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Evaluation of Circulating APOL1 in Diabetes Mellitus Patients attending Aminu Kano Teaching Hospital

Jibril, A.A.*¹, Gwarzo, M.Y.², Abdullahi, H.L.², Babale, N.S.³, Adamu, F.³, Khalid, U.M.¹, Halliru², A.H., Jamilah, I.S.³, Ahmed, A.Y.³, Mahmud, R.I.⁵, Usman, M.M.³.

NSIA Kano Diagnostic Centre (NKDC)¹, Department of medical laboratory sciences, Bayero University Kano², Department of Chemical Pathology, Aminu Kano teaching Hospital³, Department of Medical Laboratory Sciences, Kaduna State University⁴, School of Basic Medical Science, Skyline University Nigeria⁵.

Author for Correspondence*: aysheraj@gmail.com/aisha.jibril@nkdc.ng/+234-805-218-8721.

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Abstract

Apolipoprotein L1 (ApoL1) was originally recognized as a component of human high-density lipoprotein (HDL) particles. Subsequent research uncovered several highly homologous apolipoprotein L proteins encoded by genes clustered at the long arm of chromosome 22 (22q12.1–13.1). Data suggests that ApoL1 may be associated with lipid and glucose metabolisms or may exert an antioxidative effect as a distinct HDL subspecies in certain dyslipidaemia subjects. The present study aimed to evaluate the circulating APOL1 in diabetes mellitus patients attending Aminu Kano teaching Hospital. Two hundred study participants were recruited out of which 100 were Type Diabetes mellitus patients and 100 were apparently healthy individuals. Fasting plasma glucose, Glycated haemoglobin, lipid profile and serum Apolipoprotein L1 were measured in the blood samples collected from each study participants. Student t- test and Pearson correlation were employed for mean comparison and correlation of the data collected respectively. Results shows significant difference in the BMI, FPG, HbA1c, Lipid Profile and APOL1 between the T2DM and the control group (p = 0.005). Pearson's correlation shows no significant association between APOL1 and T2DM markers (FPG and HbA1c) as well as lipid profile.

Keywords: APOL1, Diabetes Mellitus, Aminu Kano Teaching Hospital

Introduction

Global estimates for the prevalence of type 2 diabetes are as high as 9.3%, affecting 463 million

people while the prevalence of type 2 diabetes is lowest in the sub-Saharan Africa (SSA) region (4.7%). The prevalence varies by country, with the highest number of people with type 2 diabetes residing in more affluent countries (Saeedi *et al.*, 2019). Africa, like the rest of the world, is experiencing an increasing prevalence of diabetes alongside other non-communicable diseases, mainly as a result of urbanization, sedentary lifestyles, obesity and population growth and ageing (Noubiap *et al.*, 2015). Estimates for 2013 by the International Diabetes Federation (IDF) indicate that the number of adults with diabetes in the world will expand by 55%, from 381.8 million in 2013 to 591.9 million in 2035 (Noubiap *et al.*, 2015). The largest increase of the population with diabetes will occur in sub-Saharan Africa, with a projected growth of 109.6%, from 19.8 million in 2013 to 41.5 million in 2035 (Noubiap *et al.*, 2015). In 2019, the estimated diabetes prevalence in Nigeria for adults aged 20-79 years was 3.9% according to the International Diabetes Federation (IDF) which corresponds to approximately 4.6 million people living with diabetes (IDF, 2019).

Apolipoprotein L1 (ApoL1) was originally recognized as a component of human high-density lipoprotein (HDL) particles (Nishimura *et al.*, 2019). Subsequent research uncovered several highly homologous apolipoprotein L proteins encoded by genes clustered at the long arm of chromosome 22 (22q12.1–13.1). Among them, human apolipoprotein L1 (APOL1) was first reported in connection with the defense against trypanosomiasis (Vanhollebeke *et al.*, 2006) and is regulated by antiviral pathways; notably, it is potently upregulated by interferons

(Ma *et al.*, 2019). Of the 6 members of the *APOL* gene family, only *APOL1* has acquired a secretory signal peptide permitting cellular export of the *APOL1* protein into the blood stream. It possesses amphipathic helices, which confer a high level of lipophilicity, and it travels in the blood entirely bound to HDL during the circulation of normolipidaemic population (Aminu *et al.*, 2021). The G0 allele of the *APOL1* gene encodes wild-type *APOL1*, a protein that protects the host against infection caused by *Trypanosome brucei* and *Trypanosome evansi*. *Trypanosome brucei rhodesiense* has evolved to inactivate wild-type *APOL1*. In turn, some African populations affected by endemic trypanosomiasis have developed variants of *APOL1* (coded by the G1 and G2 alleles) that block *APOL1* inactivation by *Trypanosome brucei rhodesiense*. Heterozygous carriage of either G1 or G2 variants protects the host against this parasite. Due to positive selection, G1 or G2 alleles virtually only exist in some human African groups and are absent in European or Asian populations (Adeva-Andany *et al.*, 2023).

