ORIGINAL ARTICLE

INVESTIGATION ON LIPID PROFILE AND ATHEROGENIC INDICES AS MARKERS OF CARDIOVASULAR DISEASE AMONG STUDENTS

| Idume, Nkechinyere I. [1] | Onyenekwe, Charles C.[1] | Anya, Kingsley K.[1] | Okoro, Godwin O.[1] | Fasogbon, Samuel A.[2*] | Oko, Patrick I.[3] |Aliu Israel I.[4] | Ayodele Kabiru O.[5] | Adeyinka, Sumbola A.[6] | Onukwue, Ifeoma G.[7] | Oguntunnbi, Damilola E. [8] | Emejuru, Chinonso F.[9] | and | Adebayo, Ahmed O.[10] |

[1.] Department of Medical Laboratory Science, Ebonyi State University | Abakaliki | Ebonyi state | Nigeria |
[2.] Public Health In-vitro Diagnostic Control Laboratory | Medical Laboratory Science Council of Nigeria (MLSCN)| Yaba-Lagos| Nigeria |
[3.] Department of Medical Laboratory Services | Asokoro District Hospital | Abuja| Nigeria |
[4.] Department of Quality Management System| PEGISOL Consultancy | Airport Road | Abuja |
[5.] Department of Chemical Pathology | Lagos State University Teaching Hospital| Ikeja| Lagos State |
[6.] Department of Anatomic Pathology | Irrua Specialist Teaching Hospital | Irrua | Edo State |
[7.] Department of External Quality Assurance| National External Quality Assessment Laboratory | MLSCN | Zaria| Nigeria |
[8.] Department of Medical Microbiology | Federal Teaching Hospital | Ido-Ekiti| Ekiti State |
[9.] Department of Medical Laboratory Services | Borngreat Healthcare and Research Laboratories| Lagos |
[10.] Department of Medical Laboratory Science | Lagos Sate College of Health Technology | Yaba | Lagos State |

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ABSTRACT

Background: Cardiovascular diseases (CVD's) are said to be chronic or acute disorders of the heart and blood vessels and it include coronary heart disease, cerebrovascular disease, peripheral artery disease, rheumatic heart disease, congenital heart disease, deep vein thrombosis and pulmonary embolism. Atherogenic index of plasma (AIP), dyslipidaemia and anthropometric indices has been said to be a strong marker that predicts the risk of cardiovascular diseases. **Aim:** To evaluate some cardiovascular risk factors among students of Faculty of Health Science and Technology Ebonyi State University, Nigeria. **Materials and Methods:** The study recruited 80 participants (males 28 (35%) and females 52 (65%)) with mean age 24.36±5.37 years old. The mean age for the male was 24.64±5.41 years, while the mean age for the female was 24.21±5.39 years. **Results:** It was observed that there was a significant difference between sexes for the following variables: TG (P=0.052), HDL-cholesterol (P=0.053), WHR (P=0.003), and AIP (P=0.003) and no significant difference between sexes for the following variables: Age (P=0.734), TC (P=0.339) and LDL-c (P=0.540). WHR showed moderate significant correlation with age while AIP was more strongly correlated with TG and HDL-c. WHR showed moderate significant correlation with age in the female participants but there was no significant correlation between WHR with age and lipid profile in the males. AIP showed strong significant correlation with TG in the males whereas it showed moderate significant correlation with age, and strong significant correlation with TG and HDL-c in females. **Conclusion:** This study support the evidences that lipid ratios could be used for identifying individual at higher risk of cardiovascular disease in the clinical practices especially, when the absolute values of lipid profile seem abnormal or higher.

