ORIGINAL ARTICLE

Association of Some Blood Group Phenotypes and Risk of Acute Myeloid Leukaemia In Kano, Nigeria

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Abstract

Background: Acute myeloid leukaemia (AML) is a heterogeneous clonal disorder characterized by abnormal proliferation of immature and non-functional cells known as blasts and subsequently results in bone marrow failure and organs infiltrations. Blood group antigens have been linked with the aetiology and pathophysiology of various communicable and non-communicable diseases.

Objective: This study therefore investigated the association of some blood group phenotypes with AML among subjects in Kano, Nigeria.

Materials and Methods: Acute myeloid leukaemia was diagnosed using the clinical and cytological criteria in Aminu Kano Teaching Hospital (AKTH). The blood groups were determined using tube method for ABO, Rh-D, Lewis, Duffy and MNS while Rh-C-c-E-e and Kell by Gel method. The Antisera and Gel cards were obtained from Lorne Laboratories in the United Kingdom. All the techniques were according to the manufacturer's instructions. Data was presented as tables and analysed using *SPSS* (version 25.0).

Results: A total of 25 AML subjects (age: 18.9±13.9 years, male/female ratio: 1.3:1) were studied with 25 healthy blood donors (controls) with mean ages of 29.2±5.3 years and male/female ratio: 11.5:1. Association of some blood groups and AML was determined using Chi-square test while Odds Ratio (OR) and Relative Risk (RR) were used to ascertain their risk values. The results showed that Le[a], Le[b], K and M antigens were significantly associated with AML (OR: 4.75, 7.11, 11.29 and 7.67 respectively).

Conclusion: We conclude that individuals with Lewis antigens (Le^a and Le^b), Kell antigen (K) and M antigen may have increased risk of developing acute myeloid leukaemia.

Keywords: AML, Blood groups, Kano, Nigeria.

INTRODUCTION

Acute myeloid leukemia (AML) is a form of malignant disorder that is characterized by uncontrolled and exaggerated growth of immature cells known as blast cells of myeloid origin (1).

Exposure to radiation, chronic exposure to high doses of benzene, chronic heavy inhalation of tobacco smoke, some drugs used in the treatment of haematological malignancies and some genetic disorders increase the incidence of AML (2).

Acute myeloid leukemia is diagnosed on the basis of clinical and microscopic examination (cellular morphology of peripheral blood and bone morrow), immunological markers (flow cytometry), cytogenetics and molecular analysis (3). In advanced centres, immunological markers, cytogenetic and molecular analysis have reduced the important of cytochemical procedures.

Since the antigen of Red blood cell (RBC) represents inherited traits, therefore their expression should be constant throughout the life of an individual (4,5). Diminished expression of ABH antigens can occur with variant ABO alleles and in hematological disorders such as leukaemia, myeloproliferative disorders, myelodysplastic syndrome and in some cases of Hodgkin's lymphoma. It has also been found in healthy elderly adults, pregnant females and neonates. Myeloid neoplasms have been reported to be more frequently associated with ABH antigenic alterations than lymphoid malignancies (6). Diminished expression of A and/or B antigens could be due to a blockage of the conversion of H substance to A and/or B substances or decreased synthesis of H antigens. Since the ABO locus encoding A and B transferases is in the 9q34 region, the chromosomal translocation (9 and 22) observed in patients with myeloid leukaemia (both chronic myeloid leukaemia and rarely AML) has also been linked to ABH red cell antigenic changes in these patients. Early recognition of the loss of ABH antigens is extremely important because it may indicate underlying sinistrous pre-leukemic states (6).

Several studies have investigated the association of blood group antigens and diseases etiology. However, no such study has been done in North-Western Nigeria. Significant results have been obtained in subjects with various diseases; For example, group A has been associated with increased risks of gallstones, colitis, and certain tumor types (7), whereas Non-O blood group individuals have increased risk of thrombo-embolic events compared to O blood groups. This is largely due to increased levels of von-willebrand's factor. These individuals have also been associated with cardiovascular diseases (8), including ischemic heart disease and atherosclerosis (9), A secretors have a higher incidence of gastric cancer (10), Non-secretors have high incidence of diseases of mouth, esophageal cancer, and epithelial dysplasia as compared to secretors(11).

This study was aimed at determining the associations of some blood group phenotypes

and risk of acute myeloid leukaemia in Northern Nigeria

MATERIALS AND METHODS
Study Location
This case-control study design was carried out at Aminu Kano Teaching Hospital (AKTH), over a period of two years (October, 2017 through October, 2019). The hospital serves as both tertiary and primary health care facility for Kano, neighbouring states and some states in the southern part of Niger Republic. Kano state is a state located in the North-western Nigeria [12]. It lies between latitude 11°30`N and longitude 8°30`E

Study Population
A total of fifty (50) participants were recruited in this study, 25 were acute myeloid leukaemia patients attending AKTH and 25 apparently healthy blood donors as controls. Both males and females were recruited in this study. Ethical clearance to conduct the research was obtained from the ethics committee of Aminu Kano Teaching Hospital. Verbal informed consent for inclusion was obtained from the selected participants.