APOL1 and Insulin resistance

Lipid droplets are lipid storage organelles derived from the endoplasmic reticulum. Neutral lipids such as triglycerides and cholesterol esters are initially deposited and clustered within the endoplasmic reticulum. A nascent lipid droplet subsequently detaches from the endoplasmic reticulum and enters the cytoplasm. The proteins involved in the separation of the emergent lipid droplet from the endoplasmic reticulum are not fully known. Wild-type *APOL1* is located in the lipid droplet, whereas mutated *APOL1* remains positioned on the endoplasmic reticulum, suggesting that normal *APOL1* may participate in the detachment of the emergent lipid droplet from the endoplasmic reticulum and may thus contribute to the normal formation of the lipid droplet. Accordingly, lipid droplet generation is impaired among carriers of *APOL1* risk variants. The defective generation of lipid droplets has been associated with insulin resistance (Chun *et al.*, 2019). Observational studies show that carriers of *APOL1* risk variants develop more frequently essential hypertension (Nadkarni *et al.*, 2017) and obesity compared to noncarriers, suggesting that individuals with an *APOL1* high-

risk genotype may endure more severe insulin resistance (Nadkarni *et al.*, 2021). Accordingly, clinical and histopathological manifestations of insulin resistance in the kidney are remarkably similar to the clinical phenotype associated with *APOL1* risk variants, namely albuminuria, CKD, faster CKD progression, glomerulomegaly, FSGS, and arterionephrosclerosis (Valdez Imbert *et al.*, 2022). In addition, plasma *APOL1* levels are increased in patients with more pronounced insulin resistance compared to those who are more insulin-sensitive (Croyal *et al.*, 2022).

In innate immunity, HDL ApoL1 is the trypanolytic factor in human serum, that forms anion channels on the lysosomal membrane of *Trypanosoma brucei* that causes sleeping sickness leading to cell death. Additionally, the association between plasma ApoL1 and the clinical characteristics including lipid-related factors in normolipidemic, dyslipidaemic, or diabetic subjects, has also been explored (Nishimura *et al.*, 2019). An independent positive correlation of ApoL1 with total cholesterol (Total-C) and TG in normolipidemia, and with TG in dyslipidemia. Moreover, it was observed that ApoL1 was elevated in the non-insulin dependent diabetic patients with hypertriglyceridemia (Duchateau, 2000). Another study reported that TG and hyperglycemia are the strongest predictors of plasma ApoL1 in dyslipidaemic coronary artery disease subjects with low HDL cholesterol (HDL-C) (Albert *et al.*, 2005). Evidence linking *APOLs* and diabetes is still very scarce but recently a positive association between basal serum *APOL1* levels and the risk of developing type 2 diabetes was reported by Croyal *et al.*, (2022).

These data suggest that ApoL1 may be associated with lipid and glucose metabolisms or may exert an antioxidative effect as a distinct HDL subspecies in certain dyslipidaemic subjects. It is as well suggested that serum ApoL1 levels may elevate and exert certain biological activity in subjects with insulin resistance that frequently presents dyslipidemia marked by hypertriglyceridemia; however, there is a paucity of data on association between ApoL1 and insulin resistance.

The present study aimed to evaluate the circulating APOL1 in diabetes mellitus patients attending Aminu Kano teaching Hospital

Materials and Method

This is a cross-sectional case control study to evaluate the serum levels of APOL1 among diabetes mellitus patients attending Aminu Kano teaching Hospital. The study was conducted in Aminu Kano teaching Hospital. Inclusion and Exclusion criteria were duly considered.

Ethical Approval/ Informed Consent

Ethical approval was sought and obtained from the ethical committee of Aminu Kano teaching Hospital with the reference number (NHREC/28/01/2020/AKTH/EC/3853). Informed consent was obtained from all participants and utmost confidentiality was maintained. Ethical consideration was in line with Helsinki declaration. Written informed consent was obtained from all the study participants prior to inclusion in the study. Self-administered questionnaires were prepared and given to all study participants. Anthropometric variables were collected as participants were enrolled. This help in obtaining their socio demographic data including Age, gender, tribe, occupation, family history of DM and duration of diabetes mellitus as well as smoking or alcoholic status.