Keywords: *Cardiovascular Diseases, Atherogenic Index, Lipid profile, Cholesterol.*

1. INTRODUCTION

Cardiovascular diseases (CVD's) are said to be chronic or acute maladies of the heart and blood vessels and it include coronary heart disease, cerebrovascular disease, peripheral artery disease, rheumatic heart disease, congenital heart disease, deep vein thrombosis and pulmonary embolism [1]. Many of these CVD's are degenerative and heavily influenced by lifestyle [2]. Cardiovascular disease is known to be a leading cause of 30 % of mortality worldwide [3] and has recently emerged as a public health problem, and it is now one of the major world's leading causes of death [4]. In 2006, the total number of cardiovascular disease (CVD) deaths (which include coronary heart disease, stroke, and rheumatic heart disease) globally increased to 17.5 million from 14.4 million as was seen in 1990, 7.6 million of these death were attributed to coronary heart disease and 5.7 million to stroke [5]. It has been noted that the prevalence of cardiovascular diseases in general and coronary heart disease in particular is on the increase in Nigeria [6].This is in tandem with the projected escalation in the global burden of cardiovascular diseases. Here in the developing countries it is seen that demographic transition is experienced more than in developed countries [7]. The World Health Organization (WHO) gave an estimated figure that there will be about 20 million CVD deaths in 2015, these will account for 30 percent of all deaths worldwide [8]. By 2030, researchers' had projected that CVD alone will be responsible for more deaths in low income countries than infectious diseases (including HIV/AIDS, tuberculosis, and malaria), maternal and prenatal conditions, and nutritional disorders combined [9]. The most basic or primary way of dealing with the epidemic of CVD is to understand the risk factors and how they interact with each other. It is seen that the factors responsible for

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cardiovascular risk include, demographic shift with altered population, age profiles, life style changes (altered decreased physical activity, dyslipidemia, obesity and tobacco use) due to urbanization and industrialization diet [10]. Among these enumerated risk factors, lipid profile of plasma is the major risk factor and predictor for CVD [11]. A number of plasma lipid parameters have been used to estimate cardiovascular risk and these risk algorithms, such as SCORE, Framingham score and Reynolds Risk score, are based on total cholesterol (TC), or low-density lipoprotein cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C), and most intervention trials have targeted the LDL-C levels [12]. Many clinical studies have made effort by introducing a better marker which is atherogenic dyslipidemia which can predict the risk of CVD and is useful for evaluating response to treatment [13]. Atherogenic index of plasma (AIP), therefore, has been said to be a strong marker that predicts the risk of atherosclerosis and coronary heart disease. AIP also reflect the true relationship between protective and atherogenic lipoprotein [13]. In the area of diagnosis, different diagnostic criteria have been used by different working groups to diagnose CVD. They have also used atherogenic index as an established marker of cardiovascular risk [13]. Atherogenic index (AI) as an emerging index is said to fulfil the criteria which can be used as a stand-alone index for cardiac risk stratification [13]. Different study group have criteria for studying cardiovascular risk but the most frequently used criteria for determining cardiovascular risk are the one suggested by National Cholesterol Education Program Adult Treatment Panel III (NCEPATP III) criteria. The NCEP ATP III criteria were based on the association of factors which causes subsequent development of coronary heart diseases in Caucasian population [14]. Looking at Nigerian statistics as it concerns CVD, it has been said that the number of persons suffering from raised blood pressure was 34.8% in 2008 and obesity 6.5% in 2014, over 7% is said to have suffered from cardiovascular disease [15]. Reddy reported that the rate of death due to CVD among 15 to 59 years of age is 3 to 8 times as high in Nigeria and Tanzania as in England and Wales. There is paucity of information on serum lipids in the northern part of Nigeria. Given the heterogeneity of the Nigerian population with different socio-cultural background including dietary habits and the impact it has with lipid metabolism [16, 17, 18]. WHO report (2002) also revealed that 80% of deaths occurred from cardiovascular diseases, and 8th of related disability from CVD occurred in developing countries and it has also been estimated that 9 million mortality were recorded from CVDs and it is expected to increase to 19 million by 2020. The prevalence of cardiovascular disease has increased to an alarming rate in Nigeria in the past 2 decades and the trend is expected to increase continuously especially if no control measures are put in place [19]. The epidemic of CVD has insidiously established themselves without attracting global attention and cardiovascular disease historically has been under-studied [20]. Many years back, heart disease had been taught to be primarily a disease of older people and adults that live in affluence and physical inactivity but abdominal obesity has been implicated in several studies of non-obese adults as best surrogate of risk factors accumulation of cardiovascular disease and death. Also substantial work have not been done in asymptomatic adults to improve their cardiovascular health, since there is increased incidence of obesity, thus increasing the rate of coronary heart disease (CHD) and death among asymptomatic young adults 18 to 50 years of age [20]. Consequently, young adults who are students in Nigeria Universities have been under represented in longitudinal studies of disease history and in clinical trials therefore describing the prevalence of dyslipidemia and those who are at risk of developing cardiovascular disease becomes imperative to reduce the burden of CVD in the future decades. Abdominal obesity and other indices of obesity such as waist-hip ratio and atherogenic index have been implicated as best surrogate of risk factors for cardiovascular disease [21, 22]. The re-emerging obesity amongst student population in the university may be an indication of a changing lifestyle that may predispose to impaired cardiovascular functions. Therefore, the need to assess asymptomatic students for lipid profiles and atherogenic index for purposes of identifying those students that may be at risk of developing obese associated ailments.