Inclusion Criteria
Only subjects confirmed to have AML attending haematology day-care unit, male and female medical wards were considered eligible and recruited into this study.

Exclusion Criteria
Non-acute myeloid leukaemic subjects, elderly adults, pregnant women, neonates and those who do not consent to participate in the study were excluded.

Sample Collection and Analysis
Acute myeloid leukaemia was diagnosed by bone marrow aspiration at the department of haematology of Aminu Kano Teaching Hospital, Kano. Diagnosis of AML was made on the basis of clinical and cytological criteria (morphological and cytochemical procedures) if blasts of myeloid origin constituted at least 20% of nucleated cells in the bone marrow (3). Blood group antigenic profiles of the AML patients were determined at the teaching laboratory of the department of Medical Laboratory Science, Bayero University, Kano, Nigeria.

Three milliliters (3ml) of venous blood was collected under aseptic condition from both controls and subjects confirmed to have AML into an ethylene diamine tetra-acetic acid (EDTA) anticoagulated bottle. Samples were thereafter washed in saline before screening for blood group antigens to ABO, Rh-D, Lewis, Duffy and MNS using tube method while Rh-C, Rh-c, Rh-E, Rh-e and Kell using Gel card techniques.

Five percent (5.0%) suspension of washed red cells was made in Phosphate Buffered Saline (PBS). In a labelled test tube: 1 drop of Lorne Anti-sera reagent and 1 drop of test red cell suspension were added. The tubes for ABO, Lewis, M and N antigens were incubated at room temperature while tubes for Rh-D, Duffy and S antigens were incubated at 37^0C temperature for 15 minutes. The tubes were then centrifuged for 20 seconds at 1000 rcf. The red cell button was gently resuspended and red macroscopically and microscopically for agglutination. AHG was added in any tube, which showed a negative or questionable result

Blood Grouping by Gel Card for Rh antigen (C, E, c, e) and K antigen (Diamed)

Red cells from each sample was prepared as 5.0% suspension in Phosphate Buffered Saline (PBS) and Rh antigen (C, E, c, e) and K antigen typing were performed by gel card. Ten micro litre of red cell suspension was added to micro tubes followed by centrifuge at 910 rpm for 10 minutes. Agglutinated cells forming a red line on the surface of gel or dispersed in gel were

considered positive. A compact button of cells on bottom of the micro tube indicated the absence of the corresponding antigen (13).

Statistical Analysis

Data was presented as tables and figures and analysed using *SPSS* (version 25.0). Association of some blood groups and leukaemias were determined using Chi-square test while Odds Ratio (OR) and Relative Risk (RR) were used to ascertain their risk values. All statistical analyses were at 5% level of significance, $p < 0.05$.

RESULTS

A total of twenty-five (25) acute myeloid leukaemic subjects and twenty-five (25) non leukaemic blood donors were studied in this research. Their ages range from 4-43 years while that of blood donors ranges from 18-40 years. The mean ages for AML was 18.9±13.9 years with male/female ratio: 1.3:1 while that of blood donors was 29.2±5.3 years with male/female ratio: 11.5:1.

AML was found to have both childhood and adulthood age distribution in this study. No AML subject was found above the age of 50 years **(Table 1).**

Table 2: Shows the association of blood group antigen phenotypes and risk of acute myeloid leukaemia (AML). Some blood group antigen phenotypes showed statistically significant association while some did not. The relative risk (RR) and odds ratio (OR) for all the statistically significant association were recorded. The Odds ratio given by odds of cases divided by odds of control function as Relative risk assessment that the given phenotype is associated with the leukaemia. Blood group Le^a, Le^b, K and M phenotypes were significantly associated with AML (OR: 4.75, 7.11, 11.29 and 7.67).

Table 1: Distribution of Acute Myeloid Leukaemia patients Based on Age Groups

Age Groups (Years)	Number of Patients (%)
1-10	13 (52.0)
11-20	3 (12.0)
21-30	2 (8.0)
31-40	5 (20.0)
41-50	2 (8.0)
Total	25 (100)

Table 2: Association of Blood Group Antigen Phenotypes and Risk of Acute Myeloid Leukaemia (AML).

