

Prevalence of Hyperlipidaemia Among Adults During the Last Three Years at Murjan Hospital, Hilla City

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ABSTRACT

Background: Hyperlipidemia is marked by reduced high-density lipoprotein and elevated triglycerides, total cholesterol, and low-density lipoprotein cholesterol. It begins with endothelial damage, triggering inflammation and lipid accumulation in artery walls, leading to fibrous plaques that may rupture and cause thrombosis or arterial blockage, worsening cardiovascular disease. Management includes lifestyle changes and drugs such as statins, fibrates, niacin, PCSK9 inhibitors, and ezetimibe.

Aim: To examine the relationship between hyperlipidemia and age and gender, identify risk factors and health consequences, and provide preventive and therapeutic insights.

Materials and Methods: This retrospective study analyzed 17,660 adults (aged 20–70) with hyperlipidemia—7,064 females and 10,596 males—using laboratory records from Murjan Hospital in Hilla City (2022–2024). Data were assessed using descriptive statistics (frequencies and percentages).

Results: Cases were distributed as 33% (2022), 33% (2023), and 34% (2024). Lipid abnormalities were notably more frequent in older than younger age groups. Of the total sample, 7,064 (40%) were female and 10,596 (60%) were male.

Conclusion: Hyperlipidemia is linked to age and gender. Aging-related metabolic changes and reduced physical activity increase risk, while men show higher harmful lipid levels during early and mid-adulthood.

Keywords: Prevalence, Hyperlipidaemia, Adults.

1. Introduction

An abnormal blood lipid level is the hallmark of dyslipidemia. Hyperlipidemia (HL), the most common form of dyslipidemia, is represented by a decrease in high-density lipoprotein (HDL) levels and an increase in peripheral blood levels of triglycerides (TG), total cholesterol (TC), and low-density lipoprotein cholesterol (LDL-C) [1].

All mammalian tissues' cell membranes rely on cholesterol, which also serves as a precursor to bile acids and steroid hormones [2]. Of all the lipids, TG are the most prevalent. Adipocytes contain a large amount of it. The TG are esters of glycerol that contain three molecules of fatty acid [3].

Actually, a number of metabolic diseases, such as type 2 diabetes (T2DM), hypertension, fatty liver, and atherosclerosis, are associated with HL [4]. Hewage and Yao stated that certain ones of endothelial dysfunctions are brought on by persistent, prolonged HL, which is the key risk factor for atherosclerosis with cardiovascular consequences [5].

Elevated atherogenic indices, considerable serum dyslipidemia (LDL, low-density lipoprotein (VLDL), and TG), ischemia-modified albumin levels, and histological analysis of the heart tissues can all be used to identify HL [6].

An accumulation of fats, or molecules that are similar to fats, are present in the blood and are mentioned to as "lipids". Triacylglycerol (TAG), fatty acids, phospholipids, cholesterol, and cholesterol esters are the main lipids in blood [7]. Because lipids are insoluble in plasma, they are carried via lipoproteins. The HL classifications additionally rely on carrier lipoproteins due to their variability [8].

2. Pathophysiology of hyperlipidemia

The HL is started from endothelial damage to blood vessels, which results in the loss of nitric oxide in the damaged area. This causes an increase in the inflammatory response surrounding the affected area and buildup of lipids in the deepest portion of the endothelial wall. Macrophages then engulf the lipids, creating what is known as a foam cell with cholesterol content [9].

Necrosis, apoptosis, as well as mitochondrial dysfunction will result from the production of foam cells. Simultaneously, the smooth muscle cells wrap the fibrotic plaque-producing foam cells and prevent their destruction. At the other hand, tissue stimuli that stimulate platelet activity can cause thrombosis and plaque rupturing [10]. Plaque can develop quickly, obstructing blood vessels, or gradually, resulting in blood vessel stenosis. In both mechanisms, lipid plaque remains the mainstay of the development of cardiovascular disease (CVD) and deterioration of patient health status [11].

