ADDIS ABABA UNIVERSITY
COLLEGE OF HEALTH SCIENCES
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Assessment of Lipid Profiles and Associated Factors among Patients with Alzheimer's and Parkinson's disease at Tikur Anbessa Specialized Hospital, Addis Ababa, Ethiopia.

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This is to certify that the thesis prepared by Abditsion Disani Gudisa, entitled:

"Assessment of Lipid Profiles and Associated Factors among Patients with Alzheimer's and Parkinson's disease at Tikur Anbessa Specialized Hospital, Addis Ababa, Ethiopia**"** and submitted in partial fulfillment of the requirements for Master of Science degree in Clinical Laboratory Sciences specialty in Medical Laboratory Sciences (Clinical Chemistry Track) complies with the regulations of the University and meets the accepted standards for originality and quality.

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Abbreviations

AD Alzheimer Disease

APP Amyloid precursor protein

BF Body fat

BMI Body Mass Index

HDL High Density Lipoprotein

LDL Low Density Lipoprotein

PD Parkinson's Disease

SOPs Standard Operating Procedures

SPSS Statistical Package for the Social Sciences

TC Total Cholesterol

TG Triglyceride

WC Waist circumference

WHO World Health Organization

Abstract

Background: Parkinson's disease and Alzheimer's disease are the most common neurodegenerative diseases with major public health impact. These neurodegenerative diseases have been associated with dysregulation of lipid metabolism. However, the monitoring of lipid profiles for those patients was not common in the study area due to the paucity of published evidence about their lipid profiles.

Objective: To assess lipid profiles and associated factors among patients with Parkinson's and Alzheimer's diseases at Tikur Anbessa Specialized Hospital, Addis Ababa, Ethiopia.

Method: A hospital-based, prospective, cross-sectional study was undertaken starting in March to June, 2024, among 172 conveniently selected study participants, which included 50 Parkinson's disease patients, 61 Alzheimer's disease patients, and 61 apparently healthy individuals. Pretested structured questionnaires were used to collect sociodemographic data and other information about the diseases. A blood sample was obtained and analyzed for lipid profiles using the Cobas c 311 automated analyzer, following the acquisition of ethical consent. Data entry and analysis were conducted by SPSS Version 27. A student independent t-test, Pearson correlation, and multivariate regression were applied to check the association and correlation among different parameters. A P value of less than 0.05 was deemed to indicate statistical significance.

Result: The mean levels of LDL (mg/dl), TG (mg/dl), and TC (mg/dl) of the Parkinson's disease patients were significantly lower (76.7 ± 21.77, 115.02 ± 24.84, and 152.2 ± 30.44) compared with controls (85.2
± 12.59, 124.12 ± 16.01, and 165.36 ± 29.33) at $P < 0.05$, respectively. On the other hand, Alzheimer's disease patients showed significantly higher levels of LDL (mg/dl) and lower HDL (mg/dl) (106.76 ± 26.55 and 37.64 ± 7.45) compared to the controls (85.2 ± 12.59 and 41.84 ± 5.83) at $P < 0.05$, respectively. In Parkinson's disease patients, lipid profile levels were significantly associated with the duration of the disease, age, and underweight status ($P < 0.05$). For those with Alzheimer's disease, significant associations with lipid profile levels were identified, including disease duration, age, physical exercise, smoking habits, and the frequency of high-fat food consumption ($P < 0.05$).

Conclusion: According to the result, the disturbance of lipid profiles in patients with Alzheimer's and Parkinson's diseases were observed. And this has been found to be associated with factors such as the duration of the disease, age of the patient, physical exercise, smoking habits, and poor compliance to diet.

Key word: Lipid profile, Alzheimer's disease, Parkinson's disease.

1. Introduction

1.1. Background

Alzheimer’s disease (AD) and Parkinson's disease (PD) are the two most prevalent neurodegenerative diseases [1]. It is estimated that Alzheimer’s disease accounts for around 60–70% of dementia cases worldwide [2]. This condition primarily affects older individuals, leading to the progressive degeneration and eventual death of neurons, particularly in brain regions that play a crucial role in memory and cognitive function [3]. The clinical presentation of Parkinson's disease encompasses both motor and non- motor disorders, characterized by symptoms such as bradykinesia, resting tremor, rigidity, behavioral changes, sleep disturbances, and autonomic dysfunction [4].

The precise etiology of Alzheimer's and Parkinson's diseases remains incompletely elucidated; however, it is believed by researchers that a combination of genetic predispositions, environmental, and lifestyle factors may play a significant role in their onset [5]. The defining characteristics of Alzheimer's disease include the presence of amyloid plaques composed of aggregated amyloid-beta(Aβ) and twisted threads of tangled tau protein [6]. Aβ is generated through the sequential action of the enzymes β- and γ- secretase on amyloid precursor protein (APP). As Aβ levels rise, it leads to the formation of insoluble amyloid plaques in the extracellular matrix, thereby contributing to the process of neurodegeneration [7].

Lipids are integral to the normal functioning of the nervous system. The brain contains around 25% of the body's total cholesterol, thereby classifying it as a cholesterol-rich organ [8]. A substantial portion of this cholesterol is located in myelin, which comprises over 80% of the cholesterol found in the adult brain. Elevated cholesterol levels enhance the permeability of the blood-brain barrier, facilitating the entry of peripheral cholesterol into the central nervous system. The lipid 27-hydroxycholesterol, which can readily traverse the blood-brain barrier, stimulates the expression of the enzyme beta-site amyloid precursor protein cleaving enzyme 1 (BACE1). This enzyme is critically involved in the formation of Aβ by initiating the cleavage of amyloid precursor protein [9].

The primary site of cholesterol accumulation is in membrane microdomains known as lipid rafts, where the amyloidogenic pathway occurs. Elevated cellular cholesterol levels bind directly to

APP, encouraging its integration into the phospholipid monolayers of lipid rafts and other organelles containing β- and γ- secretases, thus promoting the amyloidogenic pathway [10]. Additionally, cholesterol influences the polymerization of APP and the processing of APP by γ-secretase, resulting in the production of Aβ, which exacerbates the progression of Alzheimer's Disease [11].

The presence of Aβ plaques leads to neuroinflammation through the activation of microglial cells [12]. This activation is driven by pattern recognition receptors (PRRs), such as Toll-like receptors (TLRs) and scavenger receptors, which detect Aβ as a pathological factor. Upon activation, microglia release various pro-inflammatory cytokines, including tumor necrosis factor (TNF-α), interleukin-1 beta (IL-1β), interleukin-6 (IL-6), and chemokine, which contribute to a neuroinflammatory state [13]. These cytokines can worsen neuronal damage by increasing oxidative stress and triggering apoptosis. Aβ oligomers are particularly harmful to synapses, disrupting long-term potentiation (LTP) and impairing neuronal communication, thus playing a significant role in the pathogenesis of Alzheimer's disease [14].

The hallmark of Parkinson's disease is the presence of Lewy bodies (LB), which are proteinaceous inclusions primarily consisting of aggregates of α-synuclein. This small cytosolic protein is significantly expressed in the brain and is predominantly located at synaptic terminals [15]. It has a strong preference for binding to negatively charged lipids, particularly phosphatidylserine and cholesterol-rich regions like lipid rafts. Alterations in cholesterol metabolism can disrupt the structural integrity of lipid rafts and the fluidity of membranes [16].

The low levels of cholesterol, especially low-density lipoprotein cholesterol (LDL-C), may play a role in the onset of Parkinson's disease by negatively affecting the integrity and functionality of neuronal membranes [17]. Cholesterol is crucial for maintaining the structural stability and fluidity of cell membranes, which are essential for effective synaptic transmission. A deficiency in cholesterol can lead to compromised fluidity of neuronal membranes, thereby interfering with the release of neurotransmitters that are vital for motor control [18].

The biosynthetic pathways of cholesterol and coenzyme Q10 (CoQ10) share initial precursors, such as acetyl-CoA and mevalonate. A reduction in cholesterol levels leads to a decreased flux through the mevalonate pathway, which restricts the availability of isoprenoid intermediates

essential for CoQ10 synthesis. Consequently, lower levels of CoQ10 hinder mitochondrial oxidative phosphorylation by disrupting the electron transfer processes between complexes I/II and III of the electron transport chain. This impairment results in a reduction of adenosine triphosphate (ATP) production, particularly affecting the dopaminergic neurons in the substantia nigra that have high energy demands, thus rendering them more susceptible to degeneration in Parkinson's disease [19].

Several lifestyle and metabolic factors have been implicated in the development and progression of neurodegenerative diseases like Alzheimer's and Parkinson’s. Body mass index (BMI) has been linked to cognitive decline, with both obesity and underweight status potentially increasing the risk of dementia and neurodegeneration through mechanisms involving inflammation, insulin resistance, and vascular dysfunction [20].

Physical exercise, on the other hand, is widely recognized as a protective factor, as it enhances neuroplasticity, reduces oxidative stress, and improves lipid metabolism, thereby mitigating the risk of neurodegenerative diseases [21]. Additionally, smoking has been reported to have complex effects, with some studies suggesting a potential neuroprotective role in Parkinson’s disease due to nicotine’s interaction with dopaminergic pathways, while others indicate an increased risk of cognitive decline and vascular damage in Alzheimer’s disease. These factors, alongside lipid metabolism, contribute to the intricate pathophysiology of neurodegenerative disorders, underscoring the need for a multifactorial approach in understanding their etiology [22].

Alzheimer's and Parkinson's diseases represent significant public health challenges, primarily due to the absence of effective diagnostic tools and treatment strategies. These neurodegenerative disorders not only impact the quality of life for millions of individuals but also place a substantial burden on healthcare systems worldwide [24]. Despite extensive research efforts aimed at understanding these conditions, the relationship between serum lipid levels and the risk of developing Alzheimer’s and Parkinson’s diseases remains unclear and inconsistent across various studies [25]. Lipid profiles have been hypothesized to play a role in the pathogenesis of neurodegenerative diseases. Different studies suggest that certain lipid imbalances may contribute to the development or progression of Alzheimer’s and Parkinson’s diseases by influencing inflammation, oxidative stress, and neuronal health. However, findings

have varied significantly, with some research indicating a protective effect of specific lipids while other studies report no correlation or even a detrimental impact [26].

Given this backdrop of conflicting evidence, the current investigation aims to provide a comprehensive analysis of lipid profiles and the risk of Alzheimer's and Parkinson's diseases. By conducting a comprehensive analysis of serum lipid levels in individuals diagnosed with these neurodegenerative disorders compared to healthy controls, this study seeks to clarify the potential links and underlying mechanisms involved [27]. The outcomes of this research could provide valuable insights into the role of lipids in neurodegeneration, potentially guiding future diagnostic and therapeutic strategies. Ultimately, a better understanding of how lipid levels influence these diseases may pave the way for novel interventions that could improve patient outcomes and reduce the overall burden of these debilitating conditions.

1.2. Statement of the problem

Alzheimer's and Parkinson's diseases are the foremost neurodegenerative disorders, with millions of individuals affected globally. The impact of these diseases is increasingly viewed as a significant public health concern [28]. At present, there are roughly 50 million people living with Alzheimer's disease worldwide. While a cure for Alzheimer's disease has not yet been discovered, there are treatments that can assist in managing its symptoms. Moreover, Alzheimer's disease is associated with a high rate of mortality, ranking as the seventh most common cause of death among the elderly [29, 30].

It is estimated that Parkinson's disease affects roughly 6.1 million people worldwide, making it the second most common neurodegenerative disease [31]. Individuals with Parkinson's disease and their caregivers confront substantial challenges and suffering. Vulnerable populations tend to experience a greater burden of the disease and stigma, which is often exacerbated by the disparities in access to neurological care that exist globally [32].

In sub-Saharan Africa, dementia prevalence rates vary between 2.3% and 20%, with an incidence rate of 13.3 per 1,000 person-years. Specifically, in East Africa, studies have reported dementia prevalence rates ranging from 6% to 23% among individuals aged 50 to 70 years [33].

In Ethiopia, data on Alzheimer's disease prevalence are limited. However, the country has reported high rates of cognitive impairment among the elderly, with studies indicating prevalence rates of 42.1% and 43.8% among individuals aged 50 to 70 years [28]. These findings suggest a significant burden of cognitive disorders, which may correlate with undiagnosed dementia cases. For Parkinson's disease, Ethiopia reports one of the lowest prevalence rates globally, at approximately 7 per 100,000 individuals. This low figure may be attributed to underreporting, limited access to healthcare, and diagnostic challenges [34].

The underlying causes of Alzheimer's disease and Parkinson's disease remain partially understood. However, recent findings suggest that lipid metabolism could be crucial in the development and advancement of both diseases [35]. A study conducted in Iran among Parkinson's disease patients highlighted the existence of abnormal lipid profiles [36].

Furthermore, individuals in the advanced stages of Parkinson's disease are characterized by abnormal lipid profiles. This dysregulation may reflect the progression of the disease. Additionally, dyslipidemia could further accelerate the progression of Parkinson's disease, particularly through the localization of alpha- synuclein within the brain tissues of affected individuals. Alpha-synuclein is integral to the pathology of Lewy bodies [37].

Dyslipidemia may result in the development of plaque-like pathology, where elevated serum levels of non-high-density lipoproteins are significantly associated with neurotic plaques. There exists a lack of agreement on the association between lipid profile levels and degenerative diseases, highlighting the importance of exploring these factors in Alzheimer's disease and Parkinson's disease. This exploration is likely to yield new diagnostic methodologies. As a result, the simple evaluation of serum lipid parameters could serve as a significant predictive marker for neurodegenerative conditions [38].

1.3. Significance of the study

The rising incidence of Alzheimer's disease and Parkinson's disease, along with their complications, constitutes a considerable threat to global health [39, 40]. Despite being chronic illnesses, AD and PD can be effectively managed to avert complications. Abnormalities in lipid profiles have been associated with a heightened risk of chronic complications in both conditions. Numerous studies have shown that maintaining lipid profiles within normal ranges can be beneficial in delaying or preventing the emergence of late-stage complications of AD and PD [41].

Currently, the diagnostic tools for Parkinson's and Alzheimer's diseases include magnetic resonance imaging (MRI), CT scans, and histological analysis, which pose challenges in our country. Therefore, the findings of this study will offer valuable insights to support physicians and healthcare practitioners in establishing routine diagnostic cues. This study may also play a significant role in promoting awareness and facilitating public health initiatives, allowing participants to become informed about their lipid profiles and to undertake necessary corrective actions early. Finally, it will be used as a foundational dataset for subsequent large-scale studies that are intended to be conducted nationally in this sector.

