SERUM LIPID PROFILE AND ITS CORRELATION WITH IRON DEFICIENCY ANEMIA

A Dissertation submitted to

THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY

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for the award of the degree of

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DEPARTMENT OF GENERAL MEDICINE

GOVERNMENT KILPAUK MEDICAL COLLEGE AND HOSPITAL,

CHENNAI

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This is to certify that this dissertation **" SERUM LIPID PROFILE AND ITS CORRELATION WITH IRON DEFICIENCY ANEMIA"" ** is the original Bonafide work done by Dr.. **RAMYA VENKATESAN** Post graduate in General Medicine, under my overall supervision in the Department of General Medicine, Govt. Kilpauk Medical College in partial fulfillment of the regulations of The Tamilnadu Dr. M.G.R Medical University for the award of **M.D. DEGREE IN GENERAL MEDICINE.**

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DECLARATION

I solemnly declare that this dissertation titled "**SERUM LIPID PROFILE AND ITS CORRELATION WITH IRON DEFICIENCY ANEMIA**" is a Bonafide work done by me at the Department of General Medicine, Govt. Kilpauk Medical College and Hospital, Chennai, under the supervision of **Dr. P. PARANTHAMAN** M.D., Professor, Head of the Department, Department of General Medicine.This dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the University regulations for the award of the Degree of M.D. GENERAL MEDICINE examinations to be held in May 2023.

Place: Chennai

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SERUM LIPID PROFILE AND ITS CORRELATION WITH ANEMIA – A CROSS-SECTIONAL CASE-CONTROL STUDY

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ABBREVIATION

LDL	-	Low-density lipoprotein
HDL	-	High-density lipoprotein
CHD	-	Coronary heart disease
CAD	-	Coronary Artery Disease
ATGL	-	Adipose triglyceride lipase
HSL	-	Hormone-sensitive lipase
CM	-	Chylomicrons
VLDL	-	Very low-density lipoproteins
LPL	-	Lipoprotein lipase
HL	-	Hepatic lipase
LCAT	-	Lecithin cholesterol acyl transferase
CETP	-	Cholesteryl ester transfer protein
MTP	-	Microsomal triglyceride protein

ACAT	-	Acyl Co-A transferase
SCA	-	Sickle cell anemia
TC	-	Total cholesterol

INTRODUCTION

In an era where there is an increasing prevalence of atherosclerotic vascular disease, the factors that contribute to atherosclerosis need to be evaluated. Hyperlipidemia is one of the chief contributors to the atherosclerotic process. Hyperlipidemia as per an objective definition is described as levels of low-density lipoprotein (LDL), total cholesterol, triglycerides, or lipoproteins greater than the 90th percentile in comparison to the general population, or an HDL level less than the 10th percentile when compared to the general population. Lipids typically include cholesterol levels, lipoproteins, chylomicrons, VLDL, LDL, apolipoproteins, and HDL. There has been significant research on preventing and treating elevated lipid profiles, for management of the future complications such as atherosclerosis disease.

Yet another significant issue in the spotlight in India is the prevalence of anemia. Anemia, as per the World Health Organisation (WHO), is defined as Hemoglobin (Hb) levels <12.0 g/dL in women and <13.0 g/dL in men. [3] At the national level, the findings of the NFHS-5 reveal that there has been an increase

in the prevalence of anemia among women and children compared to the previous NFHS-4 survey that was conducted in 2015-16, about 4 years ago. The increase in anemia among pregnant women is by 1.8 percentage points, among all women of reproductive age is by 3.9 percentage points, and among adolescent women is by 5 percentage points. [4]

Several studies have suggested that lipid profile levels are associated with IDA in both humans and animals. A study reported severe IDA in patients with lower levels of triglyceride and total cholesterol which in turn was normal after treating the iron replacement therapy. [4]

Thus, with an increase in the prevalence of both Atherosclerotic Cardiovascular disease (ASCVD) and anemia, a correlation, if any, between the lipid profile and anemia, if established, can help in modifying preventive strategies and in also defining the target hemoglobin level while treating anemia.

With the recent research, knowledge, and data the current study aims to assess the correlation between lipid profiles and anemia.

The study also aims to assess the severity of anemia and correlate it with the lipid profiles of the patient to understand more about the direct correlation. This will enable to diagnosis and treat the risk of dyslipidemia and anemia.

LITERATURE REVIEW

WHAT IS HYPERLIPIDEMIA?

In the past, numerous research has been conducted in various demographic settings to draw a connection between anaemia and cholesterol levels. [1] Recently, non-fasting lipid profiles have been used instead of fasting lipid profiles to ease blood sampling. [2] Hyperlipidaemia is a condition when the blood's lipid levels are increased. Atherosclerosis and its associated disorders, such as coronary heart disease (CHD), ischemic cerebrovascular disease, peripheral vascular disease, and pancreatitis, are largely brought on by hyperlipidemia. Hyperlipidemia may also be caused by different genetic or environmental factors. [3]

A rise in one or more plasma lipids, such as triglycerides, cholesterol, cholesterol esters, phospholipids, and/or plasma lipoproteins, such as very low-density lipoprotein and low-density lipoprotein, along with a decline in high-density lipoprotein levels, characterises the medical condition known as hyperlipidemia. This increase in lipid levels is the major risk for cardiovascular diseases. In clinical studies, several medications have demonstrated a good

potential for treating hyperlipidaemia, including lanosterol synthase inhibitors, squalene epoxidase inhibitors, diacylglycerol acyl transferase inhibitors, and ATP citrate lyase inhibitors. [4]

CLASSIFICATION:

Hyperlipidaemia can be categorized into two types:

PRIMARY HYPERLIPIDEMIA

It is caused due to genetic defects which may be monogenic or polygenic.

SECONDARY HYPERLIPIDEMIA

Secondary hyperlipidemia is caused by other disorders such as diabetes, alcoholism, and hypothyroidism. Also the usage of drugs such as beta-blockers, corticosteroids, and oral contraceptives. [5]

PATHOPHYSIOLOGY:

One of the most common risk factors influencing the development of atherosclerosis and subsequent vascular disease is hyperlipidemia, particularly increased LDL (hypercholesterolemia). Endothelial damage, hyperlipidemia, inflammatory and immunologic variables, plaque erosion or rupture,

hypertension, and smoking are only a few of the causes that cause atherosclerosis to develop.[6]

Atherosclerosis develops as a result of underlying endothelial damage, which appears to be caused by nitric oxide loss within the endothelium. There are two different ways that atherosclerotic plaques develop: either by a slow, chronic plaque build-up that eventually results in luminal stenosis or through an acute onset of fast luminal blockage brought on by plaque rupture and thrombosis.[6, 7]

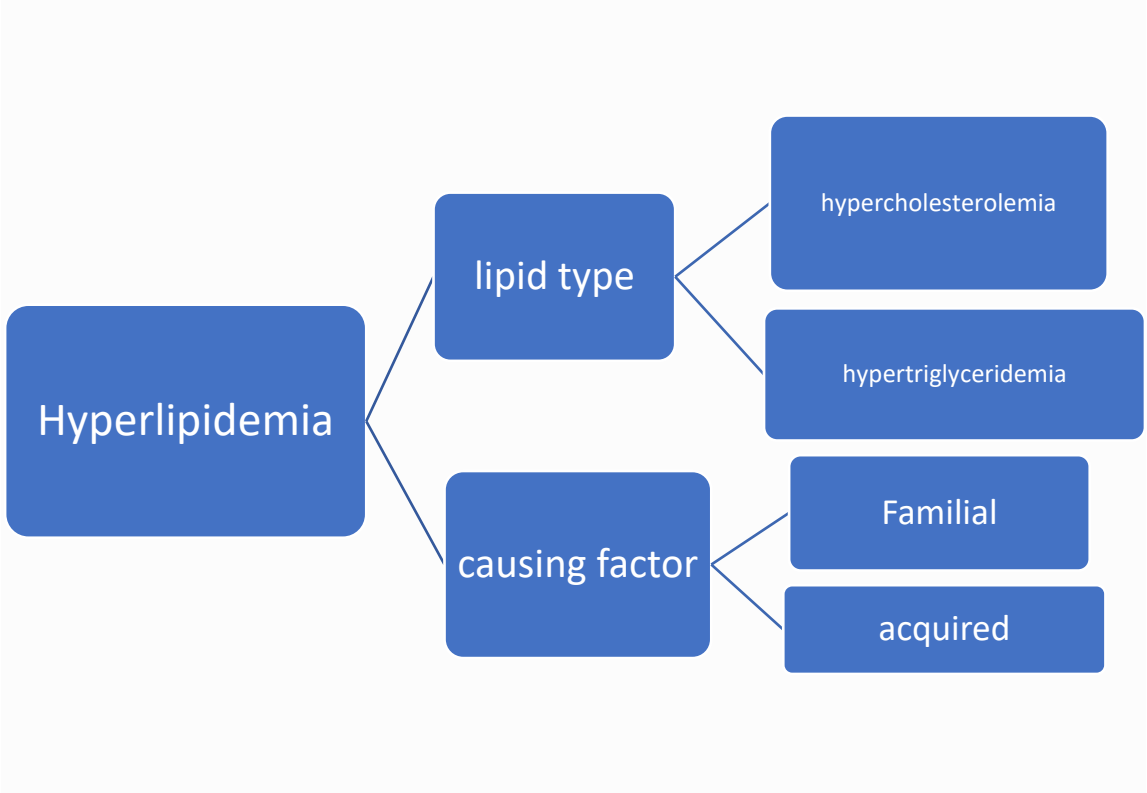
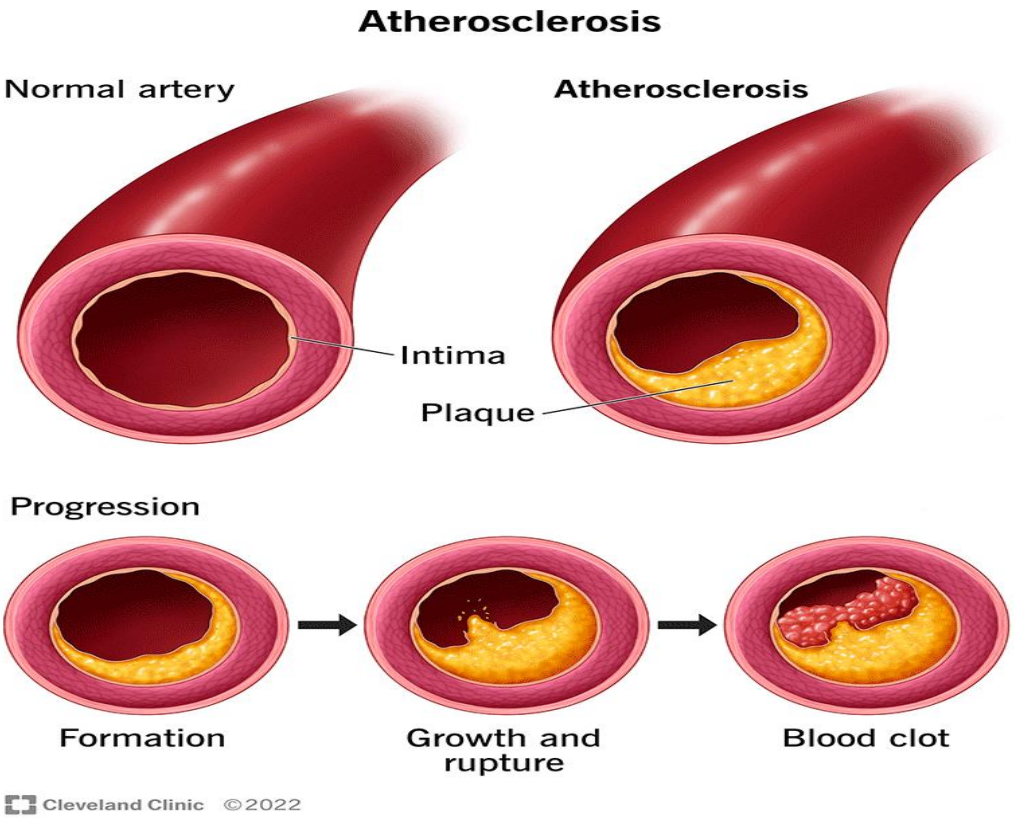


