ASSESSMENT OF LIPID PROFILE LEVELS AMONG OLDER ADULTS IN NNEWI

ABSTRACT

Background/Objective of Study: Cardiovascular diseases continue to be the main cause of morbidity and mortality and Aging is an important modifier of cardiovascular health. **Materials and Methods:** This cross-sectional study evaluated the lipid profile levels of older adults in Nnewi, Nigeria. In total, 128 subjects were enlisted for the study using simple random sampling. They were divided into two equal groups: older people (n=64) and control groups (n=64). 32 participants, both male and female, between the ages of 45 and 75 who have given written informed consent make up the test group. The control group consists of 64 students, 32 males and 32 females, between the ages of 18 and 30. Each participant gave a fasting venous blood sample of five milliliters, which was then placed in a plain container for analysis of the lipid profile levels. Total cholesterol (TC), triglyceride (TG), and high density lipoprotein cholesterol (HDL-C) were determined using enzymatic colorimetric methods while low density lipoprotein cholesterol (LDL-C) was calculated: LDL-C = (Total Cholesterol) − (HDL-C) − (TGs/5). **Results:** Results showed that the older persons had mean serum levels of TC, TG, and LDL-C that were significantly higher than those of the control group, but HDL-C levels were lower. Additionally, older male and female adults had significantly higher mean blood levels of TC, TG, and LDL-C compared to the male and female control groups, while HDL-C was significantly lower. Moreover, total cholesterol and LDL-C levels were linked with age in both the control group and the test group, while LDL-C was significantly connected with total cholesterol levels. **Conclusion:** Ageing may, thus, have a deleterious impact on lipid profile.

Key words: Older adults, Aging, Cardiovascular Health, Lipid profile.

INTRODUCTION

Worldwide, cardiovascular diseases continue to be the main cause of morbidity and mortality [1]. Atherosclerosis is the underlying disease mechanism, which is a narrowing of the arteries brought on by a buildup of plaque that results in inadequate blood flow to important organs, primarily the heart and brain [2]. Cardiovascular diseases have historically been linked to numerous lipid classes [3]. Cardiovascular disease is more common in the aging and older population and Adults' age is a risk factor for cardiovascular disease [4, 5]. Aging is defined as a progressive physiological change in an organism that leads to a reduction in biological capacities and the organism's ability to respond to metabolic stress [6]. According to World Health Organization (WHO), between 2015 and 2050, the proportion of the world's population over 60 years will nearly double from 12% to 22% and by 2050, 80% of older people will be living in low- and middle-income countries [7].

Lipids are a diverse category of molecules that are vital for the body's structural, metabolic, and functional activities [8]. Lipids serve important functions in cellular membranes, cell signaling, and cell metabolism [9]. There is an increasing evidence associating alterations in lipid profile levels with advancing age [10]. Hyperlipidaemia has been identified as a significant risk factor for atherosclerotic cardiovascular disease [11], which is very prevalent in middle-aged men and women [12]. Globally, several studies have shown varying levels of abnormalities in lipid levels (dyslipidemia) in diverse population [13-16], principally as a result of sedentary life pattern [17, 18]. It is therefore important to investigate the lipid profile levels in the apparently healthy older adults in Nnewi, especially given the paucity of studies in this area in the current study. Despite numerous studies documenting varying levels of prevalent dyslipidemia in older adults, there

seems to be conflicting results due to differences in the study participants' dietary patterns, location, and activities that characterize the study population that may influence the findings.

MATERIALS AND METHODS

Study Design and Population

This study adopted cross-sectional study design. One hundred and twenty eight (128) volunteers in all were used in this study. They were split into two equal groups: test (older adults) and control groups. The test group consists of 32 male and female volunteers between the ages of 45 and 75 who have provided written informed consent. Sixty-four (64) students, 32 males and 32 females, between the ages of 18 and 30, make up the control group.

Study Area

The study was carried out within Nnewi Metropolis, Anambra State, Nigeria.

