INTERNATIONAL JOURNAL OF PROGRESSIVE RESEARCH IN ENGINEERING MANAGEMENT AND SCIENCE (IJPREMS)

e-ISSN : 2583-1062

Impact Factor : 5.725

www.ijprems.com
editor@ijprems.com

Vol. 03, Issue 09, September 2023, pp : 362-378

PREVALENCE OF DISLIPIDEMIA AMONG SUBJECTS PRESENTED FOR LIPID PROFILE ANALYSIS AT BENAZIR BHUTTO HOSPITAL OVER 7 YEARS (2015-2022)

Farheen Shahid[1]

[1]BDNS, 5th Year, Department of Dietetics and Nutritional sciences , The University Of Lahore, Islamabad campus, Pakistan.

ABSTRACT

Background- Dyslipidemia is a prominent cause of morbidity and mortality worldwide and it is one of the modifiable risk factors for cardiovascular disease. The purpose of this study was to determine the prevalence of dyslipidemia.

Methods- A total of 9989 participants of every age were enrolled in the study. Enzymatic colorimetric techniques were used to examine the serum levels of total cholesterol and triglycerides. Dyslipidemia was defined based on serum lipids levels following the standard guidelines by National Cholesterol Education Program Adult Treatment Panel III.

Results - Data of 9989 patients presented with signs and symptoms of dyslipidemia referred to Benazir Bhutto Hospital Rawalpindi for the Lipid profile assessment in the previous 7 years (2015-2022), were retrieved from HMIS. Of the adult subjects, 4283(44 .7%) were males and 5305 (55.3%) were females while 401(4%) were subjects under 19 years of age. Females have significantly high concentrations of cholesterol median (min-max) 203.25(48-1157) vs 196.95(2-924).Mann Whitney U p value= 0.0001.No significant difference was observed for the concentrations of triglycerides and distribution of age in both genders. A highly significant difference was observed in concentrations of cholesterol and triglycerides in different age groups.Kruskal Wallise H 145.09 p-value 0.0001,Kruskal Wallise H 17151.09 p-value 0.0001,respectively.

Conclusion- Dyslipidemia is very common.These findings imply that measures to increase public awareness are necessary to prevent and manage dyslipidemia in people.

Keywords: Dyslipidemia, Cardiovascular diseases,Serum lipid level, cholesterol level, concentration of triglycerides.

1. INTRODUCTION

A lipid profile is a combination of blood tests to check the levels of lipids in the blood, such as cholesterol, HDL, LDL, and triglycerides[1]. Lipids are composed of fats and oils, yield high energy, and are responsible for different functions within the human body[2].

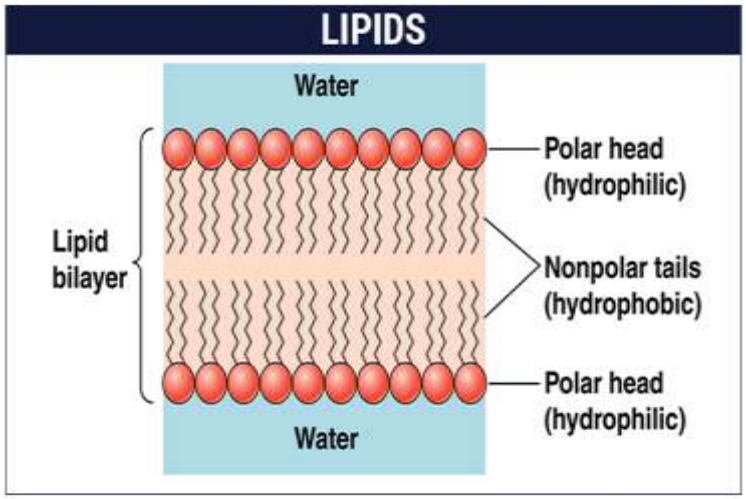

Figure 1.1: Structure of Lipid

Main Plasma lipids are cholesterol, triglycerides, phospholipids, and free fatty acids. Lipids are insoluble in water and therefore are transported in plasma in combination with proteins as lipoproteins. All lipoproteins consist of protein fraction (apolipoprotein) and varying amounts of triglycerides, cholesterol, and phospholipids.

1.1 Classification of Lipoproteins:

Based on density, lipoproteins are reclassified as:

i. Chylomicrons: The major lipid in chylomicrons is the triglycerides and the chylomicrons transport triglycerides from the gut to tissues and liver[3]. Triglycerides are a type of fat found in food that a person consumes. Blood triglyceride levels that are too high are linked to pancreatic inflammation and cardiovascular disease.

INTERNATIONAL JOURNAL OF PROGRESSIVE RESEARCH IN ENGINEERING MANAGEMENT AND SCIENCE (IJPREMS)

e-ISSN : 2583-1062

Impact Factor : 5.725

www.ijprems.com
editor@ijprems.com

Vol. 03, Issue 09, September 2023, pp : 362-378

ii.VLDL(Very low density lipoprotein):

The major lipid in VLDL is triglycerides[4].VLDL transports triglycerides from the liver to the tissues. It is typically only found in minimal concentrations in fasting blood samples since it is derived chiefly from recently consumed meals. This form of cholesterol may indicate faulty lipid metabolism if it increases in a fasting sample

iii. IDL: (Intermediate density lipoprotein)

The major lipid in IDL is cholesterol ester, and it transports it to the liver and tissues[5].

iv. LDL: (Low-density lipoprotein)

The major lipid in LDL is cholesterol ester, which transports cholesterol to tissues. Low-density lipoprotein cholesterol is the "bad cholesterol" subtype of cholesterol. A person's risk of developing the cardiovascular disease may increase if it builds up in the blood vessels.

v. HDL:(High density lipoprotein)

It mainly consists of cholesterol esters and transports cholesterol from the tissues to the liver. The so-called "good cholesterol," or high-density lipoprotein (HDL), aids in reducing LDL buildup in the blood vessels.

About 60% of plasma cholesterol is present in LDL,25% in HDL, and a small quantity in VLDL. In a fasting state, the central part of plasma triglycerides is present in VLDL since chylomicrons are absent from plasma.

Exogenous(dietary)lipids are carried in chylomicrons to the tissues and liver[7]. Endogenous lipids from the liver are incorporated in VLDL, which is metabolized to LDL through IDL.HDL removes cholesterol from the tissues to the liver; hence, it has a beneficial effect. Certain enzymes modify lipoproteins, and remnants are taken up by receptors on the cell, mainly in the liver. The metabolism of lipoproteins is controlled by their protein components Apo-lipoproteins. Apo A1 in HDL and Apo B-100 in LDL fraction are important[8].Lipoprotein(a) is also present in human plasma. It is not derived from previously mentioned lipoproteins but is synthesized in the liver. It is similar in size but denser than LDL. It has cholesterol ester as a significant lipid and is an independent risk factor for ischemic heart disease. High serum levels of LDL and possibly VLDL are associated with premature atherosclerosis and an increased risk of ischemic heart disease[9]. High serum levels of HDL are a negative risk factor and low levels are associated with an increased risk of ischemic heart disease.

Hyperlipidemia means a high level of lipids in the blood. It may be primary or secondary to other diseases like diabetes mellitus, hypothyroidism, nephrotic syndrome, etc .[10]. The nature of a lipoprotein abnormality can usually be determined by plasma cholesterol and triglyceride concentrations, as shown in the table below[11]. In primary hyperlipidemia, it may be necessary to define the lipoprotein abnormality for treatment. Different genetic defects may result in similar lipoprotein abnormalities.

Table 1.1: Different Lipoprotein Abnormalities

Type	Electrophoretic pattern	Lipoprotein Increased	Lipids Increased
I	Increased Chylomicrons	Chylomicrons	Triglycerides
IIa	Increased β-lipoproteins	LDL	Cholesterol
IIb	Increased pre- β and βlipoproteins	VLDL and IDL	Cholesterol and Triglycerides
III	'Broad β' band	IDL	Cholesterol and Triglycerides
IV	Increased pre-β lipoprotein	VLDL	Triglycerides
V	Increased pre-β lipoprotein and chylomicrons	VLDL and Chylomicrons	Cholesterol and Triglycerides

An array of blood tests known as a lipid panel or lipid profile detect abnormalities in lipids, including cholesterol and triglycerides.