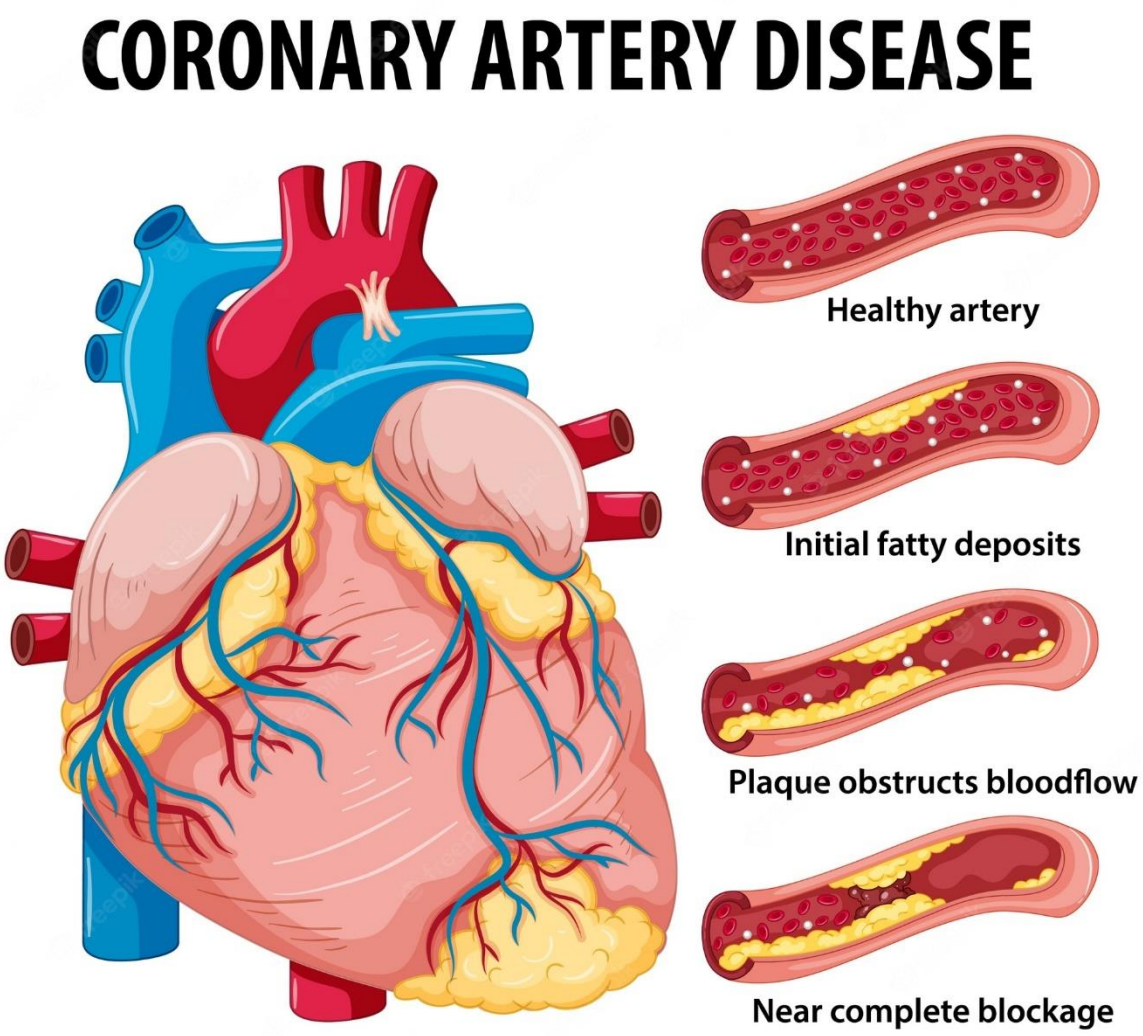
Fig 1: Classification of Hyperlipidemia

COMPLICATIONS DUE TO HYPERLIPIDEMIA:

1. **Atherosclerosis:** Fat, cholesterol, and calcium buildup in the artery linings cause this condition, which is quite prevalent. Fibrous plaques are created as a result of this deposition [8]. Three elements typically make up a plaque: 1) An atheroma, which is a fatty, soft, yellowish nodular mass made up of macrophages, cells involved in immunity, and found in the center of a bigger plaque; 2) a layer of cholesterol crystals, and 3) a calcified outer layer. The main cause of cardiovascular disease is atherosclerosis. [9]



2. **Coronary Artery Disease:** The most common cause of CAD is atherosclerosis. It is characterized by a constriction of the arteries that carry blood to the myocardium, which limits blood flow and leaves the heart with insufficient oxygen levels to meet its needs. The constriction could get so bad that the shortage of blood could hurt the heart muscle. Coronary atherosclerosis is associated with an elevated lipid profile. [9] [10]



3. Myocardial Infarction: The condition known as MI is brought on by a partial or complete blockage of the blood and oxygen supply to the cardiac arteries, which causes injury or death of heart cells. The creation of a clot in an artery is typically what causes the blockage.[9] [11]

LIPIDS:

DEFINITION:

Fatty, waxy, or oily molecules are referred to as lipids. Lipids act as an essential component in the formation of the cell membrane. Typically, the structure consists of a glycerol backbone, two hydrophobic fatty acid tails, and a phosphate group (hydrophilic). [12]

MECHANISM:

During lipid transport in plasma, the interaction between water-fearing and fat-loving cells is more visible. Triglycerides and cholesterol are both nonpolar lipid molecules. As a result, they must travel through the polar plasma with the

assistance of lipoprotein particles. Lipoprotein is made up of triglycerides, cholesterol, phospholipids, and apolipoproteins.[12]

There are two routes for plasma lipid transport. The first is an exogenous route for the transport of dietary triglycerides and cholesterol from the small intestine. The other route is the endogenous system, which transports cholesterol and triglycerides from the liver and other non-intestinal tissues into circulation. Fat can also be broken down for energy when it is required. For fatty acid liberation, glucagon (released during fasting) or epinephrine (released during exercise) activates adipose triglyceride lipase (ATGL), hormone-sensitive lipase (HSL), and monoglyceride lipase (MGL). Most tissues can then use these fatty acids for energy thanks to mitochondria and the Krebs cycle.[12] [13]]

TRIGLYCERIDES:

These are the lipids that are most prevalent. It is widely distributed in adipocytes. The majority of these are found in both plant and animal cell store lipids. Extra calories, alcohol, and sugar are turned by the body into triglycerides, which are then deposited in fat cells all over the body. Triglycerides are glycerol esters that have three fatty acid molecules added to

them. Triglycerides have a normal range of 200 mg/dl and an unhealthy range of 500 mg/dl. Ranges more than 500 mg/dl are thought to be risky for the onset of cardiovascular disease [9]

CHOLESTEROL:

It is an essential part of all tissues' mammalian cell membranes and a precursor of bile acids and steroid hormones. All animal cells contain it, either free or as many fatty esters, but it is only present in plant fat. [14]

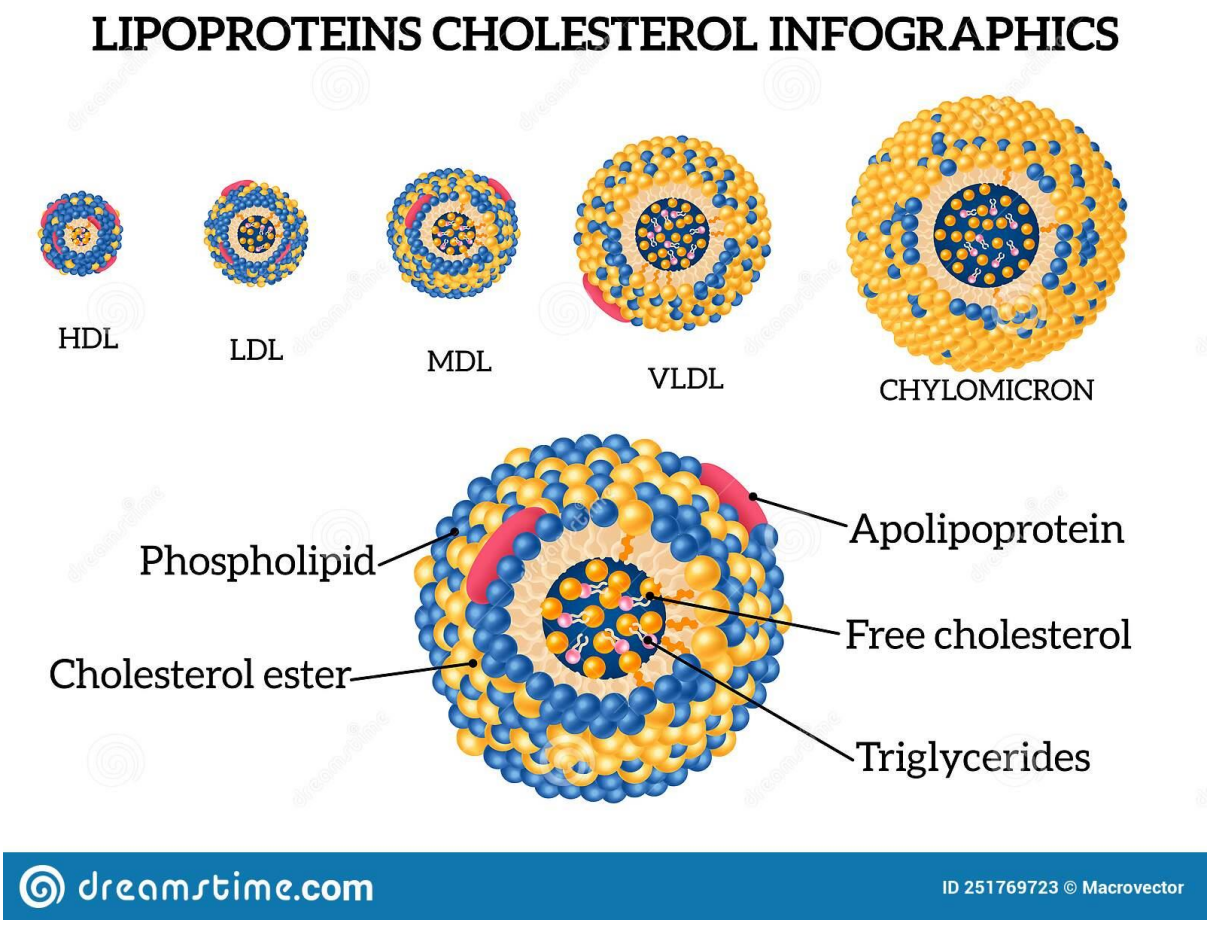
PLASMA LIPOPROTEINS:

Lipoproteins are macromolecule aggregates made up of lipids and proteins; this structure allows lipids to be compatible with aqueous body fluids. Apolipoproteins are specialised proteins found in lipoproteins that are made of polar and non-polar lipids. Non-polar lipids are composed of triglycerides and cholesteryl esters whereas polar lipids are composed of phospholipids and unesterified cholesterol. [15]

FUNCTION:

Triglycerides, an important energy source, are generated and absorbed into regions of consumption and storage. Plasma lipoproteins are crucial for lipid solubilization to transport cholesterol between various sites of absorption, synthesis, catabolism, and elimination.[16]

CLASSIFICATION OF LIPOPROTEINS:



Lipoproteins consist of five classes namely Chylomicrons (CM), very low-density lipoproteins (VLDL), low-density lipoproteins(LDL), intermediate-density lipoproteins (IDL), and high-density lipoproteins (HDL). Each one of these is heterogenous and has a different size, composition, and density. [16]

Chylomicrons: The density and size of these particles are the greatest, and their concentration is closely connected with the number of dietary triglycerides. [9]

VLDL: Very low-density lipoproteins, which are released by the liver, are smaller particles with lower triglyceride levels than chylomicrons. VLDL transports cholesterol from the liver to the body's organs and tissues. [17]

LDL: Low-density lipoproteins are produced partially in the intestinal chyle and partially following VLDL lipolysis. It has a direct connection to CHD. [18, 19]

IDL: In the capillaries of adipose tissue and muscle, the lipase enzyme breaks down VLDL particles to produce intermediate-density lipoprotein. [9]

HDL: Commonly referred to as "good cholesterol," HDL. The liver is where high-density lipoproteins are made. It transports lipids from tissues back to the

liver for breakdown, including cholesterol. HDL has anti-atherogenic properties. [20]

ENZYMES IN LIPOPROTEIN METABOLISM:

Lipoprotein lipase (LPL): The endothelial cells of the heart, muscle, adipose tissue, macrophages, and lactating mammary glands express LPL, a multifunctional enzyme. Triglyceride (TG) breakdown into two free fatty acids and monoacylglycerol depending heavily on LPL. [4] [21]

Hepatic lipase (HL): HL is a multifunctional protein that controls the metabolism of lipoproteins. In plasma lipoproteins, HL hydrolyzes phospholipids and triglycerides. HL also influences cellular lipid delivery by promoting lipoprotein absorption by cell surface receptors and proteoglycans.[22]

Lecithin cholesterol acyl transferase (LCAT): In the metabolism of HDL, lecithin cholesterol acyltransferase is a key enzyme. It turns free cholesterol into

cholesteryl esters, which are subsequently trapped inside the lipoprotein core and produce mature HDL. [23]

Cholesteryl ester transfer protein (CETP): The esterified cholesterol esters (CE) are transferred from HDLs to chylomicrons, VLDL, and LDL at a faster rate thanks to the hydrophobic plasma glycoprotein known as cholesterol ester transfer protein (CETP), also known as plasma lipid transfer protein. A lack of ACETP is associated with higher HDL levels and lower LDL levels. [24]

Microsomal triglyceride protein (MTP): The lipid transfer protein microsomal triglyceride protein (MTP), which was isolated from the liver and intestinal mucosa, catalyses the transfer of neutral lipids, triglycerides, and cholesterol esters between the membrane and the lumen of microsomes. It is well known that MTP plays a crucial role in the regulation of cholesterol ester production as well as the manufacture of glycolipid-presenting molecules.[24]

Acyl Co-A transferase (ACAT): Transferase Co-A (ACAT) Acyl Co-A transferase (ACAT) is a membrane-bound protein that converts cholesterol and long-chain fatty acyl-CoA into cholesteryl esters. ACAT has a key function in the synthesis and secretion of apolipoprotein-B-containing lipoproteins in the

liver and intestines, which explains the significance of ACAT. In many tissues, ACAT performs important functions in maintaining cellular cholesterol homeostasis and guarding against the harmful build-up of extra cholesterol inside of cells.[25] [4]

RECEPTORS:

