Lipid Profiles in Different ABO Blood Groups in Owerri Metropolis, South East Nigeria: A Preliminary Study

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ABSTRACT

Objective*: The study was set out to investigate the possibility of using blood groups as predictive indices for diseases associated with lipid metabolism.* ***Methods****: Lipid profiles were examined in 100 apparently healthy male (40) and female (60) subjects of different ABO blood groups aged between 18-30 years from Imo State University. Of these, 20 were blood group A, 30 were B blood type, 4 were AB blood type, and 46 were blood group O. Lipid profile parameters were determined according to enzymatic assay using a commercial kit from Randox Laboratories, United Kingdom and calculation using Friedewald's equation. Blood group was determined by monoclonal ABO blood grouping reagent.* ***Results****: Total Cholesterol (140.62 ± 21.66 mg/dl) and High-Density Lipoprotein (96.20 ± 40.32 mg/dl) were highest in blood group B. Blood group A had the highest level of Triglyceride (80.84 ± 18.60 mg/dl) and Very Low-Density Liporprotein (15.21 ± 6.24 mg/dl). Blood group O showed TC level of 130.60 ± 34.76 mg/dl with the highest level of LDL (70.74 ± 20.15 mg/dl) and the lowest level of HDL (51.68 ± 20.50 mg/dl) compared to non- O blood types (P<0.05).* ***Conclusion****: The study revealed that blood group O may have a higher propensity for dyslipidemia, suggesting increased risk for disease associated with lipid metabolism.*

Keywords: ABO blood group, Lipid metabolism, Lipid profile, Dyslipidemia

INTRODUCTION

ABO blood group constitute ABH-antigens which are complex carbohydrate molecules (glycoprotein and glycolipids) expressed on the extracellular surfaces of human cells and tissues, including: red cell membrane, platelets, and vascular endothelial cell [1-3]. ABO blood group has been recognized as an important system in clinical practice particularly in the field of transfusion and transplantation medicine [4]. Since its discovery, ABO group has been studied as etiological factors of many diseases [5-7]. The clinical implication of ABO blood group system may well extend beyond immunohaematology [8]. Studies in the recent past have reported a critical involvement of ABO blood group system in the development of cardiovascular diseases (CVDs) [3,9-13], certain malignancies such as gastric cancer [14-16] and malaria [7,17-19].

Considerable evidence underpins CVDs to be linked with ABO blood group system [3,4,13], although the mechanism linking ABO blood group with CVD, particularly atherosclerosis, remains unclear. However, previous studies suggest that ABH antigen act as the principal agent for endothelial cell proliferation [3], as such plays a role in the disease process by a modulation in the vascular endothelial haemostasis [1,20]. Besides, the theory that glycosyltransferases (enzymes involved in the modification of the A, B and H antigens) regulate circulation of soluble products of cellular adhesion molecules (CAMs) - biomarkers of inflammation-related diseases - may explain the relationship [4,21]. Previous evidence has reported increase in the biomarkers to be associated with cardiovascular diseases (CVDs) [22-26]. For instance, it was quantitatively demonstrated that individuals with A blood group had reduced level of some of these inflammation-related disease biomarkers compared to B or O blood groups [4]. Similar finding was reported in individual with blood group A in another study, given a decreased cleavage of CAMs with A- antigen [27]. This may explain why individuals expressing the A-antigen have been reported to be less likely associated with CVD. In the contrary, documented evidence suggest a close relation between non- O blood groups (A, B, AB) and CVD [13]. For instance, a study has shown that individuals with blood group A have a more frequent family history of CVD as opposed to other ABO blood groups [28]. Perhaps, due to increased level of von Willebrand Factor (vWF) and factor VIII, which predispose non-O subjects to greater risk of arterial and venous thrombo-embolism than individuals with O blood group [7,13]. Furthermore, established evidence demonstrates a close association of ABO blood group with lipid metabolism [29], which may further explain the linkage between CVD and ABO blood group system. In the light of this, the study was set out to investigate the possibility of using ABO blood group system as predictive indices for disease associated with lipid metabolism by assessing the plasma lipid profile.