The lipid profile typically includes the following:

- Low-density lipoprotein (LDL)
- High-density lipoprotein (HDL)
- Triglycerides
- Total Cholesterol

INTERNATIONAL JOURNAL OF PROGRESSIVE RESEARCH IN ENGINEERING MANAGEMENT AND SCIENCE (IJPREMS)

e-ISSN : 2583-1062

Impact Factor : 5.725

www.ijprems.com
editor@ijprems.com

Vol. 03, Issue 09, September 2023, pp : 362-378

By using the values of the above parameters, lab technologists can also calculate the following:

- Very low-density lipoprotein
- Cholesterol: HDL (Ratio)

So, The lipid profile tests are of 7 types:

- Total lipids
- Serum total cholesterol
- serum HDL cholesterol
- Total cholesterol/HDL cholesterol ratio
- Serum triglycerides
- Serum Phospholipids
- Electrophoretic fractionation to determine the percentage of
- Chylomicrons
- LDL
- VLDL
- HDL

This test's results can be used to identify specific hereditary diseases and estimate the risks of developing cardiovascular disease, some types of pancreatitis, and other disorders. Lipid panels are typically ordered in combination with other panels, such as the complete blood count (CBC) and basic metabolic panel(BMP), as part of a physical examination[12].

Following are the typical ranges of the lipid components present in the blood of an adult. The unit of measured Cholesterol levels is mg/dL.

Table 1.2: Typical ranges of Lipid components in Adults by National Cholesterol Education Program Adult Treatment Panel

Cholesterol Component	Normal	Borderline	Unhealthy
Total serum cholesterol	< 200mg/dL	200-239 mg/Dl	>240 mg/Dl
HDL	> 60 mg/dL	50-60 mg/Dl	<40 mg/Dl
LDL	<100 mg/dL	130-159 mg/Dl	160-189 mg/Dl
Triglycerides	< 150 mg/dL	150-199 mg/Dl	200-499 mg/Dl
VLDL	5-40mg/dL	40- 49 mg/Dl	>50 mg/Dl
Total cholesterol: HDL	4:1 (optimal)	4:1 – 5:1	>5:1

The normal ranges of lipid components observed in children are as follows:

Table 1.3: Typical ranges of Lipid components in Children National Cholesterol Education Program Treatment Panel

Cholesterol type	Acceptable	Borderline	Unhealthy
Total cholesterol	< 170 mg/dL	170-199 mg/dL	>200 mg/Dl
HDL	>45 mg/dL	~35-45 mg/dL	< 35mg/Dl
LDL	<100 mg/dL	100-129 mg/dL	>130 mg/Dl
Triglycerides	<150 mg/dL	150-199 mg/dL	>200 mg/Dl

INTERNATIONAL JOURNAL OF PROGRESSIVE RESEARCH IN ENGINEERING MANAGEMENT AND SCIENCE (IJPREMS)

e-ISSN : 2583-1062

Impact Factor : 5.725

www.ijprems.com
editor@ijprems.com

Vol. 03, Issue 09, September 2023, pp : 362-378

Disturbances in cholesterol and triglycerides are referred to as dyslipidemia. Dyslipidemia is a risk factor for many diseases, such as cardiovascular disease and pancreatitis[13]. As it's the era of modernization, most of the work done by hand in the past has been replaced with machines. And due to the modern lifestyle, people's attitudes towards physical activity and exercise are almost negligible, and they are getting obese daily. And obesity is the leading cause of dyslipidemia. There is a significant difference in total cholesterol levels between people who do physical exercise and who do not do any physical activity[16]. As males exercise more than females, high-density lipoprotein cholesterol levels were higher in males than in females. Obesity and being overweight are the risk factors for hypercholesterolemia and hypertriglyceridemia[17]. Hypolipidemia develops in increasing frequency with severe COVID-19 disease[18]. It inversely correlates with levels of acute-phase reactants, indicating SARS-COV-2 as the causative agent for lipid and thyroid levels alteration. Higher blood lipids are linked to smoking and alcohol consumption[19]. While a diet high in vegetables is associated with lower blood lipids and glucose, physical exercise is associated with lower blood lipids. It has been seen that various disturbances in cholesterol and triglyceride levels cause many diseases, including subclinical hypothyroidism, cardiovascular diseases, liver diseases, obesity, psychiatric disorders, and pancreatitis. Lipid profile screening is also helpful in detecting genetic disorders such as familial hypercholesterolemia, which can be lethal if not treated early[20].

1.2 Risks associated with High levels of lipids :

Lipids are essential for our health. However, consuming an excessive amount of them can increase the risk of developing diseases, including liver disease and heart disease. Complications of hyperlipidemia include coronary heart disease, acute coronary syndrome, heart attack, stroke, peripheral arterial diseases, diabetes, and high blood pressure. LDL cholesterol accumulation can narrow the arteries (atherosclerosis). These "clogs" can make the artery's opening smaller and cause a heart attack or stroke. High LDL levels are associated with diets high in saturated fats, which include deep-fried foods, processed foods, cheese, fatty or processed meats, cream-based sauces, and cheese.[21] The leading cause of death in the US is cardiovascular disease[22]. According to estimates from the World Health Organization (WHO), 17.9 million individuals worldwide pass away from cardiovascular disease each year.

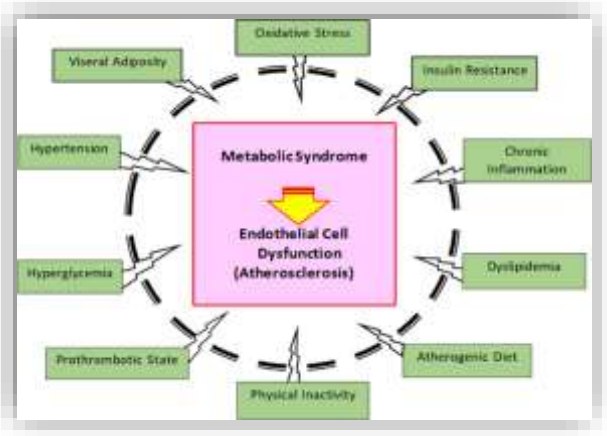

Figure 1.2: Diseases associated with Dyslipidemia

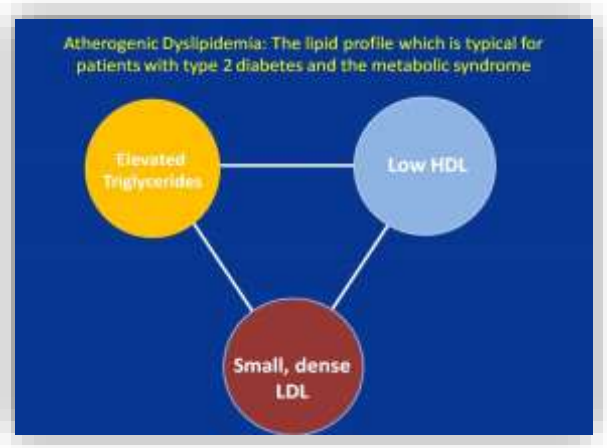

Figure1.3: Atherogenic Dyslipidemia

INTERNATIONAL JOURNAL OF PROGRESSIVE RESEARCH IN ENGINEERING MANAGEMENT AND SCIENCE (IJPREMS)

e-ISSN : 2583-1062

Impact Factor : 5.725

www.ijprems.com
editor@ijprems.com

Vol. 03, Issue 09, September 2023, pp : 362-378

1.3 Prevalence of high cholesterol in Pakistan :

Dyslipidemia is the critical factor contributing to the increased prevalence of CHD in South Asians[23]. In Pakistan, high cholesterol is a serious lifestyle illness. It is a sign of several deadly conditions[24]. According to the most recent figures, one person in Pakistan dies from a heart attack every 33 seconds[25]. It has been noted that the probability of dying from a heart attack (in young demographics) rises by 200% for every 40 points higher cholesterol levels[26]. Currently, 72% of Pakistanis have low levels of good cholesterol, and over 75% have dangerous cholesterol levels[27].

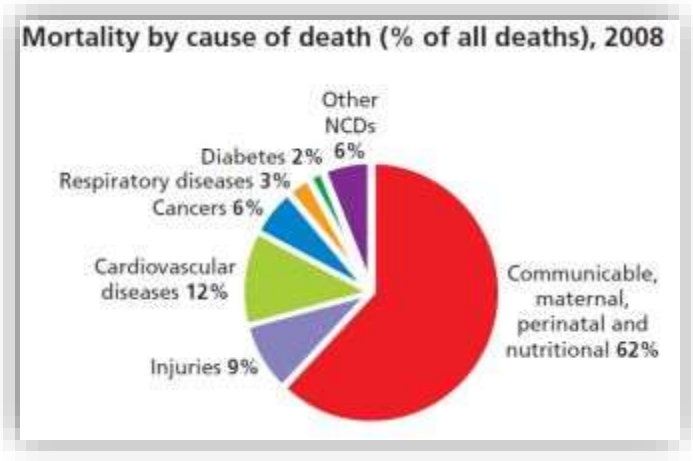

Figure 1.4: Mortality by cause of death

2. LITERATURE REVIEW

Lipids are organic compounds that contain hydrogen, carbon, and oxygen atoms, which form the framework for the structure and function of living cells. All living cells are made up of lipids, which are important for numerous bodily processes. Lipids aid in hormone regulation, nerve impulse transmission, organ cushioning, and the storage of energy in the form of body fat.