2. MATERIALS AND METHODS

2.1 Study Population

The study group was assessed for cardiovascular risk factors using NCEP: ATP III, NIDDK and WHO criteria. A total of 80 students within the age range of 18 years and above were used for the study. Present study was a cross-sectional study conducted at College of Health Science (CHS), Presco Campus Abakaliki, Nigeria between the months of July 2016 and August 2016. The subjects included in the present study were 80 students aged between 18years and above. All the subjects' anthropometric measurements were taken and when they came in the morning after 12 hours of fasting. Fasting venous samples of 7ml (blood) were collected between 7.30am to 11.30am employing standard procedures for the analysis of Lipid profile; (total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low density lipoprotein cholesterol (LDL-C). Laboratory analysis was carried out at Federal Teaching Hospital Abakaliki (FETHA 2), Ebonyi State, Nigeria.

2.2 Physical/Anthropometric measurements

Anthropometric measurements were taken prior to blood collection and analysis. Height was measured without shoes in meters and weight was measured nearest to 0.1 kg with the help of standard weighing machine with minimal clothing. The waist circumference was measured with a measuring tape in standing position over the abdomen, with measurements made halfway between the lower border of the ribs and the highest point of iliac crest (at the umbilicus

level) in the standing position, Hip circumference was taken around the maximum circumference of the buttocks [23] and blood pressure was taken with a sphygmomanometer device (Microlife BP A50, Microlife AG, Switzerland) in sitting position Three BP measurements were taken with at least three minute intervals between consecutive measurements. The mean systolic and diastolic BP from the second and third measurements was analyzed [23]. The mid upper arm circumference was taken. The body mass index (BMI) was calculated by dividing the weight in kilograms by the square of the height in meters (kg/m2) using the formula:

$$\frac{\text{Weight}}{\text{Height2}} \text{ (kg/m}^2\text{)} \qquad (1)$$

This was used to determine the obesity when BMI of participants exceeded 30 kg/m^2.

2.3 Sampling Technique

The sampling technique employed in the study was multistage, stratified sampling method. Students were selected from the various classes/levels or units in the Department of Medical Laboratory Science and Nursing Sciences that made up the faculty. The study was done during the school. A total of 28 male and 52 female students were selected making a total of 80 participants.

2.4 Informed consent

Informed consent was obtained from the subjects that participated in the study.