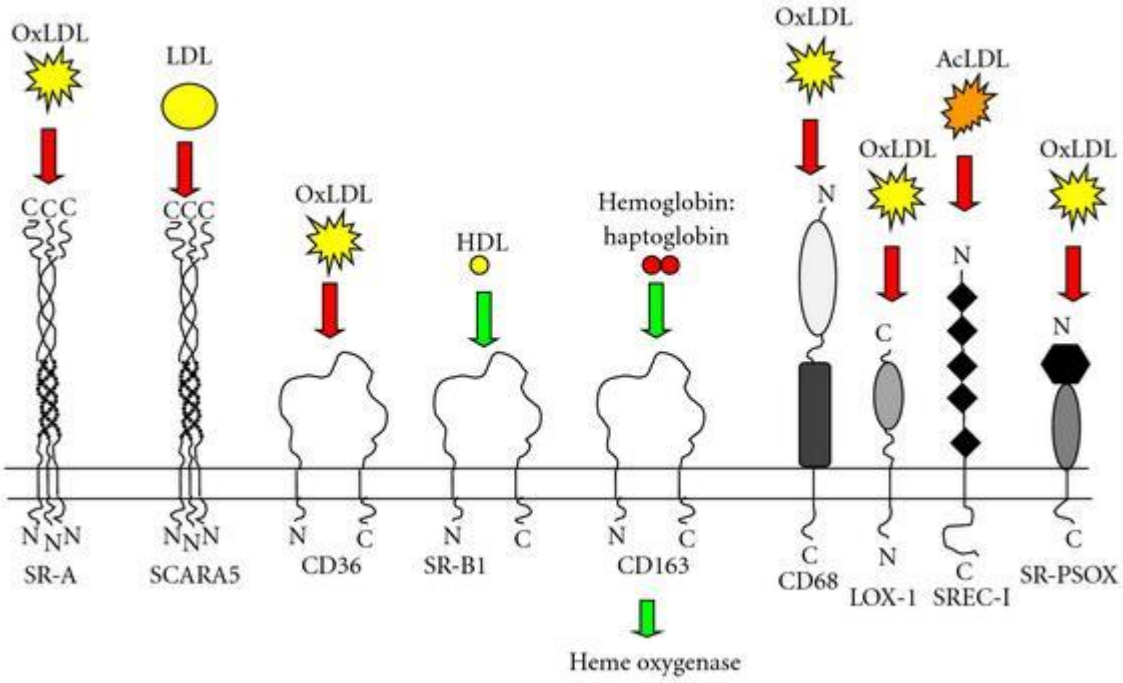
LDL Receptor: This receptor binds apo B100 and apo E Possessing Lipoproteins. Cholesterol content inside the cell regulates the expression of LDL receptors on the cell surface. [26]

LDL Receptor–related protein. This is an integral receptor of the cell membrane. It has a high affinity for apo E enriched chylomicron remnants and VLDL remnants but does not bind with LDL. [26]

VLDL Receptors: The receptor binds with apo E containing Lipoprotein. VLDL receptors are found especially in muscle, brain, and fat. It is not present in the liver. It delivers to the target organs triglyceride enriched Lipoproteins. [26]

Apolipoprotein E Receptor: This receptor is expressed in the Brain and placenta and helps in Lipoprotein metabolism in the CNS. It transduces extracellular signals and has an important role in normal brain development. [26]

Scavenger Receptors: Scavenger receptors gain significance because of their property to interact with a broad range of different kinds of ligands involved in various processes like atherosclerosis, CNS disorders, and host defense mechanisms. They are classified into five subclasses – subclass A, B, C, D, and E. Acetylated, Acetoacetylated and Malondialdehyde attached LDL particles are taken up by scavenger receptors [26]



DYSLIPIDEMIA:

India has a high prevalence of dyslipidemia regardless of socioeconomic status. Any irregularity of the lipids in the blood is referred to as dyslipidemia, which is typically linked to high levels of triglycerides and cholesterol (TGs). Low-density lipoprotein (LDL) seems to pose the greatest risk of any of them. The majority of dyslipidemias in wealthy nations are hyperlipidemias. [27]

ANEMIA

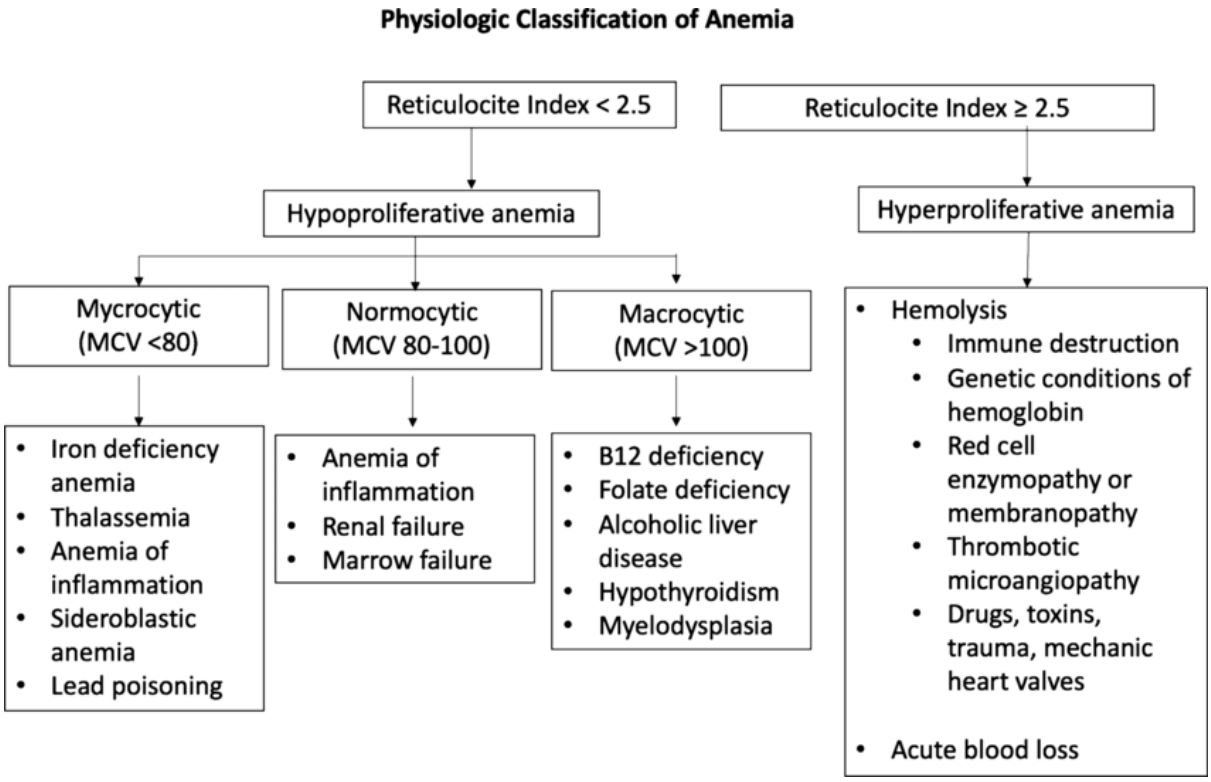
A third of the world's population suffers from anaemia, with iron deficiency accounting for 50% of cases. It is a significant issue for worldwide public health that has an impact on physical performance, referral to medical professionals, and maternal and child mortality. The diagnosis of iron deficiency anemia is more precise when serum ferritin, transferrin saturation, serum soluble transferrin receptors, and the serum soluble transferrin receptors-ferritin index are measured.[28]

The most prevalent hematological ailment to impact humans, anemia is typically seen in states of chronic disease like non-specific anaemia, which can make diagnosis challenging. The ability of the physician to correlate the possible clinical pathways of the underlying disease with the patient's Ferro kinetic state is required to diagnose this disorder. Iron deficiency anemia and dyslipidemia are both widespread public health issues, particularly among Indians. For humans, iron is a crucial micronutrient. It is required for a variety of iron-dependent enzymes to operate as well as for the creation of myoglobin

and haemoglobin in myocytes and erythrocytes, respectively. It's critical to maintain a healthy bodily iron balance for our well-being and quality of life.[28]

IDA is linked to emotional instability, depression, stress, and poorer cognitive function tests in postpartum women. Therefore, IDA may affect pregnant women in the same way. Utilizing the proper markers, primarily plasma ferritin, plasma transferrin saturation, serum "soluble" transferrin receptor (sTfR), blood haemoglobin, and a complete blood count, iron status can be evaluated before delivery.

International practice guidelines have been encouraged to give special consideration to IDA diagnosis and management as new information on the role of IDA in worsening clinical outcomes continues to mount.[28]



CAUSES:

The cause of anaemia is determined by whether the anaemia is hypo proliferative or hyperproliferative.

HYPOPROLIFERATIVE ANEMIA (corrected reticulocyte count <2%): It is further divided into microcytic, normocytic and macrocytic anemia respectively.

Microcytic Anemia: Due to decreased hemoglobin production, microcytic anemia is characterized by smaller-than-normal red blood cells.

Normocytic anemia: Smaller-than-normal red blood cells are a hallmark of microcytic anemia, which is caused by reduced hemoglobin synthesis.

Macrocytic anemia: A blood condition called macrocytic anemia develops when your bone marrow makes unusually big red blood cells.

HYPERPROLIFERATIVE ANEMIA (corrected reticulocyte count >2%) [29]

CLINICAL REPRESENTATION OF ANEMIA:

The most common way to identify anemia is through abnormal screening laboratory tests. Advanced anemia and its accompanying symptoms and signs are less frequently experienced by patients. Blood loss or hemolysis are the causes of acute anemia. A reduction in pH or an increase in CO2 cause alterations in the O2-hemoglobin dissociation curve, which boost the O2 supply if blood loss is minimal (Bohr effect). The clinical picture of acute blood loss is dominated by hypovolemia, and the levels of hematocrit and hemoglobin do not correspond to the amount of blood lost. [30]

Signs of vascular instability appear with acute losses of 10–15% of the total blood volume. When >30% of the blood volume is lost suddenly, patients are unable to compensate with the usual mechanisms of vascular contraction and changes in regional blood flow. The signs and symptoms of acute hemolysis are determined by the mechanism that causes red cell destruction. [30]

Acute back pain, free hemoglobin in the plasma and urine, and renal failure may be associated with intravascular hemolysis with the release of free hemoglobin. The symptoms of more chronic or progressive anaemia are determined by the patient's age and adequate blood supply to critical organs. Fatigue, loss of stamina, shortness of breath, and tachycardia are symptoms of moderate anaemia. [30]

Anaemia is frequently associated with certain conditions. Chronic inflammatory states (e.g., infection, rheumatoid arthritis, cancer) are associated with mild to moderate anemia, whereas lymphoproliferative disorders, such as chronic lymphocytic leukemia and certain other B cell neoplasms, may be associated with autoimmune hemolysis.[30]

ANEMIA DUE TO RBC DESTRUCTION OR ACUTE BLOOD LOSS:

BLOOD LOSS: Common causes include trauma and GI hemorrhage (which may be occult); less common causes include genitourinary sources (menorrhagia, gross hematuria), internal bleeding such as intraperitoneal from spleen or organ rupture, retroperitoneal, and iliopsoas hemorrhage (e.g., in hip fractures). Acute bleeding is associated with hypovolemia, reticulocytosis, and macrocytosis, whereas chronic bleeding is associated with iron deficiency, hypochromia, and microcytosis.[31]

IRON DEFICIENCY ANEMIA: One of the most common types of malnutrition is iron deficiency. Iron deficiency is responsible for 50% of anemia worldwide and accounts for nearly a million deaths annually. Africa and parts of Asia bear 71% of the global mortality burden, while North America accounts for only 1.4% of total morbidity and mortality due to iron deficiency. **[32]**

CAUSES OF IRON DEFICIENCY:

1. Physiologic increase in requirements

• Growth phase

• Menstruation

• Pregnancy

• Breast-feeding

2. Blood loss

• **Gastrointestinal –** Oesophagus – Varices – Ulcers – Tumours – Inflammations – Malformation (blood vessels)

• **Urogenital –** Hypermenorrhea – Birth – Tumors of the urinary tract – Calculi

• **Iatrogenic** – Laboratory testing (excessive) – Drugs – Blood donors

3. Malabsorption

• Sprue

• Gastric resections

• Chronic atrophic gastritis

• Drugs

4. Inadequate supply

- Unbalanced diet
- Age
- New vegetarians

ANEMIA OF ACUTE AND CHRONIC INFLAMMATION/INFECTION (AI): AI is one of the most common clinical forms of anemia, encompassing inflammation, infection, tissue injury, and conditions (such as cancer) associated with the release of proinflammatory cytokines. It is the most important anemia in the differential diagnosis of iron deficiency because many of the symptoms are caused by insufficient iron delivery to the marrow, despite the presence of normal or increased iron stores. Low serum iron, increased red cell protoporphyrin, hyperproliferative marrow, transferrin saturation in the range of 15-20%, and normal or increased serum ferritin all indicate this. Serum ferritin levels are one of the most important distinguishing factors between true iron-deficiency anemia and the iron-restricted erythropoiesis associated with inflammation.[32]

SICKLE CELL ANEMIA:

Sickle cell anemia (SCA), which is a kind of hereditary hemolytic anemia, has as its results the polymerization of deoxygenated hemoglobin molecules and sickle-shaped cells with a shorter average lifespan. Its etiology is linked to a single mutation in the -globin gene. [33] A persistent inflammatory process, alterations in lipid metabolism, endothelial dysfunction, and hemolysis are all part of the complicated pathophysiology of SCA.