The three main types of lipids are Phospholipids, sterols (which include the many types of cholesterol), and triglycerides (which make up more than 95% of the lipids in food)[28].

A lipid profile is a combination of blood tests performed to check the cholesterol levels and the level of triglycerides in the blood.This is done to access different diseases. The study of lipid profiles and their implications in adults is an interesting area that requires to be explored in recent times. Hyperlipidemia can be associated with cardiovascular diseases, nephrotic syndrome, obesity, and hypertension. Awareness is the first step in lifestyle modification.

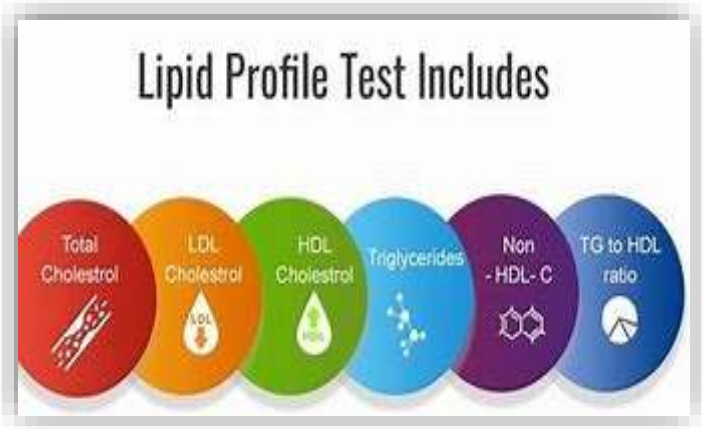

Figure2.1: Components of Lipid Profile Test

2.1 Hyperlipidemia:

Hyperlipidemia is a very big health issue worldwide. The prevalence of hyperlipidemia is increasing in developed as well as developing countries. Hyperlipidemia incidence and prevalence is increasing in Pakistan due to change in the lifestyle of the Pakistani people. Hyperlipidemia is a major risk factor for cardiovascular disorders. Cardiovascular disorders are the leading cause of death in Pakistan. Hyperlipidemia has high mortality and morbidly because it is the major risk factor for developing stroke and cardiovascular disorders.

INTERNATIONAL JOURNAL OF PROGRESSIVE RESEARCH IN ENGINEERING MANAGEMENT AND SCIENCE (IJPREMS)

e-ISSN : 2583-1062

Impact Factor : 5.725

www.ijprems.com
editor@ijprems.com

Vol. 03, Issue 09, September 2023, pp : 362-378

2.2 Dyslipidemias and cardiovascular disorders:

Blood lipid levels are modifiable risk factors for atherosclerosis and CHD. It has been observed that many lipid/lipoprotein abnormalities are prevalent in obesity and heart problems, collectively termed as dyslipidemia, however, these dyslipidemias are often hyperlipidemia wherein a majority of lipids are shifted towards the upper limits of range or higher than the range. Owing to the recent modernization of the lifestyle and availability of transportation means combined with a unique ethnicity have resulted in the high prevalence of metabolic disorders in Pakistan like the rest of the world. This not only affects daily activities, work performance, and social interactions but also poses a huge healthcare burden. Dyslipidemia is the principal cause of the excess burden of CAD in South Asians, which is characterized by increased levels of apolipoprotein (apo) B, TG, Lipoprotein Lp(a) and LDL-C, and low levels of HDL-C and apoA1[29]. The liberal use of saturated and trans fats in daily cooking especially curry-based cuisines and extensive deep frying along with lack of physical activity is one the profound features of Pakistani culture, thus leading to raised lipid levels.

Previous studies in our population have provided diverse results; Jafar et al. reported high TG levels in 34.5% .Kayani et al. documented 10% and Khan et al. found 16% of all study subjects had raised cholesterol levels. In 2011, a study in Lancet reported the mean TG levels in Asians were not the highest but they were continuously on the rise owing to the epidemiological transition towards urbanization[30].

2.3: Dyslipidemias and diabetes mellitus:

Dyslipidemia is one of the major risk factors for cardiovascular disease in diabetes mellitus. The characteristic features of diabetic dyslipidemia are a high plasma triglyceride concentration, low HDL cholesterol concentration, and increased concentration of small dense LDL-cholesterol particles. The lipid changes associated with diabetes mellitus are attributed to increased free fatty acid flux secondary to insulin resistance.

Lipid abnormalities constitute the risk factor for cardiovascular diseases. The global prevalence of various metabolic disorders, including dyslipidemia, is continuously rising at an alarming rate. Different prevalence patterns of lipid abnormalities have been reported in various countries. Uncontrolled hyperglycemic conditions also contribute to the alteration in lipid metabolism, thus posing a great risk for developing cardiovascular events. Dyslipidemia (hypertriglyceridemia being at its highest—61.9%) was observed to be higher among the Arabs with a poorly controlled glycemic status similar findings were observed in the Pakistani population.About 63% of the local subjects had shown lipid derangements. Among the varying lipid parameters, low HDL-C was observed to be frequent (17.3%). The high frequency of varying lipid parameters presents an independent risk factor for cardiovascular pathologies.Various epidemiological studies have shown a strong association between lipid variations with cardiovascular disease risk. Hypercholesterolemia and other lipid derangements have been established as significant risk factors for cardiac events and stroke. According to WHO reports, this outcome can cause 2.6 million deaths per year globally.

Several factors can cause lipid derangements, including diabetes (T2DM), hypertension, obesity, and family history. Dietary patterns, lifestyle, and various metabolic disorders can also constitute the onset of dyslipidemia.Insulin resistance has an impact on lipid metabolism modification. Generally, clinicians consider dyslipidemia as a late-onset complication of diabetes.A local study showed the presence of disturbed lipid profiles in newly diagnosed as well as known diabetics. Dyslipidemia alone and in diabetes is a risk predictor for the progression of CVDs. The growing number of individuals with deranged lipids is a matter of health concern. The earlier screening of lipid abnormalities in populations with and without hyperglycemic status might serve as an effective health management strategy. This can be achieved with the timely identification of lipid variations and the effective execution of lifestyle modification.

3. AIMS & OBJECTIVES

- The present study aims to evaluate the serum levels of Cholesterol and Triglycerides over the past 7 years in subjects presented to the Chemical pathology lab at Benazir Bhutto Hospital, Rawalpindi referred.
- To determine the trends of dyslipidemia among all age groups.

4. MATERIALS AND METHODS

4.1 STUDY DESIGN:

Retrospective study

4.2 STUDY PERIOD:

03 months after the approval of the Research Proposal

4.3 SAMPLING TECHNIQUE:

Consecutive Non-Probability Sampling

4.4 SAMPLE SIZE:

A total of 9989 subjects were included in the study.

4.5 SETTING:

Clinical Chemistry lab, Benazir Bhutto Hospital, Rawalpindi.

4.6 SAMPLE COLLECTION AND EXAMINATION:

Lipid Profile data for the year 2015-2022 was retrieved from the Hospital management information system, Benazir Bhutto Hospital

4.7 INCLUSION CRITERIA:

All patients who were referred for lipid profile analysis.

METHODOLOGY:

4.8 PREPARATIONS NEEDED FOR LIPID PROFILE TEST:

1. Requires Fasting
2. Fasting samples needs to be collected after a minimum 12-14 hour overnight fasting status.
3. Clear fluids like water can be consumed during this period. Do not consume beverages like tea, coffee, and milk in the morning until specimen collection is completed.
4. In case of diabetics on oral or injectable hypoglycemic agents, please consult your physician about continuing with these medications before specimen collection."
5. You will be required to take several precautions before the test is performed. You should:-

- Not eat high-fat foods the night before
- Not drink alcohol, and
- Not exercise strenuously before the test

6. A doctor might ask you to fast before the test, which means that you cannot eat or drink anything except water for 9 to 12 hours before the test.
7. It is recommended to take your medicines on the morning of the test, with water. While fasting is not always necessary, it might be recommended. Doctor can inform patient about any additional precautions that will be needed to take before the test. Make sure to take history about:

- Any health symptoms or problems of the patient
- Family history of heart health
- Medications and supplements of patient

4.9 MATERIALS REQUIRED FOR SAMPLING:

- Gloves
- Mask
- Tourniquet
- Antiseptic Solution
- 5ml disposable syringe
- Gel and Clot Activator Tube
- Cotton

4.10 BLOOD SAMPLING FOR LIPID PROFILE:

About 2 ml venous blood sample is taken in a gel tube after 8 -12 hours of fasting and allowed to clot.

