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***ORIGINAL ARTICLE***

# ABO Red Cell Antigens, von Willebrand Factor, Factor VIII and Platelet counts among Hypertensive Patients In Kaduna Metropolis, Kaduna.

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**Abstract**

**Background:** von Willebrand Factor and factor VIII have been implicated in the vulnerability of causing hypertension which is related/common to some of the ABO blood group antigens. However, the precise mechanism/role of the von Willebrand Factor and factor VIII in hypertension is unknown. The study is a comparative study that is aimed to determine the association of ABO red cell antigen, von Willebrand factor, Factor VIII, and Platelets among hypertensive patients in Kaduna, Nigeria.

**Methods:** To determine this association, fifty-five (55) hypertensive patients and 28 controls were recruited. A purposive sampling technique was employed in selecting the study participants. Determination of the ABO blood group, vWF, FVIII, and platelets was carried out to establish the frequency and the association of ABO blood type with hypertension.

**Results:** It was established that blood group O (43%) has the highest distribution followed by A, B, and AB (6%) the least. There was no significant difference in vWF(ng/L), FVIII(pg/ml), and platelet count(109/l) between the patients and the controls (454.9 and 456.2), (242.0 and 228.4) and (238.0 and 213.0) respectively, $p>0.05$. The correlation studies showed a strong association between vWF and FVIII (r - 0.544, $P<0.0001$). There was a significant difference between males' and females' vWF and FVIII ($P=0.0013$ and 0.0029 respectively), indicating females had a higher level of vWF and FVIII, and therefore at higher risk of developing hypertension. Women should therefore be screened for these parameters as a matter of routine.

**Conclusion:** The risk of developing hypertension based on the parameters considered in this study is independent of the ABO blood group of an individual. There is a need for further studies, to confirm or rule out these findings.

**Ket words:** von-willebrand Factor, Factor VIII, Platelet, ABO Blood Group, Hypertension.

## INTRODUCTION

The ABO blood group antigens are composed of complex carbohydrate molecules expressed on the surface of red blood cells and in a variety of other cells and tissues. As the first blood group system to be discovered, the ABO blood group system has been the subject of intensive research for more than a century. They have a pivotal role in transfusion and transplantation medicine. Also, the ABO antigens participate in many other physiological processes and, in particular, are important determinants of von Willebrand factor and factor VIII circulating plasma levels.(1) It is however been implicated in the development of several human diseases and its involvement in the pathogenesis of cardiovascular disorders, mainly through its effect on von Willebrand factor levels.(1)

The antigens of the ABO blood group system (A, B, and H determinants) consist of complex carbohydrate molecules placed on the extracellular surface of the red blood cell (RBC) membranes.(2). The A and B alleles encode slightly different glycosyltransferases that add N-acetylgalactosamine and D-galactose respectively, to a common precursor side chain, the H determinant, converting it into A- or B-antigens. The O alleles encode no functional enzyme and hence in OO carriers, the H antigen remains unmodified, with a fucose moiety attached to the precursor oligosaccharide chains.(2)

The ABH antigens are found not only on the surface of RBCs but are widely expressed on other cells and tissues including mucosal epithelial tissue, sensory neurons, and in body fluids including the genital tract. This is dependent on the activity of the enzyme, Fucosyltransferase 2 (FUT2), which is responsible for ABO biosynthesis in body fluids.(2)

Thus, ABO matching is critical not only in blood transfusion but also in cell/tissue/organ transplantation. Accordingly, the clinical significance of the ABO blood group system extends beyond transfusion medicine and several reports have suggested an important involvement in the susceptibility to infectious, neoplastic, and cardiovascular disorders.(1) It is suggested that the non-O blood types could be associated with stroke risk and that some of the excess risks would be mediated by increased FVIII levels. This suggestion was found in a study titled the ABO blood type and stroke risk; the reasons for Geographic and Racial Differences in Stroke Study.(3). It was further hypothesized that, due to racial differences in ABO blood type, blood type would mediate some of the excess stroke risk seen in blacks in the United States. While blood type is not a modifiable risk factor, it may help select a population at risk for aggressive risk factor reduction.(3)