3. Hyperlipidemia classification

Kanakavalli et al. [12] state that there are two main categories of HL:

1. Primary: The predominant form, commonly referred to as familial HL because it arises from a genetic anomaly, can be either monogenic (a single gene flaw) or polygenic (several gene flaws) [13].
2. Secondary: Other illnesses like diabetes (DM), nephritic syndrome, hypothyroidism, chronic alcohol consumption, oral contraceptives, beta blocker use, in addition to corticosteroids might cause the second form, acquired HL. Severe hypertriglyceridemia and secondary hyperlipidemia are possible causes of pancreatitis [14].

4. Causes of hyperlipidemia

The primary causes of HL include altered lifestyle behaviors, fat intake over 40% of overall calories, saturated fats exceeding 10% of total calories, cholesterol exceeding 300 mg daily, or treatable medical conditions. Elevated cholesterol values are caused by a diet high in fat, as well as other bad lifestyle choices including smoking, getting overweight, consuming too much alcohol, and not properly getting enough exercise. Other influencing variables include pregnancy, DM, polycystic ovarian syndrome, renal disease, in addition to hypothyroidism [15].

It has been discovered that elevated doses of estrogen in addition to other female hormones can change or increase cholesterol levels. Development and spread of HL are significantly influenced by age as well as gender, according to earlier research. [16].

The CVD is a major cause of mortality worldwide. Through cellular synthesis, assembly, transit, plasma levels, oxidation, and degradation, lipids and lipoproteins have a major impact on the initiation and progression of CVD [17].

The Framingham Heart Study (FHS) found that blood cholesterol was the first direct correlation between circulating lipids and CVD. A higher 10-year risk of cardiovascular mortality is linked to elevated cholesterol readings [18]. By promoting the formation of thrombi and plaque, higher plasma concentrations of LDL, VLDL, and IDL contribute to CVD. According to the World Health Report (2002), elevated cholesterol values are responsible for around 56% of cardiac disease cases worldwide and nearly one-fifth (18%) of stroke events (mainly nonfatal). This represents 2.8% of the world's disease burden and around 4.4 million fatalities (7.9% of the overall) [19].

5. Diagnosis of hyperlipidemia

Only a blood test can identify hyperlipidemia, which usually has no symptoms. A blood test known as a lipid profile is used to check for hyperlipidemia. The National Cholesterol Education Program (NECP) states that screening [20] should begin at age 20 and be repeated at least every 5 years if the results are normal. The following is a list of typical lipid profile levels [21] (Table 1).

Table 1. Normal levels for a lipid profile [22]

Lipids	Desirable value	Borderline	High risk
Cholesterol	Less than 200 mg/dl	200-239 mg/dl	240 mg/dl
Triglyceride	Less than 140 mg/dl	150-199 mg/dl	200-499 mg/dl
HDL cholesterol	60 mg/dl	40-50 mg/dl	Less than 40 mg/dl
LDL cholesterol	60-130 mg/dl	130-159 mg/dl	160-189 mg/dl
Cholesterol/ HDL ratio	4.0	5.0	6.0

6. Treatment: Non-pharmacology therapy

The initial course of action for an increased lipid profile is still a lifestyle change, which includes changing one's diet, losing weight, abstaining from tobacco use or, if one is already smoking, starting to quit, and engaging in moderate-to-intense physical activity for thirty to sixty minutes every day [23].

7. Pharmacological therapy

Dietary regimens and lipid lowering medications are key components of the care of HL, which aims to prevent atherosclerosis and minimize a possibility of acute coronary episodes in individuals with previous history of coronary or peripheral vascular illness [24]. Harvey pointed out that there are five categories of medications indicated to deal with hyperlipidemia [25]:

6.1 The 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors (statins)

Atorvastatin, fluvastatin, lovastatin, and pravastatin are examples of statins. The rate-limiting step in the production of cholesterol, HMG-CoA reductase, is competitively inhibited by statins, resulting in the reduction of cell-level cholesterol [26]. HMG CoA and statins share structural similarities. The primary substance involved in the production of cholesterol is HMG CoA. This depletion causes the cell to create additional LDL receptors. The ultimate result is a drop in plasma cholesterol. Furthermore, statins prevent the release of VLDL into the blood and indirectly raise LDL activity [27].