2. Literature review

2.1. The level of lipid profile tests and Parkinson's disease

A case-control investigation was carried out in 2014 at Sichuan University in western China, involving 555 patients diagnosed with sporadic Parkinson's disease and an equal number of control participants. The findings revealed that the mean of total cholesterol, LDL, and triglycerides were significantly lower in the Parkinson's disease patients when compared to the control group (4.5 ± 0.9 mmol/L versus 5.0 ± 0.9 mmol/L, $p < 0.001$), (2.5 ± 0.7 mmol/L versus 2.9 ± 0.7 mmol/L , $p < 0.001$), and (1.2 ± 0.8 mmol/L versus 1.5 ± 0.8 mmol/L , $p < 0.001$ respectively). However, the mean HDL was slightly lower in the PD patients compared to the controls (1.5 ± 0.4 mmol/L vs. 1.6 ± 0.4 mmol/L ; $p = 0.003$), which was also observed in the female PD patients compared with the female controls (1.6 ± 0.4 mmol/L vs. 1.7 ± 0.4 mmol/L; $p <0.001$). Finally, they concluded that High levels of total cholesterol and LDL-C may be associated with low prevalence of PD [42].

A case-control study carried out from 2016 to 2018 at Tabriz University in Iran involved 75 individuals diagnosed with Parkinson's disease and a control group of 75 healthy individuals. The findings revealed that the serum triglyceride levels were significantly lower in the PD group (111.92±8.75 mg/dL) compared to the healthy participants (123.64±9.97 mg/dL, P=0.008). Furthermore, total cholesterol (TC) levels were also notably lower in those with PD (180.33±7.40 mg/dL) in contrast to the control group (189.49±12.36 mg/dL P=0.004). However, the serum levels of high-density lipoprotein cholesterol (HDL) did not show a significant difference between the PD patients and the control group (48.06±4.62 mg/dL vs. 50.42±6.27 mg/dL, P=0.135). They conclude that serum concentration of TC, LDL-C, and TG in subjects with PD were lower than the healthy individuals. Lipid abnormalities appear to be associated with PD, as further studies can investigate the underlying mechanism in this area, which is more dependent on diet and lifestyle [36].

In a cohort study conducted in Honolulu from 1999 to 2001 with a sample of 3,233 male participants, findings indicated that the incidence of Parkinson's disease increased in a dose-dependent manner as low-density lipoprotein levels decreased (P = 0.044). This relationship was found to be significant exclusively in men aged 71 to 75 years. After adjusting for confounding factors such as age, smoking, coffee consumption, and others, the relative odds of PD for men at

the 80th percentile of LDL compared to those at the 20th percentile were determined to be 0.4. These results support the hypothesis that lower LDL cholesterol levels are associated with an increased risk of PD [43].

In a 2016 study conducted in Italy involving 150 patients with Parkinson's disease, researchers found a significant correlation between lipid levels and disease duration. After adjusting for age, gender, smoking status, and levodopa dosage, high-density lipoprotein (HDL) levels showed a positive correlation with disease duration ($r = 0.67$, $P=0.001$). Specifically, for each additional year of disease duration, HDL levels increased by approximately 5.8mg/dL. Conversely, the total cholesterol to HDL (TC/HDL) ratio demonstrated a significant negative correlation with disease duration ($r = -0.54$, $P<0.001$), with the ratio decreasing by 2 units per year of disease progression [44].

A prospective study was conducted in the United States from 1995 to 2020, involving participants from two large cohorts: the Nurses' Health Study (122,046 women) and the Health Professionals Follow-Up Study (50,833 men). Over the follow-up period, 530 new cases of Parkinson's disease were identified. After adjusting for age and smoking history, higher total cholesterol levels were associated with a modestly increased risk of PD (hazard ratio = 1.18, 95% CI: 1.05–1.33, $p = 0.008$), with an average total cholesterol level of 210 ± 35 mg/dL in individuals who developed PD. However, the use of cholesterol-lowering medications, including statins, showed no significant association with PD risk (HR = 0.97, 95% CI: 0.85–1.10, $p = 0.58$). These findings suggest that elevated total cholesterol may contribute to a slightly increased risk of Parkinson’s disease, independent of cholesterol-lowering treatment [45].

A population-based case-control study was conducted in Rotterdam, Netherlands, from 2016 to 2020, involving 7,983 participants. Initial analyses were adjusted for age and sex, followed by additional adjustments for smoking habits, vitamin E intake, coffee consumption, body mass index (BMI), and baseline use of cholesterol-lowering medications. The findings demonstrated an inverse association between serum total cholesterol levels and the risk of Parkinson’s disease (odds ratio = 0.76, 95% CI: 0.62–0.92, $p = 0.005$), with this relationship being more pronounced in women (OR = 0.70, 95% CI: 0.55–0.88, $p = 0.002$). The mean total cholesterol level among participants who developed Parkinson’s disease was 190 ± 28 mg/dL, compared to 205 ± 32 mg/dL in controls. Additionally, the study found no evidence suggesting that Parkinson’s disease

itself altered cholesterol levels, reinforcing that lower serum cholesterol may be an independent risk factor for Parkinson's disease rather than a consequence of the condition [46].

2.2. The level of lipid profile tests and Alzheimer's disease

A study conducted in China from 2018 to 2022 examined the relationship between low-density lipoprotein (LDL) cholesterol and the risk of Alzheimer's disease. The analysis included 3,500 participants, comprising 1,750 AD patients and 1,750 age- and sex-matched controls. The results showed that individuals with Alzheimer's disease had significantly higher LDL levels (AD group: 135 ± 30 mg/dL vs. Control group: 120 ± 28 mg/dL, $p < 0.001$). Meta-regression analysis identified age ($p = 0.002$) and cardiovascular disease ($p = 0.008$) as significant confounders influencing the relationship between LDL cholesterol and Alzheimer's risk. However, other factors, including body mass index, education, smoking status, hypertension, and diabetes mellitus, did not show a statistically significant impact ($p > 0.05$). The study concluded that elevated LDL cholesterol levels may serve as an independent risk factor for Alzheimer's disease, highlighting the potential role of lipid metabolism in neurodegenerative processes. [47].

A study conducted in China from 2017 to 2023 investigated the relationship between blood cholesterol levels and the incidence of Alzheimer's disease in the elderly population. The study included 4,200 participants, comprising 2,100 AD patients and 2,100 age- and sex-matched controls. After adjusting for body mass index, smoking behavior, history of stroke, hypertension, type 2 diabetes, and cardiovascular diseases, the findings indicated that higher total cholesterol and LDL levels, along with lower HDL levels, were associated with an increased risk of AD. Specifically, TC: AD group 220 ± 35 mg/dL vs. Control group 200 ± 30 mg/dL ($p < 0.001$, OR = 1.32, 95% CI: 1.15–1.50) LDL: AD group 140 ± 32 mg/dL vs. Control group 125 ± 28 mg/dL ($p = 0.002$, OR = 1.25, 95% CI: 1.10–1.42) HDL: AD group 45 ± 12 mg/dL vs. Control group 52 ± 14 mg/dL ($p = 0.007$, OR = 0.78, 95% CI: 0.65–0.93).The study concluded that dyslipidemia, particularly elevated TC and LDL levels along with reduced HDL, may contribute to an increased risk of Alzheimer's disease in the elderly population [48].

A retrospective cross-sectional study conducted in the United States from 2020 to 2023 examined the relationship between amyloid deposition and total cholesterol levels in the brain among 140 patients. The study focused on individuals aged 40 to 55 years, assessing early amyloid accumulation. The results demonstrated a significant correlation between elevated TC

levels and increased amyloid deposition in younger participants. Specifically levels: Participants with early amyloid deposition had a mean TC of 225 ± 38 mg/dL, compared to 190 ± 30 mg/dL in those without amyloid accumulation (p = 0.003).Odds of amyloid pathology: Higher cholesterol levels were associated with a 2.9-fold increased risk of amyloid deposition (OR= 2.92, 95% CI: 1.58–5.40,p=0.001).The study concluded that serum hypercholesterolemia could serve as an early indicator of increased risk for amyloid pathology associated with Alzheimer's disease, highlighting the potential role of lipid metabolism in early disease mechanisms [49].

A population-based study conducted in Ibadan, Nigeria, from 2010 to 2013 investigated the relationship between lipid profiles and Alzheimer's disease among elderly Yoruba individuals aged 70 years and above. The study included 1,075 participants, comprising 538 individuals diagnosed with AD and 537 age- and sex-matched controls. The findings revealed that individuals with AD had significantly higher total cholesterol and low-density lipoprotein levels, while their triglyceride levels were lower compared to the control group: Total cholesterol: AD group 215 ± 34 mg/dL vs. Control group 195 ± 30 mg/dL (p < 0.001, OR = 1.40)Low-density lipoprotein: AD group 140 ± 28 mg/dL vs. Control group 125 ± 26 mg/dL (p = 0.002, OR = 1.32)Triglycerides: AD group 110 ± 22 mg/dL vs. Control group 125 ± 25 mg/dL (p = 0.008, OR = 0.79)The study concluded that elevated TC and LDL levels, along with lower triglycerides, may be associated with an increased risk of Alzheimer's disease in the elderly Yoruba population, suggesting a potential role of lipid dysregulation in neurodegeneration [50].

3. Objectives

3.1. General Objective

Assessment of lipid profiles and associated factors among patients with Alzheimer's and Parkinson's diseases at Tikur Anbessa Specialized Hospital (TASH), Addis Ababa, Ethiopia. March to June 2024 G.C.

3.2. Specific Objective

- ❖ To compare lipid profile (TG, HDL, LDL, and TC) levels between Parkinson's disease patients and the control group at TASH, Addis Ababa, Ethiopia, 2024.
- ❖ To compare lipid profile (TG, HDL, LDL, and TC) levels between Alzheimer's disease patients and the control group at TASH, Addis Ababa, Ethiopia, 2024.
- ❖ To identify associated factors with the level of lipid profile test levels among Alzheimer's and Parkinson's disease patients at TASH, Addis Ababa, Ethiopia, 2024.

4. Materials and methods

4.1. Study Area

This investigation took place at Tikur Anbessa Specialized Hospital (TASH), which stands as one of Ethiopia's largest and most frequented referrals specialized medical facilities. TASH, the largest and oldest tertiary-level specialized hospital in Ethiopia, provides services to a population of almost 8 million. It plays a crucial role as the hospital affiliated with the only neurology training center in the nation. The Neurology Referral Clinic at TASH is acknowledged as one of the foremost and most technologically advanced neurology centers in Ethiopia. It operates as an integral component of the national referral system, providing expert neurological care to patients throughout the nation.

4.2. Study design and period

A hospital-based comparative cross-sectional study was carried out from March to June 2024.

4.3. Population

4.3.1. Source population

All neurodegenerative patients who visited the neurology unit in TASH during the study period.

All Tikur Anbessa Specialized Hospital Admin staff

All Parkinson’s and Alzheimer’s patient’s guardians

4.3.2. Study population

All consented patients with Alzheimer’s and Parkinson’s diseases and apparently healthy individuals who met the specified criteria for inclusion.

4.4. Eligibility criteria

4.4.1. Inclusion criteria

- All consented and volunteered Alzheimer’s and Parkinson’s disease patients and apparently healthy individuals

4.4.2. Exclusion criteria

- ❖ Individuals with other neurodegenerative diseases such as Huntington's disease, amyotrophic lateral sclerosis, and multiple sclerosis
- ❖ Alzheimer's and Parkinson's patients with a history of chronic diseases like diabetes mellitus, hypertension, cardiovascular disease, and others.
- ❖ Individuals who are on treatment of lipid lowering drugs
- ❖ Individuals who are not fasting in the morning

4.5. Study variables

4.5.1. Dependent variable

The level of serum lipid profiles (total cholesterol, triglycerides, high-density lipoprotein cholesterol, and low- density lipoprotein cholesterol).

4.5.2. Independent variables

- ❖ Socio-demographic factors (age, marital status, educational level, and others)
- ❖ BMI
- ❖ Smoking status
- ❖ Coffee consumption
- ❖ Alcohol consumption
- ❖ Physical activity
- ❖ Nutrition status
- ❖ Duration of disease

4.6. Sample size determination and sampling method

4.6.1. Sample size calculation

The sample size was calculated through a comparative-based study design for quantitative data, taking into account a 95% confidence interval and an 80% power, as indicated by the formula below [51].

$$N = \frac{S1^2 + S2^2 \, (Z\beta + Z\alpha/_2)^2}{d^2}$$

Where **S1**= standard deviation of case group, **S2**= standard deviation of healthy group, **d** = Expected mean difference between two groups, $\mathbf{Z\alpha/_2}$=1.96, **Zβ** = power = 0.84. According to the authors' understanding, there has been no prior publication addressing the lipid profile levels in individuals with Parkinson's and Alzheimer's diseases in the Ethiopian. Consequently, data from a study conducted on the Yoruba population in Nigeria was used [50]. A study revealed that the average total cholesterol levels in the case group and the control group were 192.2 mg/dl and 170.9 mg/dl, respectively. The standard deviation for total cholesterol in both groups was found to be 36 mg/dl.

$$N = \frac{36^2 + 36^2\,(0.84 + 1.96)}{192.2 - 170.9^{\,2}} = \frac{2592 * (2.8)^2}{(21.3)^{\,2}} = \frac{2592 * 7.84}{453.69}$$

$$N = \frac{0321.28}{453.69} = \mathbf{44.79,\ approximate\ 45}$$

N.B.: In this study, a sample size of 50 participants was established for each group, accounting for a 10% non-response rate. The study involved a sample of 50 patients diagnosed with Parkinson's disease, 61 patients diagnosed with Alzheimer's disease, and 61 healthy controls. The sample sizes for the Alzheimer's and control groups were increased due to the availability of additional cases beyond the calculated sample size, thereby allowing for a more generalized conclusion regarding the findings [52, 53].

4.6.2. Sampling method

Quota sampling techniques were applied to recruit study participants. Over a period of four months, Parkinson's disease and Alzheimer's disease patients were identified and recruited if they met the exclusion criteria.

4.7. Measurement and Data collection

4.7.1. Data collection procedure

The study's obje**c**tives were clearly articulated to the participants, who then offered their written consent to engage in the study. Data collection was carried out using a pre-tested structured questionnaire, designed based on a comprehensive analysis of literature from a range of sources. A preliminary test of the questionnaire was conducted with 5% of the individuals participating in the study.

The collection of anthropometric data's such as height, weight was carried out by trained personnel with a standardized measurement scale. BMI (kg/m2) was determined by dividing the weight of an individual by the square of their height. Prior to obtaining a blood sample, volunteers were instructed to refrain from eating for a duration of 8 to 12 hours overnight.

4.7.2. Blood sample collection, processing and analysis

Complying with antiseptic techniques, a lab technologist collected 5 ml of venous blood in a BD serum separator tube, which contained a clot activator and gel for serum separation, from the antecubital vein of a voluntary study participant who had fasted overnight for 8–12 hr. To promote clot formation, the blood specimen was left to stand for a period of 10 to 20 minutes. The sample was centrifuged at 3,000 revolutions per minute (rpm) for 10 minutes to separate the serum from whole blood. Then, 1.5 mL of serum was transferred to a Nunc tube and stored at −20°C until analysis.