ABO blood groups have been linked to various diseases such as diabetes, many types of cancers, infections, hematological disorders, cognitive disorders, circulatory diseases, and cardiovascular diseases.(4)

Several later studies elucidated that ABO blood groups, particularly non-O blood groups, are associated with major cardiovascular risk factors and/or increased rates of cardiovascular events.(4) However, there is limited consensus regarding the magnitude and significance of the ABO antigen effects at the population level.(5)

ABO antigens are also expressed on vascular endothelium and through unclear mechanisms, associated with levels of the procoagulant proteins such as factor VIII (FVIII) and von Willebrand factor (vWF) as well as markers of endothelial function such as

p-selectin and soluble intracellular adhesion molecule 1. vWF is a critical determinant of thrombus formation at high arterial shear rate conditions(6) It is a large glycoprotein that circulates in plasma as a series of heterogeneous multimers, mediating platelet tethering, translocation, and finally adhesion to areas of injured endothelium under physiologically high arterial blood flow conditions above a critical threshold of 500 - 1,000 s-1 shear rates. (7) Furthermore, vWF protects coagulation FVIII from rapid proteolytic inactivation. It plays a critical role in platelet adhesion and aggregation after vascular injury and at sites of high shear rate.(7)

Also, vWF plays a key role in vascular inflammation, favoring leukocyte recruitment and extravasation as well as activating complement cascade. In cardiovascular disease, including coronary artery disease and stroke, vWF could be a predictor of future cardiovascular (CV) events.(8) However, a previous study has shown that the prevalence of CVD in vWD patients was less than in non-vWD patients.(9)

Indeed, it is now clearly acknowledged that individuals in the non-O blood group have circulating levels of both vWF and FVIII that are approximately 25% higher than O blood group subjects. The underlying mechanism resides in the positive influence on vWF levels and activity of the addition of A and B antigens, by the specific glycosyltransferase enzymes, on the existing vWF H oligosaccharides.(10). The non-O related increased levels of vWF and FVIII, in addition to those of several inflammatory cytokines such as tumor necrosis factor-alpha, soluble intercellular adhesion molecule 1, P-selectin, interleukin-6 and increased cholesterol levels, have been suggested as the most likely mechanisms for explaining the association between ABO blood group and cardiovascular diseases.(11)

In addition, the leading underlying mechanism that has been put forward to explain this association involves the profound influence that the ABO blood group system exerts on hemostasis, particularly on the von Willebrand factor and, consequently, on coagulation FVIII plasma levels, which are both well recognized prothrombotic risk factors.(12)] The molecular basis of this phenomenon has been precisely identified with the presence of ABH antigenic structures on circulating vWF, which modulate the activity of this multifunctional protein through different degrees of glycosylation (12). The aim of the research is to determine the association of ABO blood group antigens, von Willebrand factor and Platelets in hypertensive patients.

## Materials and Methods

Study area: The study is a comparative study conducted in Sokoto while study participants selected in Kaduna State of Nigeria.

Sampling technique: A purposive sampling technique was used to select the study participants. It was designed to determine the relationship between the ABO red cell antigens, VWF level, FVI, II, and Platelets of patients with hypertensive heart disease and the control group.

**Study Population:** A total of 83 subjects were recruited for this study, of this number, 55 were hypertensive patients and the remaining 28 were healthy individuals. Both the patients and control subjects were between the age ranges of 30 to 60 years and attended the hypertensive clinic of the study facilities.