4.11 SAMPLE TRANSPORTATION:

The sample was transported immediately to the lab without any further delay. In case of delay, serum was stored at -20°C.

4.12 SAMPLE PROCESSING:

4.12.1 ESTIMATION OF TOTAL CHOLESTEROL

An enzymatic endpoint method (CHOD-PAP) was used to estimate cholesterol.

Principle:

Cholesterol esters are hydrolyzed by cholinesterase esterase into cholesterol and fatty acids. Cholesterol is oxidized by cholesterol oxidase into cholestenone and H2O2. H202 in the presence of peroxidase reacts with phenol and 4-aminophenazone to form a red-colored substance(quinone). It was measured at 505 nm.

Specimens:

Serum or plasma stable at 2-8℃ for 7 days.

Reagents:

- Kits for the enzymatic determination of total cholesterol contain the following;
- Buffer and surfactant
- Enzymes
- The working reagent is obtained after reconstituting above mentioned and contains phosphate buffer, phenol,4 amino antipyrine, cholesterol oxidase, cholesterol esterase, and peroxidase.

Procedure:

1. Bring all the reagents, samples, and controls to room temperature.

2. Add 1.0 ml of working reagent to the tubes marked as 'test', 'standard and 'blank'.

3. Add 10 ul of serum to the tube marked 'test',10 ul of cholesterol standard to the 'standard' tube, and 10 ul of water to the 'blank' tube.

4. Mix and incubate for 5 minutes at 37℃ in a water bath.

5. Read the absorbance of the test and standard against the reagent blank at 505nm, after setting the instrument to zero with the blank.

6. Total cholesterol in mg/dl =Absorbance of test / Absorbance of standard *Concentration of standard.

4.12.2 HDL - CHOLESTEROL ESTIMATION

Routinely it is measured by the enzymatic method. It can also be measured by ultracentrifugation and electrophoresis.

Principle:

The chylomicrons, VLDL and LDL contained in the sample are precipitated by phosphotungstic acid in the presence of divalent cations (Mg++). The supernatant obtained after centrifugation contains HDL-Cholesterol, and it is measured by the enzymatic method.

Specimen:

Serum or plasma is stable for 7 days at room temperature.

Reagents:

The commercial kit contains:

- The precipitant solution consists of phosphotungstic acid and magnesium chloride.
- GOD-PAP reagent consisting of 4-aminophenazone, glucose oxidase, and peroxidase.
- Standard solution – 50mg/dl

Procedure:

1. Take 1 ml of precipitant solution in a centrifuge tube.

2. Add 0.5 ml of the serum, mix, and allow to stand for ten minutes at room temperature.

3. Then centrifuge for 10 min at 4000 rpm.

4. Label the tubes as 'test', 'standard', and 'blank' and pipette 1 ml of prepared working reagent in all the tubes.

5. Add 0.1ml of supernatant into the labeled 'test'.

6. Add 0.1ml of water and standard solution to the tubes labeled 'blank' and 'standard' respectively.

7. Mix and incubate for 5 minutes in a water bath at 37℃.

8. Read the absorbance of the 'test' and the 'standard' against the reagent 'blank' at 500nm or 546nm.

9.HDL-C (mg/dl) = Absorbance of test / Concentration of standard * Absorbance of standard

4.12.3 NON-HDL CHOLESTEROL ESTIMATION

By subtracting HDL Cholesterol from the total cholesterol, we get the value of Non-HDL Cholesterol.

4.12.4 LDL CHOLESTEROL ESTIMATION

Routinely it is measured by enzymatic and calculation methods. But it can also be measured by ultracentrifugation and electrophoresis.

Principle:

LDL cholesterol is precipitated. The supernatant is removed by centrifugation and the precipitate is re-suspended. The LDL cholesterol is measured by an enzymatic method.

Specimen:

Serum or plasma stable for 7 days at room temperature.

Calculation:

The establishment of a formula in 1972 by Friedwald has led to the use of a calculated LDL cholesterol value. The formula is based on the assumption that VLDL is only a carrier of TG and that the ratio of triglycerides/cholesterol is constant (2.2 / 1).

LDL-Chol = (Total Chol) - ((HDL-Chol) + (TG / 2.2))

4.12.5 ESTIMATION OF TRIGLYCERIDES

In routine practice, enzymatic endpoint methods(GPO-PAP) are used to estimate TG.

Principle:

Triglycerides are hydrolyzed by lipases to release glycerol and fatty acids. Glycerol is converted by glycerol kinase to glycerol phosphate. Glycerol Phosphate is oxidized by glycerol phosphate oxidase to dihydroxyacetone phosphate. The hydrogen peroxide released during this reaction is exposed to phenol and 4-aminophenazone in the presence of peroxidase. A colored compound (quinone) is formed which is measured at 505nm.

Specimens:

Serum or plasma is stable in the sample for 7 days at 2-8℃.

Reagents:

The commercial reagent kit contains:

- Buffer: Pipes buffer pH 7.5 $ 4-chlorophenol.
- Enzyme reagents: 4 aminophenazone, ATP, Lipases, glycerol kinase, glycerol 3 phosphate oxidase, peroxidase.
- Standard: 2.29 mmol/L (200 mg/dl)
- The enzyme reagent bottle is reconstituted with buffer to form a working reagent.

Procedure:

1. Bring all the reagents, samples, and controls to room temperature.

2. Add 1.0ml of working reagent to the tubes marked as ‘test’, ‘standard’, and ‘blank’.

3. Add 10 ul of the serum to the tube marked ‘test’, 10 ul of the standard to the ‘standard’ tube, and 10 ul of water to the ‘blank’ tube.

5. Mix and incubate for 5 minutes at 37℃ in a water bath.

6. Read the absorbance of the ‘test’ and ‘standard’ against the reagent ‘blank’ at 505nm, after setting the instrument to zero with the blank.

7.Total triglyceridesin mg/dl= Absorbance of test / Absorbance of standard * Concentration of Standard.

Reference Range:

<1.6mmol / L.

Conversion Factor:

Conventional unit (mg/dl)*factor (0.0114) = SI unit (mmol / L).

SI Unit (mmol/L) * factor (88.57) = Conventional unit (mg / dl)

4.13 DATA ANALYSIS :

Research protocols were reviewed and approved by Ethical Review Board at Rawalpindi Medical University. The personal information of the subjects was kept confidential.

4.13.1 STATISTICAL ANALYSIS:

The data were tested for normality by the Shapiro-Wilk test. Patients were stratified by gender and age for analysis. Continuous variables were presented as mean ± Standard deviation (SD) and median interquartile range (IQR). Categorical variables were expressed as frequency (percentages). A two-tailed p-value of less than 0.05 was considered statistically significant. All analyses were performed with SPSS Statistics, version 22.0 (IBM SPSS Inc., Chicago, IL) and \Graph Pad Prism 7.

i.Descriptive Data Analysis:

A. Tables (Frequencies, Percentages)

B. Statistical Figures (Histogram, Scatterplot)

C.Statistical mean and standard deviation

INTERNATIONAL JOURNAL OF PROGRESSIVE RESEARCH IN ENGINEERING MANAGEMENT AND SCIENCE (IJPREMS)

e-ISSN : 2583-1062

Impact Factor : 5.725

www.ijprems.com
editor@ijprems.com

Vol. 03, Issue 09, September 2023, pp : 362-378

ii. Inferential Data Analysis:

This approach is used to test the statistical hypothesis and Spearman correlative – factor for measuring the association between the quantitative variables.

5. RESULTS

A total of 9989 participants (4500 males and 5489 females) from the previous 7 years (2015-2022) were included in this study. All subjects were tested for lipid profile at Benazir Bhutto Hospital. Frequencies and percentages of baseline characteristics are shown in table 5.1.