Statins have been shown to lower the risk of cardiac disease, though this benefit appears to be primarily driven by a lower risk of acute coronary syndrome [28]. Nonetheless, some research indicates that statin treatment is associated with a lower risk of stroke and coronary heart disease in those with low to moderate cardiovascular risk [29]. By reducing platelet aggregation, lowering LDL cholesterol, and lowering C-reactive protein, using statin is predicted to reduce the risk of CVD. All people with T2DM, high LDL cholesterol, and additional CVD risk factors who are deemed to be at moderate risk with respect to age and medical history are advised to take statins as an outcome of these findings [30].

6.2 Fibrates

Fibrates include clofibrate, gemfibrozil, and fenofibrate. Fibrates employed to treat HL are TG-lowering drugs that reduce TG-rich lipoproteins and increase the development of the lipoprotein lipase and the level of apolipoprotein CII, which reduces the TAG level within the blood [31]. In one study, fibrates significantly reduced the risk of CVD when taken with a statin medication when compared to a statin by itself [32].

6.3 Niacin Derivatives

For a number of years, HL has been managed with niacin, it is called as vitamin B3, a water-soluble vitamin. Substances that share structural similarities with niacin and have comparable effects on lipid metabolism are recognized as niacin derivatives. Among these substances are nicotinamide, nicotinic acid, and inositol hexanicotinate [33]. Nicotinic acid is the niacin derivative that is frequently employed for managing HL. It works by blocking the action of lipoprotein lipase, which increases the formation of high-density lipoprotein HDL and decreases the formation of VLDL [34].

6.4 Bile Acid Sequestrants (BAS)

Bile acid sequestrants (BAS) are large, positively charged, non-absorbable polymers that are recommended medications to deal with HL. As a result of binding to bile acids in the colon and blocking their reabsorption, they lower the amount of cholesterol in the blood [35]. A resin called cholestyramine attaches itself to bile acids in the colon to stop them from being reabsorbed into blood. It is available for oral use as a powder or granule that can be mixed up with liquids like water [36].

Cholestyramine has been shown to reduce TG levels by up to 40%, LDL cholesterol by up to 30%, and TC by up to 20%. Common unwanted side effects include nausea, bloating, abdominal pain, and constipation [37]. An additional resin that attaches itself to bile acids in the intestine and stops them from being reabsorbed into the blood is colestipol [38]. Colesevelam is a synthetic polymer that stops bile acids from being reabsorbed into blood by binding them in the intestine [39].

6.5 Cholesterol absorption inhibitors (Ezetimibe)

One class of medications intended to deal with high cholesterol, or HL, is called an inhibitor of cholesterol absorption. These medications reduce the quantity of cholesterol that gets the bloodstream by preventing the intestines from absorbing dietary cholesterol. Ezetimibe is a good example of a cholesterol absorption inhibitor [40]. A synthetic drug called ezetimibe stops the small intestine from absorbing cholesterol from food [41].

8. Aim of the study

To comprehend the connection between age as well as hyperlipidemia, investigate gender variations in hyperlipidemia, identify risk variables and health consequences, and offer preventative and therapeutic insights.

9. Materials and methods

This study comprised 17,660 cases of hyperlipidemia in people between the ages of 20 and 70 (7,064 females and 10,596 males). The laboratory records of Murjan Hospital in Hilla City were used to retrospectively gather the patient data. Retrospective metadata from routine hyperlipidemia monitoring data reported between 2022 and the completion of 2024 were used to statistically assess the study's findings. Annually, patient data was gathered and examined using descriptive statistical techniques, such as frequencies and percentages.

10. Result

Information from 2022 to the end of 2024. Every year, patient data was gathered, and the percentages for 2022, 2023, and 2024 are 33%, 33%, and 34%, respectively.