2.5 Assay Procedure

Assay Procedure for Total Cholesterol (TC) [24]

Using a micropipette, 10 µl of distilled water, 10 µl of the standard and 10 µl of the test sample was added to the test tubes designated (properly labeled test tubes) for the reagent blank, standard and test sample respectively. 1000 µl of reagent was added to each of the three (3) test tubes mentioned above. After which these test tubes were mixed respectively and incubated for 10 minutes at 25 ºC. After incubation, small volumes of the reagent blank and test sample were transferred into the cuvettes respectively. The concentration of the test sample was read against the reagent blank at 500 nm wavelength.

Assay Procedure for Triglycerides (TGs) [24]

Using a micropipette, 10 µl of the standard and 10 µl of the test sample were added to the test tubes designated for the standard and test sample respectively. 1000 µl of the reagent were added each to the two (2) test tubes mentioned above and also to the test tube designated for the reagent blank. After which these test tubes were mixed respectively and incubated for 10 minutes at 25 ºC. After incubation, small volumes of the reagent blank and test sample were transferred into the cuvettes respectively. The concentration of the test sample was read against the reagent blank at 500 nm wavelength.

Assay Procedure for HDL-Cholesterol (HDL-C) [24]

The procedure for analyzing **HDL-Cholesterol was** made up of two stages- the first stage involved precipitation while the second stage did not.

Stage I: Using a micropipette, 200 µl of the standard and 200 µl of the test sample were added into the centrifuge tubes designated for the standard and test sample respectively. 500 µl of diluted precipitant (RI) were added to the two (2) centrifuge tube mentioned above**.** After which these centrifuge tubes were mixed respectively and allowed for 10 minutes at room temperature. The test sample and standard were centrifuged for 10 minutes at 4.00 rpm. After centrifuging the clear supernatant was separated using a micropipette.

Stage II: Using a micropipette, 100 µl of distilled water, 100 µl of the standard and 100 µl of the supernatant were added to the test tubes designated (properly labeled test tubes) for the reagent blank, standard and test sample respectively. 1000 µl of reagent was added to each of the three (3) test tubes mentioned above. After which these test tubes were mixed respectively and incubated for 10 minutes at 25 ºC. After incubation, small volumes of the reagent blank and test sample were transferred into the cuvettes respectively. The concentration of the test sample was read against the reagent blank at 500 nm wavelength.

Calculation of Low Density Lipoprotein Cholesterol (LDL-C) [25]

LDL-C was determined using Friedwald's formula.

$$\text{From the formula; LDL-C= TC-HDL-C-}(\tfrac{\text{TG}}{2.2}) \qquad (2)$$

$$\text{Reference Ranges for LDL-C = <3.4 mmol/L)} \qquad (3)$$

Calculation of atherogenic index of plasma (AIP)

$$\text{From the formula, AIP} = \log\left(\frac{\text{TG}}{\text{HDL-C}}\right) \quad (4)$$

Reference Ranges for AIP = > 0.21

2.6 Statistical methods

SPSS (version 20.0) was used for all statistical analyses. Student's *t*-tests were used to evaluate differences in mean for study groups. Continuous variables were expressed as mean ± standard deviation. Correlations between lipid profile and anthropometric indices were examined using Pearson's correlation coefficients. Using previously described methods, 95% confidence intervals for prevalence estimates were determined [26]. The statistical significance was set at the *p* value of $*p<0.05$; $**p<0.01$.