It was then centrifuged at a rate of 3000 revolutions per minute (rpm) for 10 minutes, allowing for the separation of serum from the whole blood, which was stored in Nunc tube at −20 °C until it was analyzed. Subsequent to successfully completing all phases of the standard procedure outlined in the standard operating procedure manual, lipid panel analyses were conducted using the Cobas c311 automated analyzer. Following this, the results of all tests were meticulously recorded on the data collection sheet.

4.7.3. Laboratory Analysis

Clinical chemistry analyzer: Cobasc311.

4.7.3.1. High Density Lipoprotein-Cholesterol (HDL)

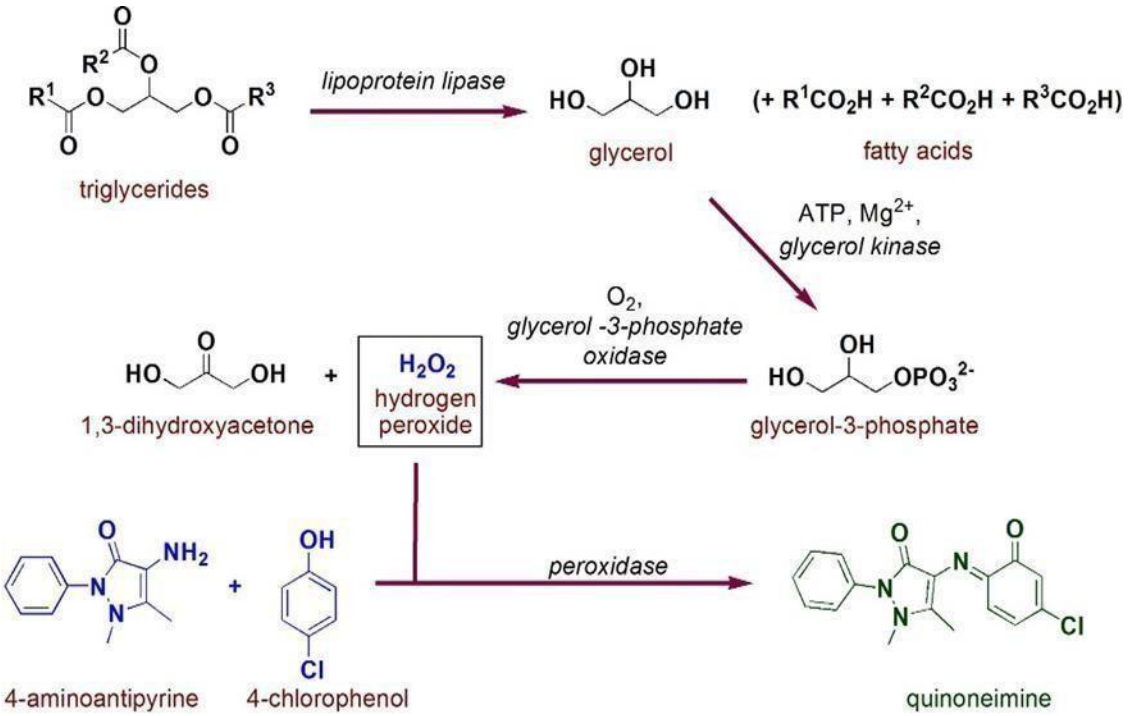
Homogeneous enzymatic colorimetric test: The interaction of non-HDL lipoproteins, such as LDL, VLDL, and chylomicrons, with polyanions and a detergent result in the formation of a water-soluble complex. This complex effectively inhibits the enzymatic action of cholesterol esterase on non-HDL lipoproteins. As a result, only HDL particles are able to participate in reactions with cholesterol esterase (CHOR) and cholesterol oxidase (CHOD). The color intensity produced is directly proportional to the cholesterol concentration and is assessed photometrically at 700/600 nm.

4.7.3.2. Low Density Lipoprotein-Cholesterol (LDL)

Homogeneous enzymatic colorimetric assay: The quantification of cholesterol esters and free cholesterol within low-density lipoprotein is conducted through an enzymatic assay that employs cholesterol esterase and cholesterol oxidase, facilitated by surfactants that specifically solubilize LDL. Surfactants, along with a sugar compound, inhibit enzymatic reactions with lipoproteins other than LDL. The resulting color intensity of the dye produced is directly proportional to the concentration of cholesterol and is assessed using photometric.

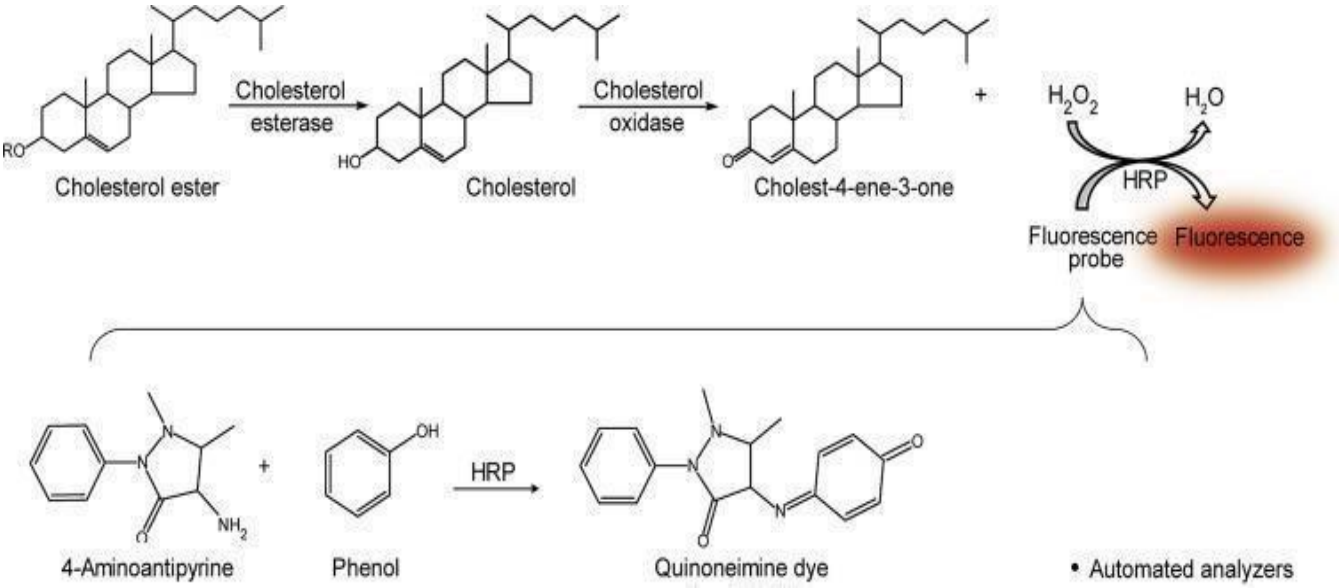
4.7.3.3. Triglyceride (TG)

This method is based on the work by Wahlefeld using a lipoprotein lipase from microorganisms for the rapid and complete hydrolysis of triglycerides to glycerol followed by oxidation to dihydroxyacetone phosphate and hydrogen peroxide. The hydrogen peroxide produced then reacts with 4-aminophenazone and 4-chlorophenol under the catalytic action of peroxidase to form a red dye. The color intensity of the red dye formed is directly proportional to the triglyceride concentration and can be measured photometrically (700 / 505 nm).



4.7.3.4.Total cholesterol (TC)

Enzymatic colorimetric method: The colour intensity of the dye formed is directly proportional to the cholesterol concentration. It is determined by measuring the increase in absorbance (700/505 nm).



4.8. Data Quality Assurance

The questionnaire was initially designed in English and subsequently translated into Amharic, the indigenous language. To evaluate its consistency, a back-translation into English was executed. A pretest was conducted with 5% of the study subjects before the data collection took place.

After the interview had been completed, thorough cross-checking was conducted on a daily basis to ensure the entirety of the collected information. The measurement of height was done using a standard height measuring meter in an upright position, and weight was recorded after calibrating the scale to zero.

The daily verification of instruments and laboratory reagents was conducted through the use of quality control samples (human sera) prior to the analysis of patient samples. This verification process was reiterated whenever results deviated from the predetermined values. All requisite procedures, protocols, and comprehensive safety measures were adhered to in accordance with the manufacturer's guidelines.

4.8.1. Pre-analytical phase

All procedures, including the collection, transportation, and storage of blood samples, were conducted in accordance with standard operating procedures (SOPs) to maintain data integrity. Participants were adequately prepared, and meticulous labeling was performed on the questionnaire and the SST test tube.

Blood samples were collected from the antecubital fossa of the forearm. The area was first disinfected using 70% alcohol, after which the samples were placed into serum separate tubes containing separator gel. The specimens were then stored and transported to the laboratory for analysis in compliance with established SOPs.

4.8.2. Analytical phase

The analysis of lipid profiles was conducted in the clinical chemistry unit of the MKH laboratory department. Monthly calibration of the equipment was performed. Additionally, two levels of internal quality control (IQC) samples, representing both normal and pathological states, were processed alongside the serum sample. The results of the control samples were evaluated according to the Westgard multi-rule algorithm. The sample analysis commenced only after the principal investigator and senior laboratory technologists had thoroughly reviewed the instructions for each analyte.

4.8.3. Post Analytical Test

The results were reviewed for all post-analytical factors, including the reporting unit and the accuracy of the serial number assigned by the investigator. The responsible laboratory technologist subsequently approved the results. The results were then promptly attached to the questionnaire. If printer paper was not available, the results were diligently recorded in the specified area with the corresponding ID on the data collection sheet.

4.9. Data interpretation and Analysis

The data entry and analysis were performed using IBM SPSS, version 27. To assess the normality of the data, both the Kolmogorov-Smirnov and Shapiro-Wilk tests were employed with p-values exceeding 0.05, indicating a normal distribution. Quantitative variables were reported as mean ± standard deviation, and an independent t-test was utilized to compare the lipid profile test levels between the case and control groups.

In this study, Pearson’s correlation alongside bivariate and multivariate logistic regression

analyses was utilized to ascertain the risk factors linked to lipid profile test levels. The model's goodness of fit was evaluated using the Hosmer and Lemeshow test. The multivariate logistic regression analysis was conducted on those variables that had a p-value under 0.25 in the bivariate logistic regression analysis.

The adjusted odds ratio (AOR) was calculated to demonstrate the strength of the associations, with a p-value of less than 0.05 indicating statistical significance. The result was organized and displayed in tables, accompanied by figures and narrative explanations.

4.10. Ethical consideration

The study received ethical approval from the Department Research and Ethical Review Committee (DRERC) of the Department of Medical Laboratory Sciences, College of Health Sciences, Addis Ababa University. The study's purpose was communicated in detail to each participant, who subsequently provided signed informed consent. Confidentiality was stringently maintained throughout the study.

4.11. Dissemination of Result

The results of the study is conveyed to Addis Ababa University, College of Health Sciences, Department of Medical Laboratory Sciences. Additionally, the findings will be shared with the study site, Tikur Anbessa Specialized Hospital, Addis Ababa, Ethiopia. Furthermore, the study results was submitted for publication in a national or international peer-reviewed journal. Moreover, these findings may serve as foundational data for future research endeavors.

4.12. Operational definitions

- **Apparently healthy:** an individual who don't have a history of any systemic diseases such as Hypertension, Cardiac problem, Renal disease and Diabetic mellitus
- **Lipid profile:** includes HDL cholesterol, LDL cholesterol, triglyceride, and total cholesterol.
- **Dyslipidaemia:** defined as having elevated levels of total cholesterol (>200 mg/dL), LDL cholesterol (>100 mg/dL), or triglycerides (>150 mg/dL), and/or decreased levels of HDL cholesterol (<40 mg/dL for men and <50 mg/dL for women).
- **Parkinson's disease**: Diagnosis is usually based on clinical criteria, including the presence of motor symptoms (tremor, rigidity, bradykinesia) and the exclusion of other conditions that may mimic PD.
- **Alzheimer's disease**: A confirmed diagnosis typically involves clinical assessment, cognitive testing.

5. Work flows of the study

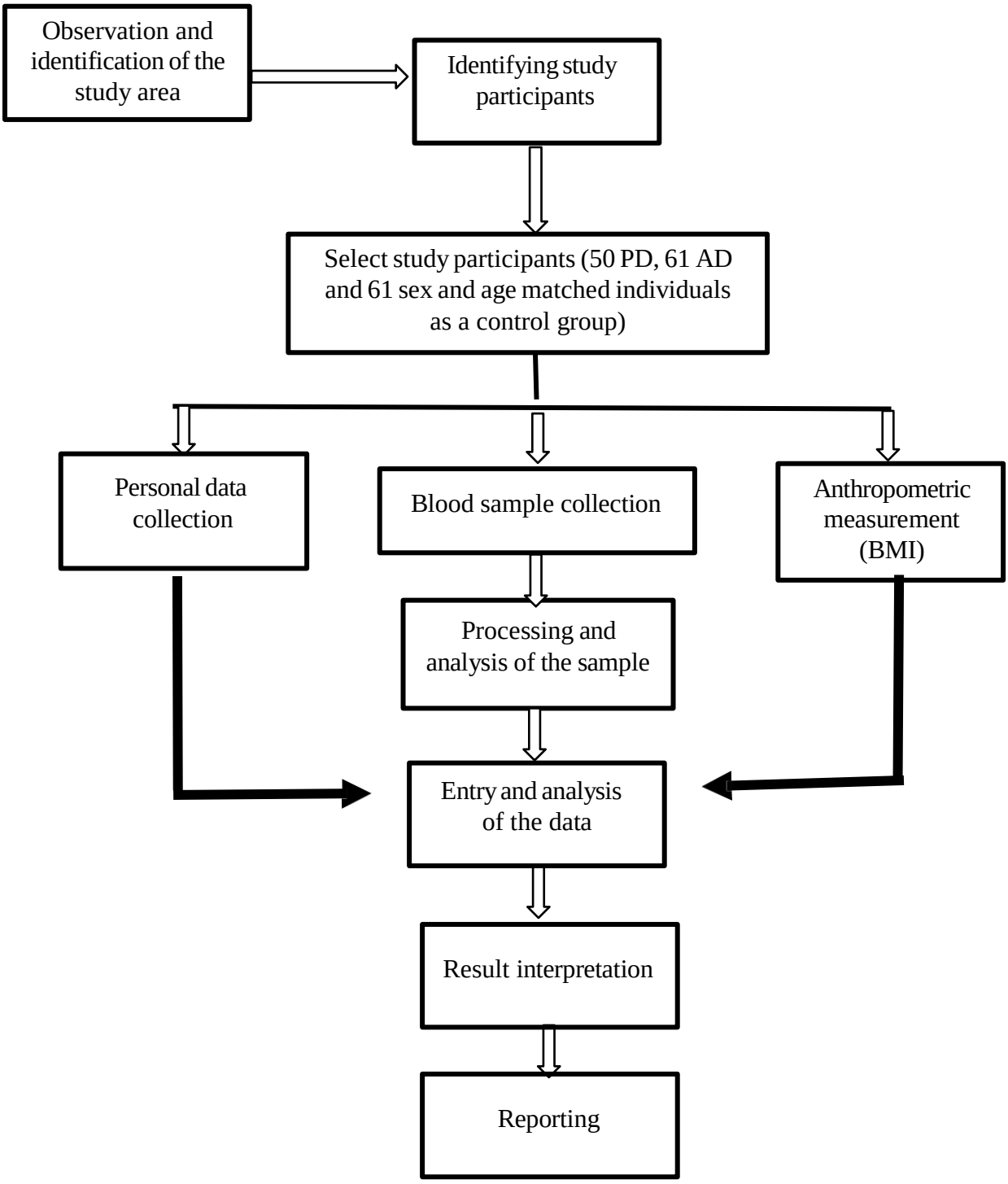


Figure 1: **Work flow**

6. Result

6.1. Descriptive characteristics of the study participants

The study involved 172 participants, who were divided into three categories: 50 individuals with Parkinson's disease, 61 individuals with Alzheimer's disease, and a control group consisting of 61 participants. The majority of the study participants were under 65 years of age: 49 (80.3%) for Alzheimer's patients, 33 (66%) for Parkinson's patients, and 49 (80.3%) for the control group.