MATERIALS AND METHODS

This cross-sectional study utilized a convenience sampling procedure to recruit one hundred (100) students, comprising forty (40) males and sixty (60) females. Subjects recruited were students of Imo State University aged between 18 and 30 years. Approval for the study was obtained from Department of Medical Laboratory Sciences on behalf of Students Affairs Department of Imo State University Owerri, Nigeria. Each study participants completed a well structured questionnaire to help exclude participants with possible conditions and/or behavioral risk factors that may affect lipid metabolism. Among students recruited for the study, those who gave full consent were included whereas those with known cases of smoking, obesity, diabetes, hypertension and other related disorders or those on medication that could affect lipid metabolism were excluded from the study. Total cholesterol and triglycerides were measured by enzymatic colorimetric method as described by Trinder [30], using a commercial kit by Randox laboratories, United Kingdom. HDL was measured enzymatically after all non HDL lipoproteins were removed. LDL-C was calculated using Friedewald's equation:

$$LDL = total\ cholesterol - \{HDL + (TG/5)\}$$

Blood grouping was done using monoclonal ABO blood grouping reagents by CLAS Technology, United Kingdom.

Data analysis

Both the descriptive and inferential statistics was carried out using the SPSS version 22. The results obtained were expressed in mean ± standard deviation (SD). Analysis of variance (ANOVA) was used to calculate the difference in mean of each lipid profile fractions between different ABO blood groups. Within two blood groups comparison was determined using t-test. A statistically significant difference was set at $P < 0.05$.

RESULTS

Out of the a total of 100 students recruited for the study, 20% of the subjects were of blood group A, 30% were of blood group B, 4% were of blood group AB and 46% were of blood group O. Table 1 shows the blood groups of the subjects with their lipid profile levels. The mean result showed that total cholesterol (TC) (140.62 ± 21.66) and High density cholesterol (HDL) (96.20 ± 40.32) were highest in blood group B while Triglyceride(TG) (80.84 ± 18.60) and very low density lipoprotein (VLDL) (15.21 ± 6.24) was highest in group A. LDL level was lowest (29.71 ± 15.17) in AB blood group. Blood group O showed TC level of 130.60 ± 34.76 with highest level of LDL (70.74 ± 20.15) and lowest level of HDL (51.68 ± 20.50) compared to A, B, and AB blood type respectively ($P < 0.05$).

Table 1: Mean and Standard deviation of lipid profile in different ABO blood group systems

	A	**B**	**O**	**AB**
TC (mg/dl)	129.89 ± 34.83	140.62 ± 21.66**	130.60 ± 34.76	115.20 ± 25.30
TG (mg/dl)	80.84±18.60	60.50 ± 25.18	68.84 ± 30.50	70.00 ± 35.50
HDL (mg/dl)	74.61 ± 37.62	96.20 ± 40.32**	51.68 ± 20.50*	55.41 ± 18.20
LDL (mg/dl)	49.85 ± 10.55	60.20 ± 18.50	70.74 ± 20.15**	29.71 ± 15.17
VLDL (mg/dl)	15.21 ± 6.24	12.64 ± 5.21	14.43 ± 4.57	13.50 ± 4.10

(*) statistically significantly low, $p < 0.05$ across blood group types; (**) statistically significantly high, $p < 0.05$ across blood group types

Within two blood groups comparison

TG: A *vs.* B; $p < 0.05$, B *vs.* O; $P > 0.05$, O *vs.* A; $p > 0.05$
HDL: A *vs.* B, B *vs.* O, O *vs.* A; $p < 0.05$
LDL: A *vs.* B, B *vs.* O, O *vs.* A; $p < 0.05$
VLDL: A *vs.* B, B *vs.* O, O *vs.* A; $p < 0.05$
TC: A *vs.* B, B *vs*. O, O *vs.* A; $p < 0.05$