Table 5.1: Frequencies and percentages of Baseline Characteristics

Characteristic	Groups	Frequency (Total = 9989)	Percentage
Age	0-13	207	2.1
	14-26	1044	10.5
	27-36	2335	23.4
	40-52	3529	35.3
	53-65	2168	21.7
	66-79	583	5.8
	80-100	123	1.2
Gender	Male	4500	45.0
	Female	5489	55.0
Before Covid	Normal	3214	53.2
	Borderline	1646	27.25
	High risk	1179	19.52
After Covid	Normal	1739	44.0
	Borderline	1119	28.3
	High risk	1092	27.6
Triglycerides	Normal	4011	40.2
	Borderline	2145	21.5
	Unhealthy	3833	38.4
Cholesterol	Normal	4953	49.5
	Borderline	2765	27.6
	Unhealthy	2270	22.7

The following tables show the mean values of triglyceride and cholesterol in study participants.

Table 5.2: Mean value Of Cholesterol among different age groups

Age groups	Mean Cholesterol	χ^2 value, p-value
0-13	227.05	124.233 0.0001
14-26	194.31	
27-39	208.06	
40-52	209.66	
53-65	206.58	
66-79	195.26	
80-100	188.32	

INTERNATIONAL JOURNAL OF PROGRESSIVE RESEARCH IN ENGINEERING MANAGEMENT AND SCIENCE (IJPREMS)

e-ISSN : 2583-1062

Impact Factor : 5.725

www.ijprems.com
editor@ijprems.com

Vol. 03, Issue 09, September 2023, pp : 362-378

Table 5.3: Mean value Of Triglyceride among different age groups

Age	Mean Triglycerides	χ^2 value, p-value
0-13	234.7784	117.13 0.0001
14-26	175.3168	
27-36	207.7346	
40-52	212.0707	
53-65	206.9516	
66-79	193.8499	
80-100	180.2960	

Average cholesterol levels in men and women were found to be 203.1169 and 208.8669 mg/dl, and average triglyceride levels in men and women were 206.7403 and 203.7927, respectively. Regarding triglyceride levels, 40.2 % were normal,21.5% were borderline, and 38.4% were unhealthy,while cholesterol levels, 49.6 % were normal,27.7% were borderline, and 22.7% were unhealthy.

5.1 Age Wise Distribution(n=9989):

Patients were divided into seven groups according to their age.2.1% (n=207)of patients were 1-13 years in age,10.5%(n=1044) of patients were 14-26 years in age, 23.4% (n=2335) patients were 27-39 years in age,35.3%(n=3529) patients were 40-52 years in age, 21.7%(n=2168) patients were 53-65 years in age, 5.8%(n=583) patients were 66-79 years in age and 1.2%(n=123) patients were 80-100 years in age. Dislipidemia is mostly seen in 40-52 years of patients.

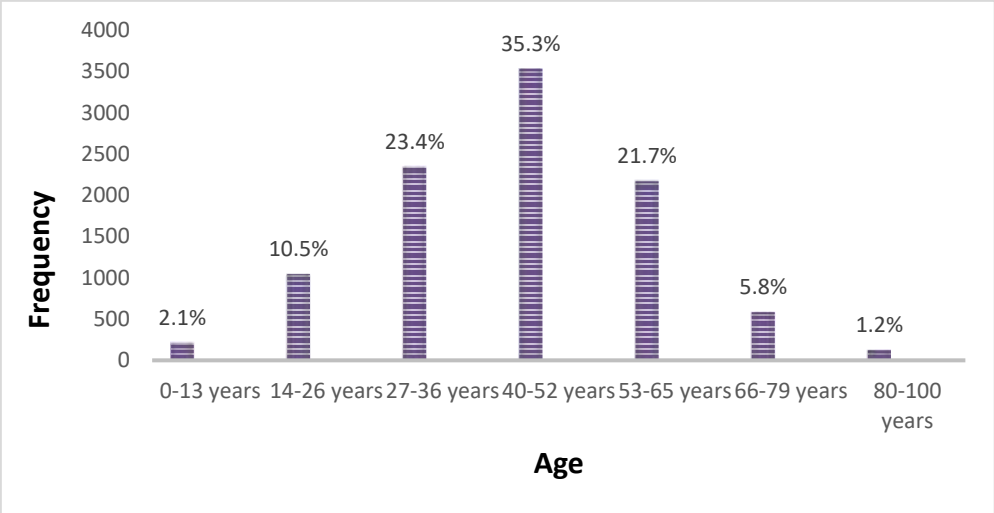

Fig.5.1 Bar chart showing the Age Wise distribution of patients presented for lipid profile

5.2: Gender-Wise Distribution:

This graph shows the gender-wise distribution of the Lipid profile of patients who were prescribed at Benazir Bhutto Hospital from 2015-2022.

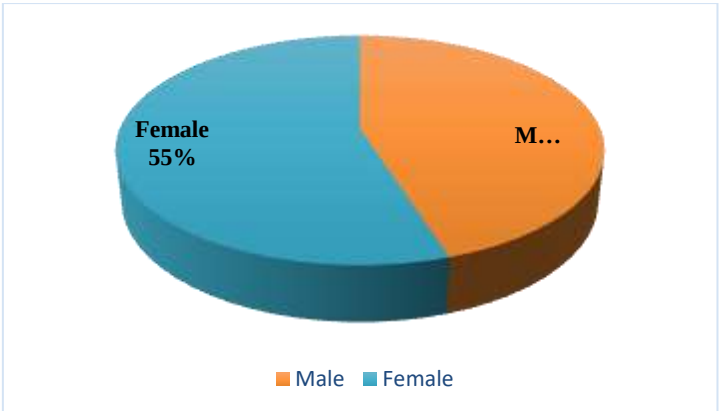

Fig.5.2 Pie chart showing the Gender Wise distribution of patients presented for lipid profile

INTERNATIONAL JOURNAL OF PROGRESSIVE RESEARCH IN ENGINEERING MANAGEMENT AND SCIENCE (IJPREMS)

e-ISSN : 2583-1062

Impact Factor : 5.725

www.ijprems.com
editor@ijprems.com

Vol. 03, Issue 09, September 2023, pp : 362-378

5.3 Year-wise Distribution:

A graph showing the frequency of patients every year is shown below. The lipid profile of patients was also stratified into two groups; before covid and after covid and we came to know that dyslipidemia is more common in individuals after Covid.

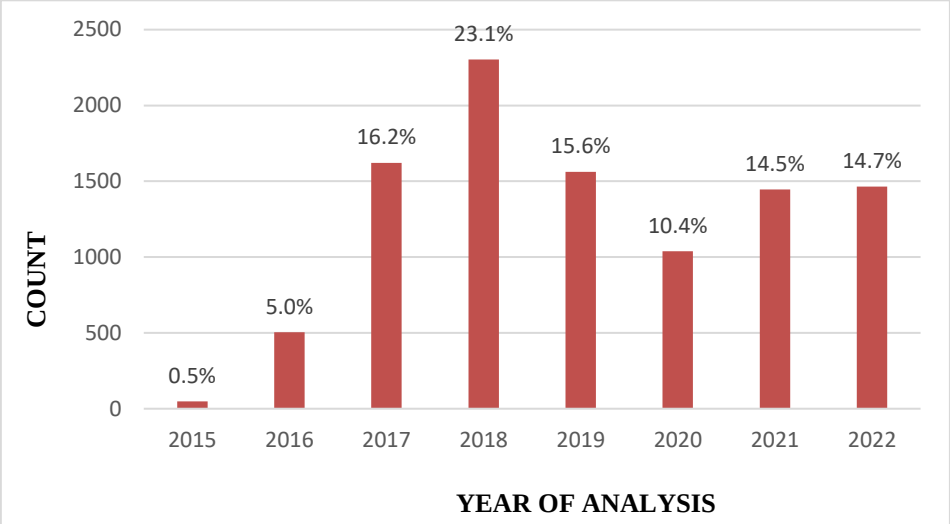

Fig. 5.3 Bar chart showing the Year Wise distribution of patients presented for lipid profile

Table 5.4: Dunnett t-test Dunnet T-test to compare TG and cholesterol conc. in different years

Multiple Comparisons

Dependent Variable	(I) Year_of_analysis	Std. Error	Sig.	95% Confidence Interval	
				Lower Bound	Upper Bound
Cholesterol	2015	9.14209	.636	-36.7196	11.5196
	2016	3.21851	.092	-16.2333	.7495
	2017	2.24641	.000	-16.2044	-4.3510
	2018	2.08284	.000	-20.1283	-9.1380
	2019	2.26680	.009	-13.2411	-1.2801
	2020	2.52788	.234	-1.6254	11.7132
	2021	2.31038	.995	-4.8468	7.3442
Triglycerides	2015	20.41448	.993	-42.1203	65.5989
	2016	7.18699	.979	-13.9120	24.0109
	2017	5.01629	1.000	-13.2712	13.1977
	2018	4.65102	.820	-17.4539	7.0877
	2019	5.06180	.033	-27.4672	-.7581
	2020	5.64480	.785	-21.4957	8.2897
	2021	5.15913	.717	-20.1557	7.0670

The mean difference is significant at the 0.05 level.