**Sample collection and Transportation:** 2.5 ml of blood was collected by venipuncture into a $K_3$EDTA anticoagulant bottle (at a concentration of 1.2mg/ml), already labelled with subject's name, sex and age. Venous blood sample collected into citrate anticoagulant was used for vWF and FVIII while EDTA was used for Platelet count and ABO Blood group. ELISA technique was used for the determination of vWF and FVIII levels while Sysmex (KX21N) hematology autoanalyzer was used for the platelet count. Period of Sample Collection

was between February through August, 2019.

## Ethical Approval

The ethical clearance and permission to conduct the study was obtained from the Ethics Committee of Ministry of Health and Human Resources, Kaduna-Ref number: MOH/ADM/744/VOL.1/503 (Appendix I). The data was collected after obtaining an informed consent from the study respondents.

## Informed Consent

Informed consent was obtained from the study participants (patients and controls) and the consent form was duly filled and signed by all the consenting subjects.

## Procedures

### von Willebrand Factor Assay

a. Standard Dilution: Serial dilution of the kit standard was made as described by the manufacturer (Melsin Medical Co., Limited, china).

b) Blank well and sample wells were set (precaution was taken not to add sample and HRP Conjugate. Only chromogen solution A and B, and stop solution are allowed; the other steps werethe same).

c) Forty microlitres (40 µl) of sample diluent was added to sample wells and 10 µl of the sample was added (final sample diluted is 5-fold). Care was taken to avoid touching the wall and bottom of the wells during pipetting. It was gently mixed.

d) HRP Conjugate reagent was added: 50 µl of HRP Conjugate reagent was added to each ofthe standard and test wells, except blank well.

e) The plate was closed with closure plate membrane and incubated for 30 mins at 37□.

f) Wash Solution Concentrate was diluted (30 x) with distilled water to prepare 600 ml of Wash Solution (1 x).

g) The plate was washed three times using the wash solution and blotted after each wash.

h) Fifty micro litres (50 µl)of Chromogen Solution A and 50µl Chromogen Solution B was added to each well; the plate was covered and incubated for 10 mins at 37□.

i) Fifty micro litres (50 µl) stop solution was added into each well to stop the reaction (the blue colour changed into yellowimmediately).

j) The plate was read out using micro titre plate reader machine (Set at 450 nm wavelength) and the reading of each well was taken and recorded

### Factor VIII Assay

a) Serial dilution of the standard was made with the standard diluent provided as described by the manufacturer (Melsin Medical Co., Limited, china).

b) Addition of Sample: Blank well and sample wells were set up, 40µl of sample diluent was added to sample well, and 10µl of the sample was also added (final sample diluted was 5-fold). Care was taken to avoid touching the wall and bottom of the wells duringadding of samples. It was gently mixed.

c) Fifty microlitres (50 µl) of HRP Conjugate reagent was added to each well, except blank well.

d) The plate was closed with Closure plate membrane, and incubated for 30 mins at 37□.

e) Wash Solution Concentrate was diluted (30 x) in distilled water to prepare 600 ml of WashSolution (1 x).

f) The plate was washed three times using the wash solution and blotted after each wash.

g) Fifty microlitres (50µl) of Chromogen Solution A and 50µl Chromogen Solution B was added to each well, it was covered and incubated for 10mins at 37□.

h) Fifty microlitres (50µl) of stop solution was added into each well to stop the reaction (the blue colour changes to yellowimmediately).

i) The plate was read out using micro titre plate reader machine (Set at 450 nm wavelength) and the reading of each well was taken and recorded

### Platelet Count

Platelet count was carried out using Sysmex KX21N- an automated Haematology analyser.

### Statistical Analysis

Graph prism version 5.0 Software was used to analyze the data generated from this research work. The data were presented as median and interquartile ratio (IQR) using tables, frequency, and percentages. Test of significance was carried out using ANOVA

(Kruskal-Wallis test analysis) and student t-test (Mann-Whitney U Test). A p-value of ≤ 0.05 was considered significant in the statistical analysis. A correlation study was carried out using spearman correlation analysis.