DISCUSSION

The present study attempted the evaluation of the impact of ABO blood groups on lipid profile of apparently healthy subjects. Subjects with O blood group have higher level of LDL-C and lower HDL-C compared to non-O groups. This may suggests that subjects with O blood group may have higher propensity for diseases associated with lipid metabolism than non-O blood group types. However, this contradicts the study by Li et al. [21], which reported that non-O subjects (A, B, AB) had higher level of LDL-C compared with O subjects as such does not favour the report by previous researchers [3,12,13,31] who suggest that non-O blood type are at significantly greater risk for developing CVDs compared to O blood group. At this same time, the study does not underpin the report that only non-O subjects (A, B, AB) have close relation with CVD as suggested by other researcher [7,13]. However, the current study seems to underpin previous report by Anvari et al. [32], who demonstrated that the prevalence of coronary heart disease in blood group O is markedly higher than in all other ABO blood groups. Perhaps due to elevated level of LDL-C in O blood type as shown in the present study. The mechanism underlining such blood-type differences in association with lipid profile levels is still unclear; however, it may be attributed to the fact that, there is a distinct ABO genetic association pattern with plasma lipids [33]. Moreover, given that ABO is associated with cholesterol metabolism [21], CVDs may not only be conferred on non-O blood types as shown by previous studies. The fact that blood group B had highest TC and HDL while O has highest LDL is suggestive that both blood groups B and O might be predisposed to diseases associated with lipid disorders; however the consequence of increased level of TC in blood group B also could be ameliorated by elevated level of HDL present.

However, the findings should be considered in the context of the study limitations. First, selection bias may be likely; given that the sample was not randomly selected as such may not be a representative sample for the population. Secondly, as a single centre study (among students) with relatively small size and convenience sampling technique, generalization of result is limited and should be undertaken with caution. Findings are only association, not causation as this study was a cross-sectional survey which cannot ascertain causal link between ABO blood group and dyslipidemia. The use of self-administered questionnaire as a method of excluding those with possible conditions/behavioral risk factors likely to impact lipid metabolism may be associate with a number of bias such as recall bias and social desirability bias [34], given that most participants may fail to fully remember all cases or decide to conceal some for fear of being stigmatized among their fellow students. Therefore, further study with large sample size, perhaps population-based survey across all age, may be necessary to validate the level of significant shown in this study with small sample size. However, taken together, the study had made important contribution in that, to the researchers' best knowledge, this is among the first study that shows the association of lipid profile parameters among ABO blood group system in adult Nigerian.

CONCLUSION

The study, therefore, demonstrates that among different ABO blood group studied, subjects with blood group O is associated with reduced level of HDL-C and elevated level of LDL-C as opposed to non-O blood types. This suggests that individuals with blood group O may be most at risk for developing disease associated with lipid metabolism.

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REFERENCES

[1] S. Zhou, I. Welsby, *World J. Cardiology.*, **2014**, 6(9), 985-992.

[2] S. Karabuva, V. Carevic, M. Radic, D. Fabijaniic, *Biochem. Med. Zagreb.*, **2013**, 23(3), 351-359.

[3] H. Nafakhi, H. Al-Nafakh, A. Al-Mosawi, *Artery Research.*, **2016**, 14, 36-40.

[4] Y. Chen, X. Zhuo, Y. Lin, W. Huang, J. Xiao, J. Zeng, L. Jiang, C. Chen, H. Lin, M. Dettke, *Transfusion.*, **2015**, 55, 2759-2765.

[5] G. Garraty, W. Dzik, P. Issitt, D. Lubin, *Transfusion.*, **2000**, 40, 477-489.

[6] P. Bloomfield, A. Bradbury, N. Grubb, D. Newby, Davidsons Principles and Practice of Medicine, Elsevier Churchill Livingstone, Edinburg, **2009**, 20, 519-648.

[7] D. Anstee, *Blood.*, **2010**, 115(23), 4635-4643.

[8] G. Liumbruno, M. Franchini, *Blood transfusion.*, **2013**, 11, 491-499.

[9] F. Dentali, A. Sironi, W. Ageno, *Seminars In Thrombosis Hemostasis.*, **2012**, 38, 535-548.

[10] M. Franchini, E. Favaloro, G. Targher, G. Lippi, *Critical Revision Clinical Laboratory Science.*, **2012**, 49, 137-149.

[11] M. Franchini, M. Makris, *Blood Transfusion.*, **2013**, 11, 164-165.