Sample size and Sample Size Calculation

The G-Power software, version 3.1.9.2.25, was used to calculate the sample size and power of this study. The predicted sample size of 128 participants has an error probability of 0.05 and an 80% power to detect variations in replies as small as 0.5 (effect size). Simple random sampling was used to select 128 consecutive consenting adult participants and control subjects (64 males and 64 females). The control group was comprised of individuals between the ages of 18 and 30, whereas the test group ranged in age from 45 to 75.

Inclusion Criteria

Apparently healthy older adults (aged 45 to 75) and control volunteers (aged 18 to 30) were recruited in this investigation.

Exclusion Criteria

The current study excluded people with known disorders such as Diabetes mellitus, kidney disease, cardiovascular diseases (heart disease), pregnant women, nursing mothers, alcoholics, and smokers.

Approval on ethical grounds

Ethical clearance for the study was obtained from Nnamdi Azikiwe University Teaching Hospital Ethics Committee (NAUTH/CS/66/VOL. 16/VER. 3/07/2023/07).

Participants' informed consent

Prior to the start of the study, the participants' written informed consent was sought and obtained.

Collection of samples and analysis

After 10–12 hours of fasting, each participant was venipunctured through the antecubital vein using a five milliliter plastic syringe with the least amount of stasis possible into plain containers for the measurement of lipid profile levels. The blood sample was spun at 3000 rpm for 10 minutes after being given time to clot and retract. The serum was separated and used for study of the lipid profile levels. Serum samples that were not immediately analyzed were kept frozen at-20ºC.

Laboratory Methods

Estimation of Total Cholesterol (Cholesterol Oxidase/Peroxidase Reaction)

Total cholesterol (TC) was determined by enzymatic colourimetric method according to Roeschlaw *et al.* [19].

Determination of Serum Triglycerides

Triglycerides (TG) level was assayed using enzymatic Glycerol Phosphate Oxidase/ Peroxidase method described by Schettler and Nussel [20].

Estimation of high density lipoprotein cholesterol (HDL-C)

HDL Cholesterol (HDL-C) level was estimated by Phosphotungstate/$MgCl_2$ Cholesterol Oxidase/Peroxidase method described by Assmann *et al*. [21].

Determination of Low density lipoprotein cholesterol (LDL-C)

LDL-C was estimated by computation, according to the method described by Friedewald *et al*. [22].

Calculation

LDL-C = (Total Cholesterol) − (HDL-C) − (TGs/5).

STATISTICAL ANALYSIS

Statistical program for social sciences (SPSS) (Version 26) software was used to compare the mean values of the control and test groups using the Students t-test and Pearson's correlation coefficient. The results were reported as mean±SD and a p-value of < 0.05 was taken as the threshold for statistical significance.

RESULTS

The mean serum total cholesterol (TC), triglyceride (TG), and low density lipoprotein cholesterol (LDL-C) levels were statistically higher in the older adults compared to the control group, while the mean serum high density lipoprotein cholesterol (HDL-C) levels were lower (P-value = 0.000) for each. Table 1.

When older adults male and female groups were compared to control male and female groups, there were statistically significantly higher mean serum total cholesterol (TC), triglyceride (TG), and low density lipoprotein cholesterol (LDL-C) levels with lower mean serum high density lipoprotein cholesterol (HDL-C) levels (P-value= 0.000) in each case. Table 2 and 3.

In the control group, there were significant positive correlations between TC and age, LDL-C and age, and LDL-C and TC (*P*-value <0.05). Additionally, in older persons, there was a significant positive correlation between age and TG, TC and TG, and TC and LDL-C (*P*-value <0.05). Table 4 and 5 respectively.

Table 1: Serum Levels of Parameters Studied in the older adults and Control Subjects (Mean±SD).