Increased triglycerides indicate hypertriglyceridemia, while low levels of total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) identify hypocholesterolemia. [33 34]]

ANEMIA IN AGING:

Anemia is common in people over the age of 65. It is estimated that it affects 11% of community-dwelling older adults and up to 40% of nursing home residents. In general, erythropoietin levels rise with age in older people who maintain normal hemoglobin levels. This compensatory increase to maintain

normal oxygen delivery appears to be due to erythropoietin resistance; studies of red cell life span in older people have not revealed a decrease in red cell survival. The significance of this unexplained aging anemia is that low hemoglobin levels are linked to an increase in falls, hospitalizations, frailty development, and mortality.[32]

UREMIC ANEMIA:

Normochromic normocytic anemia has a significant specific variant known as uremic anemia. The clinical finding that plasma erythropoietin levels are lower in uremic patients than in non-uremic individuals with a comparable level of anemia shows that renal failure-related erythropoietin production is insufficient and contributes to uremic anemia. The causes of uremic anaemia are undoubtedly complex, and while adequate hemodialysis therapy can help in some cases, it is important to recognise the importance of the extracorporeal hemolytic component. Hexose monophosphate shunt defects have been observed in a large number of patients, and hemodialysis-related factors like mechanical intravascular hemolysis and pump stress are becoming more important.[32]

IRON OVERLOAD:

Iron storage proteins ferritin and hemosiderin are more prevalent when there is an excess of iron in the body. In the parenchymatous organs, deposition occurs if the storage capacity is surpassed. Cell death and functional impairment of the organ issue arise from the ensuing cell damage An elevated ferritin level should always be considered a sign of iron overload, and the possibility of true iron overload should be investigated using differential diagnosis [35]

Causes of iron overload

1. **Primary, hereditary hemochromatosis**

2. **Secondary acquired hemochromatosis**

- Ineffective erythropoiesis

– Thalassemia major

– Sideroblastic anemias

– Aplastic anemias

• Transfusion

3. Diet-related hemochromatosis

• Excessive iron intake

– Chronic alcoholism

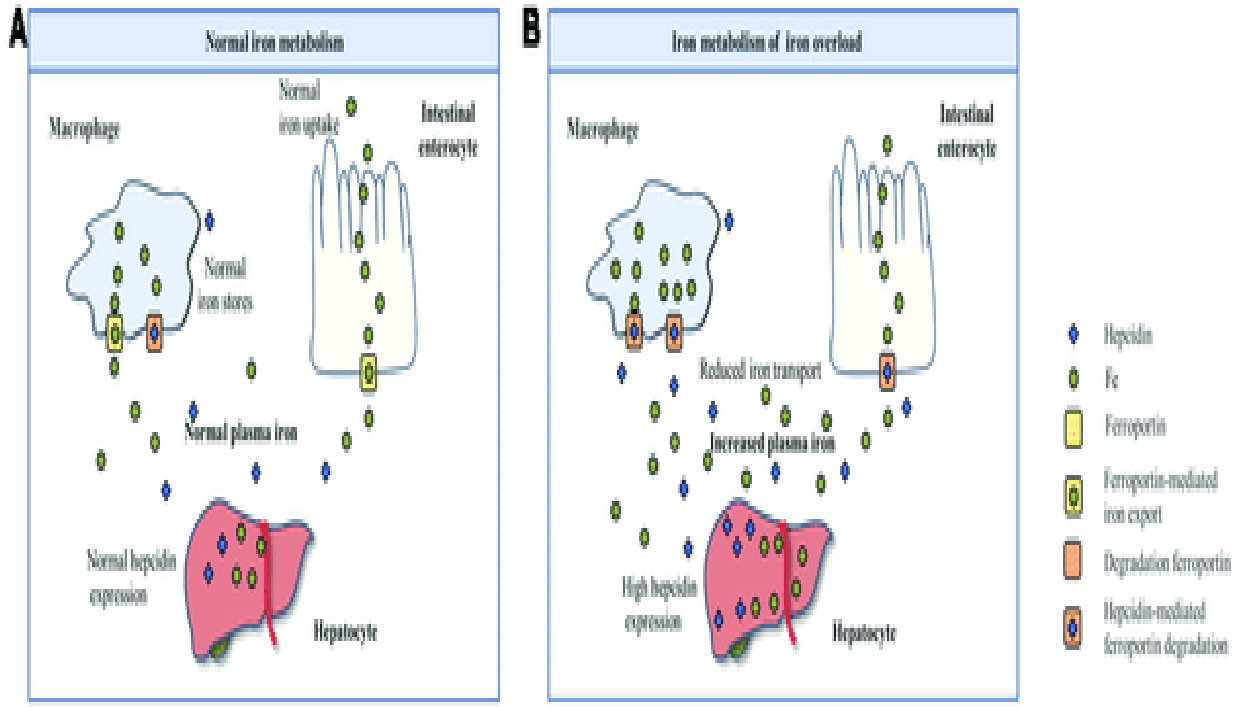


Fig 2: Iron metabolism Process.

FOLIC ACID:

Folic acid is required for the biosynthesis of purines and pyrimidines, which are required for the synthesis of DNA and RNA, and thus for all growth and cell division processes. Under the influence of ascorbic acid and vitamin B12, folic acid is reduced in the body to tetrahydrofolic acid (THF). Tetrahydrofolic acid, as a transporter of activated C-1 groups (methyl-, formyl-, formiate-, and hydroxymethyl groups), plays an important role in amino acid and nucleotide metabolism, as well as in the methylation of homocysteine to methionine. [35]

The body's total folic acid reserves are estimated to be 5-10 mg.

The German Society for Nutrition recommends 400 mg of folic acid per day (dietary folate). Women who are pregnant or breastfeeding should consume 600 mg of folic acid (dietary folate) per day.

Causes of folic acid deficiency

1. Inadequate intake

– Alcoholism

– Unbalanced diet

2. Increased requirement

– Pregnancy

– Adolescence

– Malignancy Increased cell turnover (hemolysis and chronic exfoliative skin diseases) Hemodialysis, patients

3. Malabsorption

– Sprue

– Drugs (barbiturates and phenytoin)

4. Disturbed folic acid metabolism

– Inhibition of dihydrofolic acid reductase (e.g., methotrexate, pyrimethamine, and triamterene)

– Congenital enzyme defects

Patients with folic acid deficiency are usually malnourished and have a variety of gastrointestinal symptoms such as diarrhoea, cheilosis, and glossitis.

Disturbances in the absorption of folic acid occur in both tropical sprue and gluten enteropathy. Manifest macrocytic anemia may develop in both clinical pictures. Other signs of malabsorption may also occur. Alcohol-induced folic acid deficiency may to a certain extent also be caused by malabsorption. Diseases of the small intestine may also prevent the absorption of folic acid.[35]

VITAMIN B12:

Food-borne cobalamin is absorbed as deoxyadenosine-cobalamin, methyl-cobalamin, and hydroxy-cobalamin. The dietary proteins bound to the R-protein, which is made by the salivary glands and is present in gastric juice, are liberated from cobalamin in the acid environment of the stomach. The affinity of cobalamin for R protein is much higher than for the intrinsic factor. A lack of vitamin B12 can cause both hematologic and gastroenterological symptoms. In many cases, neurologic symptoms are seen regardless of how long a vitamin B12 deficit has existed.[34] [35]

Causes of vitamin B12 deficiency
Inadequate intake
• – Vegetarians (rare) Malabsorption
• Deficiency of intrinsic factor
– Pernicious anemia
– Gastrectomy
– Congenital (extremely rare)
• Diseases of the terminal ileum
– Sprue
– Crohn’s disease
– Extensive resections
– Selective malabsorption (Imerslund syndrome – extremely rare)
• Parasite-induced deficiencies
– Fish tapeworm

– Bacteria (“blind loop syndrome”)
• Drug-induced
– PAS, neomycin

TREATMENT:

Over the last 30 years, therapeutic options for treating anemias have grown dramatically. Blood component therapy is a viable and risk-free option. Recombinant EPO as an anemia management adjunct has improved the lives of dialysis patients with chronic renal failure and reduced transfusion needs in anemic cancer patients receiving chemotherapy. Patients with inherited globin synthesis disorders or globin gene mutations, such as sickle cell disease, may eventually benefit from the successful implementation of targeted genetic therapy. [34]

The documented cause(s) of the anemia determines the appropriate treatment, whether the anemia is acute or gradual in onset. The cause of anemia is frequently multifactorial. A patient with severe rheumatoid arthritis who has

been taking anti-inflammatory drugs, for example, may have hypoproliferative anemia due to chronic inflammation as well as chronic blood loss due to intermittent gastrointestinal bleeding. Before and during the treatment of any anemia, it is critical to thoroughly assess the patient's iron status.[35]

AIM AND OBJECTIVES

1. To look for a correlation between the lipid profile and anemia.

2. If a correlation that is statistically significant is established, look for the variations of the lipid profile with the different types and the various grades of anemia based on severity.

METHODOLOGY

Study Design: Cross-sectional case-control study

Study Period: 6 months

Study Centre: Dept of General Medicine, Government Kilpauk Medical College, Chennai.

Total sample size: 146

Inclusion criteria:

Cases: Patients with anemia, irrespective of the type, matched for age, sex, BMI., without the conditions mentioned in the exclusion criteria

Controls: Patients without anemia, matched for age, sex, and BMI., without the conditions mentioned in the exclusion criteria.

Exclusion criteria:

- Individuals with chronic diseases like systemic hypertension, diabetes mellitus, prior myocardial infarction, prior cerebrovascular accidents, hypothyroidism, and nephrotic syndrome.
- Patients with liver diseases.

- Patients on lipid-lowering medications, glucocorticoids, diuretics, and beta blockers were excluded from the study.
- Patients with retroviral diseases.
- Patients with a BMI>25.

Methodology:

The participants are enrolled in the study after obtaining their written informed consent. All proven cases of anemia, irrespective of the cause and those satisfying the exclusion criteria, were included as cases.

Case definition: Anemia, as per the World Health Organisation (WHO), is defined as Hemoglobin (Hb) levels <12.0 g/dL in women and <13.0 g/dL in men

Classification	Normal	Mild	Moderate	Severe
MEN	13 g/dl	12-12.9 g/dl	9-11.9 g/dl	<9 g/dl
WOMEN	12g/dl	10-11.9 g/dl	7-9.9 g/dl	<7 g/dl

The following investigations are done in all participants: complete hemogram, random blood glucose, serum creatinine, liver function tests, TSH, fasting lipid profile, viral markers, and peripheral smear. Total cholesterol, triglycerides, HDL, and LDL values are obtained. VLDL obtained by formula TGL/5.

Statistical Analysis: For categorical variables, frequencies/percentages will be calculated and continuous variables will be expressed as mean ± standard deviation. Comparisons among groups will be performed using ANOVA/student t-test. All tests will be performed using a two-tailed test at a significance level of 0.05. SPSS for Windows version 20 (SPSS, Inc., Chicago, IL, USA) will be employed for all statistical analyses.

RESULTS

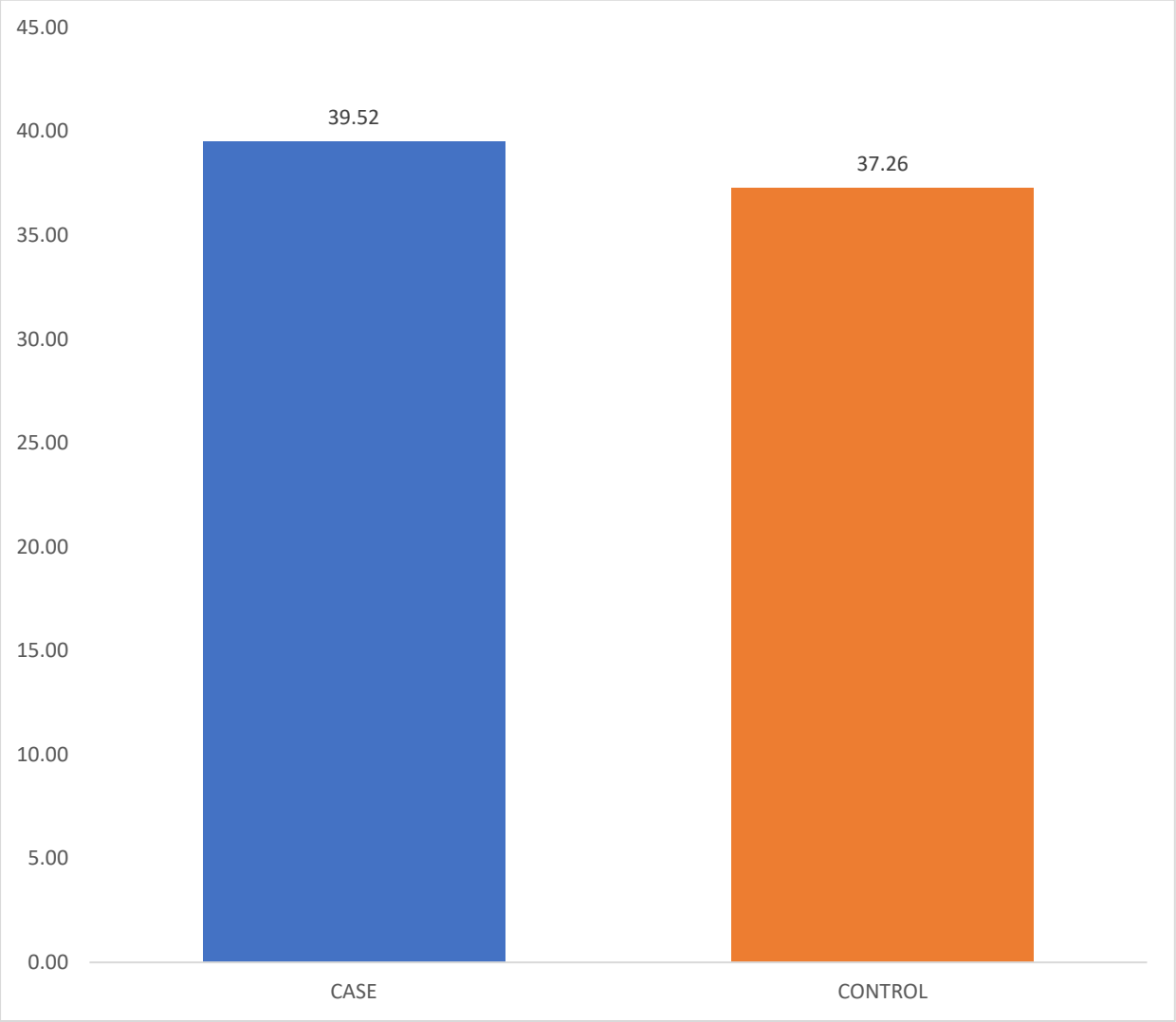
1. Age distribution

A mean age of 39.52 (SD = 17.08) was reported in the case group whereas, a mean age of 37.26 (SD = 15.75) was seen in the control group (p-value = 0.407).

