. Also

present in the blood are antibodies or agglutinins which are present in individuals that lack the corresponding agglutinin[1].

The ABO antibodies are routinely produced in the serum of individuals who lack the corresponding antigen and these are developed during infancy (4-8 months), attaining a level in late childhood at which it is maintained with little variation throughout life and only decreasing in old age[1].

A number of studies have shown that these antibodies occur in varying proportions in different populations. In a screening done on a South American Indian population, all were found to be in group O while a population of Vietnamese showed 45% in group O, 21.4% in group A, 29.1% in group B and 4.5% in group AB[2]. Among the Australian aborigines the ratio was found to be 44.4% in group O and 55.6% in group A. Studies on a German population[3] showed 42.8% in group O, 32.5% in group A 9.4% in group A₂, 11.0% in group B, 3.1% in group AB and 1.1% in A₂B.

While screening for potential donors for the production of ABO antisera, we decided to investigate the pattern and frequency of each group in the staff of NIMR and in normal blood donors from LUTH. We also deemed it contributory to knowledge to test for any differences in avidity between the sera of NIMR staff most of whom have never donated blood before and those of regular LUTH donors, some of whom are touts. The results which are distinct from the pattern reported in other populations[2,3], form the basis of this paper.

Materials and methods

Collection of samples

About 5-10 ml of whole fasting blood was collected from each of the 111 staff of the NIMR and 1239 donors who reported at LUTH for blood donation between 1986 and 1988. These samples were centrifuged for 5 minutes at 4,000 RPM at 8°C in order to obtain clear supernatant sera for the tests.

Screening for avidity

The WHO recommended method[4,5] was used. Briefly, one drop of serum was added to one drop of saline on a clear dry Coomb's tile and was mixed together. To this mixture was added 10% group A or group B cells that had been washed thrice in physiological saline. This was rapidly mixed and the

timer immediately started. The time taken for the haemagglutination to occur was recorded in seconds and those sera that reacted strongly within 35 seconds were screened for potency.

Screening for potency (i.e. Titre)

The WHO recommended method[4] was also applied. Ten Kahn test tubes were arranged in a row and, with a pasteur pipette marked to deliver a constant unit (e.g. 0.1 ml), the unit volume of isotonic saline was placed in all tubes except the first. The unit volume of the test serum was placed in the 1st and 2nd tubes and the contents of the 2nd tube were mixed. The same unit volume was transferred from tube 2 to tube 3 and the process was repeated up to tube 10 when the extra mixture was discarded. Thus the doubling dilution was carried out from neat to 1/512.

An equal volume of the appropriate 3% red cell suspension in saline was added to these dilutions. The serum/cell mixture was then shaken to facilitate intimate antibody-antigen contact (for testing anti-A, pooled group A red cells were used while for anti-B pooled B red cells). These test tubes were then centrifuged at 1000 RPM for 2 mins at 8°C[5]. For group O serum, 2 rows of 10 tubes were set up. Group A cells and group B cells were added to each respective row of tubes. Commercial antisera served as the controls and the readings were made macroscopically.

Results

Table 1 shows the frequency of the ABO blood groups in the subjects. In LUTH donors, group A was found to be 17.76%, group B 22.76%, group AB 6.86% and group O 52.62%. The pattern was not different from those obtained in NIMR staff: A (18.0%), B (22.5%), AB (7.2%) and O (52.3%).

The tile avidity test results (Table 2a) showed that 87 (7.02%) of LUTH donors showed evidence of agglutination within 10 seconds, 399 (32.20%) within 20 seconds and 789 (63.68%) of the 1239 donors reacted within 35 seconds. The rest reacted after 35 seconds. On the other hand, none of the 111 sera belonging to NIMR staff reacted within 10 seconds; 2 (1.8%) reacted within 20 seconds, 11 (9.9%) reacted within 35 seconds. Agglutination was observed in the rest only after 35 seconds.