From a total of 172 study participants, 89 (51.7%) were female, 127 (73.8%) were married, and 71 (41.3%) practiced Orthodox Christianity. Among them, 56 participants (32.6%) were government employees, and 61 (35.4%) had completed higher education. It was found that a majority of the participants, 91 out of 172 (52.9%), did not engage in physical activity on a regular basis. Additionally, a considerable number of participants reported not consuming alcohol or smoking cigarettes, 115 (66.9%) and 100 (58.1%), respectively (Table 1).

Table 1: Descriptive characteristics of study participants at TASH in Addis Ababa, Ethiopia, 2024 (N=172).

Parameters	Categories	Study groups			Total
		PD (n=50)	AD (n=61)	CG (n=61)	
		N (%)	N (%)	N (%)	N (%)
Age (year)	< 65	33 (66%)	49 (80.3%)	49 (80.3%)	131 (76.2%)
	≥ 65	17 (34%)	12 (19.7%)	12 (19.7%)	41 (23.8%)
Gender	Female	25 (50%)	35 (57.4%)	29 (47.5%)	89 (51.7%)
	Male	25 (50%)	26 (42.6%)	32 (52.5%)	83 (48.3%)
Marital status	Single	4 (8%)	7 (11.5%)	4 (6.6%)	15 (8.7%)
	Married	36 (72%)	43 (70.5%)	48 (78.7%)	127 (73.8%)
	Divorced	10 (20%)	11 (18%)	9 (14.7%)	30 (17.5%)
Religion	Orthodox	22 (44%)	23 (37.7%)	26 (42.6%)	71 (41.3%)
	Muslim	15 (30%)	16 (26.2%)	17 (27.9%)	48 (27.9%)
	Protestant	8 (16%)	13 (21.3%)	10 (16.4%)	31 (18%)
	Others	5 (10%)	9 (14.8%)	8 (13.1%)	22 (12.8%)
Occupation	Government employee	18 (36%)	18 (29.5%)	20 (32.8%)	56 (32.6%)
	Private employee	8 (16%)	12 (19.7%)	12 (19.7%)	30 (18.6%)
	House wife	16 (32%)	19 (31.1%)	18 (29.5%)	53 (30.8%)
	Others	8 (16%)	12 (19.7%)	11 (18%)	31 (18%)
Educational level	No formal education	16 (32%)	19 (31.1%)	13 (21.3%)	48 (27.9%)
	Primary school	11 (22%)	14 (23%)	15 (24.6%)	40 (23.3%)
	Secondary school	8 (16%)	9 (14.8%)	6 (9.8%)	23 (13.4%)
	Higher level	15 (30%)	19 (31.1%)	27 (44.3%)	61 (35.4%)
Residence	Urban	40 (80%)	41 (67.2%)	52 (85.2%)	133 (77.3%)
	Rural	10 (20%)	20 (32.8%)	9 (14.8%)	39 (22.7%)
Physical exercise	> 3 days per week	17 (34%)	19 (31.1%)	23 (37.7%)	59 (34.3%)
	1-3 days per week	2 (4%)	3 (5%)	17 (27.9%)	22 (12.8%)
	Sedentary	31 (62%)	39 (63.9%)	21 (34.4%)	91 (52.9%)
Coffee intake	Yes	9 (18%)	12 (19.7%)	26 (42.6%)	47 (27.3%)
	No	41 (82%)	49 (80.3%)	35 (57.4%)	125 (72.7%)
Alcohol intake	Yes	14 (28%)	32 (52.5%)	11 (18%)	57 (33.1%)
	No	36 (72%)	29 (47.5%)	50 (82%)	115 (66.9%)
Smoke cigarette	Yes	20 (40%)	35 (57.4%)	17 (27.9%)	72 (41.9%)
	No	30 (60%)	26 (42.6%)	44 (72.1%)	100 (58.1%)

Abbreviations: PD, Parkinson's disease patients; **AD,** Alzheimer's disease patients and **CG**, control group

The Body Mass Index (BMI) of most study participants was ranging from 18.5 to 24.9. Of this majority of the participants were individuals with Alzheimer's disease: 38 (62.3%) and the control group 54 (88.5%). In contrast, patients with Parkinson's disease displayed a BMI of less than 18.5, accounting for 22 (44%), while 18 (36%) had a BMI between 18.5 and 24.9. The mean duration of the diseases in Alzheimer's patients and Parkinson's patients was 10.48 and 11.24 years, respectively (Table 2).

Table 2: The deity habit and clinical characteristics of the study participants at TASH in Addis Ababa, Ethiopia, 2024 (N=172).

Parameters	Categories	Study groups			Total
		PD (n=50)	AD (n=61)	CG (n=61)	
		N (%)	N (%)	N (%)	N (%)
High fat foods intake	< 3 times per week	28 (56%)	27 (44.3%)	32 (52.5%)	92 (53.5%)
	≥3 times per week	22 (44%)	34 (55.7%)	29 (47.5%)	80 (46.5%)
BMI (kg/m^2)	< 18.5	22 (44%)	1 (1.6%)	2 (3.3%)	5 (2.9%)
	18.5 – 24.9	18 (36%)	38 (62.3%)	54 (88.5%)	123 (71.5%)
	25 – 29.9	10 (20%)	22 (36.1%)	5 (8.2%)	44 (25.6%)
Duration of disease	Mean ± SD, (year)	11.24 ± 4.62	10.48 ± 4.18	-	-

Abbreviations: BMI, body mass index; **PD**, Parkinson's disease patients; **AD,** Alzheimer's disease patients and **CG,** control group

6.2. The lipid profiles of the study participants and the control group

The levels of LDL were significantly lower in patients with Parkinson's disease (76.7 ± 21.77 mg/dl) compared to the control group (85.2 ± 12.59 mg/dl), P = 0.012. Significantly lower levels of TG were observed in patients with Parkinson's disease (115.02 ± 24.84 mg/dl) compared to the control group (124.12 ± 16.01 mg/dl), P = 0.028. Furthermore, the level of TC was significantly lower in patients with Parkinson's disease (152.2 ± 30.44 mg/dl) compared to the control group TC (165.36 ± 29.33 mg/dl), P =0.023 (Table 3).

The mean HDL levels were found to be significantly lower in Alzheimer's disease patients (37.64 ± 7.45 mg/dl) compared to the control group (41.84 ± 5.83 mg/dl, P = 0.001). Conversely, LDL levels were found to be significantly higher in the Alzheimer's disease patients (106.76 ± 26.55

mg/dl) than in the control group (85.2 ± 12.59 mg/dl, P < 0.001). However, the analysis revealed no significant differences in TC (167.43 ± 28.36 mg/dl vs. 165.36 ± 29.33 mg/dl, P = 0.693) and TG (127.25 ± 23.64 mg/dl vs. 124.12 ±

16.01 mg/dl, P = 0.394) in Alzheimer's disease patients and the control group, respectively (see Table 3).

Table 3: The lipid profiles of the study participants and the control group at TASH in Addis Ababa, Ethiopia, 2024 (N=172).

Lipid profiles (mg/dl)	Parkinson's disease patients		Alzheimer's disease patients		Control group
	Mean ± SD	P value	Mean ± SD	P value	
LDL	76.7 ± 21.77	**0.012**	106.76 ± 26.55	**< 0.001**	85.2 ± 12.59
TG	115.02 ± 24.84	**0.028**	127.25 ± 23.64	0.394	124.12 ± 16.01
HDL	42.38 ± 8.08	0.691	37.64 ± 7.45	**0.001**	41.84 ± 5.83
TC	152.2 ± 30.44	**0.023**	167.43 ± 28.36	0.693	165.36 ± 29.33

Note: The P value is based on comparison with control group using the independent sample t-test. **Abbreviations: HDL,** high-density lipoprotein; **TG,** triglyceride; **LDL,** low-density lipoprotein; **TC,** total cholesterol; **mg/dl,** milligram per deciliter.

In female patients diagnosed with Parkinson's disease, the levels of total cholesterol were significantly lower (150.48 ± 27.63 mg/dl) when compared to the female control group (169.86 ± 26.3 mg/dl), P = 0.011. Furthermore, male Parkinson's disease patients demonstrated significantly lower triglyceride and low-density lipoprotein levels, recorded at 110.56 ± 28.18 mg/dl and 74.28 ± 20.84 mg/dl, respectively, in contrast to the male control group (122.28 ± 15.29 mg/dl and 86.38 ± 12.94 mg/dl), with P values of 0.050 and 0.015, respectively (see Table 4).

The study revealed that female Alzheimer's disease patients had significantly lower mean HDL levels (37.06 ± 6.57 mg/dl) compared to the female control group (41.86 ± 6.9 mg/dl, P = 0.006). Conversely, the LDL levels were significantly higher in the female Alzheimer's cohort (109.23 ± 25.66 mg/dl) than in the female control group (83.9 ± 12.3 mg/dl, P < 0.001). Additionally, male patients with Alzheimer's disease also showed significantly increased LDL levels (103.42 ± 27.85 mg/dl) compared to their male control counterparts (86.38 ± 12.94 mg/dl, P = 0.007) (Table 4).

Table 4: Gender-Adjusted lipid profile levels of the study participants and the control group at TASH in Addis Ababa, Ethiopia, 2024 (N=172).

Lipid profiles (mg/dl)	Female Respondents				
	Parkinson's disease patients (N = 25)		Alzheimer's disease patients (N=35)		Control group (N =29)
	Mean ± SD	P value	Mean ± SD	P value	
LDL	79.12 ± 22.83	0.356	109.23 ± 25.66	< **0.001**	83.9 ± 12.3
TG	119.48 ± 20.6	0.204	123.43 ± 23.95	0.598	126.14 ± 16.8
HDL	41.32 ± 8.96	0.807	37.06 ± 6.57	**0.006**	41.86 ± 6.9
TC	150.48 ± 27.63	**0.011**	163.83 ± 29.38	0.390	169.86 ± 26.3
					Male Respondents
Lipid profiles (mg/dl)	Parkinson's disease patients (N =25)		Alzheimer's disease patients (N=26)		Control group (N =32)
	Mean ± SD	P value	Mean ± SD	P value	
LDL	74.28 ± 20.84	**0.015**	103.42 ± 27.85	**0.007**	86.38 ± 12.94
TG	110.56 ± 28.18	**0.050**	132.39 ± 22.66	0.059	122.28 ± 15.29
HDL	43.44 ± 7.12	0.331	38.42 ± 8.56	0.079	41.81 ± 4.77
TC	153.92 ± 33.49	0.403	172.27 ± 26.73	0.158	161.28 ± 31.69

6.3. Analysis of Factors Associated with the Level of Lipid Profile among the Study Participants

The results showed that there are significant negative correlations between LDL ($r = -0.610$, $P < 0.001$), TG ($r = -0.594$, $P < 0.001$), and TC ($r = -0.622$, $P < 0.001$) levels and the duration of Parkinson's disease (Figures 2, A and B). In contrast, there is a significant positive correlation between the duration of Alzheimer's disease and LDL ($r = 0.604$, $P < 0.001$) levels, whereas HDL levels reveal a negative correlation ($r = -0.567$, $P < 0.001$) (Figure 2, C and D).

The relationship between age and lipid profile test levels revealed that patients with Alzheimer's disease have a positive correlation with LDL levels ($r = 0.274$, $P = 0.032$), whereas patients with Parkinson's disease have a negative correlation with TG ($r = -0.320$, $P = 0.023$) (Table 5). Additionally, the study indicated that in patients with Parkinson's disease, a significant positive correlation was observed between body mass index and triglyceride ($r = 0.421$, $P = 0.002$), total cholesterol ($r = 0.385$, $P = 0.006$), and low-density lipoprotein ($r = 0.431$, $P = 0.002$) as presented in Table 5.