Parameters	Older adults (n=64)	Control (n=64)	t-value	*P*-value
TC (mmol/L)	4.65±0.66	3.90±0.60	6.646	0.000
TG (mmol/L)	1.52±0.44	1.08±0.26	6.952	0.000
HDL-C (mmol/L)	1.16±0.08	1.34±0.09	10.918	0.000
LDL-C (mmol/L)	2.79±0.62	2.06±0.59	6.674	0.000

**P*-value is statistically significant at <0.05. TC= total cholesterol; TG= triglyceride; HDL-C= high-density lipoprotein cholesterol, LDL-C= low-density lipoprotein cholesterol.

Table 2: Serum levels of the parameters studied in older adult male participants and male control subjects (Mean±SD).

Parameters	older adult male participants (n=31)	Male Control (n=32)	*P*-value
TC (mmol/L)	4.66±0.64	3.87±0.58	0.000
TG (mmol/L)	1.55±0.43	1.09±0.24	0.000
HDL-C (mmol/L)	1.15±0.08	1.35±0.09	0.000
LDL-C (mmol/L)	2.79±0.61	2.02±0.53	0.000

*_P_-value is statistically significant at <0.05. TC= total cholesterol; TG= triglyceride; HDL-C= high-density lipoprotein cholesterol, LDL-C= low-density lipoprotein cholesterol.

Table 3: Serum levels of the parameters studied in older adult female participants and female control subjects (Mean±SD).

Parameters	**older adult female participants (n=33)**	**female Control (n=32)**	***P*-value**
TC (mmol/L)	4.63±0.68	3.92±0.63	0.000
TG (mmol/L)	1.50±0.45	1.06±0.28	0.000
HDL-C (mmol/L)	1.16±0.09	1.33±0.09	0.000
LDL-C (mmol/L)	2.78±0.65	2.10±0.66	0.000

*_P_-value is statistically significant at <0.05. TC= total cholesterol; TG= triglyceride; HDL-C= high-density lipoprotein cholesterol, LDL-C= low-density lipoprotein cholesterol.

Table 4: Levels of Associations between parameters studied in control group (n=64)

Parameters	**Pearson r coefficient**	***P*-value**
TC Vs Age	0.312	0.012*
LDL-C Vs Age	0.302	0.015*
LDL-C Vs TC	0.968	0.015*

*_P_-value is statistically significant at <0.05. TC= total cholesterol; TG= triglyceride; HDL-C= high-density lipoprotein cholesterol, LDL-C= low-density lipoprotein cholesterol.

Table 5: Levels of Associations between parameters studied in test group (n=64)

Parameters	Pearson r coefficient	*P*-value
Age Vs TG	0.260	0.038*
TC Vs TG	0.375	0.002*
TC Vs LDL-C	0.940	0.000*

**P*-value is statistically significant at <0.05. TC= total cholesterol; TG= triglyceride; HDL-C= high-density lipoprotein cholesterol, LDL-C= low-density lipoprotein cholesterol.

DISCUSSION

The mean serum level of total cholesterol in the current investigation was significantly higher in the aged participants in this study when compared to the values observed in the control participants. The aged participants' higher mean serum cholesterol may be caused by a variety of factors. First, it could be due to aging, which is associated with a decline in physiological and biochemical function and frequently results in a change in homeostasis and dysregulation of important metabolic processes [23-25]. The primary age-related chronic diseases and decline in physiologic functions, which include atherosclerosis, hypertension, diabetes, hyperlipidemia, obesity, sarcopenia, osteoporosis, thrombogenesis, chronic inflammation, and decline in immune functions, are greatly influenced by numerous hormonal and metabolic changes that occur with aging [23]. Second, the majority of low-density lipoprotein cholesterol is removed from circulation by low-density lipoprotein receptor expressed in the liver, and this decreases with advancing age. Low-density lipoprotein receptor is necessary for the cellular uptake of low-density lipoprotein. Because hepatic LDL receptors are fewer due to intracellular cholesterol buildup, less LDL enters cells, resulting in an increase in plasma LDL concentration [26].