[12] L. Spiezia, E. Campello, M. Bon, *Blood Transfusion.*, **2013**, 11, 250-253.

[13] H. Takagi, T. Umemoto, *American J. Cardiology.*, **2015**, 116, 699-704.

[14] G. Edgren, H. Hjalgrim, K. Rostgaard, *Am. J. Epidemiology.*, **2010**, 172, 1280-1285.

[15] Z. Wang, L. Liu, J. Ji, *Inter. J. Molecular Sci.*, **2012**, 13, 13308-13321.

[16] C. Rizzato, I. Kato, M. Plummer, *Inte. J. Cancer.*, **2013**, 133, 315-322.

[17] C. Cserti-Gazdewich, W. Mayr, W. Dzik, *Vox Sanguinis.*, **2011**, 100, 99-111.

[18] A. Panda, S. Panda, A. Sahu, *Malaria J.*, **2011**, 10, 309.

[19] C. Timmann, T. Thye, M. Vens, *Nature.*, **2012**, 489, 443-446.

[20] H. Lee, Y. Lin, C. Lin, C. Wang, C. Chang, L. Hsu, *Internal Medicine.*, **2012**, 51, 1815-1820.

[21] S. Li, R. Xu, Y. Guo, Y. Zhang, C. Zhu, J. Sun, J. Li, *Nutrition, Metabolism & Cardiovascular Diseases.*, **2014**, 25, 411-417.

[22] P. Trinder, *Annals of Clinical Biochem.*, **1969**, 6, 24-27.

[23] N. Larson, E. Bell, P. Decker, M. Pike, C. Wassel, M. Tsai, J. Pankow, W. Tang, N. Hanson, K. Alexander, N. Zakai, M. Cushman, S. Bielinski, *Atherosclerosis.*, **2016**, 251, 422-429.

[24] S. Barbaux, S. Blankenberg, H. Rupprecht, C. Francomme, C. Bickel, G. Hafner, V. Nicaud, J. Meyer, F. Cambien, L. Tiret, *Arteriosclerosis, Thrombosis, and Vascular Biology.,* **2001**, 21, 1668-1673.

[25] P. Ridker, C. Hennekens, B. Roitman-Johnson, M. Stampfer, J. Allen, *Lancet.*, **1998**, 351, 88-92.

[26] S. Hwang, C. Ballantyne, A. Sharrett, L. Smith, C. Davis, A. Gotto Jr, E. Boerwinkle, *Circulation.*, **1997**, 96, 4219-4225.

[27] P. Ridker, *European Heart J.*, **1998**, 19, 1119-1121.

[28] J. Polgar, J. Matuskova, D. Wagner, *J Thrombosis and Haemostasis.*, **2005**, 3, 1590-1596.

[29] M. Barbalic, J. Dupuis, A. Dehghan, J. Bis, R. Hoogeveen, R. Schnabel, V. Nambi, M. Bretler, N. Smith, A. Peters, C. Lu, R. Tracy, N. Aleksic, J. Heeriga, J. Keaney Jr., K. Rice, G. Lip, R. Vasan, N. Glazer, M. Larson, A. Uitterlinden, J. Yamamoto, P. Durda, T. Haritunians, B. Psaty, E. Boerwinkle, A. Hofman, W. Koenig, N. Jenny, J. Witteman, C. Ballantyne, E. Benjamin, *Human Molecular Genetic.*, **2010**, 19, 1863-1872.

[30] A. Abdollahi, M. Qorbani, H. Asayesh, M. Nouroozi, M. Mansourian, *Scientific J. Iran Blood Transfusion Organisation.,* **2012,** 8(4), 293-297.

[31] C. Petrochko, Cardiology, 2012. Available from: http://www.medpagetoday.com/cardiology/myocardialinfarction/34205

[32] M. Anvari, M. Boroumand, B. Emami, A. Karimi, M. Soleymanzadeh, S. Abbasi, S. Saadat, *Labmedicine.*, **2009**, 40(9), 528-530.

[33] C. Carpeggiani, M. Coceani, P. Landi, *Atherosclerosis.*, **2010**, 211, 461-466.

[34] A. Bowling, Research *Methods in Health*, Open University Press, New York, **2009**, 3.