Dunnett t-tests treat one group(2022) as a control and compare all other groups against it.

INTERNATIONAL JOURNAL OF PROGRESSIVE RESEARCH IN ENGINEERING MANAGEMENT AND SCIENCE (IJPREMS)

e-ISSN : 2583-1062

Impact Factor : 5.725

www.ijprems.com
editor@ijprems.com

Vol. 03, Issue 09, September 2023, pp : 362-378

Table 5.5 Mann Whitney U Test to compare Age, TG and cholesterol in both genders

Ranks

	Gender	N	Mean Rank	Sum of Ranks
Cholesterol	Male	4500	4778.80	21504619.00
	female	5488	5171.36	28380447.00
	Total	9988		
Triglycerides	Male	4500	4983.52	22425850.50
	female	5488	5003.50	27459215.50
	Total	9988		
Age	Male	4500	4971.54	22371937.00
	female	5488	5013.33	27513129.00
	Total	9988		

Test Statistics

	Cholesterol	Triglycerides	Age
Mann-Whitney U	11377369.000	12298600.500	12244687.000
Wilcoxon W	21504619.000	22425850.500	22371937.000
Z	-6.770	-.345	-.721
Asymp. Sig. (2-tailed)	.000	.730	.471
a. Grouping Variable: Gender			

5.4 Frequency and Percentage Distribution of Cholesterol and Triglyceride Level:

Following is the Line chart showing the frequency and percentage distribution of patients presented for lipid profile.

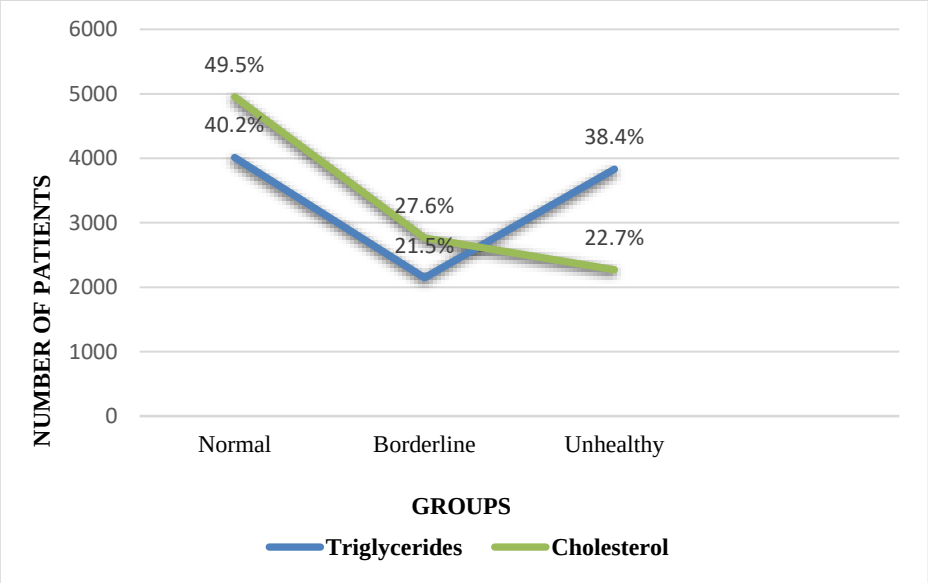

Fig.5.4 Line Chart showing the Group Wise distribution of Cholesterol and Triglyceride Levels

5.5. HYPOTHESIS TESTING RESULTS

The distribution of Triglycerides is the same across categories of Gender".

According to the results evaluated from the Independent Samples of the Kruskal-Wallis Test, the developed hypothesis has been approved. The result shows both genders have the same distribution of triglycerides. The sig. value by the Kruskal-Wallis test is .861**. It demonstrates that hypertriglyceridemia or hypotriglyceridemia does not depend upon gender. Thus, the hypothesis is accepted.

INTERNATIONAL JOURNAL OF PROGRESSIVE RESEARCH IN ENGINEERING MANAGEMENT AND SCIENCE (IJPREMS)

e-ISSN : 2583-1062

Impact Factor : 5.725

www.ijprems.com
editor@ijprems.com

Vol. 03, Issue 09, September 2023, pp : 362-378

The distribution of Cholesterol is the same across categories of Gender".

According to the results evaluated from Independent Samples Kruskal-Wallis Test, the developed hypothesis has been rejected. The result shows the different distribution of cholesterol among both genders. The sig. value by the Kruskal-Wallis test is .000**. It demonstrates that hypercholesterolemia or hypocholesterolemia depends upon gender due to different lifestyles. Thus, the hypothesis is rejected.

T-Test Results:

By applying the T-test, we came to know about the mean value of cholesterol and triglycerides in both genders and the mean age of both genders.

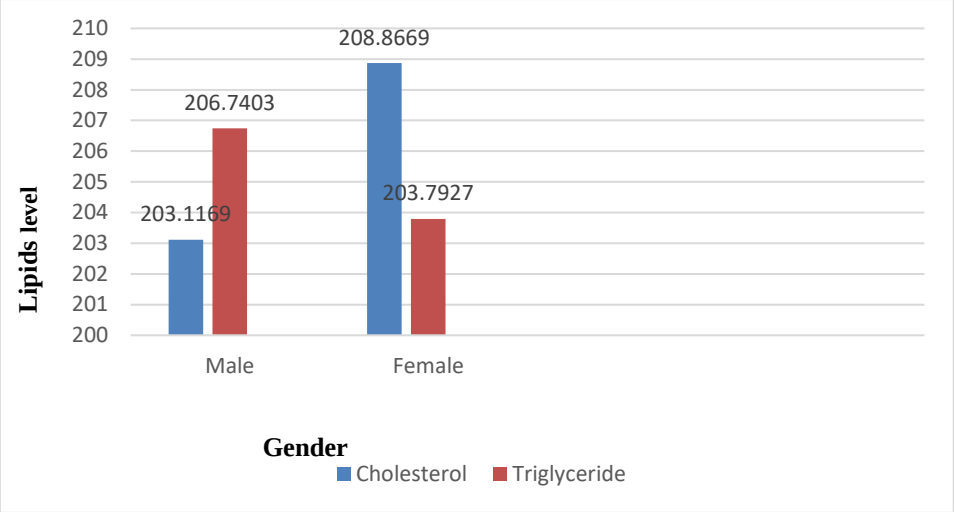

Fig5.5 Bar Chart showing the mean values of Cholesterol and Triglycerides in both genders

6. CONCLUSION, LIMITATIONS, AND RECOMMENDATIONS

6.1 CONCLUSION:

Dyslipidemia, a major risk factor for many systemic diseases, should be detected early in life so that effective management is carried out before the start of actual manifestations. This study gave a high prevalence of dyslipidemia in asymptomatic adults. Furthermore, lipid-lowering and anti-diabetic drugs should be affordable to patients to avoid sudden complications.

6.2 LIMITATIONS:

1. This study was conducted only in a single Benazir Bhutto Hospital in Rawalpindi. There is a need to conduct this study all across Pakistan.
2. Potential risk factors of dyslipidemia were not taken into consideration.