Additionally, intracellular buildup can still happen if dietary cholesterol intake is too high, especially if the diet is high in saturated fat. This is due to the fact that a high saturated fat diet can inhibit LDL receptor function, which is a key activator of cholesterol metabolism [27, 28]. Thus, there is hepatic LDL receptor expression, which ultimately results in a reduction in LDL clearance. Also, age-related increases in serum cholesterol are also influenced by a decline in the conversion of cholesterol to bile acid. A significant cardiovascular risk factor that raises the prevalence of atherosclerotic diseases in adults is hypercholesterolemia [29]. It may be challenging to draw direct comparisons because the majority of earlier investigations in the seemingly healthy aged persons were prevalence studies that also used diverse settings. Nevertheless, the current finding is consistent with the findings of numerous investigations that showed that among apparently healthy adult Nigerians, dyslipidemia occurred at various rates [30-32]. In contrast, Ahaneku *et al*. observed no significant change in the mean blood total cholesterol in their study on the assessment of lipid and other cardiovascular risk factors in a rural population in eastern Nigeria [33].

However, aged participants had significantly higher mean serum triglyceride levels than did the control group. This is consistent with research conducted by Ahaneku *et al*. [33] on the assessment of lipid and other cardiovascular risk factors in a rural population in eastern Nigeria. They observed that aged individuals had significantly higher triglyceride levels than younger people. In their study on the anthropometric status and lipid profile of older persons in Dekina Local Government Area of Kogi State, Nigeria, Emmanuel *et al*. [34] found that consumption of high-risk foods (processed foods) is one of the main contributing factors to elevated triglyceride levels in older adults. They also found that older adults who consume low-risk foods are protected from disorders related to nutrition. Nevertheless, This increase in TG level in the aged

individuals in this study may be attributable to dysfunctional lipoprotein lipase (LPL)-mediated lipolysis, impaired residual clearance, and abnormalities in the synthesis of intestinal chylomicrons and hepatic very-low density lipoprotein (VLDL) in aged people and these are the principal causes of hypertriglyceridemia in aged persons [35, 36]. A vital part of the clearance of the triglyceride-rich lipoproteins is the endothelium-bound lipoprotein lipase (LPL), which hydrolyzes triglycerides [37]. For plasma triglycerides (TGs) to be properly cleared, LPL is essential. The TGs of TG-rich lipoproteins are hydrolyzed by LPL bound to the arterial lumen, releasing fatty acids for absorption by tissues. Delayed TG clearance has been documented in aged people previously [38]. The delayed clearance of plasma TGs in elderly adults raises the possibility that aging may affect LPL activity [39]. Furthermore, another contributory factor to higher TG level in aged people is adipose tissue lipolysis, in which TGs held in adipose are released as nonesterified fatty acids into the bloodstream. Adipose tissue lipolysis is altered in older people, similar to plasma TG clearance [39]. It has been shown previously in mice and rats, that lipolysis declines with advancing age [40]. In humans, aging raises plasma TG levels, lowers plasma TG clearance rates, lowers LPL activity, and lowers lipolysis [38]. More so, age is also linked to increased fat deposition at visceral adipose tissue. All of these alterations indicate a higher risk of metabolic diseases. Also, an essential connection between obesity and dyslipidemia in aging is inflammation, which is connected to the malfunction of adipose tissue [41], which worsens morbidity and mortality in aged persons [42].

Notably, the mean serum level of low density lipoprotein cholesterol (LDL-C) was significantly higher in the aged individuals compared to the control. Age-related increases in LDL are typically explained by a decreased ability to remove it; however, it is also possible that this increase is the result of decreased hepatic LDL receptor expression. With aging, there is a

reduction in the clearance of LDL, which causes an increase in LDL plasma concentration due to the fact that aging also reduces the conversion of cholesterol to bile acid.