Table 1 – Mean age of patients

Group		Mean	Std. Deviation	P value
AGE	CASE	39.52	17.08	0.407
	CONTROL	37.26	15.75	

Figure 1 – Participant's mean age



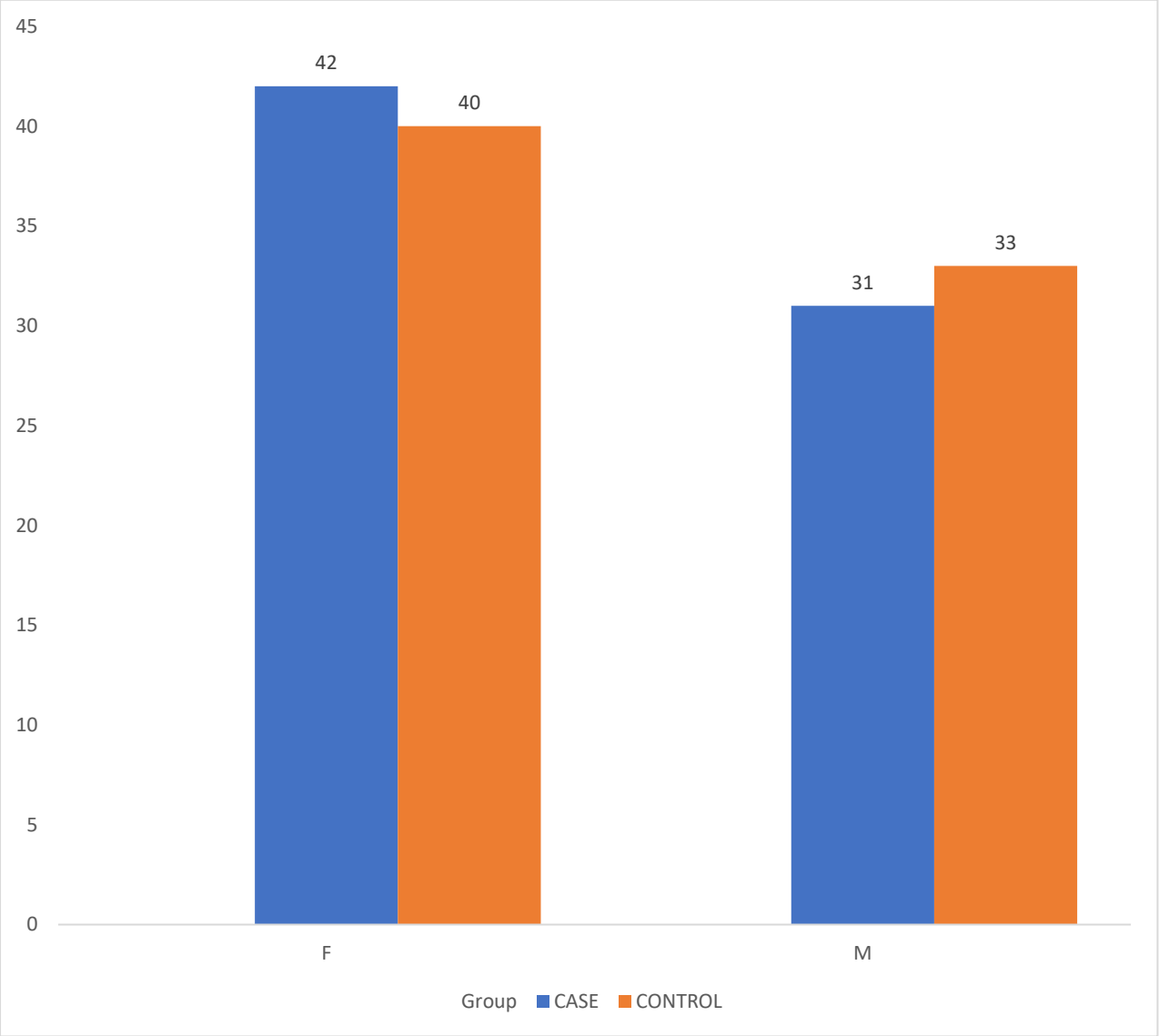
2. Gender distribution of patients

A female predominance was reported in the current study with 82 females (56.2%) and males were 64 (43.8%). In the case group, 42 females were seen and 40 males were included, followed by 31 females and 33 males in the control group.

Table 2 – Gender distribution of patients

			Group		Total	P value
			CASE	CONTROL		
GENDER	F	Count	42	40	82	0.739
		% within Group	57.5%	54.8%	56.2%	
	M	Count	31	33	64	
		% within Group	42.5%	45.2%	43.8%	
Total		Count	73	73	146	
		% within Group	100.0%	100.0%	100.0%	

Figure 2 – Participant's gender distribution in case and control group



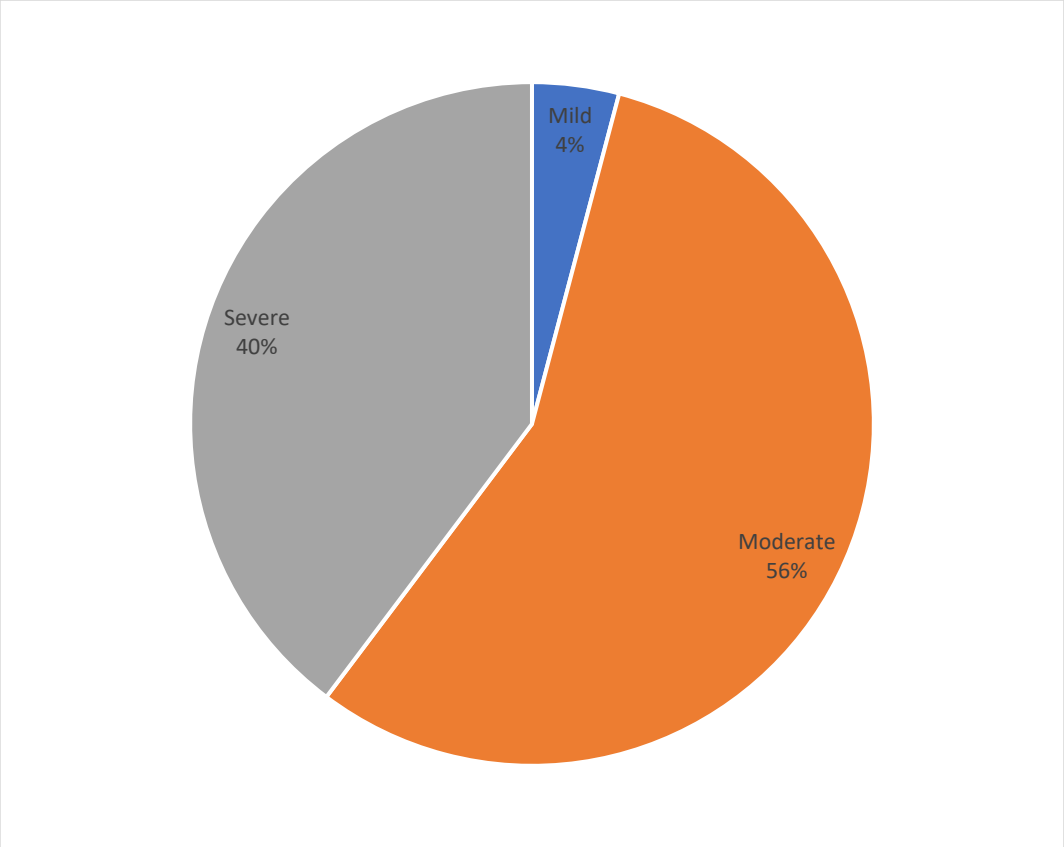
3. Anemia severity in the case group

Out of 73 patients in the case group, 41 patients presented with moderate anemia (56.2%), followed by severe anemia in 29 patients (39.7%).

Table 3 – Anemia severity

ANEMIA SEVERITY	Frequency	Percent
Mild	3	4.1%
Moderate	41	56.2%
Severe	29	39.7%
Total	73	100.0%

Figure 3 – Anemia severity % distribution



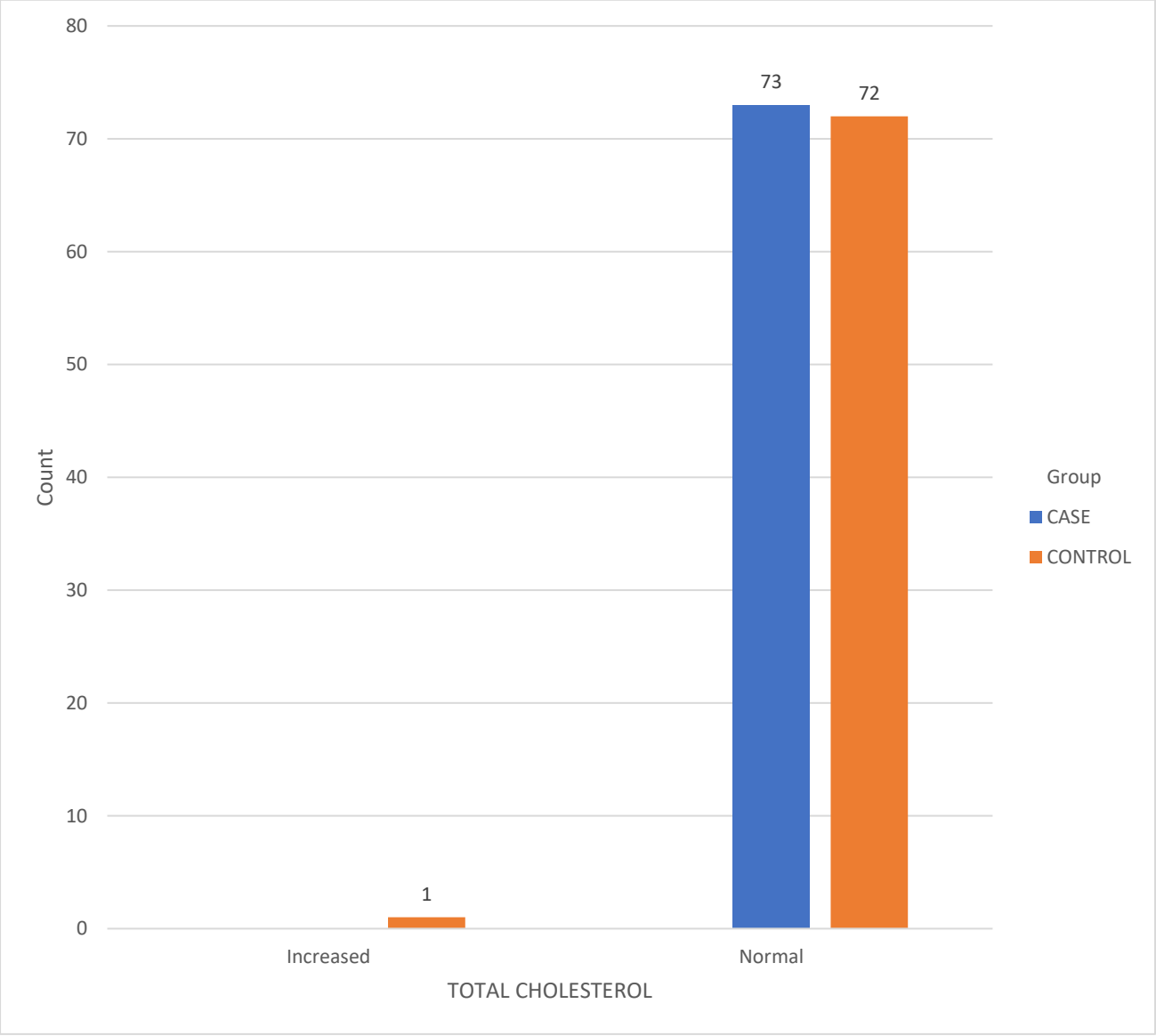
4. **Total cholesterol levels**

The study did not report a significant difference in cholesterol levels, as 73 patients in the case group were reported with normal cholesterol levels. In addition, 72 patients in the control group were reported with normal cholesterol levels.