	Patients and Controls (n=25 each)				
Blood Groups	Patients: Number (%)	Controls: Number (%)	P-value	OR	RR
A	12 (48.0)	5 (20.0)	0.0718	3.69	1.79
B	2 (8.0)	6 (24.0)	0.2467	0.28	0.46
AB	0 (0.0)	3 (12.0)	0.2347	0.13	0.00
O	11 (44.0)	11 (44.0)	1.2239	1.00	1.00
D	23 (92.0)	24 (96.0)	1.0000	0.48	0.73
C	9 (36.0)	5 (20.0)	0.3451	2.25	1.45
c	21 (84.0)	21 (84.0)	1.2981	1.00	1.00
E	16 (64.0)	10 (40.0)	0.1564	2.67	1.64
e	20 (80.0)	24 (96.0)	0.1895	0.17	0.55
Le^a	15 (60.0)	6 (24.0)	0.0209*	4.75	2.07
Le^b	20 (80.0)	9 (36.0)	0.0037*	7.11	2.90
Fy^a	6 (24.0)	2 (8.0)	0.2467	3.63	1.66
Fy^b	6 (24.0)	1 (4.0)	0.0983	7.58	1.94
K	8 (32.0)	1 (4.0)	0.0232*	11.29	2.14
M	10 (40.0)	2 (8.0)	0.0181*	7.67	2.11
N	23 (92.0)	25 (100)	0.4898	0.18	0.48
S	0 (0.0)	0 (0.0)	Nil	Nil	Nil

Legend: P<0.05 indicates statistically significant difference, OR=Odds Ratio, OR<1.00 indicates protection, OR>1.00 indicates risk exposure, OR=1.00 indicates that the antigen is not a risk factor, RR=Relative Risk, D= Rhesus D antigen, C= Rhesus C antigen, c= Rhesus c antigen, E= Rhesus E antigen, e= Rhesus e antigen, Lea= Lewis A antigen, Leb= Lewis B antigen, Fya= Duffy A antigen, Fyb= Duffy B antigen, K= Kell antigen, M= MNS antigen, N= MNS antigen, S= MNS antigen.

DISCUSSION

The clinical application of blood group antigens is mainly in the field of blood transfusion, organ donation and research purposes since the discovery of these blood group antigens. However, the use of blood group antigens in diagnosis and prognosis of various health problems is rapidly expanding as researchers had proven the association between blood group type and certain disease conditions, examples are the association between incidence of peptic ulcer and blood group O and group A with gastric carcinoma (14).

The mean ages for AML patients in this study was 18.9±13.9 years. These mean ages lesser than 44 years was reported in Jos, North Central Nigeria by *Damulak et al., (15)*. While less than 52.1 years was reported in the South region of Nigeria by Nwannadi *et al.,* (16). The differences in our result compared with the previous ones may be due to early development of western civilization with better health care, economic opportunities, and a resultant longer life expectancy in the Southern part of the country as postulated by *Damulak et al.,* (15). Also, advancement in medical diagnosis as well as possible genetic predisposition may partly account for the early onset as seen in this study. However, it may no longer be true that acute leukaemias only dominates in childhood as documented earlier by Hoffbrand *et al.,* (17). This is because AML was recorded in both children and adults in our study.

In AML, the ABO antigenic distribution is slightly deviated from the distribution seen in normal healthy non leukaemic subjects. One interesting finding was noticed on the absence of AB antigenic phenotype in AML with distribution of A antigen phenotype of 12(48.0), O phenotype of 11 (44.0) and B antigenic phenotype of 2(8.0%) while AB phenotype was 0(0%). In many other studies among blood donors, blood group O has been found to be the most common blood group and AB as least common blood group which is slightly different from our findings where we found more A than O blood groups and no AB subjects. Eru *et al.,* (18) reported group 52.5% and 2.2% in groups O and AB respectively amongst their participants in Benue. Looking at the distribution of ABO among leukaemic patients in our study and other studies within the country and elsewhere in the world, our finding is similar to that of Safaa (14) who found that blood group A has the highest frequency in acute non-lymphoblastic leukemia. It was also in agreement with the findings of Shirley and Desai (19) who reviewed several previously published data and found no statistically significant difference in the distribution of A blood group with respect to O blood group in patients with acute leukemias.

The Rhesus D prevalence in this study (92.0%) is consistent with reports from previous studies among different sets of Nigerian blood donors by Olubayode *et al.,* (20). The prevalence of Rh D was also reported by Jeremiah, (21) as 96.7%, 94% by Adeyomo and Soboye` (22), 97.7% by Bakare *et al.,* (23), 93.2% by Akhigbe *et al.,* (24) and 93%. by Erhabor *et al.,*(25). Safaa,(14), also showed that Rh D positive blood type was detected in 91.5% of AML while Rh D negative blood type comprised only 8.5% of AML patients. The Rhesus antigens (c, C, e and E) prevalence from our study showed some dissimilarity when compared with the prevalence of Rhesus antigens in a study done previously in Kano by Baffa and Shehu (26). They reported the prevalence of the Rh phenotypes as follows e>>c>>E>>C e (96.1%, 85.4%, 34% and 28,2%, respectively) (26).