### Results

Table 1 shows the distribution and percentage of ABO blood groups among the study subjects and the control. It is showing that blood group O has the highest percentage followed by A, B, and AB respectively. $\chi 2=2.079$, df=1, p=0.5561.

Table 2 shows the Mann-Whitney U test analysis between the patients and controls group for the study parameters. The analysis of all the parameters indicated no statistically significance differences, P-value > 0.05.

Table 3 shows the Kruskal-Wallis test analysis among the different blood groups to determine the effect of red cell antigens, the association between ABO blood groups, and the level of procoagulants. It indicates no significant statistical difference among all the blood groups (P-value > 0.05). However, blood group B is associated with an insignificant increase in vWF level, and blood group AB is associated with an insignificant increase in FVIII.

Table 4 shows the correlation study using the Spearman correlation between vWF and the other study parameters. The p.value of all the correlations shows no statistical association, except between vWF and FVIII, which shows a strong positive association (P < 0.0001)

Table 5 shows the Mann-Whitney U test analysis of vWF between the male and female patients and controls. The result shows a strong statistical difference between the male and female patients, p.value of 0.0013

Table 6 shows the Mann-Whitney U test analysis of FVIII between the male and female patients and controls. The result shows a strong statistical difference between the male and female subjects, p.value of 0.0029.

**Table 1: Distribution of different ABO Blood groups among the study subjects**

| Blood group | Patients (%) | Control (%) | Total (%) |
|---|---|---|---|
| A | 14 (25.5) | 9 (32.1) | 23 (27.7) |
| B | 14 (25.5) | 4 (14.2) | 18 (21.7) |
| AB | 3 (5.5) | 3 (10.7) | 6 (7.2) |
| O | 24 (43.5) | 12 (42.9) | 36 (43.4) |
| Total | 55 (100) | 28 (100) | 83 (100) |

$\chi^2=2.079$, df=1, p=0.5561

**Table 2: Platelet and Procoagulants among the patients and controls.**

| Variables | Median (IQR) | | p-value# |
|---|---|---|---|
| | Patients (n=55) | Controls (n=28) | |
| **Platelet ($10^9$/l)** | 238.0 (178.0-334.0) | 213.0 (177.3-245.0) | 0.0750 |
| **vWF (ng/L)** | 454.9 (395.6-503.6) | 456.2 (391.1-511.2) | 0.9905 |
| **FVIII (pg/ml)** 242.0 (177.8-281.5) 228.4 (183.3-283.3) 0.8726 | | | |

*Key: # =Mann-Whitney U test; vWF = von Willebrand factor; FVIII = Factor VIII; T.Chol. = Total Cholesterol; Tg = Triglyceride; HDL= High density lipoprotein; LDL= Low density lipoprotein; n= Number, IQR = Inter quartile Ratio.*

**Table 3: Platelet and Procoagulants among different ABO Blood Groups**

| Variables | Median value (IQR) | | | p-value# |
|---|---|---|---|---|
| A (n=14) B (n=14) | AB (n=3) | O (n=24) | | |
| vWF (ng/L) | 424 (390.5-497.2) | 469.1 (398.2-501.8) | 462.6 (411.1-1585.0) | 451.0 (376.9-507.9) | 0.7101 |
| FVIIIpg/ml) | 243.2 (128.4-333.3) | 251.9 (165.4-561.6) | 325.9 (212.4-1304.0) | 225.9 (170.4-300.6) | 0.5402 |
| Platelet ($10^9$/l) | 227 (188.5-249.0) | 211.5 (157.3-352.5) | 291.0 (217.0-301.0) | 264.0 (197.0-409.0) | 0.3003 |

Key: # = *Kruskal-Wallis test; vWF = von Willebrand factor; FVIII = Factor VIII; T.Chol. = Total Cholesterol; Tg = Triglyceride; HDL= High density lipoprotein; LDL= Low density lipoprotein; n= Number, IQR = Inter quartile Ratio.*