Table 1: Frequency of ABO blood groups in the blood donors

Blood group	Distribution		% in population screened	
	LUTH donors	NIMR Staff	LUTH donors	NIMR Staff
A	220	20	17.76	18.0
B	282	25	22.76	22.5
AB	85	8	6.86	7.20
O	652	58	52.62	52.3
Total	1239	111	100%	100%

Table 2a: Tile avidity test of ABO antibodies in the blood donors from LUTH and NIMR

Source	No Screened	Reaction time in sec.			
		Within 10 Sec	20 Sec	35 Sec	>35 Sec
NIMR Staff	111	0(0.0%)	2(1.8%)	11(9.9%)	100(90%)
LUTH Donors	1239	87(7.02%)	399(32.20%)	789(63.68%)	450

Table 2b: Mean tile avidity time for individual blood groups in LUTH donors

Blood Group	Antibody	No. Reacting Within 35 Sec	Mean Reaction Time In Sec \pm SD
A	Anti-B	153	19.6 \pm 8.0
B	Anti-A	168	27.1 \pm 8.1
O	Anti A+B	468	26.1 \pm 7.9

Table 3: Potency test of the ABO antibodies in the blood donors

	Neat	2	4	8	16	32	64	128	256	512	>512
Anti-A	—	1	2	5	11	22	21	15	24	16	21
Anti-B	—	2	2	4	6	4	17	15	20	25	22
Anti-A +	—	—	1	8	21	31	21	35	14	8	10
B	—	5	6	21	41	47	80	63	46	34	30

Table 2b shows that of the 789 LUTH donors reacting within 35 seconds, 153 were in group A, 168 in group B and 468 in group O (anti A + B). Blood group B donors seemed to have the most prolonged mean reaction time although this was not statistically significant.

On the potency test of the ABO antibodies in the blood donors, it was observed that there were bimodal peaks at titres 32 and 256 respectively of anti-A antibodies (Table 3). This repeated itself in the anti-A component of group O sera (i.e. anti - A + B) but this time, the peaks occurred at 32 and 128.

Discussion

It was found in this study that 17.76% of the donors from LUTH and 18.0% of NIMR staff were in group A. This differs from the 44% found in Europeans[6]. Furthermore, group B was 22.76% in LUTH and 22.50% in NIMR sera whereas the reported findings in an European population was 7%. Group AB was found to be 6.86% (LUTH) and 7.20% (NIMR) as against the reported 3% in Europeans. The figure for group O was 52.62% LUTH and 52.30% NIMR as against 46% in Europeans[6].

Our figures however seem to agree with those of Kassim and Ejezie[7] who in their studies on ABO blood groups in malaria and haematobium schistosomiasis, found that with group A, B and O, the percentages were 18.5%, 22.3% and 56.7% respectively. However, there appears to be a substantial difference with respect to AB (6.86% versus 2.5%) when our findings are compared with those of Martins *et al*[8].

It can be seen from Table 2a that, using a tile avidity of 35 seconds, more than half of the LUTH donors could be selected as having highly avid agglutinins for antiserum production. Whereas using 20 seconds, only 33% of the LUTH population fell within the time (Table 2b). The situation was different in the sera of NIMR staff where only 9.9% reacted within 35 seconds, 1.8% within 20 seconds and none within 10 seconds. It should however be recalled that most blood donors from LUTH are touts, who tend to donate blood regularly for commercial purposes. NIMR staff on the other hand are conventional civil servants, who volunteered to give their blood for this study. It is therefore suggested that socio-economic status of the donors influenced avidity. Some workers have suggested that sera for antisera production should have a tile avidity time of 10 seconds[9] but in this environment, we suggest that a minimum acceptable avidity should be 24.27 ± 8 seconds.

Aubert *et al*[10] have found that in their own studied population, the commonest anti-A titre was 256 and the commonest anti-B titre was 64. Our findings at LUTH and NIMR showed that the commonest titre for group B (anti-A) sera was 256 and that for group A (anti-B) was 512. Here anti-B titres tended to be consistently higher than anti-A, even when comparing the two components in group O. The latter however agrees with Ichikawa[11].

The commonest anti-A titre in group O was found to be 128 while anti-B was found to be 64. One can say that a titre of 64 (Table 3) is the potency that cuts across anti-A, anti-B and anti-AB with nearly equal number of sera falling into it, even though the anti-B of group O serum surpassed the rest. From the potency test, the anti-A from group B serum showed bimodal peaks at titres 32 and 256. A similar pattern was reflected in the anti-A component of the group O serum, but here, the peaks were at titres 32 and 128. Since A cells were used, it is probable that the 2 peaks may be due to the presence of anti-A and anti-A₂ in the same anti-A sera. Work is still in

progress to answer this and other questions that have arisen from our investigations.

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