Table 4 – Total cholesterol levels in patients

			Group		Total	P value
			CASE	CONTROL		
TOTAL CHOLESTEROL	Increased	Count	0	1	1	0.316
		% within Group	0.0%	1.4%	0.7%	
	Normal	Count	73	72	145	
		% within Group	100.0%	98.6%	99.3%	
Total		Count	73	73	146	
		% within Group	100.0%	100.0%	100.0 %	

Figure 4 – Total cholesterol levels in patients



5. Triglycerides levels

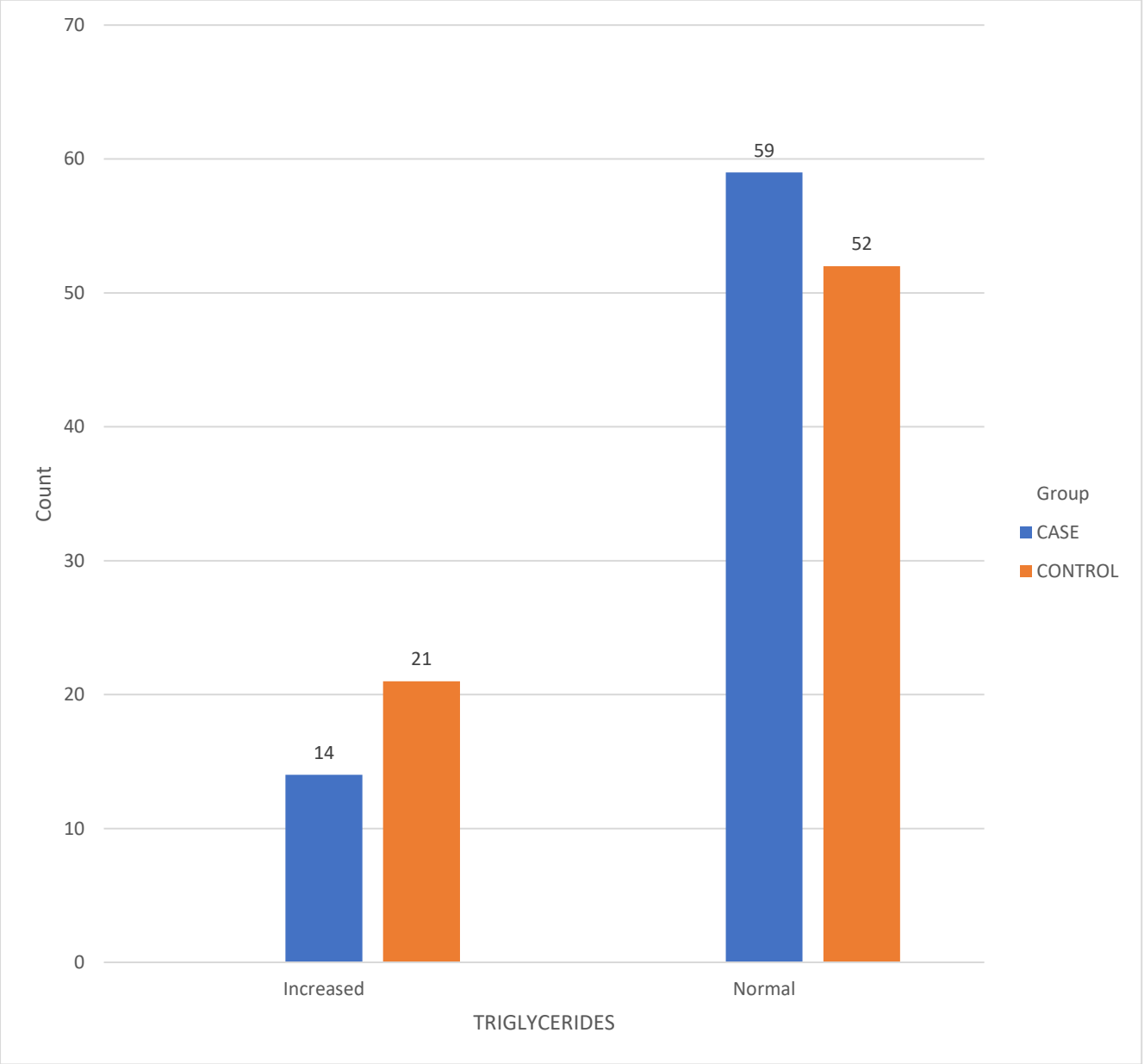
A total of 14 patients were reported with increased triglyceride levels in the case study group and 21 patients in the control group were reported with

high levels of triglycerides. The study did not report a significant difference in triglyceride levels in patients.

Table 5 – Triglycerides in patients

			Group		Total	P value
			CASE	CONTROL		
TRIGLYCERIDES	Increased	Count	14	21	35	0.175
		% within Group	19.2%	28.8%	24.0 %	
	Normal	Count	59	52	111	
		% within Group	80.8%	71.2%	76.0 %	
Total		Count	73	73	146	
		% within Group	100.0 %	100.0%	100.0 %	

Figure 5 – Triglycerides level in patients



6. HDL, LDL, and VLDL levels in patients

All participants in the current study were observed with normal levels of HDL in both groups.

Table 6 – HDL levels in patients

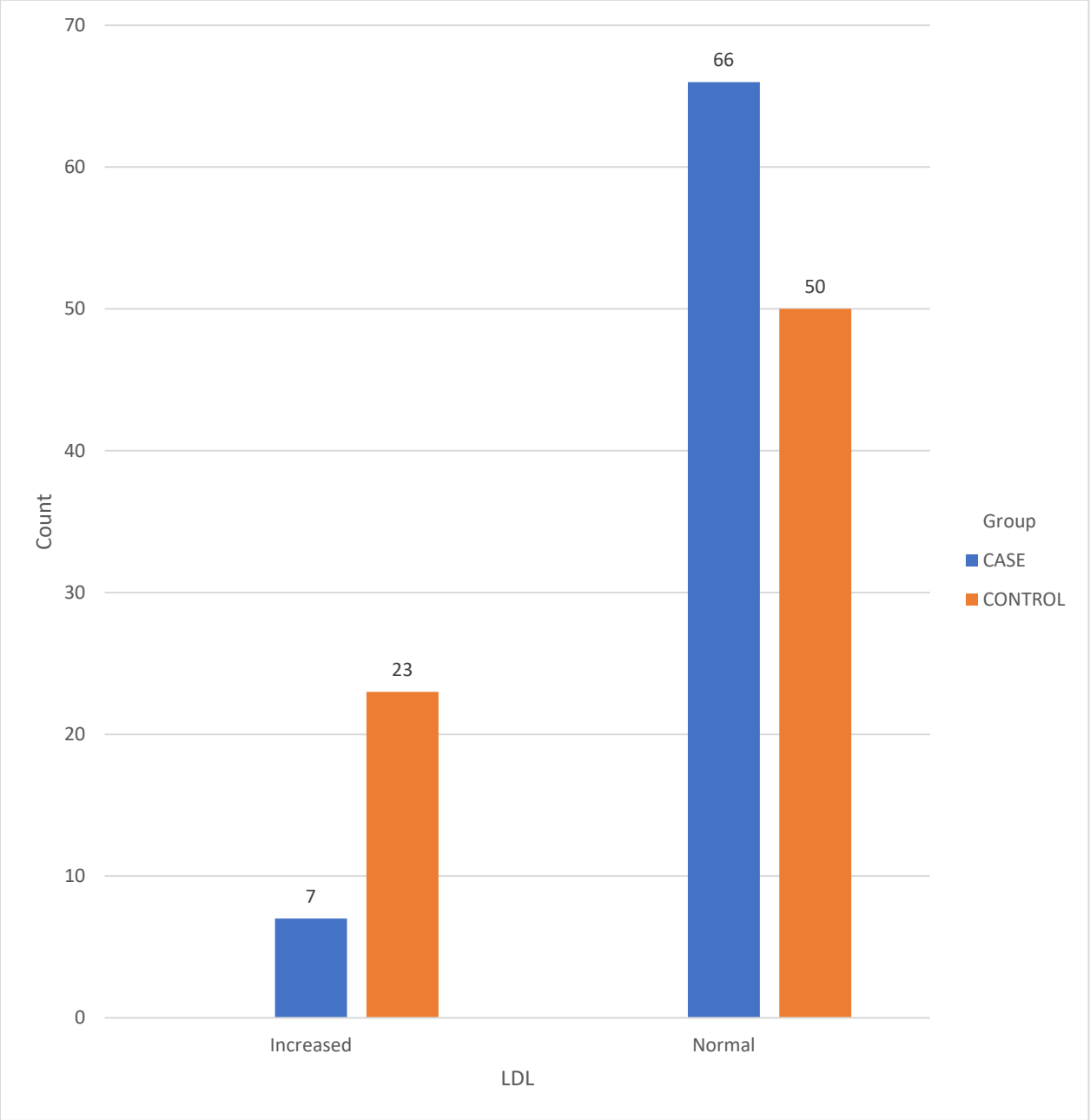
			Group		Total
			CASE	CONTROL	
HDL	Normal	Count	73	73	146
		% within Group	100.0%	100.0%	100.0%
Total		Count	73	73	146
		% within Group	100.0%	100.0%	100.0%

Table 7 – LDL level in patients

			Group		Total	P value
			CASE	CONTROL		
LDL	Increased	Count	7	23	30	0.001
		% within Group	9.6%	31.5%	20.5%	
	Normal	Count	66	50	116	
		% within Group	90.4%	68.5%	79.5%	
Total		Count	73	73	146	
		% within Group	100.0%	100.0%	100.0 %	

The study reports a significant difference in the LDL levels in patients in the case and control groups (p-value = 0.001). Elevated levels of LDL were reported in 7 patients in the case group and 23patientss in the control group.

Figure 6 – LDL levels in patients



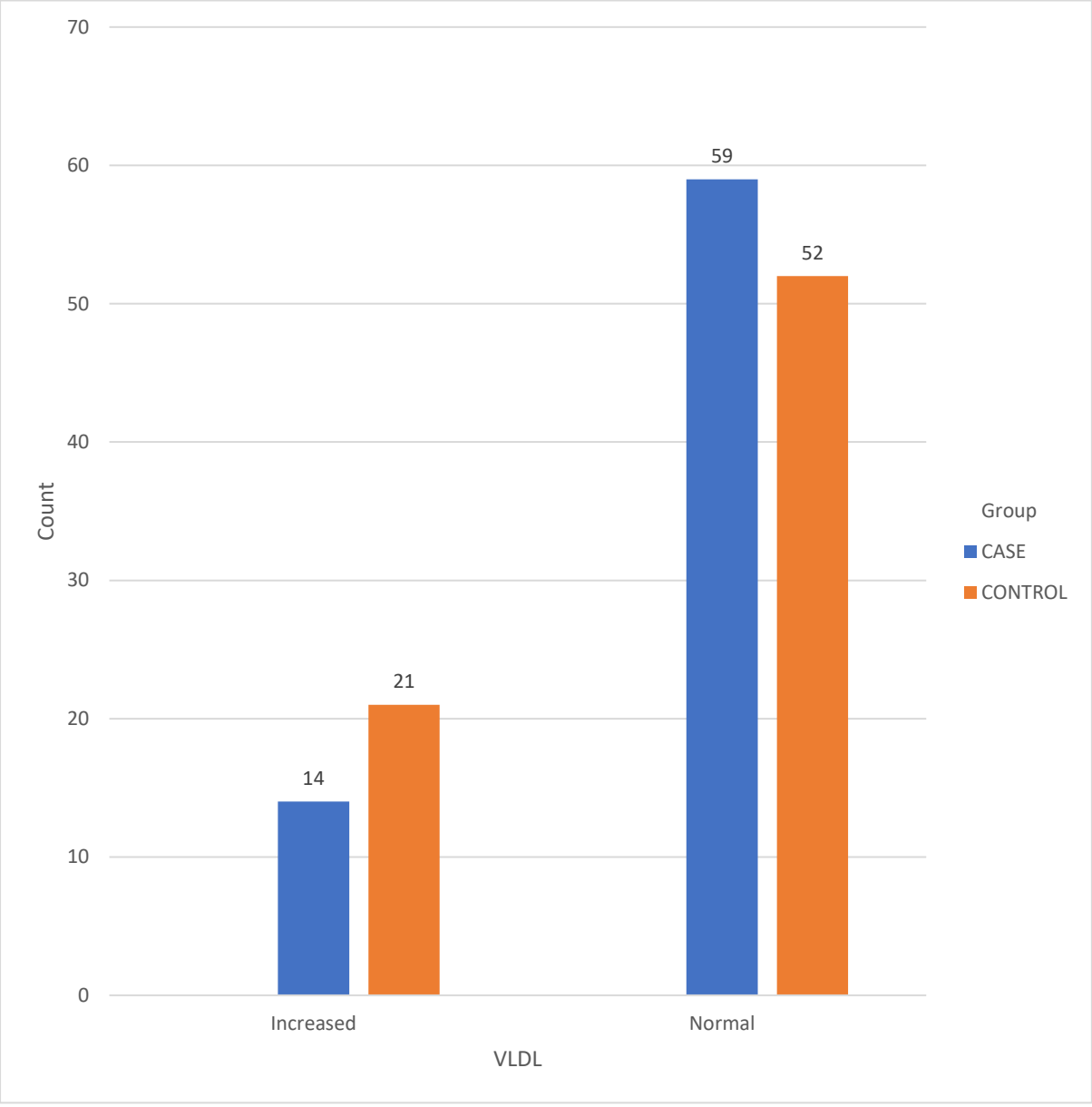
An increased level of VLDL was reported in 14 patients in the case group and 21 patients with high levels of VLDL were reported in the control group.

However, the study did not reports a significant difference between the two groups.