3. RESULTS

3.1 Anthropometric and biochemical findings

The study recruited 80 participants (28 males [35%] and 52 [65%] females) with mean age 24.36±5.37 years old. The mean age for the male was 24.64±5.41 years, while the mean age for the female was 24.21±5.39 years. The participants mean ±SD TC concentration (mmol/l) was 4.36±1.07 (male was 4.20±1.19, while female was 4.44±1.00). The mean ±SD TG concentration (mmol/l) was 0.78±0.54 (male was 0.98 ±0.81 while female was 0.66±0.25). The mean ±SD for the HDL-c concentration (mmol/l) was 1.64±0.56 (male was 1.46 ±0.48, while female was 1.72 ±0.59). The mean ±SD LDL-c concentration (mmol/l) was 2.37±1.05 (male was 2.27±1.18 while female was 2.42±0.98). The mean ±SD for the WHR was 0.82±0.06 (male was 0.84 ±0.04, while female was 0.81 ±0.06). The mean ±SD for the AIP was -0.35±0.26 (male was -0.42±0.25, while female was -0.24±0.22). In Table 4.1, it was observed that there was a significant difference between sexes for the following variables: TG (*P*=0.052), HDL-cholesterol (*P*=0.053), WHR (*P*=0.003), and AIP (*P*=0.003) and no significant difference between sexes for the following variables: Age (*P*=0.734), TC (*P*=0.339) and LDL-c (*P*=0.540).

Table 4.1: General characteristics and anthropometric indices between Male and Female students.

Variables	**Gender**			***P*- value** [a]
	Total Mean ±SD	Male (n=28) Mean ±SD	Female (n=52) Mean ±SD	
Age	24.36±5.37	24.64±5.41	24.21±5.39	0.734
Total cholesterol (mmol/l)	4.36±1.07	4.20±1.19	4.44±1.00	0.339
Triglycerides (mmol/l)	0.78±0.54	0.98 ±0.81	0.66±0.25	0.072
High-density lipoprotein (mmol/l)	1.64±0.56	1.46 ±0.48	1.72 ±0.59	0.053
Low-density lipoprotein (mmol/l)	2.37±1.05	2.27±1.18	2.42±0.98	0.540
Waist-Hip Ratio (WHR)	0.82±0.06	0.84 ±0.04	0.81 ±0.06	0.003
Atherogenic index in plasma (AIP)	-0.35±0.26	-0.42±0.25	-0.24±0.22	0.003

a: Differences determined by using 2-tailed t tests following Levene's test for equality of variance.

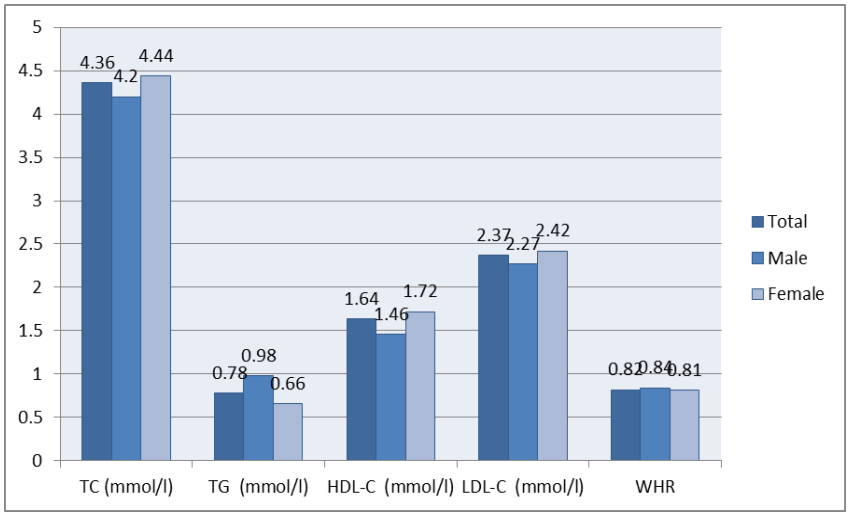

Figure 4.1: Mean (SD) of Lipid profile and WHR between sexes (* *P*<0.05). TC=Total cholesterol; HDL-C=High-density lipoprotein cholesterol; LDL-C=Low-density lipoprotein cholesterol, WHR; Waist-Hip Ratio.

The mean of lipid profile, WHR and AIP is presented in Figure 4.1. Overall, the mean TG was higher and the mean HDL-C was lower in male participants than the female participants. However, only the mean of HDL-C was statistically significant (1.46 ±0.48 mmol/l in the male participants *vs.* 1.72 ±0.59 mmol/l in the female participants, Figure 1; *P*=0.053). The mean WHR was higher in the male participants than the female participants.