6.3 RECOMMENDATIONS:

This study shows that dyslipidemia should not be neglected as its consequences can be severe. Future Public Health strategies involving education and food fortification programs with diet to reduce appreciable levels of lipids in Pakistan should also involve all key stakeholder groups working together. This includes government personnel, physicians involved with Medical Colleges, clinical pharmacologists, and relevant patient organizations working urgently to address the current rates of Dyslipidemia in Pakistan. Several Western countries have lipid food fortification policies serving as an example. Consequently, Pakistan should follow this lead and include other strategies in its forthcoming National Health Plan to reduce the extent of Dyslipidemia and its consequences on morbidity and mortality. Secondly, A national program on the yearly Lipid assessment is urgently needed. This builds on patient concerns regarding developing chronic heart diseases and obesity, etc. Dyslipidemia has become a major public health concern, and its prevalence is rising at a steady pace, particularly in developing countries[31]. The increasing prevalence of dyslipidemia is associated with several factors, many of which are modifiable and dependent on socioeconomic, cultural, and ethnic characteristics [32]. Changes in lifestyle and diet contribute to a significant portion of risk factors associated with dyslipidemia. Risk factors for dyslipidemia are lack of physical activity, obesity, particularly central obesity, metabolic syndrome, hypertension, old age, and a diet rich in saturated fats and cholesterol[33]. Pharmacological intervention can effectively control dyslipidemia but, more importantly, through dietary and lifestyle modification[34].Unfortunately, a lack of awareness and appropriate therapeutic intervention and management are emerging as barriers to preventing complications related to dyslipidemia. Dyslipidemia is a major pathogenic risk factor for atherosclerotic cardiovascular disease (CVD). Various cardiovascular incidents reported are all associated with uncontrolled dyslipidemia[35]. Surveys

conducted in China showed that the incidence of ischemic CVD is significantly reduced by controlling lipid profile among individuals, decreasing the mortality and morbidity associated with it[36]. The asymptomatic nature of dyslipidemia is also a concerning issue. Hence, early screening and preventive measures are equally important in controlling it[37]. A National Health and Nutrition Examination Survey was conducted in the United States (U.S.) from 2003 through 2006. The proportion of the U.S. population with abnormalities in the lipid profile was assessed. In this study, it was stated by Toth et al. that about 53 percent of U.S. adults have at least one lipid abnormality[38]. A similar survey in China reported that approximately 41.9 percent of the Chinese population was found to have dyslipidemia [39]. These studies reflected the threat dyslipidemia poses to the population's general health. Our study showed that approximately 50% of the population had dyslipidemia, which is significantly greater than the prevalence described in the above countries. The prevalence of dyslipidemia increases with age; however, it is essential to note that it also affects younger adults [40]. Moreover, the process of atherosclerosis starts early in life. A compilation of various observations made by the Bogalusa Heart Study established an association between coronary atherosclerosis and cardiovascular risk factors in young people, among which serum LDL and serum triglyceride concentration were significant factors, along with many others[41]. Therefore, young adults with abnormal levels of lipids are at an increased risk of developing cardiovascular complications, such as coronary heart disease, later in life. Considering the lack of literature on dyslipidemias and its importance in the early detection of cardiovascular risks, we have highlighted the significance of identifying dyslipidemia in adults. Our study showed that the prevalence of dyslipidemia is higher in females than males. Our result is coherent with a cross-sectional survey conducted in Chongqing, China. In this study, the prevalence of dyslipidemia in women was 55% as compared to 45% in men. This was primarily due to differences in the frequency of overweight, obesity, and central obesity[42].In a study conducted in Iran, the incidence of dyslipidemia was 37.4% for males and 55.4% for females and, therefore, higher in females, which is consistent with the result of our study[43]. However, a vast amount of literature provides evidence that the prevalence of dyslipidemia is much higher in men than women. This was shown by a cross-sectional study by Pan et al. in which the incidence of dyslipidemia was higher in males than in females(41.92% vs. 32.47%).[44]Likewise in 2002, the Chinese national nutrition and health survey concluded the prevalence of dyslipidemia in Chinese adults as 22.2% and 15.9% in males and females respectively[45]. Moreover, a systemic review on dyslipidemia in Chinese adults published between 2003 and 2013 stated that dyslipidemia was more common in men than women.27 Prior research has established that abnormal lipid profile is more prevalent in men than women.[46] This could be due to a higher frequency of cigarette smoking and more consumption of alcohol and high-cholesterol food in men.[47]The prevalence of isolated hypercholesterolemia and isolated hypertriglyceridemia in the total population of our study was 47.12% and 59.9%, respectively. Pan et al.'s study showed that this prevalence was 2.9% and 11.9%, respectively.19 Extensive surveys in China organized from 2002 to 2010 revealed that low HDL-C and hypertriglyceridemia were the two major types of dyslipidemia in Chinese adults.[48]These, findings were reinforced in a meta-analysis by Huang et al.[49] However, these trends are different in western countries where high cholesterol and high LDL-C were more common forms of dyslipidemia.[50] This was largely due to a high dietary fat and cholesterol intake in American residents. Furthermore, according to our study, isolated hypertriglyceridemia was higher in males than females. The results are consistent with the Korean National Health and Nutrition Survey. The large difference in HDL-C across six countries was secondary to smoking, alcohol consumption, age, fat intake, BMI, and educational level. High levels of estrogen in women partly explain the gender difference in HDL-C. This explains the lower mortality rate due to cardiovascular diseases in the female gender. Hence HDL-C is believed to be protective[51]. The early detection of dyslipidemia is important for implementing management strategies. These strategies can reduce the risk of cardiovascular diseases that may manifest later in life. Certain lifestyle modifications and lipid-lowering drugs can effectively treat individuals with abnormal lipid profiles. However, literature regarding the benefits of early screening and initiation of treatment is sparse. A systematic review in the United States by Chou et al. stated that no prior study could provide strong evidence for the effects of screening or treatment on clinical outcomes in younger adults. Moreover, the benefit of early treatment was not clear. Therefore trials conducted on a big scale are needed to find evidence for the benefits and harms of dyslipidemia screening or treatment in younger adults.

7. REFERENCES

[1] Mann S, Beedie C, Jimenez A. Differential effects of aerobic exercise, resistance training and combined exercise modalities on cholesterol and the lipid profile: review, synthesis, and recommendations. Sports medicine. 2014 Feb;44:211-21.

[2] Gulcin İ. Antioxidants and antioxidant methods: An updated overview. Archives of toxicology. 2020 Mar;94(3):651-715.

[3] Feingold KR. Introduction to lipids and lipoproteins. end text [Internet]. 2021 Jan 19.

[4] Nielsen S, Karpe F. Determinants of VLDL-triglycerides production. Current opinion in lipidology. 2012 Aug 1;23(4):321-6.

[5] Schwartz CC, VandenBroek JM, Cooper PS. Lipoprotein cholesteryl ester production, transfer, and output in vivo in humans. Journal of lipid research. 2004 Sep 1;45(9):1594-607.

[6] Kakadiya J. Causes, symptoms, pathophysiology and diagnosis of atherosclerosis–a review. PharmacologyOnline. 2009;3:420-42.

[7] Feingold KR Lipid and Lipoprotein Metabolism Endocrinology and Metabolism Clinics 2022 Sep 1;51(3.437-58.

[8] Cohn JS, Wagner DA, Cohn SD, Millar JS, Schaefer EJ. Measurement of very low-density and low-density lipoprotein apolipoprotein (Apo) B-100 and high-density lipoprotein Apo AI production in human subjects using deuterated leucine. Effect of fasting and feeding. The Journal of clinical investigation. 1990 Mar 1;85(3):804-11.

[9] Newman III WP, Freedman DS, Voors AW, Gard PD, Srinivasan SR, Cresanta JL, Williamson GD, Webber LS, Berenson GS. Relation of serum lipoprotein levels and systolic blood pressure to early atherosclerosis. New England Journal of Medicine. 1986 Jan 16;314(3):138-44.

[10] Hill MF, Bordoni B. Hyperlipidemia. InStatPearls [Internet] 2022 Feb 8. StatPearls Publishing.

[11] Sniderman A, Couture P, De Graaf J. Diagnosis and treatment of apolipoprotein B dyslipoproteinemias. Nature Reviews Endocrinology. 2010 Jun;6(6):335-46.

[12] Spotnitz M, Patterson J, Huser V, Weng C, Natarajan K. Harmonization of Measurement Codes for Concept-Oriented Lab Data Retrieval. InMEDINFO 2021: One World, One Health–Global Partnership for Digital Innovation 2022 Jun 1 (pp. 12-16). IOS Press.

[13] Hernandez P, Passi N, Modarressi T, Kulkarni V, Soni M, Burke F, Bajaj A, Soffer D. Clinical management of hypertriglyceridemia in the prevention of cardiovascular disease and pancreatitis. Current atherosclerosis reports. 2021 Nov;23:1-3.

[14] Ko DT, Alter DA, Guo H, Koh M, Lau G, Austin PC, Booth GL, Hogg W, Jackevicius CA, Lee DS, Wijeysundera HC. High-density lipoprotein cholesterol and cause-specific mortality in individuals without previous cardiovascular conditions: the CANHEART study. Journal of the American College of Cardiology. 2016 Nov 8;68(19):2073-83.

[15] Castañer O, Pintó X, Subirana I, Amor AJ, Ros E, Hernáez Á, Martínez-González MÁ, Corella D, Salas-Salvadó J, Estruch R, Lapetra J. Remnant cholesterol, not LDL cholesterol, is associated with incident cardiovascular disease. Journal of the American College of Cardiology. 2020 Dec 8;76(23):2712-24.