Table 8 – VLDL levels in patients

			Group		Total	P value
			CASE	CONTROL		
VLDL	Increased	Count	14	21	35	0.175
		% within Group	19.2%	28.8%	24.0%	
	Normal	Count	59	52	111	
		% within Group	80.8%	71.2%	76.0%	
Total		Count	73	73	146	
		% within Group	100.0 %	100.0 %	100.0 %	

Figure 7 – VLDL level in participants



7. **Anemia severity correlation with cholesterol levels**

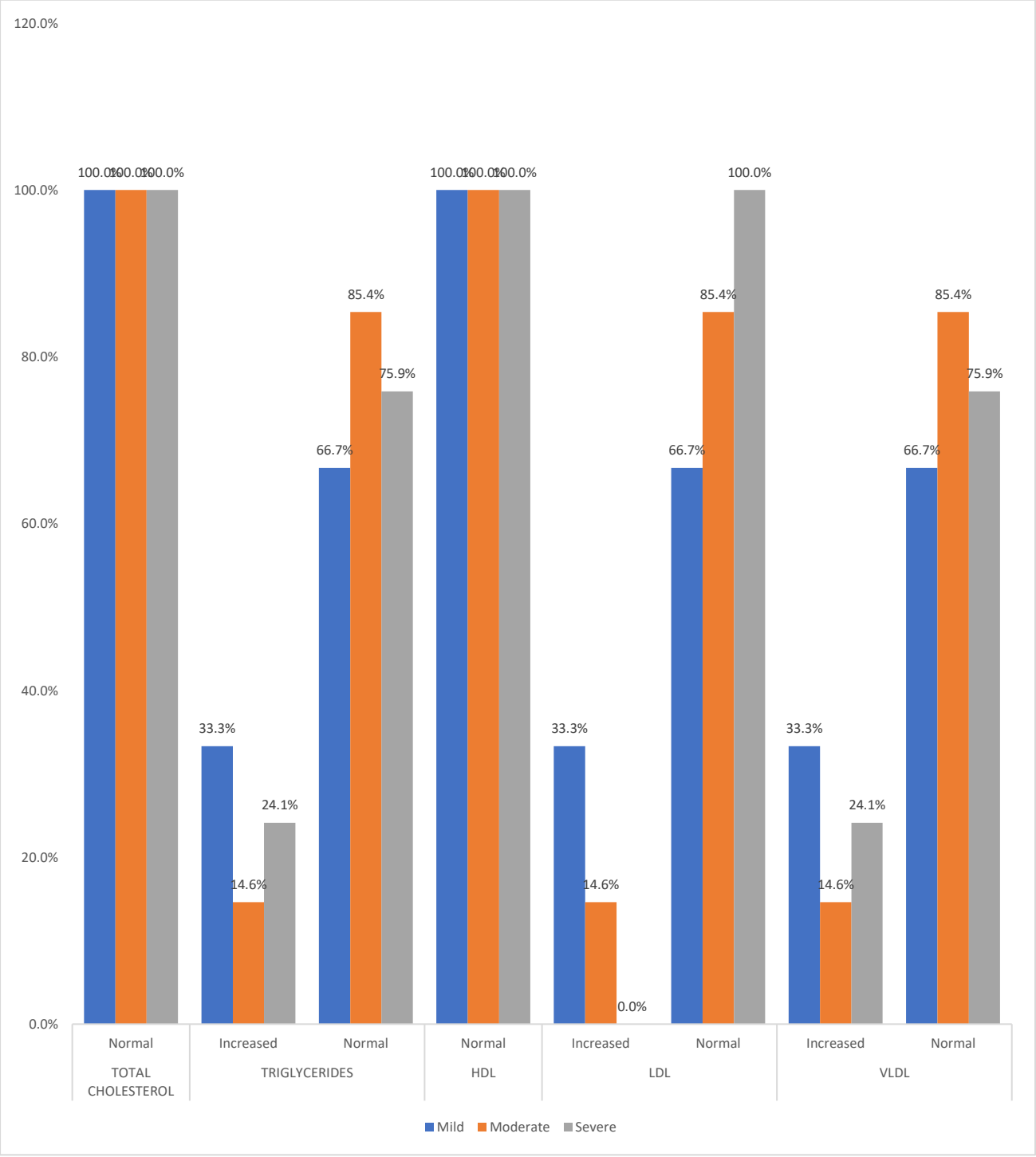
The current study reports that severe anemia patients were seen with increased triglyceride levels (24.1%), followed by increased VLDL levels in

24.1% of the patients. Patients with moderate anemic conditions were seen with elevated levels of triglycerides (14.6%). An increased level of LDL was seen in patients with moderate anemia (14.6%).

Table 9 – Anemia severity correlation with cholesterol levels

Case		ANEMIA SEVERITY						P value
		Mild		Moderate		Severe		
		Count	Column N %	Count	Column N %	Count	Column N %	
TOTAL CHOLESTEROL	Normal	3	100.0%	41	100.0%	29	100.0%	n.a
TRIGLYCERIDES	Increased	1	33.3%	6	14.6%	7	24.1%	0.498
	Normal	2	66.7%	35	85.4%	22	75.9%	
HDL	Normal	3	100.0%	41	100.0%	29	100.0%	n/a
LDL	Increased	1	33.3%	6	14.6%	0	0.0%	0.044
	Normal	2	66.7%	35	85.4%	29	100.0%	
VLDL	Increased	1	33.3%	6	14.6%	7	24.1%	0.498
	Normal	2	66.7%	35	85.4%	22	75.9%	

Figure 8 – Anemia correlation with cholesterol levels



8. Mean findings for cholesterol levels in the case and control group

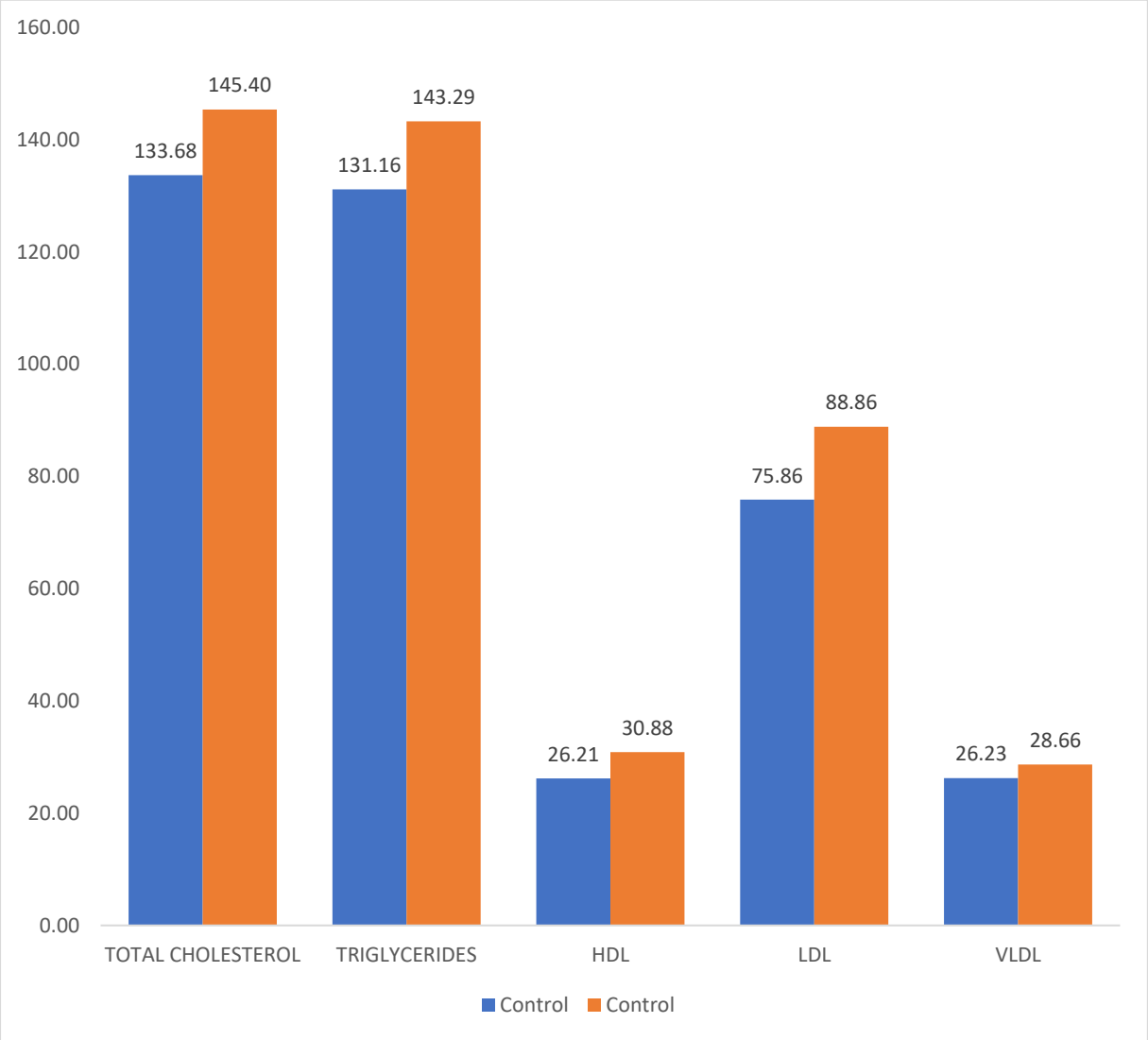
Patients in the case group were observed with mean total cholesterol of 133.68 (SD = 19.03), triglycerides 131.16 (25.32), HDL 26.21 (5.53), LDL of 75.86 (SD = 20.70), and VLDL of 26.23 (SD = 5.06). In contrast, the control group reports reported total cholesterol mean of 145.40 (SD = 25.81), triglycerides level of 143.29 (SD = 40.45), HDL level of 30.88 (SD = 6.52), LDL levels of 88.86 (SD = 17.77), and VLDL level of 28.66 (SD = 8.09).

The study finds that the control group significantly had elevated levels of cholesterol levels when compared with the case group and reports a statistical difference in HDL and LDL levels for the case and control group respectively (p-value <0.0001).

Table 10 – Comparison of case and control group cholesterol levels

	Group				P value
	CASE		CONTROL		
	Mean	Standard Deviation	Mean	Standard Deviation	
TOTAL CHOLESTEROL	133.68	19.03	145.40	25.81	0.002
TRIGLYCERIDES	131.16	25.32	143.29	40.45	0.032
HDL	26.21	5.53	30.88	6.52	<0.0001
LDL	75.86	20.70	88.86	17.77	<0.0001
VLDL	26.23	5.06	28.66	8.09	0.032

Figure 9 – Comparison of case and control group



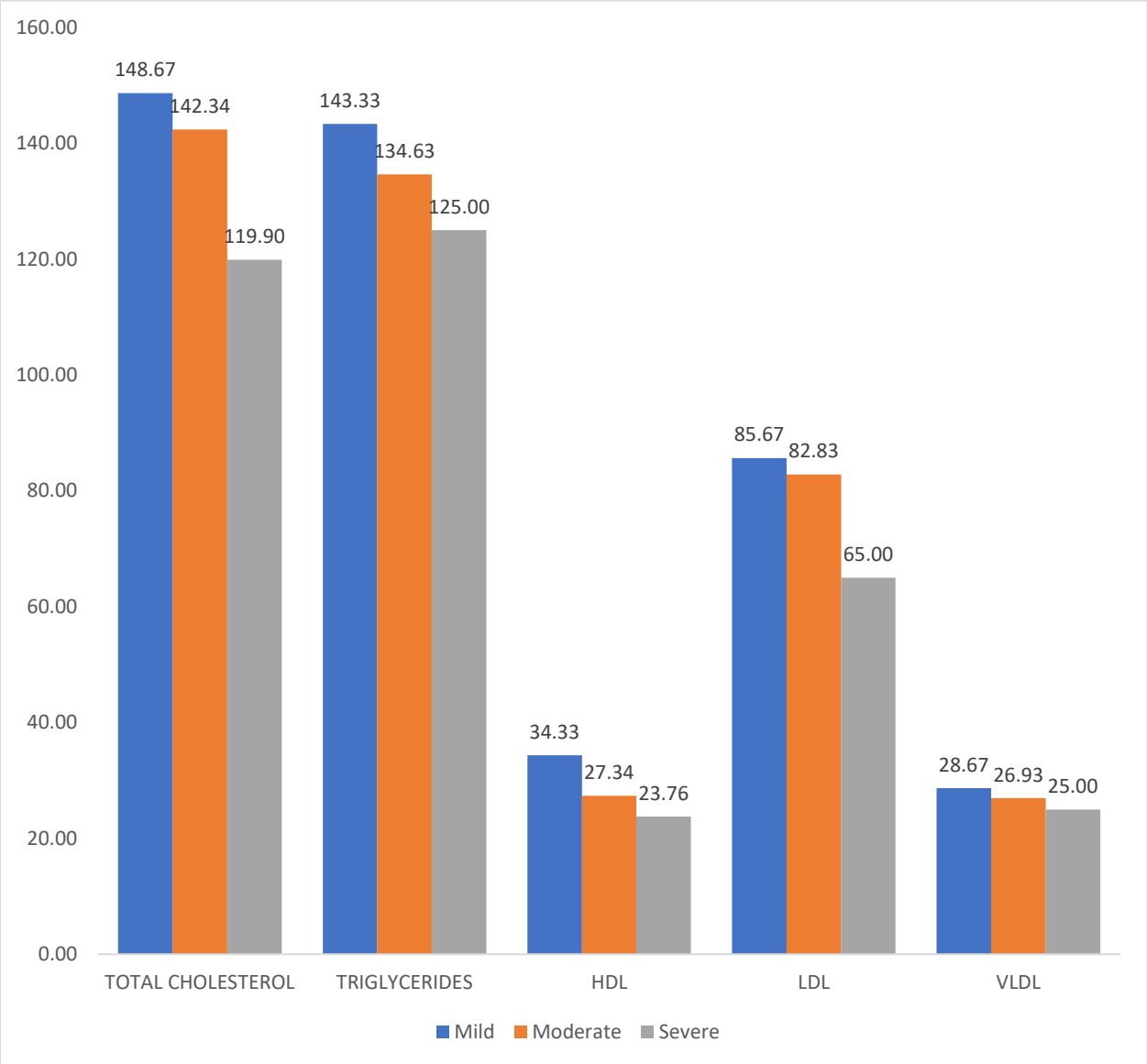
9. Comparison of anemia severity in the case group

The study reports a significant difference concerning the total cholesterol levels among patients with mild, moderate, and severe anemia (p-value <0.0001). In addition, the study also reports a significant difference in the HDL and LDL levels in the case group with a p-value of 0.001.