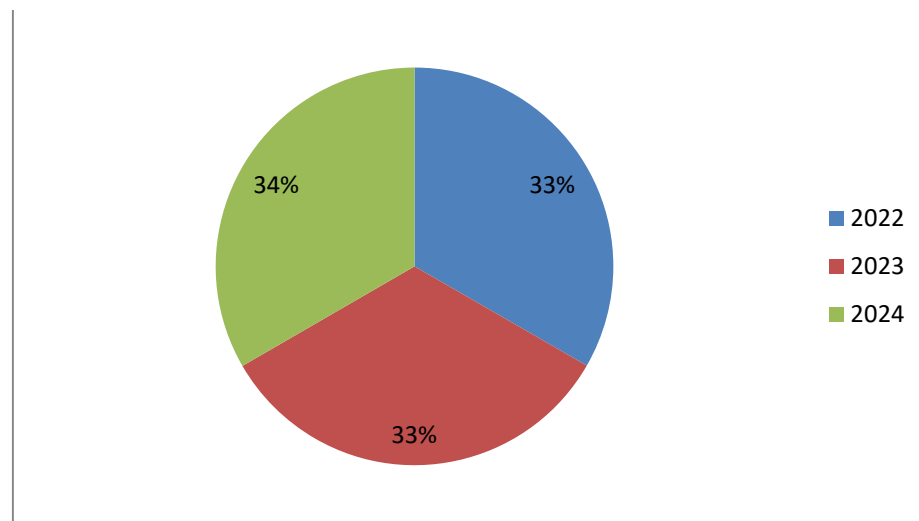


Figure1. Distribution of study sample by years of prevalence

When comparing the older age group to the younger age group, the percentage of any lipid abnormalities was significantly higher.

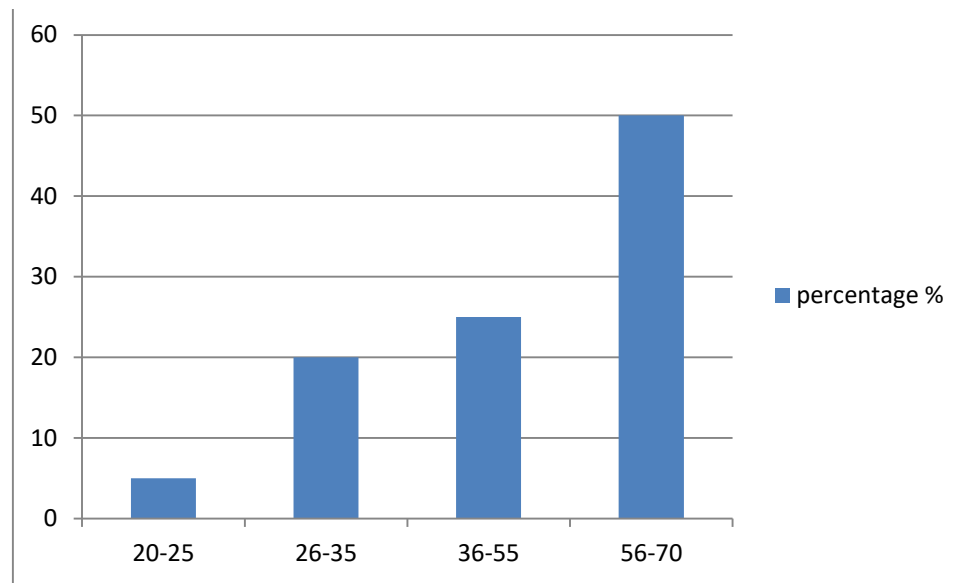


Figure2. Distribution of study sample by their age

A total of 17,660 people were included in the study; their ages ranged from 20 to 70 years, with 7,064 (40%) being female and 10,596 (60%) being male.