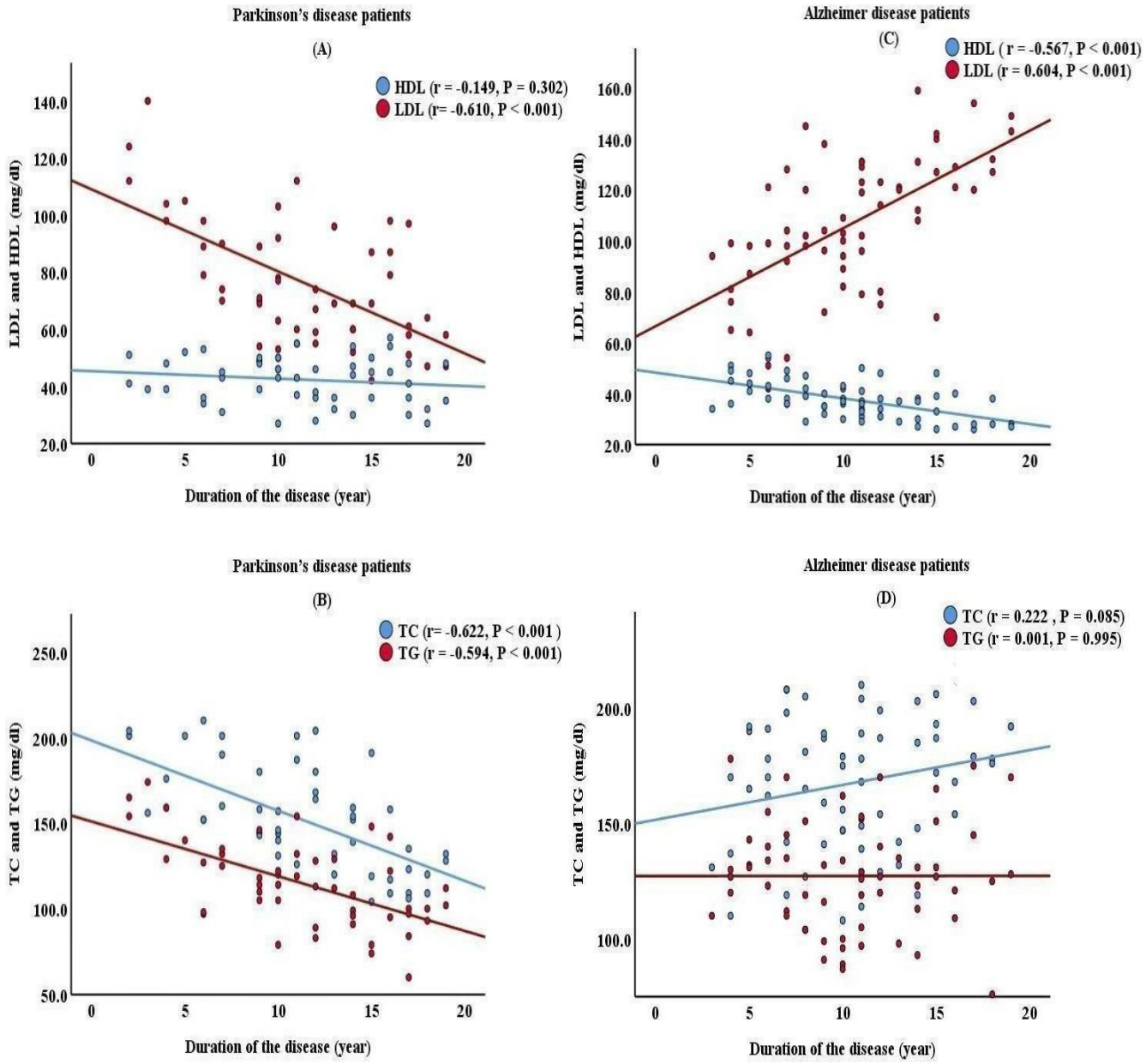


Figure 2: The correlation between lipid profile levels and the duration of Parkinson's and Alzheimer's diseases, as determined by Pearson correlation analysis.

Table 5: The correlation of lipid profile test levels with age, BMI and duration of the diseases in study participants at TASH in Addis Ababa, Ethiopia, 2024 (N=172).

Lipid profiles (mg/dl)	Study groups	Associated factors					
		Age (year)		Duration of the disease		BMI (Kg/m^2)	
		r	P value	r	P value	r	P value
HDL (mg/dl)	**PD**	-0.146	0.312	-0.149	0.302	0.062	0.671
	AD	-0.160	0.219	-0.567	< **0.001**	0.179	0.166
	CG	0.147	0.260	-	-	-0.101	0.439
LDL (mg/dl)	**PD**	-0.116	0.421	-0.610	< **0.001**	0.431	**0.002**
	AD	0.274	**0.032**	0.604	< **0.001**	-0.200	0.123
	CG	0.196	0.131	-	-	0.130	0.316
TG (mg/dl)	**PD**	-0.320	**0.023**	-0.594	< **0.001**	0.421	**0.002**
	AD	0.265	0.39	0.001	0.995	0.009	0.947
	CG	0.168	0.196	-	-	0.038	0.771
TC (mg/dl)	**PD**	-0.032	0.828	-0.622	< **0.001**	0.385	**0.006**
	AD	-0.078	0.549	0.222	0.085	-0.038	0.771
	CG	-0.067	0.610	-	-	-0.176	0.175

Abbreviations: BMI, Body mass index; **PD,** Parkinson's disease patients; **AD,** Alzheimer's disease patients; **CG,** control group and P value is based on Pearson correlation.

6.4. The factors associated with the lipid profile of the study participants

A bivariate analysis was performed to ascertain candidate variables suitable for multivariate logistic regression, guided by the Wald test results from logistic regression and a p-value less than 0.25 for at least one variable within the group. Consequently, the variables of gender, physical exercise, cigarette smoking, and high-fat food consumption were identified as candidates for the multivariate logistic regression model (refer to Tables 6 and 7).

Table 6: Bivariate logistic regression showing factors associated with HDL and LDL levels among Alzheimer disease patients at TASH in Addis Ababa, Ethiopia, 2024 (N=61).

Parameters	Categories	HDL (< 40 mg/dl)		COR (95%CI)	P value	LDL (≥ 100 mg/dl)		COR (95%CI)	P value
		Low	Normal			High	Normal		
Age (year)	< 65	33	16	1		27	22	1	
	> 65	7	5	0.68 (0.19-2.48)	0.557	9	3	2.45 (0.59-10.14)	0.28
Gender	Female	26	9	2.48 (0.84-7.3)	0.1*	24	11	2.55 (0.89-7.28)	0.081*
	Male	14	12	1		12	14	1	
Marital status	Single	5	2	0.94 (0.11-7.73)	0.952	3	4	0.63 (0.09-4.22)	0.630
	Married	27	16	0.63 (0.15-2.74)	0.540	27	16	1.41 (0.37-5.36)	0.618
	Divorced	8	3	1		6	5	1	
Religion	Orthodox	14	9	1		13	10	1	
	Muslim	11	5	1.41 90.37-5.45)		9	7		
	Protestant	10	3	2.14 (0.46-9.98)	0.614	9	4	0.99 (0.27-3.58)	0.987
	Others	5	4	0.8 (0.17-3.82)	0.331	5	4	1.73 (0.41-7.29)	0.455
					0.783			0.96 (0.2-4.54)	0.960
Occupation	Government employee	11	7	1		11	7	1	
	Private employee	9	3	1.91 (0.38-9.59)		8	4	1.27 (0.28-5.87)	
	House wife	11	8	0.88 (0.24-3.26)	0.432	9	10	0.57 (0.16-2.12)	0.757
	Others	9	3	1.91 (0.38-9.59)	0.842	8	4	1.27 (0.28-5.87)	0.403
					0.432				0.757
Educational level	No formal education	13	6	0.58 (1.33-2.51)	0.464	11	8	0.64 (0.17-2.4)	0.502
	Primary school	8	6	0.36 (0.08-1.64)	0.85	6	8	0.35 (0.08-1.45)	0.470
	Secondary school	4	5	0.21 (0.04-1.19)	0.78	6	3	0.92 (0.17-5.00)	0.926
	Higher level	15	4	1		13	6	1	
Residence	Urban	28	13	1		25	16	1	
	Rural	12	8	0.70 (0.23-2.11)	0.523	11	9	0.78 (0.27-2.31)	0.656
Physical exercise	> 3 days per week	7	12	1		6	13	1	0.952
	1-3 days per week	1	2	0.86 (0.07-11.26)	0.907	1	2	1.08 (0.08-14.41)	0.003*
	Sedentary	32	7	7.84 (2.27-27.08)	0.001*	29	10	6.28 (1.88-20.97)	
Coffee intake	Yes	30	19	0.32 (0.06-1.6)	0.64	27	22	0.41 (0.1-1.7)	0.28
	No	10	2	1		9	3	1	
Alcohol intake	Yes	20	12	0.75 (0.26-2.17)	0.596	17	15	0.6 (0.21-1.68)	0.327
	No	20	9	1		19	10	1	
Smoke cigarette	Yes	29	6	6.59 (2.04-21.32)	0.002*	26	9	4.62 (1.55-13.82)	0.006*
	No	11	15	1		10	16	1	
High fat foods intake	< 3 times per week	12	15	1		9	18	1	<0.001*
	≥ 3 times per week	28	6	5.83 (1.82-18.67)	0.003*	27	7	7.71 (2.43-24.46)	

* Candidate variables for multivariate logistic regression model

Table 7: Bivariate logistic regression showing factors associated with TG and TC levels among Alzheimer disease patients at TASH in Addis Ababa, Ethiopia, 2024 (N=61).

Parameters	Categories	TG (≥ 150 mg/dl)		COR (95%CI)	P value	TC (≥ 200 mg/dl)		COR (95%CI)	P value
		High	Normal			High	Normal		
Age (year)	< 65	10	39	1		4	45	1	
	≥ 65	2	10	0.78 (0.15-4.14)	0.771	4	8	5.63 (1.16-27.22)	0.32
Gender	Female	6	29	0.69 (0.19-2.45)	0.565	3	32	0.39 (0.09-1.83)	0.234*
	Male	6	20	1		5	21		
Marital status	Single	1	6	0.75 (0.06-10.23)	0.829	2	5	4 (0.29-55.47)	0.301
	Married	9	34	1.19 (0.22-6.52)	0.840	5	38	1.32 (0.14-12.57)	0.812
	Divorced	2	9	1		1	10	1	
Religion	Orthodox	4	19	1		4	19	1	
	Muslim	3	13	1.1 (0.21-5.74)	0.913	3	13	1.1 (0.21-5.74)	0.913
	Protestant	4	9	2.11 (0.43-10.42)	0.359	1	12	0.4 (0.04-3.98)	0.431
	Others	1	8	0.6 (0.56-6.18)	0.663	0	9	0.00 (0.00)	0.999
Occupation	Government employee	3	15	1			15	1	
	Private employee	2	10	1.0 (0.14-7.1)	1.00	3	9	1.67 (0.28-10.09)	0.578
	House wife	3	16	0.94 (0.16-5.39)	0.942	3	18	0.28 (0.03-2.96)	0.288
	Others	4	8	2.5 (0.45-14.04)	0.298	1	11	0.46 (0.04-4.98)	0.518
						1			
Educational level	No formal education	5	14	1.34 (0.3-6.02)	0.703	1	18	0.21 (0.02-2.07)	0.81
	Primary school	3	11	1.02 (0.19-5.23)	0.979	2	12	0.63 (0.1-4.01)	0.620
	Secondary school	0	9	0.00 (0.00)	0.999	1	8	0.47 (0.05-4.93)	0.528
	Higher level	4	15	1		4	15	1	
Residence	Urban	6	35	1		6	35	1	0.617
	Rural	6	14	2.5 (0.69-9.08)	0.264	2	18	0.65 (0.12-3.54)	
Physical Exercise	> 3 days per week	5	14	1		3	16	1	
	1-3 days per week	0	3	0.00 (0.00)	0.999	0	3	0.00 (0.00)	0.999
	Sedentary	7	32	0.61 (0.17-2.27)	0.463	5	34	0.78 (0.17-3.69)	0.759
Coffee intake	Yes	9	40	0.68 (0.15-3.01)	0.606	5	44	0.34 (0.07-1.69)	0.88
	No	3	9	1		3	9	1	
Alcohol Intake	Yes	5	27	0.58 (0.16-2.09)	0.407	4	28	0.89 (0.2-3.95)	0.881
	No	7	22	1		4	25	1	
Smoke Cigarette	Yes	7	28	1.05 (0.29-3.78)	0.940	6	29	2.48 (0.46-13.45)	0.291
	No	5	21	1		2	24	1	
High fat foods Intake	< 3 times per week	4	23	1		1	26	1	
	≥ 3 times per week	8	26	1.77 (0.47-6.66)	0.399	7	27	6.74 (0.78-58.65)	0.084*

* Candidate variables for multivariate logistic regression model

A bivariate analysis was undertaken to pinpoint candidate variables for the multivariate logistic regression model. Among the variables tested, gender and the consumption of high-fat foods were highlighted as candidate variables for the multivariate logistic regression, as indicated in Tables 8 and 9.

Table 8: Bivariate logistic regression showing factors associated with HDL and LDL levels among Parkinson's disease patients at TASH in Addis Ababa, Ethiopia, 2024 (N=50).

Parameters	Categories	HDL (< 40 mg/dl)		COR (95%CI)	P value	LDL (≥ 100 mg/dl)		COR (95%CI)	P value
		Low	Normal			High	Normal		
Age (year)	< 65	14	19	1		5	28	1	
	≥ 65	6	11	0.74 (0.22-2.48)	0.626	2	15	0.75 (0.13-4.32)	0.744
Gender	Female	12	13	1.96 (0.62-6.19)	0.25*	4	21	1.4 (0.28-7)	0.684
	Male	8	17	1		3	22		
Marital status	Single	0	4	0.000 (0.000)	0.999	0	4	0.000 (0.000)	0.999
	Married	15	21	0.71 (0.18-2.91)	0.639	6	30	1.8 (0.19-16.98)	0.608
	Divorced	5	5	1		1	9	1	
Religion	Orthodox	7	15	1		3	19	1	
	Muslim	4	11	0.78 (0.18-3.34)	0.737	3	12	1.58 (0.27-9.17)	0.608
	Protestant	4	4	2.14 (0.41-11.17)	0.366	0	8	0.00 (0.00)	0.999
	Others	5	0	0.00 (0.00)	0.999	1	4	1.58 (0.13-19.42)	0.719
Occupation	Government employee	5	13	1		1	17	1	
	Private employee	4	4	2.6 (0.46-14.63)	0.278	2	6	5.67 (0.43-74.38)	0.87
	House wife	8	8	2.6 (0.63-10.79)	0.88	2	14	2.43 (0.2-29.66)	0.487
	Others	3	5	1.56 (0.27-9.11)	0.621	2	6	5.67 (0.43-74.38)	0.87
Educational level	No formal education	7	9	1.17 (0.28-4.87)	0.833	4	12	4.67 (0.46-47.63)	0.94
	Primary school	5	6	1.25 (0.26-6.03)	0.781	1	10	1.4 (0.08-25.14)	0.819
	Secondary school	2	6	0.5 (0.07-3.36)	0.476	1	7	2 (0.11-36.95)	0.641
	Higher level	6	9	1		1	14	1	
Residence	Urban	20	20	1		4	36	1	
	Rural	0	10	0.00 (0.00)	0.999	3	7	3.86 (0.7-21.15)	0.720
Physical Exercise	> 3 days per week	7	10	1		2	15	1	
	1-3 days per week	0	2	0.00 (0.00)	0.999	0	2	0.00 (0.00)	0.999
	Sedentary	13	18	1.03 (0.31-3.43)	0.959	5	26	1.44 (0.25-8.37)	0.683
Coffee intake	Yes	16	25	0.8 (0.19-3.43)	0.764	6	35	1.37 (0.14-13.04)	0.783
	No	4	5	1		1	8	1	
Alcohol Intake	Yes	6	8	1.18 (0.34-4.13)	0.797	2	12	1.03 (0.18-6.07)	0.971
	No	14	22	1		5	31	1	
Smoke Cigarette	Yes	9	11	1.41 (0.45-4.47)	0.556	7	13	0.00 (0.00)	0.999
	No	11	19	1		0	30	1	
High fat foods Intake	< 3 times per week	11	17	1		4	24	1	
	≥ 3 times per week	9	13	1.07 (0.34-3.34)	0.907	3	19	0.95 (0.19-4.76)	0.948

* Candidate variables for multivariate logistic regression model

Table 9: Bivariate logistic regression showing factors associated with TG and TC levels among Parkinson's disease patients at TASH in Addis Ababa, Ethiopia, 2024 (N=50).