In Table 4.2, on the overall, WHR showed moderate significant positive correlation with age (*r*=0.371, *P*=0.001) while there was a strong significant positive correlation between AIP and TG (*r*=0.665, *P*=0.00), strong significant negative correlation between AIP and HDL-c (*r*= -0.604, *P*=0.00).

Table 4.2: Pearson correlation coefficients for the Overall associations between lipid profile, WHR and atherogenic index of plasma.

Variables		**Total No. of Students (*n*=80)**				
		AGE	TC	TG	HDL-C	LDL-C
WHR	*r*-value	0.371**	0.051	-0.163	-0.114	0.150
	P-value	0.001	0.655	0.150	0.313	0.184
AIP	*r*-value	0.058	-0.132	0.665**	-0.604**	0.031
	P-value	0.608	0.245	0.000	0.000	0.784

Abbreviations: WHR, waist hip ratio: AIP, atherogenic index of plasma.
**. Correlation is significant at the 0.01 level (2-tailed).
*. Correlation is significant at the 0.05 level (2-tailed).

In Table 4.3, WHR showed moderate significant correlation with age (*r*=0.46, *P*=0.00) and TG (*r*=0.34, *P*=0.02) in females but WHR did not significantly correlate with age and lipid profile in the males. AIP showed strong significant correlation with TG (*r*=0.74, *P*=0.00) in the males whereas it showed moderate significant correlation with age (*r*=0.31, *P*=0.02), and there was a strong significant correlation with TG (*r*=0.77, *P*=0.00) and HDL-C (*r*=-0.70, *P*=0.00) in females.

Table 4.3: Pearson correlation coefficients for the associations between lipid profile, WHR and atherogenic index

Variables		**Male students (n=28)**					**Female students (n=52)**				
		AGE	TC	TG	HDL-C	LDL-C	AGE	TC	TG	HDL-C	LDL-C
WHR	*r*-value	0.22	0.32	0.17	0.03	0.27	0.46**	0.14	0.34*	-0.15	0.18
	P-value	0.25	0.10	0.40	0.90	0.17	0.00	0.32	0.02	0.29	0.20
AIP	*r*-value	0.04	-0.15	0.74**	-0.25	-0.29	0.31*	-0.11	0.77**	-0.70**	0.19
	P-value	0.84	0.45	0.00	0.20	0.13	0.02	0.44	0.00	0.00	0.17

of plasma by gender.

4. DISCUSSION

Epidemiological studies have showed a clear correlation between obesity and cardiovascular risk factors [27]. The results of this study demonstrate that waist-related indicator of abdominal obesity such as WHR was a significant risk factor for CVD. This index (i.e., WHR) was strongly associated with cardiovascular risk in both genders. Previous reports suggested that, anthropometric index (WHR) of abdominal obesity appear to be more strongly associated with risk factors incident CVS disorders and death than the BMI [28, 29]. Lawrence *et al.,* (2007) suggested that 1cm increase in WC is associated with a 2% increase in risk of future CVD and 0.01 increase in WHR is associated with a 5% increase in risk [30]. World health organization (WHO) also states that abdominal obesity is defined as a waist–hip ratio above 0.90 for males and above 0.85 for females, or BMI above 30.0.The National Institute of Diabetes, Digestive and Kidney Diseases (NIDDK) states that women with waist–hip ratios of more than 0.8, and men with more than 1.0, are at increased health risk because of their fat distribution. Another research revealed that, female representations are most often in the 0.6-0.7 range for WHR, suggesting a preference towards lower WHR [31]. According to the National cholesterol education programme (NCEP) waist circumstance cut of values is >88 cm. In the study, WHR was significantly different between the two groups. The increase in abdominal fat was determined to be distributed as increase in trunk fat, subcutaneous fat and visceral fat [32] Several studies indicate that even with a 'normal' BMI, those with an elevated WC can have a two-to-threefold increase in cardiovascular disease risk and premature death [32, 33]. It was observed that males presented higher values of TG but slightly lower values of HDL-c and LDL-c than females. Males, however, presented higher mean values of WHR than females, which suggest an excess of intra-abdominal adipose tissue. Such data may have contributed