However, the aged people had significantly lower mean serum levels of high-density lipoprotein cholesterol (HDL-C) than the control group. The decreased functioning of HDL-C particles that result in decreased LDL-C clearance through reversed transport mechanisms is likely the cause of the lower levels of HDL-C found in the aged participants in this study. The increased visceral adiposity that is frequently seen as people age owing to dietary changes and sedentary lifestyles may make this situation even worse. One possible method by which HDL may be anti-antherogenic is the essential function HDL-C play in the reverse cholesterol transfer from peripheral tissues to the liver [43, 44]. The anti-oxidant, anti-inflammatory, anti-thrombotic, and anti-apoptotic qualities of HDL particles may also aid in their capacity to prevent atherosclerosis [45, 46]. A significant risk factor for metabolic syndrome is low levels of high-density lipoprotein cholesterol [47]. The hallmarks of dyslipidemia, which is a well-known symptom of diabetes, cardiovascular disease, and brain disease, including stroke [48], are low HDL-C levels and/or decreased HDL functionality. In order to reduce the occurrence of age-related disorders such diabetes and cardiovascular diseases, it is crucial to maintain HDL-C in optimal quantities and qualities. HDL-C may prevent low-density lipoproteins (LDL-C) from oxidation and glycation. A low HDL-C level is usually linked to a reduction in HDL-C functionality [49], such as a reduction in anti-oxidant and anti-inflammatory effects and cholesterol efflux. The current finding is consistent with the study of Cho *et al.*, which demonstrated that older age is linked to decreased HDL-C levels [50]. In their study on the anthropometric status and lipid profile of older persons in Dekina Local Government Area of Kogi State, Nigeria, Emmanuel *et al.* [34] reported a low HDL level in older people, which is consistent with the current data. Low HDL-C

levels (<1.1 mmol/L) have been linked to an increased risk of ischemic heart disease and all-cause mortality in individuals 60 years of age and older, while high TC levels have been linked to an increased chance of having heart failure [51]. However, Feng *et al*. [10] found an uneven pattern of HDL-C with aging that is inconsistent with the present report in their investigation on Age-related patterns in lipid levels: a large-scale cross-sectional study of the general Chinese population. Contrary to the current finding, Rosada *et al*. [52] observed no statistically significant difference between the mean HDL-C level of elderly people and the control group.

Additionally, when compared to the observed values in the male control participants, the aged male participants' mean serum levels of total cholesterol, triglycerides, and low density lipoprotein cholesterol were significantly higher, while HDL-C was significantly lower. This could be attributed to eating pattern changes, sedentary lifestyle changes, and common trends in increased visceral tissue composition and insulin resistance that come with aging.

Similar to this, when aged female participants were compared to the observed values in the female control individuals, the mean serum levels of total cholesterol, triglycerides, and low density lipoprotein cholesterol were significantly higher, whereas the high density lipoprotein cholesterol (HDL-C) was significantly lower. The oestrogen levels, which are typically greater in menstruation females than in menopausal females and which are a key driver of HDL-C synthesis, may be the cause of the decreased HDL-C level seen in the aged female participants.

In the control group as well as the test group, age was positively linked with total cholesterol and LDL-C levels while LDL-C demonstrated a strong positive correlation with total cholesterol levels. This suggests that levels of both total cholesterol and LDL-C rise along with aging, with LDL-C levels rising with a rise in total cholesterol.

CONCLUSION

In this study, it was found that older persons had significantly higher mean serum total cholesterol, triglyceride, and low density lipoprotein cholesterol, while having lower mean serum high density lipoprotein cholesterol. Similar to the older male and older female participants, there were significantly higher mean serum total cholesterol, triglyceride, and low density lipoprotein cholesterol with lower mean serum high density lipoprotein cholesterol compared to the male and female control individuals, respectively. Additionally, both in the control group and the test group, total cholesterol and LDL-C levels were significantly correlated with age, while LDL-C showed a significant positive association with total cholesterol levels. Thus, lipid profile may be negatively affected by aging.

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