Parameters	Categories	TG (≥ 150 mg/dl)		COR (95%CI)	P value	TC (≥ 200 mg/dl)		COR (95%CI)	P value
		High	Normal			High	Normal		
Age (year)	< 65	4	29	1		3	30	1	
	≥ 65	1	16	0.45 (0.05-4.41)	0.495	4	13	3.08 (0.6-15.74)	0.77
Gender	Female	3	22	1.57 (0.24-10.3)	0.639	3	22	0.72 (0.14-3.59)	0.684
	Male	2	23	1		4	21	1	
Marital status	Single	0	4	1 (0.000)	1	0	4	0.00 (0.00)	0.999
	Married	5	31	0.00 (0.00)	0.999	5	31	0.65 (0.11-3.96)	0.636
	Divorced	0	10	1		2	8	1	
Religion	Orthodox	2	20	1		2	20	1	
	Muslim	2	13	1.54 (0.19-12.32)	0.685	3	12	2.5 (0.36-17.17)	0.351
	Protestant	0	8	0.00 (0.00)	0.999	0	8	0.00 (0.00)	0.999
	Others	1	4	2.5 (0.18-34.67)	0.495	2	3	6.67 (0.67-66.84)	0.707
Occupation	Government employee	1	17	1		2	16	1	
	Private employee	1	7	2.43 (0.13-44.5)	0.55	1	7	1.14 (0.09-14.78)	0.919
	House wife	2	14	2.43 (0.2-29.66)	0.487	2	14	1.14 (0.14-9.21)	0.9
	Others	1	7	2.43 (0.13-44.5)	0.55	2	6	2.67 (0.3-23.43)	0.376
Educational level	No formal education	3	13	3.23 (0.3-35.11)	0.335	3	13	3.23 (0.3-35.11)	0.335
	Primary school	0	11	0.00 (0.00)	0.999	3	8	5.25 (0.47-59.29)	0.8
	Secondary school	1	7	2 (0.11-36.95)	0.641	0	8	0.00 (0.00)	0.999
	Higher level	1	14	1		1	14	1	
Residence	Urban	3	37	1		6	34	1	
	Rural	2	8	3.08 (0.44-21.58)	0.26	1	9	0.63 (0.07-5.92)	0.686
Physical Exercise	> 3 days per week	1	16	1		3	14	1	
	1-3 days per week	0	2	0.00 (0.00)	0.999	0	2	0.00 (0.00)	0.999
	Sedentary	4	27	2.37 (0.24-23.1)	0.458	4	27	0.69 (0.14-3.53)	0.657
Coffee intake	Yes	5	36	0.00 (0.00)	0.999	6	35	1.37 (0.14-13.04)	0.783
	No	0	9	1		1	8	1	
Alcohol Intake	Yes	2	12	1.83 (0.27-12.35)	0.533	1	13	0.39 (0.04-3.52)	0.398
	No	3	33	1		6	30	1	
Smoke Cigarette	Yes	5	15	0.00 (0.00)	0.999	6	14	12.43 (1.36-113.41)	0.52
	No	0	30	1		1	29	1	
High fat foods Intake	< 3 times per week	3	25	1		2	26	1	
	≥ 3 times per week	2	20	0.83 (0.13-5.48)	0.850	5	17	3.82 (0.66-22)	0.133*

* Candidate variables for multivariate logistic regression model

As shown in Table 10, multivariable logistic regression analyses were used to identify the risk factors that were associated with the levels of lipid profiles among patients with Alzheimer's disease.

In the multivariable logistic regression analysis, factors such as physical activity, smoking cigarettes, and the frequency of high-fat diets consumed per week were found to be statistically significant with HDL, LDL, and TC levels, indicated by a p-value below 0.05 and a 95% confidence interval.

Alzheimer's disease patients who did not have habits of doing physical exercise were 5.03 more likely to have decreased HDL levels (AOR: 5.03, 95% CI: 1.17 - 21.65, P = 0.03); on the other hand, they were

3.84 more likely to have increased LDL levels (AOR: 3.84, 95% CI: 1.58 - 15.93, P = 0.023) compared with those who scheduled physical exercises (> 3 days per week). In addition, those patients who smoked cigarettes had more chance of developing decreased levels of HDL (AOR: 5.35, 95% CI: 1.24-23.06, P- value = 0.025) compared to their counterparts.

Furthermore, this study found that individuals with Alzheimer's disease who consume fat-containing food in their diet for 3 or more days per week were 4.75 times more likely to have decreased HDL levels (AOR=4.75, 95% CI(1.09-20.71), p=0.038); conversely, 6.88 times more likely to have increased LDL (AOR=6.88, 95% CI(1.73-27.3), p=0.006) and 3.19 times more likely to have increased TC (AOR=3.19, 95% CI(1.05-6.54), p=0.046) levels as compared with those who consume fat-containing food for less than three days per week (Table 10).

The multivariate regression analysis indicated that there is no significant risk factors associated with Parkinson's disease, as neither gender nor the consumption of high-fat foods demonstrated a statistical correlation with lipid profile levels in patients with Parkinson's disease (Table 11).

Table 10: The relationship between lipid profile test levels and various risk factors in Alzheimer disease patients at TASH in Addis Ababa, Ethiopia, 2024 (N=61).

Parameters	Categories	HDL (< 40 mg/dl)		AOR (95%CI)	P value	LDL (≥ 100 mg/dl)		AOR (95%CI)	P value
		Low	Normal			High	Normal		
Physical activity	>3 days per week	7	12	1		6	13	1	
	1-3 days per week	1	2	0.11 (0.01 – 2.18)	0.144	1	2	0.14 (0.01-2.66)	0.191
	Sedentary	32	7	5.03 (1.17–21.65)	**0.03**	29	10	3.84 (1.58-15.93)	**0.023**
Smoking Cigarette	Yes	29	6	5.35 (1.24 –23.06)	**0.025**	26	9	2.95 (0.77-11.35)	0.115
	No	11	15	1		10	16	1	
Gender	Female	26	9	3.17 (0.72-13.89)	0.127	24	11	3.05 (0.76-12.28)	0.117
	Male	14	12	1		12	14	1	
Consume High fat diet	< 3 times per week	12	15	1		9	18	1	
	≥3 times per week	28	6	4.75 (1.09-20.71)	**0.038**	27	7	6.88 (1.73-27.3)	**0.006**
Parameters	**Categories**	**Triglyceride (≥ 150 mg/dl)**		**AOR (95%CI)**	**P value**	**TC (≥ 200 mg/dl)**		**AOR (95%CI)**	**P value**
		High	**Normal**			**High**	**Normal**		
Physical activity	> 3 days per week	5	14	1		3	16	1	
	1-3 days per week	0	3	0.42 (0.04-1.22)	0.999	0	3	0.2 (0.01-1.82)	0.999
	Sedentary	7	32	0.46 (0.1-2.01)	0.299	5	34	0.3 (0.04-2.14)	0.230
Smoking cigarette	Yes	7	28	0.95 (0.22-4.05)	0.942	6	29	1.82 (0.27-12.43)	0.543
	No	5	21	1		2	24	1	
Gender	Female	6	29	0.74 (0.2-2.79)	0.656	3	32	0.31 (0.06-1.65)	0.168
	Male	6	20	1		5	21	1	
Consume High fat diet	< 3 times per week	4	23	1		1	26	1	
	≥ 3 times per week	8	26	2.8 (0.6-13.06)	0.190	7	27	3.19 (1.05-6.54)	**0.046**

1 = reference groups

Table 11: The relationship between lipid profile test levels and various risk factors in Parkinson's disease patients at TASH in Addis Ababa, Ethiopia, 2024 (N=50).

Parameters	Categories	HDL (< 40 mg/dl)		AOR (95%CI)	P value	LDL (≥ 100 mg/dl)		AOR (95%CI)	P value
		Low	Normal			High	Normal		
Gender	Female	12	13	1.98 (0.62 – 6.36)	0.251	4	21	1.22 (0.19 – 7.64)	0.831
	Male	8	17	1		3	22	1	
Consume High fat diet	< 3 times per week	11	17	1		4	24	1	
	≥3 times per week	9	13	0.96 (0.3 – 3.09)	0.944	3	19	0.96 (0.15 - 6.03)	0.965
Parameters	**Categories**	**TG (≥ 150 mg/dl)**		**AOR (95%CI)**	**P value**	**TC (≥ 200 mg/dl)**		**AOR (95%CI)**	**P value**
		High	**Normal**			**High**	**Normal**		
Gender	Female	3	22	1.43 (0.19- 10.92)	0.727	3	22	0.4 (0.06 – 2.93)	0.369
	Male	2	23	1		4	21	1	
Consume High fat diet	< 3 times per week	3	25	1		2	26	1	
	≥ 3 times per week	2	20	0.80 (0.11 – 6.15)	0.834	5	17	6.52 (0.82 – 51.95)	0.076

1 = reference groups

7. Discussion

This study found that Parkinson's disease patients had significantly lower LDL, TG, and TC levels than the control group. Those lipid profiles may decrease due to the progressive degeneration of dopaminergic neurons in the substantia nigra and alterations in brain-liver communication may impair the biosynthesis of cholesterol and triglycerides, leading to reduced circulating lipid levels. Dopaminergic signaling plays a role in lipid homeostasis. Studies suggest that the loss of dopamine in PD affects peripheral lipid metabolism, leading to lower cholesterol and triglyceride levels in circulation [54, 55].

According to the current study, patients with Parkinson's disease had significantly lower serum LDL levels than the control group. However, Parkinson's disease patients and controls did not significantly differ in their HDL levels. This result is in line with studies done in Bangladesh, India, China, Iran, Italy, and the United States. According to all of these studies, the LDL levels of Parkinson's disease patients were significantly lower than those of the control group, while the HDL levels of the two groups did not differ significantly. Some studies suggest that higher LDL levels might be associated with a lower risk of PD, possibly because cholesterol plays a role in maintaining neuronal integrity and synaptic function. Lower LDL levels in PD patients could reflect a predisposition to neurodegeneration [42, 56-60].

The significantly lower triglyceride levels observed in patients with Parkinson's disease, as compared to the control group in our study, align with findings from earlier studies conducted in countries such as Iran, Spain, China, Brazil, and Sweden. Previous studies have consistently reported that serum triglyceride levels in individuals with Parkinson's disease are lower than those in the control group [36, 61-64]. The lower triglyceride levels in PD patients are likely due to a combination of metabolic changes, increased fat utilization, inflammation and dietary differences. In contrast to our results, the studies conducted in Iran and Jordan reported no significant difference in triglyceride levels between Parkinson's disease patients and the control group [62, 65]. This discrepancy may be attributed to differences in methodology or variations in sample size.

The total cholesterol level in our study was significantly lower in Parkinson's disease patients than in the control group; this is in agreement with studies done in China and the USA, which show that individuals with Parkinson's disease have significantly lower levels of total cholesterol

than the control group [42, 66- 70]. However, this study's results were inconsistent with those of studies conducted in Iran and Brazil, which found that Parkinson's disease patients had significantly higher levels of total cholesterol than the control group [58, 63]. According to a Jordanian study, the total cholesterol level did not differ significantly from controls [65]. Such discrepancies might be explained by variations in the methodologies applied, the genetic backgrounds of the participants, or the difference in sample sizes.

In comparison to the control group, the current study's findings demonstrated that Alzheimer's disease patients had significantly higher levels of LDL and significantly lower levels of HDL. The metabolism of amyloid precursor protein in nerve cells is affected by elevated LDL and TC levels, which also hasten the deposition of amyloid-β protein in the brain, a hallmark of Alzheimer's pathology [71, 72]. This makes it more difficult for amyloid-beta peptides to be cleared and hindering neuronal synaptic connections in the brain raises the risk of AD [73].

Lipoprotein apolipoprotein E (ApoE), which is primarily associated with low-density lipoprotein (LDL), plays a crucial role in lipid transport and metabolism in the brain. Among its three major genetic variants (ε2, ε3, and ε4), the ApoE ε4 allele is widely recognized as a major genetic risk factor for developing Alzheimer's disease (AD) [74]. Individuals carrying one or two copies of the ε4 allele have a significantly higher likelihood of developing AD compared to those with the more common ε3 allele. This increased risk is attributed to the ApoE ε4 variant's reduced efficiency in clearing amyloid-beta (Aβ) peptides from the brain, leading to their accumulation and the formation of amyloid plaques a key pathological feature of AD [75].

HDL has antioxidant and anti-inflammatory properties, which can affect the brain's neuroinflammatory responses and contribute significantly to enhanced cognitive function. Additionally, it reduces oxidative stress and eliminates amyloid plaques, which has neuroprotective effects. A decreased capacity to transport lipids and get rid of amyloid may be indicated by decreased HDL levels [76].

In this study, Alzheimer disease patients' HDL levels were significantly lower than those of the comparison group. Studies conducted in Croatia, China, Pakistan, and Poland have found similar results. In contrast to the control group, all of which documented that Alzheimer disease patients had significantly lower HDL levels [48, 77-79].

This study found that the LDL levels of Alzheimer disease patients were significantly higher than those of the comparison group. These results are aligned with studies done in China, Pakistan, Japan, and Poland [48, 78-81]. On the other hand, studies carried out in Israel and Turkey found no significant differences in LDL and HDL levels between the two groups, which contrasted with our study's findings of significantly higher LDL and lower HDL levels among Alzheimer disease patients when compared to the control group [82, 83]. The inconsistencies found in the findings may be attributed to the health status of the participants, including factors such as comorbidities, genetic differences, or variations in sample sizes.

The findings of this study suggest that there is no notable difference in total cholesterol and triglyceride levels between patients with Alzheimer's disease and the control group. This aligns with studies conducted in Israel and the United States [82, 84]. Conversely, studies from China and Japan indicates that Alzheimer's disease patients exhibit significantly higher total cholesterol levels and lower triglyceride levels when compared to the control group [48, 80]. Several factors may account for the observed variation in findings, including methodological differences, variations in sample sizes, individual health conditions, particularly the presence of comorbidities, and lifestyle differences among the study participants.

The study demonstrates that there is a significant decrease in LDL, triglycerides, and total cholesterol levels in relation to the increasing duration of Parkinson's disease. Furthermore, TG levels showed a significant decline with age among patients diagnosed with Parkinson's disease. These findings are in agreement with a study from Iran, which reported an inverse relationship between disease duration and LDL and total cholesterol levels [58].

Additionally, investigations in China and the United States have revealed a significant association between prolonged disease duration and reduced total cholesterol and LDL levels in patients with Parkinson's disease [85, 86]. The observed lower lipid levels correlated with deteriorating motor functions and cognitive impairments, particularly in the more advanced stage of the disease.