to the alterations observed in lipid profile. Cercato *et al.,* (2004) obtained similar results after studying a sample of 1,213 Brazilian adults of both sexes, as men presented higher mean values of WC and WHR and lower mean values of HDL-c [34]. As plasma lipids can be divided into the proatherogenic lipoproteins and antiatherogenic HDL-c. Assessment of the relative proportions of cholesterol in these two fractions can be valuable than the individual lipid measurements. In both subjects a significant difference was observed in their plasma TG and HDL-c levels, but higher levels TG and HDL-c was observed in the female participants. AIP is a powerful indicator of the risk assessment of coronary artery diseases. The higher the values, the higher the risk of developing cardiovascular diseases and *vice versa* [35]. The atherogenic link between high TG and HDL-c is due to the higher plasma concentration of triglyceride rich, very low-density lipoprotein (VLDL) that generates small, dense LDL-c during lipid exchange and lipolysis. These LDL-c particles accumulate in the circulation and form small, dense HDL-c particles, which undergo accelerated catabolism, thus closing the atherogenic circle [36]. Protasio *et al.,* (2008) explained that ratio of triglycerides to HDL-c was found to be a powerful independent indicator of extensive coronary disease [37]. The ratio TG/HDL-c, initially proposed by Gaziano et *al.*, (1997) is an atherogenic index that has proven to be a highly significant independent predictor of myocardial infarction, even stronger than TC/HDL-c and LDL-c/HDL-c [2]. Angela Bacelar *et al.,* (2009) reported that TG/HDL-c ratio is possible to approximately determine the presence and extent of coronary artery disease (CAD) by non-invasive methods [38]. Atherogenic Index of Plasma (AIP) show an inverse relationship that exist between TG and HDL-c and that the ratio of TG to HDL-c is a strong predictor of infarction and it was used by some practitioners as significant predictor of atherosclerosis [2]. Some of the researchers suggested that, AIP is a highly sensitive marker of difference of lipoprotein in patients. AIP values of -0.3 to 0.1 are associated with low, 0.1 to 0.24 with medium and above 0.24 with high cardiovascular risk [39]. A significant (P=0.003) AIP values were observed and its mean value was lower in males when compared (-0.42±0.25) and females (-0.24±0.22). WHR showed moderate significant correlation with age while AIP was more strongly correlated with TG and HDL-c. WHR showed moderate significant correlation with age in the female participants but there was no significant correlation between WHR with age and lipid profile in the males. AIP showed strong significant correlation with TG in the males whereas it showed moderate significant correlation with age, and strong significant correlation with TG and HDL-c in females.

5. CONCLUSION

The general lipid profile status of WHR and AIP of the study population were normal but the comparison between males and females showed significant higher values of TG, HDL-c with WHR and AIP. In case of lipid profile, elevated levels of TC, HDL-c, and LDL-c as was observed in females while a higher TG was observed in males. There was no correlation between WHR with lipid profiles whereas correlation with AIP was observed in females. Significant higher AIP were observed in both groups, however; AIP was negatively correlated with HDL-c in females. AIP was associated more with age in females than males. Elevated levels of lipid profiles alongside with raised AIP in females could indicate cardiovascular risk and can be seen as strong sovereign predictors of cardiovascular and global risk, suggesting that both may be suitable for inclusion in national screening strategies. The above observations suggest that, lipid ratios could be used for identifying individual at higher risk of cardiovascular disease in the clinical practices especially when the absolute values of lipid profile seem abnormal or higher.

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