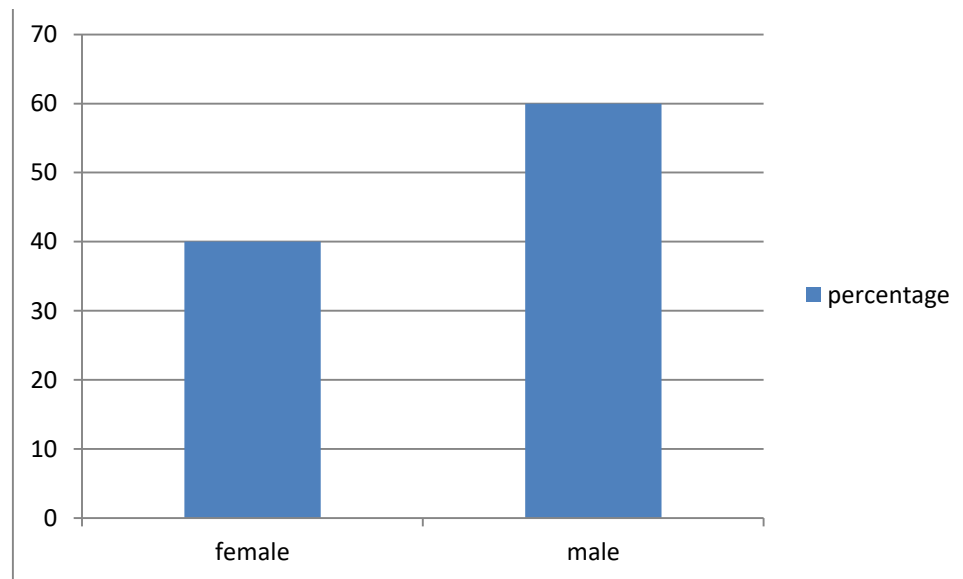


Figure 3. Distribution of study sample by their Gender

11. Discussion

The evaluation of dyslipidemia in Hilla has advanced with the current study. It offers a unique chance to examine the majority of all blood lipid profile indicators within a certain age range (20 to 70 years of age). Over the past three years, the frequency of hyperlipidemia seems to have approached a very low occurrence in 2024 [42].

The results of the current investigation made it abundantly evident that the prevalence of hyperlipidemia appears to be greater in men than in women. The primary reason for the greater prevalence rate in the male population is that there were more male patients with hyperlipidemia than female patients.

Males have a greater prevalence rate due to metabolic alterations and sedentary lifestyles, which have been shown to grow with age. These findings demonstrate that metabolic abnormalities, which are typically linked to a lower quality of life, are far too prevalent in male populations. [43].

Numerous studies have indicated lower incidence rates in females, which can be attributed to their additional preventive mechanisms during the premenopausal stage. [44]. Whilst the incidence rate of hyperlipidemia and its related consequences were found to be about equal, the prevalence rate increased along with age.

According to the ATP I guideline [45], women in the study populations were more likely to enter into high-risk groups for all lipoprotein classes. This is owing to postmenopausal women had nearly equal or higher frequency than premenopausal women [46].

The frequency of patients with hyperlipidemia was shown to rise with age according to the age category. This indicates that the consequences linked to hyperlipidemia are directly correlated with age. Furthermore, the high-risk ratio of CVDs in younger age groups is evident from the greater prevalence rate of hyperlipidemia in younger individuals. Age was found to be extremely significant in the current investigation and is typically considered a non-changeable risk indicator in the progression of hyperlipidemia associated with CVDs. The characteristics of coronary arteries and myocardial contractility decrease with age; hence, the accumulation of lipids in the blood in the coronary arteries will contribute to myocardial atherosclerosis and ischemia [46].

12. Conclusion

High blood lipid levels, including cholesterol and triglycerides, are the hallmark of hyperlipidemia, a prevalent ailment that is a major risk factor for cardiovascular diseases and other illnesses. With respect to earlier research and current analyzes, this study examines the connection between hyperlipidemia and demographic characteristics including age and gender.

The results show that aging-related metabolism alterations and decreased physical activity raise the chance of hyperlipidemia. Contrary to women, men often have more levels of dangerous lipids in their earliest and mid adult years. However, hormonal fluctuations during menopause, especially the drop in estrogen levels, greatly increase the potential of hyperlipidemia in women.

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