The correlation analysis demonstrated a significant positive association between body mass index (BMI) and serum levels of low-density lipoprotein, triglycerides, and total cholesterol. It is noteworthy that being underweight is associated with an elevated risk of incidence of Parkinson's

disease. This decrease in BMI might be associated with swallowing difficulties (dysphagia), a lack of appetite (anorexia), gastrointestinal issues, and increased energy expenditure resulting from tremors and muscle rigidity [87]. The hypothesis proposes that elevated BMIs could impact the levels of circulating and central insulin, potentially providing a protective effect against neurodegenerative disorders [88]. Our findings align with research conducted in Germany and Korea, which demonstrated an inverse relationship between overall BMI and PD risk. Furthermore, being underweight was found to heighten the risk of PD incidence [89, 90].

The duration of Alzheimer's disease has been found to have a significant positive correlation with LDL cholesterol levels, while a negative correlation is evident with HDL cholesterol levels. Additionally, there is a positive correlation between age and LDL cholesterol levels among individuals with Alzheimer's disease. This finding is consistent with a study from Germany that demonstrated an increase in LDL levels as individuals age [91].

Furthermore, studies conducted in South Korea and the Netherlands have revealed a significant association between increased LDL levels and the rapid progression of the disease [92, 93]. The higher levels of LDL and lower levels of HDL observed in Alzheimer's disease patients suggest that the severity of the disease may be linked to their involvement in the deposition of amyloid-beta and tau proteins [71, 72].

Our investigation reveals that individuals with Alzheimer's disease who do not engage in physical exercise are more likely to have lower HDL levels and higher LDL levels compared to those who exercise regularly. Regular physical activity promotes efficient lipid metabolism, leading to increased HDL production and improved clearance of LDL cholesterol from the bloodstream. This association was also observed in studies conducted in Denmark, where Alzheimer's patients who participated in moderate-to-high-intensity aerobic exercise for 16 weeks, with three 60-minute sessions per week, exhibited a significant increase in HDL levels compared to those who were physically inactive [94, 95]. These results indicate that an elevation in HDL plays a crucial role in reverse cholesterol transport, a process that removes excess cholesterol from cells, including neurons, and transports it to the liver for excretion. Exercise enhances this process, leading to higher HDL levels and lower LDL levels

Our investigation showed that Alzheimer's disease patients who smoked cigarettes had a significantly higher likelihood of exhibiting lower HDL levels in contrast to those who did not smoke. Smoking damages, the liver's ability to produce apolipoprotein A-I, a key component of HDL, leading to lower circulating HDL levels. Cigarette smoke contains harmful chemicals that promote oxidative stress and systemic inflammation, which disrupt HDL metabolism and reduce its protective effects. Findings from studies in Netherlands, Canada, South Korea, and Italy align with our results, demonstrating that Alzheimer's disease patients who smoke had significantly reduced HDL levels in comparison to those who do not smoke [82, 93, 96, 97].

The findings of this study further revealed that Alzheimer's disease patients who consume foods high in fat for three or more days per week are more likely to have lower levels of HDL and higher levels of LDL and total cholesterol compared to their counterparts who consume fat-containing foods for less than three days per week. A high intake of dietary fats stimulates excess cholesterol production in the liver, increasing circulating LDL levels while decreasing HDL efficiency. High-fat diets impair the reverse cholesterol transport mechanism, reducing HDL's ability to remove excess cholesterol from the bloodstream and leading to an accumulation of LDL. This study's outcomes are in agreement with findings from investigations performed in both the USA and China. The studies indicate that a high-fat dietary intake is associated with higher LDL levels and lower HDL levels [98, 99]. Which may contribute to the progression of Alzheimer's disease pathology.

8. Strength and limitation of the study

8.1. Strength of the study

- The findings of the current study strengthen the importance of lipid profile evaluation for patients with Parkinson's and Alzheimer's disease for better clinical and dietary management.

8.2. Limitation of the study

- The investigation was constrained by a cross-sectional study, unlike a longitudinal design, which did not show a well-established cause-and-effect relationship.

9. Conclusion and recommendation

9.1. Conclusion

This study highlights significant alterations in lipid profiles among patients with Parkinson's disease and Alzheimer's disease compared to healthy controls. Patients with Parkinson's disease exhibited significantly lower levels of LDL, TG, and TC, which may be attributed to neurodegenerative processes affecting lipid metabolism, including impaired cholesterol biosynthesis and increased metabolic demands. Additionally, lipid profile disturbances in Parkinson's disease were significantly associated with disease duration, age, and underweight status.

Conversely, Alzheimer's disease patients demonstrated significantly higher LDL levels and lower HDL levels than controls, which may contribute to amyloid-beta accumulation and disease progression. Factors such as disease duration, age, physical inactivity, smoking habits, and frequent consumption of high-fat foods were significantly associated with lipid profile alterations in Alzheimer's disease.

The study underscores the potential role of lipid metabolism in the pathophysiology of neurodegenerative diseases. Regular monitoring of lipid profiles, along with lifestyle modifications such as increased physical activity and dietary adjustments, may help mitigate disease progression. Further longitudinal studies are recommended to explore the causal relationship between lipid metabolism and neurodegeneration, as well as the potential benefits of lipid-modifying interventions in these patient populations.

9.2. Recommendation

- ❖ It is important to persistently monitor lipid profile tests in patients, as this enables early detection of potential problems and the development of strategies to mitigate further complications.
- ❖ To overcome the limitations of cross-sectional studies, it is recommended to conduct a comprehensive case-control study in order to investigate cause-and-effect relationships.
- ❖ Further pharmacological studies are needed to evaluate the effect of medical treatments on lipid profile test levels in AD and PD patients, which would support the provision of early and appropriate interventions for those receiving medication.

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ANNEX

Annex I: Participant's information sheet (English version)

Research Title: Determination of Lipid Profiles and Associated Factors Among Parkinson's and Alzheimer's Disease Patients Attending Tikur Anbessa Specialised Hospital, Addis Ababa, Ethiopia: 2024.

Principal Investigator: Abditsion Disani (BSc, MSc candidate)

Name of the Organization: Addis Ababa University, College of Health Sciences; Department of Medical Laboratory science.

Introduction: Hello! My name is Abditsion Disani, and I am an MSc student at Addis Ababa University, College of Health Science, Department of Medical Laboratory Science. I invite you to participate in research as a study subject. Please read or listen to the information sheet.

Purpose of the research: The purpose of this study is to assess lipid profiles and associated factors among Parkinson's and Alzheimer's disease patients.

Procedures and the expected participation: - After agreeing that you can take part, one or more of our research staff will ask you some questions, which will take up to 10 minutes. Your weight and height will be measured. And also, you will be asked to provide a blood sample, and we will collect 5 ml of venous blood from you by sterile, disposable vacutainer tube and needle. Then we will conduct laboratory analysis to determine lipid profiles.

Confidentiality: This study upholds privacy and confidentiality; any information that identifies you will not be shared with anyone else outside the study team.

Potential risks and discomfort: During a collection of specimens from you, appropriate precautions will be taken, and all samples will be collected by trained health professionals. If anything happened, appropriate medical care will be provided to you.

Potential benefits to subjects or to the society: You will not receive any payment for your participation in this research study as compensation. However, you will have the chance to know your lipid profile for free.

Right to refuse or withdraw: We like to freely inform you that the involvement of this study depends only on your voluntarism.

If you have additional questions about the study, you can contact:

Abditsion Disani: Phone: +251994294664

E-mail :dhiisaniitsichuu@gmail.com Mr. Abebe Edao +251913555319

abenegeso@gmail.com Mr. Gobena Dedefo

Gobenadedefo@gmail.com

Mrs. Mekdes Alem Mk.alem12@gmail.com

Thank you for your cooperation. If you are willing to participate in the study, I kindly request that you provide your response to the questionnaire on the next page.

Annex II: - Informed consent form English version

Card no/ID No_____

I had been informed that the objective of this study is to assess lipid profile tests among Alzheimer's and Parkinson's disease patients attending at Tikur Anbesa Specialized Hospital (TASH), Addis Ababa, Ethiopia. The results of this study are important to treat me and other patients and to be used as an input for diagnosing lipid alteration-induced neurodegenerative disease in Ethiopia. I had also been informed about the confidentiality of this study. The principal investigator requested that I participate in the study that would require my willingness to provide the required data that include blood and filling out a questionnaire. Therefore, with full understanding of the importance of the study, I agreed voluntarily to provide the requested samples, and my benefit will be only from the free laboratory investigation results.

I_____hereby give my consent for providing the requested information and specimens as the doctors find best for me.

Signature:_____Date_____

Annex III: Guardian consent form English version

Your parent has agreed that you take part in our study where we are assessing the level of lipid profiles among Alzheimer's and Parkinson's disease patients at TASH, Addis Ababa, Ethiopia. This study will help to improve health care services given to you and other neurodegenerative patients. Your participation in the study is fully voluntary. Any information you provided will not be given to anyone else.

If respondent agree to participate continue

Name of interviewer_____signature _____date_____

Annex IV: Information consent sheet in Amharic version

የተሳታፊዎች ፈቃድና መተማመኛ ቅፅ (Amharic version)

የጥናቱ ርዕስ: Determination of Lipid Profiles and Associated Factors Among Parkinson's and Alzheimer's Disease Patients Attending Tikur Anbessa Specialised Hospital, Addis Ababa, Ethiopia: 2024.

ዋና ተመራማሪ፡ አብዲጽዮን ዲሳኒ

የድርጅት ስም፡-አዲስ አበባ ዩኒቨርሲቲ፣ ሕክምና ላቦራቶሪ ሳይንስ ት/ክፍል

መግቢያ: ሀሎ! አብዲጽዮን ዲሳኒ እባላለሁ በአዲስ አበባ ዩኒቨርሲቲ የጤና ሳይንስ ኮሌጅ የሜዲካል ላቦራቶሪ ሳይንስ ትምህርት ክፍል የMSc ተማሪ ነኝ። ስለሆነም እርሰዎ በዚህ ጥናት ላይ እዲሳተፉ ተጋብዘዋል። እባክዎ በዚህ ጥናት ለመሳተፍ ከመስማማትዎ በፊት ከዚህ ቀጥሎ የሚገኘዉን ምንባብ በጥሞና ያንብቡና ግልጽ ያልሆነልዎትን ማንኛዉም ሃሳብ ይጠይቁ።

ጥናቱ ዓላማ፡ የዚህ ጥናት ዓላማ በፓርኪንሰን እና በአልዛይመርስ በሽታ ታማሚዎች መካከል ያለውን የሊፒድ መገለጫዎችን እና ተያያዥ ምክንያቶችን መገምገም ነው።

የጥናቱ ሂደቶች እና የሚጠበቀው ተሳትፎ፡መሳተፍ እንደሚችሉ ከተስማሙ በኋላ አንድ ወይም ከዚያ በላይ የኛ የጥናት ሰራተኛ አንዳንድ ጥያቄዎችን ይጠይቅዎታል ይህም እስከ 10 ደቂቃ ድረስ ይወስዳል። ክብደትዎ እና ቁመትዎ ይለካሉ. እና ደግሞ፣ የደም ናሙና እንዲያቀርቡ ይጠየቃሉ፣ እና 5 ሚ. ሊትር ደም ከርስዎ እንወስዳለን። ከዚያም የሊፕቲድ ፕሮፋይሎችን በላብራቶሪ እንሰራለን።

ሚስጢር ስለመጠበቅ: ይህ ጥናት የእርሶን ግላዊ ሚስጥር ይጠብቃል። እርስዎን የሚለይ ማንኛውም መረጃ ከአጥኚው ቡድን ውጭ ለሌላ ለማንም አይጋራም።

ጥናቱ የሚያስከትለው ችግርና አለመመቸት፡- የደም ናሙና በሚወሰድበት ግዜ አነስተኛ አለመመቸት ሊያስከትል ይችላል ነገር ግን ልምድ ያላቸው የላቦራቶሪ ቴክኖሎጂ ባለሙያዎች በማናቸውም ጉዳዮች ላይ አስፈላጊውን ጥንቃቄ ያደርጋሉ እና ተገቢውን የህክምና አገልግሎት ይደረግሎታል።።።

ለማህበረሰቡ ወይም ለግለሰብ የሚኖረው ጥቅም: በዚህ የምርምር ጥናት ላይ ለመሳተፍ እንደ ማካካሻ ምንም አይነት ክፍያ አያገኙም። ሆኖም ግን፣ የሊፒድ ፕሮፋይልዎን በነጻ የማወቅ እድል ይኖርዎታል።

እምቢ የማለት ወይም የመውጣት መብት፡ የዚህ ጥናት ተሳትፎ በእርስዎ ፍቃደኝነት ላይ ብቻ የተመሰረተ ነው።

ስለ ጥናቱ ተጨማሪ ጥያቄዎች ካሉዎት የሚከተለውን አድራሻ ይጠቀሙ፡-

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ለትብብርዎ እናመሰግናለን. በጥናቱ ለመሳተፍ ፈቃደኛ ከሆናችሁ በሚቀጥለው ገጽ ላይ ለመጠይቁ ምላሽ እንድትሰጡ በትህትና እጠይቃለሁ።

Annex V.
Informed consent form in Amharic version

የተሳታፊዎች ስምምነት ማረጋገጫ (የአማርኛ ቅጂ)

የካርድ ቁጥር / መታወቂያ ቁጥር _____

የዚህ ጥናት አላማ በጥቁር አንበሳ ስፔሻላይዝድ ሆስፒታል፣ አዲስ አበባ፣ ኢትዮጵያ ውስጥ በሚገኙ የአልዛይመር እና የፓርኪንሰን ህመም ህመማን ላይ የሚደረገውን የሊፒድ ፕሮፋይል ምርመራ ለመገምገም እንደሆነ ተነግሮኛል። የዚህ ጥናት ውጤት እኔን እና ሌሎች ታማሚዎችን ለማከም እና በኢትዮጵያ ውስጥ የሊፒድ ለውጥን የሚያመጣውን የነርቭ ዲጄኔሬቲቭ በሽታን ለመመርመር እንደ ግብአት ጥቅም ላይ ይውላል። የዚህ ጥናት ምስጢራዊነትም ተነግሮኛል። የጥናቱ ተመርማሪ በጥናቱ ላይ እንድሳተፍ ጠየቀኝ ይህም ደም እና መጠይቁን የሚያካትት አስፈላጊውን መረጃ ለማቅረብ ፈቃደኛ መሆንን ይጠይቃል። ስለዚህ የጥናቱን አስፈላጊነት ሙሉ በሙሉ በመረዳት የተጠየቁትን ናሙናዎች ለማቅረብ በፈቃደኝነት ተስማምቻለሁ። የዚህ ጥናት ተሳታፊ በመሆኔ የማገኘው ጥቅም የሁሉንም ምርመራ ውጤት በነጻ ማግኘት እንደሆነ ተረድቻለሁ።

በአጠቃላይ እኔ _____ከላይ በመተማመኛ ቅፅ የተጠቀሱትን ሁሉ በሚገባና በተረጋጋ መንፈስ አንብቤዋለሁኝ። ስለዚህ በዚህ ጥናት ለመሳተፍ ፈቃደኛ መሆኔን በፊርማዬ አረጋግጣለሁ።