Sample collection

Under aseptic condition, 3mls of blood was collected in SST from both subject and control group. The SST blood was centrifuged at 4000rpm for 5mins to separate the serum. The SST was barcoded and ready to be analyzed.

Sample size

The sample size calculation was done according to the formular below. The prevalence of T2DM form Uloko *et al.* (2018) was used.

The sample size was calculated using the formula below (Niang *et al.*, 2006).

$$n = \frac{Z^2 pq}{d^2}$$

Were

n= number of samples

Z= confidence interval of 95%= 1.96

p= Prevalence of Type 2 diabetes mellitus = 5.77% (Uloko *et al.*, 2018)

q= (1-p)

d= Error margin taken to be 5% (0.05)

$$n = \frac{Z^2 pq}{d^2} = \frac{(1.96)^2 \times 0.0577 \times (1-0.0577)}{0.05 \times 0.05} = 83.5$$

The minimum sample size was therefore 83.5, plus 10% attrition rate, which was round up to 100.

Therefore, the study was done on 100 T2DM against 100 controls.

Method of Sample Analysis

The serum APOL1 was detected using sandwich ELISA technique from Cusabio Technology LLC, USA. Fasting plasma glucose was determined via the principle of Glucose oxidase method. The glycated hemoglobin, HbA1c was determined using the principle of fluorescence immunoassay technology. Total cholesterol, triglyceride and HDL were determined using enzymatic method. LDL was calculated using the Friedewald Equation.

Statistical analysis

The data obtained were analyzed using Statistical Package for Social Scientific (SPSS) version 16. The data were expressed as mean ± standard deviation (Mean ± SD). Students' T test and Pearson's correlation were used to compare means and determine correlation respectively.

Results

Table 1: Mean Comparison Of BMI between DM And Control Group

Parameters	DM	Control	P-value
Age (Years)	50	38	<0.001**
Weight (Kg)	64.42±14.64	61.28±5.67	0.021*
Height (cm)	1.60.99±5.06	1.63.04±4.16	0.079
BMI	24.79±5.33	22.81±1.86	<0.001**

Key: DN= Diabetic Nephropathy, DM= Diabetes mellitus, BMI, Body mass index. P-value<0.05

Table 2: Mean Comparison of Fasting Plasma Glucose and HbA1c Between The 2 Groups.

Parameters	DM	Control	P-value
HbA1C	6.91±5.77	4.50±0.49	<0.001*
FPG	6.86±1.53	4.10±0.77	0.011*

Key: DN= Diabetic Nephropathy, DM= Diabetes mellitus, HbA1c= Glycated haemoglobin, FPG= Fasting plasma glucose. P-value<0.05

Table 3: Mean comparison of Serum APOL1 and Lipid Profile (TCHOL, HDL, TRIG and LDL) Diabetes Mellitus and Control Group.

Parameters	DM	Control	P-value
TCHOL	5.01±0.89	4.65±0.35	<0.001*
HDL	0.87±0.25	1.45±0.13	<0.001*
TRIG	1.66±0.65	1.30±0.33	0.565
LDL	3.48±0.81	2.86±0.23	<0.001*
APOL1	25.14±7.61	20.58±2.98	<0.000*

Key: DN= Diabetic Nephropathy, DM= Diabetes mellitus, TCHOL= Total cholesterol, HDL=high density lipoprotein, TG=triglyceride, LDL=low density lipoprotein, APOL1=Apolipoprotein L1. P-value<0.05

Table 4: Shows Correlation Between Serum APOL1 and Lipid Profile.

APOL1	R	p-value
FPG	0.04	0.967
HbA1c	-0.147	0.143
TCHOL	-0.108	0.285
HDL	0.039	0.698
TRIG	0.024	0.815
LDL	0.009	0.903

Key: FPG=Fasting plasma glucose, HbA1c= glycated hemoglobin, TCHOL=Total cholesterol, TG=Triglyceride, HDL= High density lipoprotein, LDL= Low density lipoprotein, r=pearson correlation coefficient, level of significance=p 0.05

Discussion

Apolipoprotein L1 (ApoL1) was originally recognized as a component of human high-density lipoprotein (HDL) particles and its levels correlate with the plasma triglyceride (TG) levels, and increases in subjects with a deficiency of cholesteryl ester transfer protein (CETP) that exchanges cholesterol and TG between the TG-rich lipoprotein and HDL.