**Table 4: Correlation analysis between vWF and other study parameters**

| Pairing variables | Correlation coefficient (*r*) | p-value |
|---|---|---|
| **VWF Vs FVIII** | 0.544 | <0.0001* |
| **vWF Vs Platelets** | 0.142 | 0.302 |
| **FVIII Vs Platelets** | 0.006 | 0.966 |

***Key:*** *vWF = von Willebrand factor; FVIII = Factor VIII; * = Significant finding.*

**Table 5: Level of vWF among male and female Patients and Controls**

| Subjects | Median (IQR) | p-value# |
|---|---|---|
| | Male | Female |
| Patients | 400.8 (295.1-507.9) | 479.4 (351.8-1585.0) |
| 0.0013 | | |
| Control | 393.0 (328.6 – 741.7) | 462.6 (202.7 – 875.9) |
| .1077 | | |

**Key:** # = Mann-Whitney U test; IQR = Inter quartile Ratio.

**Table 6: Level of FVIII among male and female Patients and Controls.**

| Subjects | Median (IQR) | p-value# |
|---|---|---|
| | Male | Female |
| Patients | 192.6 (96.30 – 281.5) | 250.6 (64.20 – 2435.0) |
| 0.0029 | | |
| Control | 196.3 (118.5 – 555.6) | 260.5 (76.54 – 876.4) |
| 0.0807 | | |

***Key: # = Mann-Whitney U test; IQR= Inter quartile Ratio.***

## Discussion

Several studies have elucidated that some ABO blood groups are associated with major cardiovascular risk and an increased rate of hypertension. This research was carried out to determine the distribution of ABO blood groups among hypertensive subjects and to assess the level of vWF, FVIII, and platelet among the study participants to provide a clear view of whether the parameters predispose one to develop hypertension or not.

It was observed that blood group O individuals have the highest percentage, followed by A, B, and AB the least. There was no significant difference in the blood group distribution between hypertensive patients and normal controls. This finding is similar to the study by Farshori and Kaur (13,14). Both studies showed a higher occurrence of hypertensive patients with blood group O and the least with blood group AB.(14) However, the findings of this study are in contrast with findings from Chandra, Gupta, Tulika and Ashish(15,16) respectively who reported a higher percentage of blood group B patients being more susceptible to developing hypertension.

The finding of this study indicates that there was no significant difference in the level of vWF, FVIII, and platelet number between the patients and the controls. These factors (vWF and FVIII) are known to be prothrombotic proteins. However, our research shows that WF and FVIII levels are not determining factors of developing hypertension in our population and this concurred with Folsom and Smith(17) findings, reported that two population-based studies showed no significant association between high vWF levels and hypertensive mortality.(17,18) However, this is in contrast with Varizi and associates(19) who reported a high level of vWF among hypertensive patients. Blann (20) also reported that the level of vWF was significantly increased in patients with hypertension, but was normalized in patients in whom hypertension was successfully treated. Ogbenna and others (21) reported that, mean vWF: Ag levels were significantly higher among hypertensives when compared with non-hypertensives. Jenkins and co-workers in 2012 has reported that, people with a high level of FVIII are at increased risk of developing deep vein thrombosis and pulmonary embolism. With regards to platelets, Gang in 2017 stated that individuals with increased MPV had a noticeably greater risk of developing hypertension, but our finding shows no significant difference between the patient's platelet count and that of the controls.(22,23) This may suggest that platelet counts may not be associated with the development of hypertension in our population. Therefore, environmental, racial, and genetic factors should be investigated.(23)