ፊርማ: _____ቀን / /

Annex VI. የጠባቂ ስምምነት ማረጋገጫ (የአማርኛ ቅጂ)

በአዲስ አበባ፣ ኢትዮጵያ የአልዛይመር እና የፓርኪንሰን ህመም ታማሚዎች መካከል ያለውን የሊፕድ ፕሮፋይል ደረጃ በምንገመግምበት ጥናታችን ላይ እንድትሳተፉ ወላጅዎ ተስማምተዋል። ይህ ጥናት ለእርስዎ እና ለሌሎች የነርቭ ዲጄኔሬቲቭ ታካሚዎች የሚሰጠውን የጤና አጠባበቅ አገልግሎት ለማሻሻል ይረዳል። በጥናቱ ውስጥ ያለዎት ተሳትፎ ሙሉ በሙሉ በፈቃደኝነት ነው። ያቀረቡት ማንኛውም መረጃ ለሌላ ሰው አይሰጥም።

ምላሽ ሰጪው ለመሳተፍ ከተስማሙ ይቀጥሉ

የቃለ-መጠይቅ ጠያቂው ስም _____ፊርማ _____ቀን_____

Annex VII: Questionnaire in English Version

Addis Ababa University,

College of Health Science, Department of Medical Laboratory Sciences

Instruction: Please try to complete all information

PART-I Socio-demographic characteristics of the study participants

No	ID No _____ Date	
	Questions	Categories
1.	Age of respondent	
2.	Gender 1) Male 2) Female	
3.	Marital status	1) Single 2) Married 3) Divorced
4.	Religion	1) Orthodox 2) Muslim 3) Protestant 4) Others
5.	Occupation	1) Government employee 2) Private employee 3) House wife 4) Others
6.	Educational Status	1) No formal education 2) Primary school 3) Secondary school 4) Higher level
7.	Residence	1) Urban 2) Rural
8.	Income	1) Low income
		2) Middle income 3) High income

Part II: Behavioral measurements

9.	Do you smoke Cigarette?	1) Yes 2) No
10.	Do you take alcohol?	1) Yes 2) No
11.	Do you drink coffee?	1) Yes 2) No
Part III: Physical activity		
12.	Do you do any physical activities?	1) Yes 2) No
13.	If yes, the frequency of physical activity?	Days per week_____
Part IV: Nutrition status		
14.	Do you use fat contained foods like meat, egg and dairy products?	1) Yes 2) No
15.	If yes, how often do you take?	Day/s per week
16.	Duration of the diseases (year)	_____

Part V: Biochemical and anthropometric measurements

15.	Height (meter)	
16.	Weight (Kg)	
17.	BMI (Kg/m^2)	
18.	High density Lipoprotein (mg/dl)	
19.	Low density Lipoprotein (mg/dl)	
20.	Triglycerides (mg/dl)	
21.	Total Cholesterol (mg/dl)	

THANK YOU FOR YOUR PARTICIPATION!!!

Annex VIII. Questionnaire Amharic version

Addis Ababa University, College of Health Science, Department ofMedical Laboratory

Sciences

መመሪያ፡ እባክህ ሁሉንም መረጃዎች ይሙሉ ክፍል-I ማህበራዊ ስነ-ሕዝብ ባህሪያት

ተ. ቁ	የመለያ ቁጥር_____ቀን	
	ጥያቄዎች	የምላሽ ምድቦች
1.	የምላሽ ሰጪው ዕድሜ	
2.	ጾታ	1) ወንድ 2) ሴት
3.	የጋብቻ ሁኔታ	1) ያላገባ 2) ያገባ 3) የተፋታ
4.	ሃይማኖት	1) ኦርቶዶክስ 2) ሙስሊም 3) ፕሮቴስታንት 4) ሌላ
5.	የሥራ አይነት	1) የመንግስት ሰራተኛ 2) የግል ሰራተኛ 3) የቤት አመቤት 4) ሌላ
6.	የትምህርት ሁኔታ	1) መደበኛ ትምህርት የለም 2) የመጀመሪያ ደረጃ ትምህርት ቤት 3) የሁለተኛ ደረጃ ትምህርት ቤት 4) ከፍተኛ ደረጃ
7.	መኖሪያ	1) ከተማ 2) ገጠር
8.	የገቢ ሁኔታ	1) ዝቅተኛ ገቢ 2) መካከለኛ ገቢ 3) ከፍተኛ ገቢ

ክፍል II: የባህሪ መለኪያዎች		
9.	ሲጋራ ያጨሳሉ?	1) አዎ 2) አይ
10.	አልኮል ይጠጣሉ?	1) አዎ 2) አይ
11.	ቡና ይጠጣሉ?	1) አዎ 2) አይ
ክፍል III: አካላዊ እንቅስቃሴ በተመለከተ		
12.	ማንኛውንም የአካል ብቃት እንቅስቃሴዎችን ያደርጋሉ?	1) አዎ 2) አይ
13.	መልሶ አዎ ከሆነ፣ የሚያደርጉት የአካል ብቃት እንቅስቃሴ መጠን?	ቀናት/በሳምንት_____
ክፍል IV: የአመጋገብ ሁኔታ በተመለከተ		
14.	እንደስጋ፣ እንቁላል እና የወተት ተዋጽኦዎች ያሉ ስብ የያዙ ምግቦችን በምግብነት ይጠቀማሉ?	1) አዎ 2) አይ
15.	መልሶ አዎ ከሆነ፣ በሳምንት ለስንት ቀናት ይመገባሉ?	ቀናት/በሳምንት_____
16.	የበሽታው ቆይታ (ዓመት)	_____

ክፍል V:ባዮኬሚካል እና አንትሮፖሜትሪክ መለኪያዎች

15.	Height (meter)	
16.	Weight (Kg)	
17.	BMI (Kg/m^2)	
18.	High density Lipoprotein (mg/dl)	
19.	Low density Lipoprotein (mg/dl)	
20.	Triglycerides (mg/dl)	
21.	Total Cholesterol (mg/dl)	

ስለ ተሳትፎ እናመሰግናለን!!!

Annex IX: Principle of each test (Cobas c311)

Clinical chemistry analyzer: Cobasc311.

1. High Density Lipoprotein-Cholesterol (HDL)

Homogeneous enzymatic colorimetric test: Non-HDL lipoproteins such as LDL, VLDL and chylomicrons are combined with polyanions and a detergent forming a water-soluble complex. In this complex the enzymatic reaction of and cholesterol esterase towards non-HDL lipoproteins is blocked. Finally, only HDL-particles can react with cholesterol esterase (CHOR) and cholesterol oxidase (CHOD). The color intensity of this dye is directly proportional to the cholesterol concentration and is measured photometrically (700/600 nm).

HDL- cholesterol esters + $H_20 \xrightarrow{\text{Cholesterol esterase}}$ > HDL-cholesterol + RCOOH

HDL-cholesterol + $O_2 \xrightarrow{\text{Cholesterol Oxidase}}$ > Δ^4- cholestenone + H_2O_2

$2H_2O_2$ + 4-amino-antipyrine+ EMSE + H+ + H_2O $\xrightarrow{\text{Peroxidasease}}$ > colored pigment + 5 H_2O

2. Low Density Lipoprotein-Cholesterol (LDL)

Homogeneous enzymatic colorimetric assay: Cholesterol esters and free cholesterol in LDL are measured on the basis of a cholesterol enzymatic method using cholesterol esterase and cholesterol oxidase in the presence of surfactants which selectively solubilize only LDL. The enzyme reactions to the lipoproteins other than LDL are inhibited by surfactants and a sugar compound. The color intensity of this dye is directly proportional to the cholesterol concentration and is measured photometrically (700/600 nm).

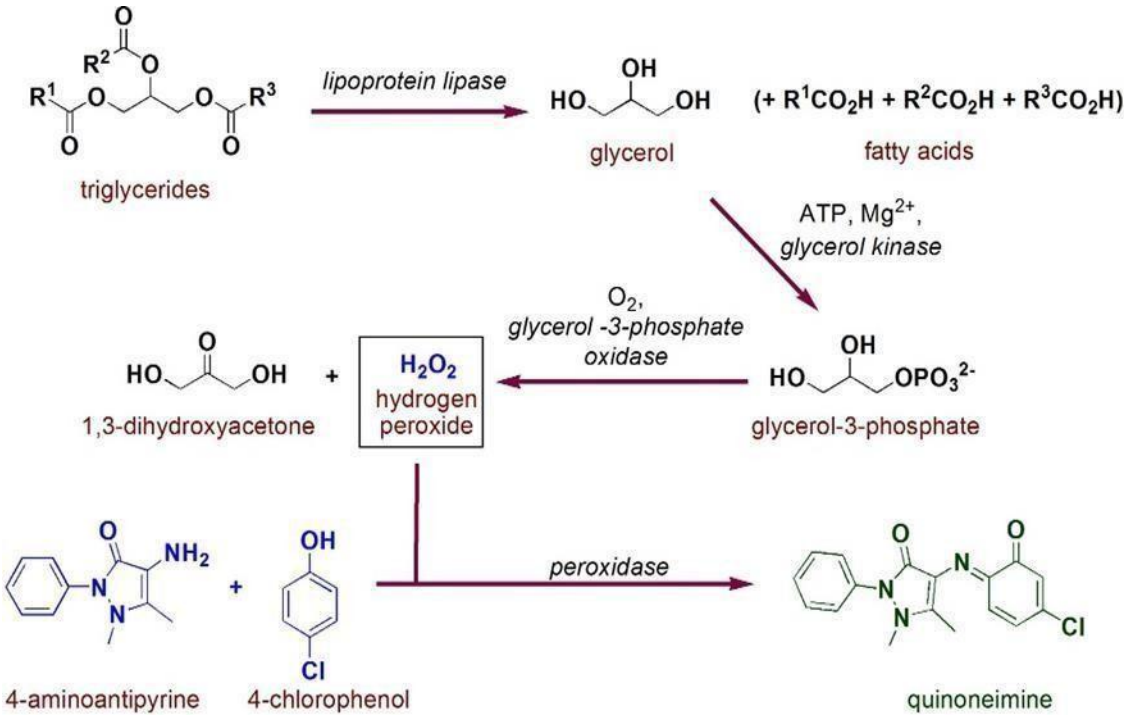
LDL-cholesterol esters + H_2O $\xrightarrow[\text{cholesterol esterase}]{\text{Detergent 1}}$ —> cholesterol + free fatty acids (selective micellary solubilization)

LDL-cholesterol + $O_2 \xrightarrow{\text{Cholesterol oxidase}}$ >Δ 4 -cholestenone + H_2O_2

$2H_2O_2$ + 4-aminoantipyrine + EMSE) + H_2O + H+ $\xrightarrow{\text{Peroxidase}}$ > red purple pigment + $5H_2O$

3. Triglyceride (TG)

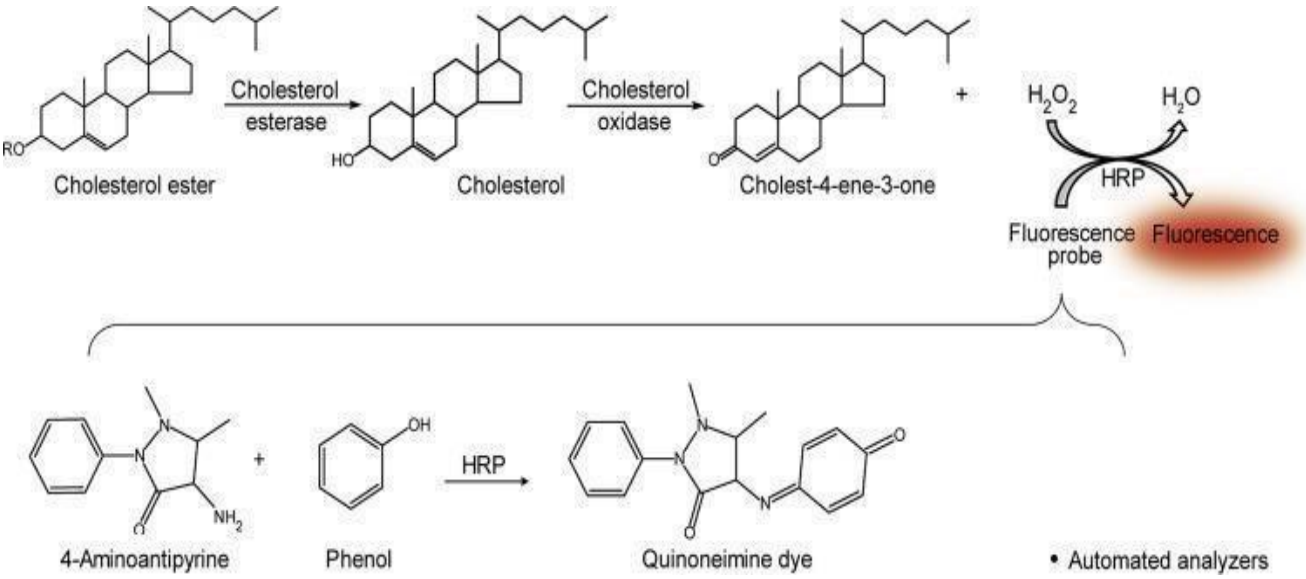
This method is based on the work by Wahlefeld using a lipoprotein lipase from microorganisms for the rapid and complete hydrolysis of triglycerides to glycerol followed by oxidation to dihydroxyacetone phosphate and hydrogen peroxide. The hydrogen peroxide produced then reacts with 4-aminophenazone and 4-chlorophenol under the catalytic action of peroxidase to form a red dye. The color intensity of the red dye formed is directly proportional to the triglyceride concentration and can be measured photometrically (700 / 505 nm).



4. Total cholesterol (TC)

Enzymatic colorimetric method: Cholesterol esters are cleaved by the action of cholesterol esterase to yield free cholesterol and fatty acids. Cholesterol oxidase then catalyzes the oxidation of cholesterol to cholest-4-en-3-one and hydrogen peroxide. In the presence of peroxidase, the hydrogen peroxide formed effects the oxidative coupling of phenol and 4-aminophenazone to form a red quinone-imine dye.

The color intensity of the dye formed is directly proportional to the cholesterol concentration. It is determined by measuring the increase in absorbance (700/505 nm).



Annex X: DECLARATION

I, the undersigned, declare that this M.Sc. thesis is my original work, has not been presented for a degree in this or any other university and that all sources of materials used for the thesis have been duly acknowledged.

M.Sc. candidate: Abditsion Disani Gudisa (BSc.)

Signature: _____

Date: _____

This thesis has been submitted with our approval as advisors. **Advisor: Abebe Edao Negesso (MSc, Assistant professor)**

Signature: _____

Date: _____

Place: Addis Ababa, Ethiopia.

Advisor: Gobena Dedefo Dekebo (MSc)

Signature: _____

Date: _____

Place: Addis Ababa, Ethiopia.

Advisor: Mekdes Alem (MSc)

Signature: _____

Date: _____

Place: Addis Ababa, Ethiopia.