Our result shows that the BMI is higher among the DM group when compared with the control group. This may be because T2DM accounts for more than 90% of DM worldwide, fueled by relative insulin deficiency owing to pancreatic β -cell dysfunction and insulin resistance is strongly connected to overweight and obesity, aging, ethnicity, and family history as reported by Ruxe *et al.* (2023). This is in line with the findings of Jaja and Yarhere (2015) who reported high BMI as a risk factor of DM in adolescents and that of xx who reported higher fasting plasma glucose levels in diabetes patients with high BMI in comparison with those with normal BMI (Riaz *et al.*, 2009). Another study supporting ours is that of Abdullahi *et al.*, 2019. Who reported similar findings in their report on Inter-relationship between type-2 diabetes mellitus, obesity and Hypertension in Nigeria.

The American Diabetic Association (ADA) reports glycosylated hemoglobin (HbA1c) as the best tool for measuring glycemic control to prevent complications and reduce its cost for management (Ahmad *et al.*, 2014). The fasting plasma glucose and glycated hemoglobin was found to be higher among the DM group when compared with that of the control even though the glycated haemoglobin seems to be of poor control according to A recent study from Ethiopia conducted on diabetic patients which defined good glycemic control at HbA1c less than 7% (Bereda and Bereda, 2021). Our report is similar to that of Alqareem *et al.*, 2022 who reported hbA1c of less than 7% for the majority of his study participants with DM and is better than the glycated hemoglobin levels of $7.9\% \pm 2.4$ to $8.3\% \pm 2.2$ reported by Oghabon (2014).

Reports show that Poor glycemic control with persistent hyperglycemia are associated with diabetic

complications including nephropathy, neuropathy and cardiovascular disease Lawal *et al.* (2018).

The lipid profile shows dyslipidemia with high levels of TCHOL LDL and low levels of HDL, among the DM group when compared with the DN group with significance of 0.01. while the trig is higher among the DM group when compared with the control but not statistically significance. It is reported that the prevalence of dyslipidemia among Nigerians living with T2D is high and Insulin resistance has been a key concept propounded behind the high prevalence of dyslipidemia in T2D (Chehade *et al.*, 2013). Our findings are in line with the report from Azeez *et al.*, (2021) who reported that the most common lipid disorders among patients living with T2D in Nigeria are hypertriglyceridemia and low HDL-C. It is also consistent with the findings of Omodanisi *et al.* (2020) in South Africa and report from a meta-analysis done among Ethiopians living with T2D which also reported low HDL-C respectively (Dagneu *et al.*, 2021). These maybe because Insulin resistance enhances the increased production of TRLs which exchange triglyceride with cholesterol on HDL-C, making the HDL-C more susceptible to increased clearance from the bloodstream which invariably leads to low HDL-C (Azeeza *et al.*, 2021).

Most recent studies correlate APOL1 risk variants instead of circulating APOL1 with either kidney markers, cardiovascular markers or Diabetic markers. While some of these studies report strong association with these markers, many others do not Madhavan *et al.*, 2014. In contrast to our study, we determine the circulating APOL1 instead of the variants.

The circulating APOL1 is higher among The DM when compared with control which suggests its association with DM. This is similar to the findings of Nishimura *et al.*, (2019) which report higher circulating APOL1 among patients with more pronounced insulin resistance, though they also reported that Subjects with higher plasma APOL1 had greater waist circumference and BMI, compared with participants with a lower APOL1 level, which is similar to our findings on BMI.

But the correlation shows no significant association between APOL1 and HbA1c, fasting plasma glucose and lipid profile. This is in contrast with the findings of Nishimura *et al.* (2021) which report an association between APOL1 and HDL suggesting that circulating ApoL1 may be associated with abnormal HDL metabolism in insulin-resistant states, indicating a potential regulatory role of insulin signaling on APOL1 levels. Other findings reported that patients with increased plasma APOL1 levels show more pronounced insulin resistance than those with lower circulating APOL1 (Croyal *et al.*, 2022). In another cross-sectional study that recruited 126 non-diabetic volunteers and 36 T2D patients, circulating levels of APOL1 were increased in participants with more pronounced insulin resistance, independent of confounding variables.

In line with our findings is that of Weckerle *et al.*, who from his study on characterization of circulating APOL1 protein complexes in African Americans reported no significant correlation between Serum APOL1 and HDL.

Conclusion

We found no significant association on serum APOL1 with Diabetic markers and Lipid profile suggesting that APOL1 does not seem to influence the lipid parameters in this cohort. Further studies may be needed to explore the role of APOL1 in other metabolic conditions.

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Conflicts of Interest: Nil.

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