Furthermore, this research found no significant difference between vWF and FVIII in non-blood group O and blood group O individuals. It has been established in other researches that; vWF is higher in non-blood group O when compared with blood group O type.(24) These findings suggest that vWF may not be a predisposing factor for developing hypertension among non-blood group O individuals. This finding is in agreement with Agnes in 1999 who reported that the association between vWF and the risk of hypertension mortality was independent of blood group (O versus non-O). [27] However, it disagrees with the research of Asuquo among others in 2014 who reported that vWF is significantly lower in individuals of blood group O than in non-group O individuals. It is also at variance with Ohira and associates in 2007 who reported that non-O blood types are associated with higher levels of vWF and FVIII all of which are procoagulant proteins that circulate as a complex in blood. Also,Meade et al (26) reported that not all studies agreed with the findings that non-O group individuals have an increased thrombotic risk compared with blood group O individuals via having higher vWF-FVIII levels.(24-26) Some authors found that the association between vWF and the risk of hypertensive mortality was independent of blood groups.(26)

The present study revealed an insignificant differ-

ence in the level of vWF, FVIII, and platelet among different blood groups (A, B, AB, and O). This is an indication that these procoagulant proteins have no influence on the development of hypertension in different ABO blood groups in our population. This finding tallies with Agnes and colleagues in 1999 who reported that the association between vWF and risk of hypertensive mortality was similar among subjects with different blood groups, but at variance with Asuquo et al.,(24) who reported that vWf: Ag is higher in non-group O than blood group O individuals.(24,27) Our finding tends to also suggest that the ABO blood group antigens do not exercise a strong influence on the expression and activity of FVIII in the study of the population. It has been known that platelet plays a key role in both thrombosis and hemostasis and their involvement in atherosclerosis and occlusion of arterial vessels are known in hypertension. However, this present study observed no significant difference in platelet number and ABO blood group distribution. Barbalic and co-workers in 2010 reported that, in large-scale genomic studies, plasma levels of sP-selectin and sICAM-1 were associated with ABO gene variants, but no association was found between the platelet-bound P selectin levels and ABO blood group, suggesting that the ABO blood group may influence proteolysis and clearance from the circulation, rather than its production and cellular presentation.(28)

This present research observed a significant statistical correlation between vWF and FVIII levels. This is an indication that the two proteins may be associated and work together. vWF serves as a carrier for FVIII. It binds to the D'/D3 domain of vWF and protects FVIII from proteolytic inactivation through activated protein C. Patients lacking vWF exhibit reduced FVIII plasma levels due to rapid clearance. Lippi and associated reported in 2014 stated that changes in FVIII levels are likely dependent on the changes in vWF as FVIII circulates in a complex with vWF, which protects FVIII from early degradation. In addition, deficiency of vWF is accompanied by reduced levels of FVIII, while usually, an increase in vWF levels drives an increase in FVIII levels. However, there was no significant correlation between vWF and the remaining study parameters.(29-31)

The current study also observed that the level of vWF and FVIII was more among females than in male subjects. Females may therefore be more prone to developing hypertension due to the increase of these procoagulants. Our finding is consistent with that of Asuquo et al., (24) work who reported that the level of vWF was significantly higher among the female subjects compared to their male counterparts.(24) It is also similar to that of Folsom et al., (17) who found higher relative risks of mortality associated with vWF among women than among men. However; Asuquo et al., (24) in a research conducted on apparently healthy individuals, reported that a comparison of the levels of vWf: Ag between the male and female subjects did not show a significant difference. In other words, that sex does not affect vWf: Ag.(17,24)

## Conclusion

From our study, hypertension was more prevalent among blood group O individuals. However, the risk of developing hypertension is independent of the ABO blood group of an individual. Increased levels of vWF and FVIII are associated with developing hypertension in individuals who are females.

## Limitations

This study is limited to patients with hypertension along side their controls; as such researchers concentrated on sufficiently similar cases of control that did not into account a wide array of possible factors. Secondly; it has led to generalized conclusion and lastly data selection predetermined the hypothesis.

## Recommendations

Routine screening for vWF and FVIII especially among female patients should be adopted for early diagnosis of hypertension.

## Conflict of interest

All authors declared no conflict of interest while carrying out the research.

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