[16] Fan AZ, Ham SA, Muppidi SR, Mokdad AH. Validation of reported physical activity for cholesterol control using two different physical activity instruments. Vascular health and risk management. 2009 Aug 6:649-61.

[17] Rizk NM, Yousef M. Association of lipid profile and waist circumference as cardiovascular risk factors for overweight and obesity among school children in Qatar. Diabetes, metabolic syndrome and obesity: targets and therapy. 2012 Dec 20:425-32.

[18] Malik J, Laique T, Ishaq U, Ashraf A, Malik A, Ali M, Zaidi SM, Javaid M, Mehmood A. Effect of COVID-19 on lipid profile and its correlation with acute phase reactants. MedRxiv. 2021 Apr 14:2021-04.

[19] Ruidavets JB, Ducimetiere P, Arveiler D, Amouyel P, Bingham A, Wagner A, Cottel D, Perret B, Ferrières J. Types of alcoholic beverages and blood lipids in a French population. Journal of Epidemiology & Community Health. 2002 Jan 1;56(1):24-8.

[20] Fahed AC, Nemer GM. Familial hypercholesterolemia: the lipids or the genes?. Nutrition & Metabolism. 2011 Dec;8(1):1-2.

[21] Rekha R, Kumari U, Kumar A. Nutritional Aspect of Type-2 Diabetes Mellitus. Bulletin of Pure & Applied Sciences-Zoology. 2019;38(2):126-31.

[22] Ward KK, Shah NR, Saenz CC, McHale MT, Alvarez EA, Plaxe SC. Cardiovascular disease is the leading cause of death among endometrial cancer patients. Gynecologic oncology. 2012 Aug 1;126(2):176-9.

[23] Misra A, Shrivastava U. Obesity and dyslipidemia in South Asians. Nutrients. 2013 Jul 16;5(7):2708-33.

[24] Singh KR, Fernandes M, Sarkar T, Sridevi P. Assessment and analysis of lifestyle disease burden in tribes of central India. J Infect Non Infect Dis. 2019;4:027.

[25] Einarson TR, Acs A, Ludwig C, Panton UH. Prevalence of cardiovascular disease in type 2 diabetes: a systematic literature review of scientific evidence from across the world in 2007–2017. Cardiovascular diabetology. 2018 Dec;17(1):1-9.

[26] Writing Group Members, Lloyd-Jones D, Adams RJ, Brown TM, Carnethon M, Dai S, De Simone G, Ferguson TB, Ford E, Furie K, Gillespie C. Heart disease and stroke statistics—2010 update: a report from the American Heart Association. Circulation. 2010 Feb 23;121(7):e46-215.

[27] Pirillo A, Norata GD, Catapano AL. Worldwide Changes in Total Cholesterol and Non-HDL-Cholesterol Trends Indicate Where the Challenges Are for the Coming Years. Clinical Chemistry. 2021 Jan;67(1):30-2.

[28] Bacle A, Gautier R, Jackson CL, Fuchs PF, Vanni S. Interdigitation between triglycerides and lipids modulates surface properties of lipid droplets. Biophysical journal. 2017 Apr 11;112(7):1417-30.

[29] Kayani AM, Bakht N, Munir R, Abid I. The mosaic of CVD risk factors–A study on 10,000 Pakistani cardiac patients. CVD Prevention and Control. 2011 Jan 1;6(1):1-7.

[30] Liaquat A, Javed Q. Current trends of cardiovascular risk determinants in Pakistan. Cureus. 2018 Oct 4;10(10).

[31] Sun G-Z, Li Z, Guo L, Zhou Y, Yang H-M, Sun Y-X. High prevalence of dyslipidemia and associated risk factors among rural Chinese adults. Lipids Health Dis. 2014; 13:189.

[32] Qi L, Ding X, Tang W, Li Q, Mao D, Wang Y. Prevalence and risk factors associated with dyslipidemia in Chongqing, China. Int J Environ Res Public Health. 2015 Oct; 12(10):13455–65

[33] Toth PP, Potter D, Ming EE. Prevalence of lipid abnormalities in the United States: The National Health and Nutrition Examination Survey 2003-2006.J Clin Lipidol. 2012; 6(4):325–30.

[34] Grundy SM, Cleeman JI, Merz CNB, Brewer HBJ, Clark LT, Hunninghake DB, et al. Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III Guidelines. J Am Coll Cardiol. 2004; 44(3):720–32.

[35] Penalva RA, Huoya M de O, Correia LCL, Feitosa GS, Ladeia AMT. Lipid profile and intensity of atherosclerosis disease in acute coronary syndrome.Arq Bras Cardiol. 2008; 90(1):24–30.

[36] Chinese guidelines on prevention and treatment of dyslipidemia in adults]. Zhonghua Xin Xue Guan Bing Za Zhi. 2007; 35(5):390–419

[37] Pan L, Yang Z, Wu Y, Yin R-X, Liao Y, Wang J, et al. The prevalence, awareness, treatment, and control of dyslipidemia among adults in China. Atherosclerosis. 2016; 248:2–9

[38] Toth PP, Potter D, Ming EE. Prevalence of lipid abnormalities in the United States: The National Health and Nutrition Examination Survey 2003-2006.J Clin Lipidol. 2012; 6(4):325–30

[39] Zhao W, Zhang J, You Y, Man Q, Li H, Wang C, et al. [Epidemiologic characteristics of dyslipidemia in people aged 18 years and over in China]. Zhonghua Yu Fang Yi Xue Za Zhi. 2005; 39(5):306–10.

[40] Chou R, Dana T, Blazina I, Daeges M, Bougatsos C, Jeanne TL. Screening for dyslipidemia in younger adults A systematic review for the U.S. preventive services task force. Ann Intern Med. 2016; 165(8):560–4.

[41] Loria CM, Liu K, Lewis CE, Hulley SB, Sidney S, Schreiner PJ, et al. Early adult risk factor levels and subsequent coronary artery calcification: The CARDIA Study. J Am Coll Cardiol. 2007; 49(20):2013–20.

[42] Qi L, Ding X, Tang W, Li Q, Mao D, Wang Y. Prevalence and risk factors associated with dyslipidemia in Chongqing, China. Int J Environ Res Public Health. 2015 Oct; 12(10):13455–65.

[43] Latifi SM, Karandish M, Shahbazian HB, Chinipardaz R, Sabet A, Pirani N. A survey of the incidence of dyslipidemia and its components in people over 20 years old in Ahvaz: A cohort study 2009-2014.Diabetes Metab Syndr. 2017; 11 Suppl 2:S751–4.

[44] Pan L, Yang Z, Wu Y, Yin R-X, Liao Y, Wang J, et al. The prevalence, awareness, treatment, and control of dyslipidemia among adults in China. Atherosclerosis. 2016; 248:2–9.

[45] Wu Y, Huxley R, Li L, Anna V, Xie G, Yao C, et al. Prevalence, awareness, treatment, and control of hypertension in China: Data from the China National Nutrition and Health Survey 2002. Circulation. 2008; 118(25):2679–86

[46] Liu X, Yu S, Mao Z, Li Y, Zhang H, Yang K, et al. Dyslipidemia prevalence, awareness, treatment, control, and risk factors in Chinese rural population: the Henan rural cohort study. Lipids Health Dis. 2018; 17(1):119.

[47] Huang Y, Gao L, Xie X, Tan SC. Epidemiology of dyslipidemia in Chinese adults: a meta-analysis of prevalence, awareness, treatment, and control. Popul Health Metr. 2014; 12(1):28.

[48] J, Wang L, Li Y, Bi Y, Jiang Y, Mi S, et al. [Epidemiologic characteristics of dyslipidemia in Chinese adults 2010]. Zhonghua Yu Fang Yi Xue Za Zhi. 2012; 46(5):414–8.

[49] Huang Y, Gao L, Xie X, Tan SC. Epidemiology of dyslipidemia in Chinese adults: a meta-analysis of prevalence, awareness, treatment, and control. Popul Health Metr. 2014; 12(1):28.

[50] Song PK, Man QQ, Hong LI, Pang SJ, Jia SS, Li YQ, Li HE, Zhao WH, Zhang J. Trends in lipids level and dyslipidemia among Chinese adults, 2002-2015. Biomedical and Environmental Sciences. 2019 Aug 1;32(8):559-70.

[51] Assmann G, Gotto Jr AM. HDL cholesterol and protective factors in atherosclerosis. Circulation. 2004 Jun 15;109(23_suppl_1):III-8.