The Lewis phenotype expression is determined by two genetic systems closely related on the short arm of chromosome 19, the Lewis genes *Le* and *le*, and the Secretor genes, *Se* and *se*. The gene product consists of various

fucosyl transferases, which from tissue liquids are adsorbed onto cell surfaces (27).

In this current study, we noticed prevalence of Le^a and Le^b as 60% and 80% respectively among AML subjects much higher than that of blood donors studied as controls (24% and 36% respectively). We observed statistically significant associations in both Le^a and Le^b antigens with AML in this study. The Odds ratio of Le^b with AML was greater than 1.0. It indicated that Le^b positivity had greater risk than Le^a positivity. However, the development of these antigens after the onset of leukaemia needs to be evaluated. The critical question here is how to unravel the mechanism responsible for these predispositions.

The Duffy protein is organized on the RBC membrane as an N-glycosylated protein that spans the red cell membrane seven times (28). Fy^a and Fy^b differ by a single amino acid change at position 42 on the extracellular domain, with glycine resulting in Fy^a expression and aspartic acid resulting in Fy^b expression. Both Fy^a and Fy^b are sensitive to destruction when RBCs are treated with proteolytic enzymes such as papain or ficin (29). The prevalence of Fy^a in AML subjects (24%) is significantly higher compared with that of blood donors (8%) studied (odds ratio = 3.63 and relative risk = 1.66). Likewise, the distribution of Fy^b is higher in AML subjects (24%) while that of blood donors was (10%) (odds ratio = 7.58 and relative risk = 1.94). Isaac et al. (30), reported prevalence of Fy^a and Fy^b as 7.4% and 4.4% respectively among malaria subjects in Sokoto, Erhabor et al.,(31) observed Fy^a and Fyb prevalence of 4.3% and 5.6% respectively among pregnant women in Sokoto as compared with the prevalence of Fy^a(66.0%, 99.0% and 10.0%) and Fy^b antigen of (83.0%, 9.2%, 23.0%) among Caucasians, Chinese and Blacks blood donors respectively.

AML had shown statistically significant association with with both Fy^a (p-value = 0.2467, OR = 3.63 and RR = 1.66) and Fy^b (p-value = 0.0983, OR= 7.58 and RR = 1.94) antigen phenotypes in this study. Also, the mechanism involved could not be suggested in this study

The Kell antigens are located on a 93-kD RBC membrane glycoprotein that consists of 731 amino acids and spans the membrane once. The N-terminal domain is intracellular, and the large external C-terminal domain is highly folded by disulfide linkages. The Kell glycoprotein is covalently linked with another protein, called Kx, by a single disulfide bond. The Kx protein (440 amino acids and 37 kD) is predicted to span the RBC membrane ten times (32). The prevalence of Kell antigen in this study was 32% while that of blood donors studied was 4%. The Kell antigen prevalence was reported by Erhabor et al.(33) as 2%among healthy individuals in Sokoto, North Western Nigeria. Ugboma and Nwauche (34) in Port Harcourt investigated the prevalence of Kell antigen and reported a Kell antigen prevalence of 2%. Lamba et al.,(35) observed a Kell antigen prevalence of 2.8% among healthy blood donors in India.

AML had statistically significant association with Kell antigen phenotype in this study (AML:p-value=0.0232, OR=11.29 and RR=2.14). This is the only blood group that recorded highest Odds ratio and serious attention should be given to find out the mechanism of this risk exposure and the possibility of acquiring it following the onset of AML.

The prevalence of MNS antigens in this current study among AML patients was recorded as M phenotype; 40%, N phenotypes; 92% and S phenotype; 0%. The observed prevalence from our study was at variance with the prevalence of MNS antigens among blood donors studied (M phenotype; 15%, N phenotype; 100% and S phenotype; 0%).

Makroo et al.,(36) reported M prevalence

of 88.8%, N prevalence of 65.4% and S prevalence of 54.8% among Indian blood donors.

AML had statistically significant association with M antigen phenotype in this study (AML:p-value=0.0181, OR=7.67 and RR=2.11).

CONCLUSION

We conclude that individuals with Lewis antigens (Lea and Leb), Kell (K) and M antigens have close associations with AML and may also be predisposed further to this disease. The possibility of acquiring these antigens during the disease state and the mechanisms involved need to be elucidated as markers of prognosis or otherwise.

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