Table 11 – Case group comparison with anemia

Case	ANEMIA SEVERITY						P value
	Mild		Moderate		Severe		
	Mean	Standard Deviation	Mean	Standard Deviation	Mean	Standard Deviation	
TOTAL CHOLESTEROL	148.67	18.45	142.34	16.98	119.90	12.82	<0.0001
TRIGLYCERIDE	143.33	22.55	134.63	25.18	125.00	25.18	0.206
HDL	34.33	5.69	27.34	5.74	23.76	3.73	0.001
LDL	85.67	17.62	82.83	19.74	65.00	17.89	0.001
VLDL	28.67	4.51	26.93	5.04	25.00	5.04	206

Figure 10 – Comparison of anemia severity in the case group



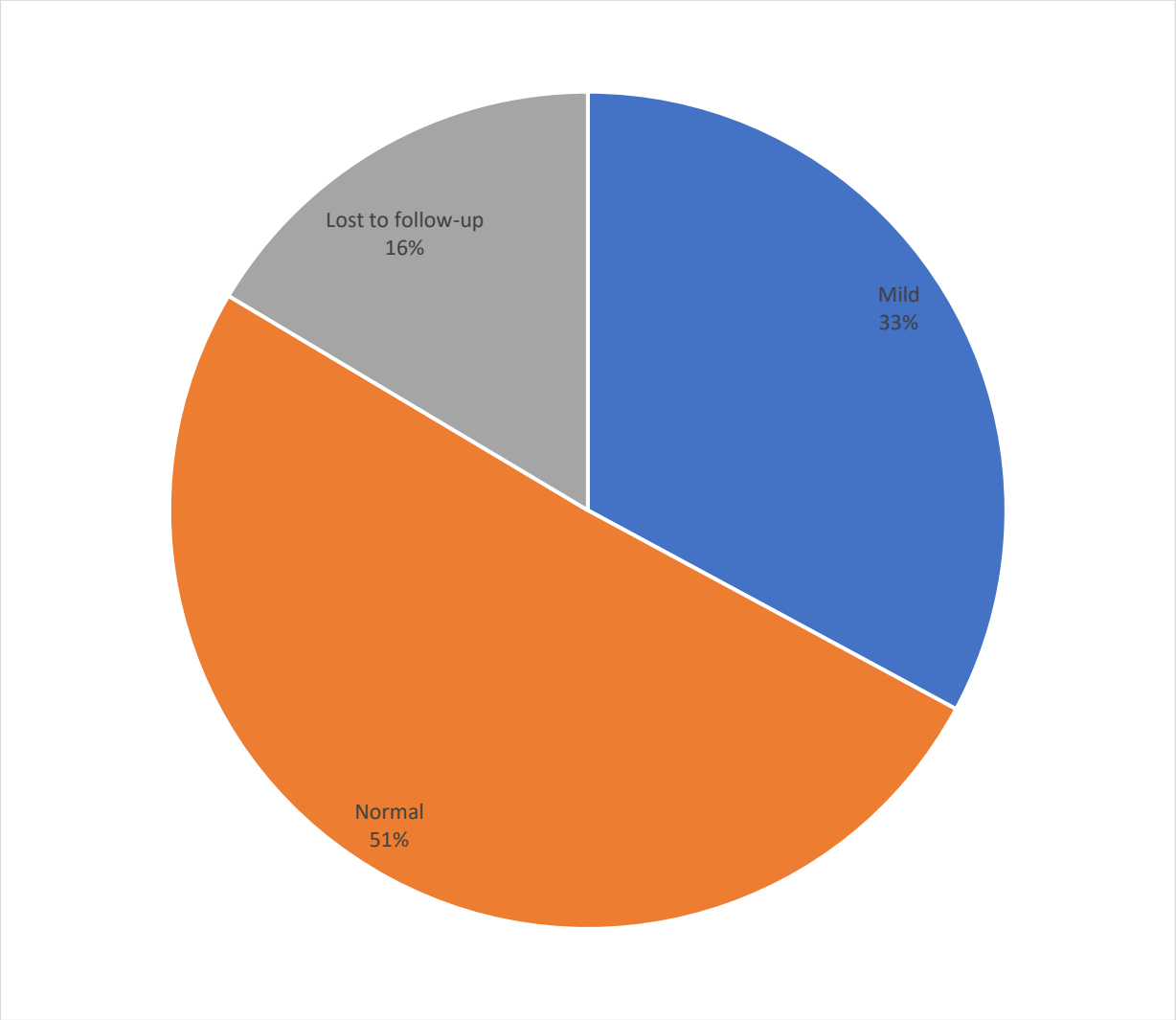
10.Follow-up of anemia severity in the case group

A 3-month follow-up revealed mild anemia in 32.9% of the patients, and 50.7% of patients were not anemia anymore. However, a loss to follow-up was reported in 12 patients (16.4%).

Table 12 – Anemia severity follow-up

ANEMIA SEVERITY_ 3 MONTHS	Frequency	Percent
Mild	24	32.9%
Normal	37	50.7%
Lost to follow-up	12	16.4%
Total	73	100.0%

Figure 11 - % distribution of 3 months follow-up



11. **Correlation of anemia severity (3 months) with cholesterol levels**

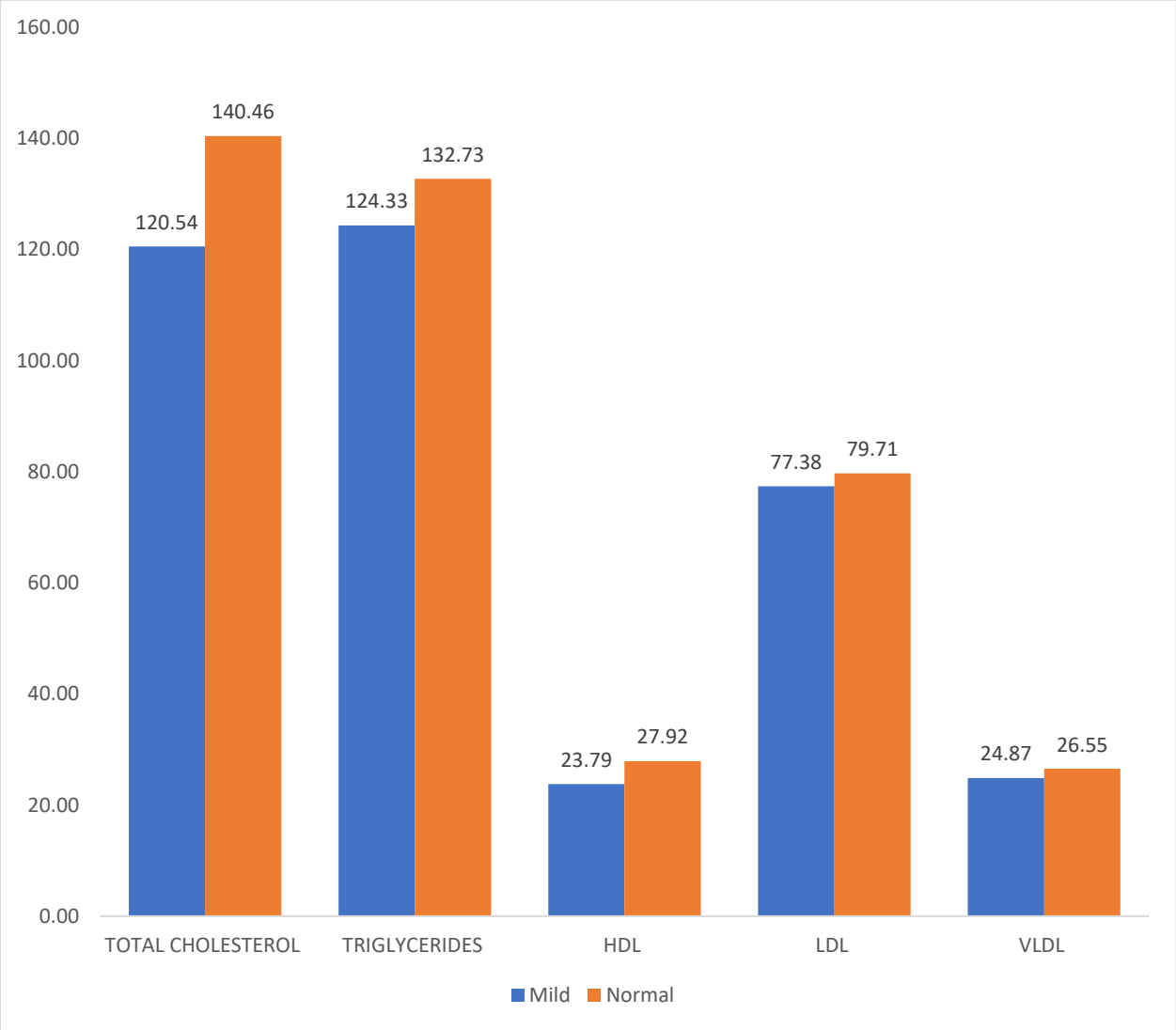
50.7% of patients after a 3-month follow-up were reported with no amenia, however, the prevalence of mild anemia was seen with a mean of 120.54

(SD = 14.44) in 32.9% of patients. The study reports a significant difference in total cholesterol levels with the severity of anemia after 3 months.

Table 13 – Anemia severity correlation with 3 months follow-up

Case	ANEMIA SEVERITY_3 MONTHS				P value
	Mild		Normal		
	Mean	Standard Deviation	Mean	Standard Deviation	
TOTAL CHOLESTEROL	120.54	14.44	140.46	17.59	<0.0001
TRIGLYCERIDES	124.33	21.22	132.73	22.49	0.151
HDL	23.79	3.96	27.92	6.34	0.006
LDL	77.38	17.08	79.71	16.84	0.603
VLDL	24.87	4.24	26.55	4.50	0.151

Figure 12 – Severity of anemia comparison with 3 months follow-up



DISCUSSION

The current study aimed to analyse the lipid profile in anemic patients compared to healthy patients. The current study was conducted among 146 patients out of which the mean of patients was reported 39.52 (SD = 17.08) in the case group and 37.26 (SD = 15.75) in the control group. A female predominance was reported with a total of 82 females (56.2%) out of which 42 females were in the case group and 31 females were in the control group. The participants included in the current study reported severe anemic conditions (39.7%), moderate anemia in 56.2%, and mild 4.1%. During the diagnosis of the lipid profile, an increased triglyceride level was seen in 14 patients in the case group and 21 in the control group, followed by an elevated level of LDL in 7 patients within the case group and 23 patients in the control group. The study reports a significant statistical difference in the LDL levels of the patients comparing the case and control group (p-value = 0.001). In addition, increased VLDL levels were reported in 14 case patients and 21 control group patients.

Elevated levels of triglycerides and VLDL levels were reported in the participants out of which severe anemic patients consist of 24.1% each

respectively, followed by moderate anemic patients in which high levels of triglycerides (14.6%), LDL (14.6%), and VLDL (14.6%) was observed. The comparison of the case and control group revealed a significant difference in the concentration of HDL and LDL levels with a p-value <0.0001. The current study findings state that patients with anemia affect the overall lipid profile levels in a patient depending on the severity of the anemic condition. The levels of lipid profile have been decreased depending on the severity of the disease, for instance, the mean HDL levels reported in mild anemia was 34.33 (SD = 5.69) whereas, in severely anemic patients HDL was reported with a mean of 23.76 (SD = 3.73). This study finding was reported in every lipid profile including, total cholesterol, triglycerides, LDL, and VLDL. The severity of anemia can lead to decreased levels of lipid profile which was also reported by Choi et.al. who reported a correlation between iron deficiency anemia, lipid profile, and hemoglobin levels. [36]

The study findings by Elwood and Mahler among 4070 women also reported a significant difference in cholesterol levels in patients with hemoglobin levels below 10.5 g/dL. [37]

A follow-up was conducted to better correlate the relationship between anemia severity and lipid profile. A 3-month follow-up revealed that 50.7% of the patients were not anemic and a prevalence of anemia was seen in 32.9% with a mean of 120.54 (SD = 14.44) in mild anemic condition. A significant difference was seen between the total cholesterol levels with a p-value <0.0001. This finding signifies that as the severity of anemia decreases the lipid profiles in patients were normalized adequately.

The clinical study conducted by Ohira et.al also reported that patients with low levels of hemoglobin (Hb = < 9.0 g/100ml) were reported with decreased levels of cholesterol levels, which was not reported in patients with normal Hb levels. [38]

A direct correlation was reported in our study with a significant statistical difference in the anemic condition and lipid profile. The study conducted by Mashid et.al. also reported that patients with iron deficiency anemia had lower levels of cholesterol levels when compared with the control group. [39] The study reported that lipid profiles were more affected in elderly patients with low hemoglobin levels.

In conclusion, a direct correlation can be seen in patients with anemia which adversely affects the lipid profile. A direct relationship has been observed concerning the severity of anemia and the lipid profiles of patients.

The lipid profile can be used as a prognostic marker to assess the anemic condition in patients and to determine the possible therapeutic outcome among patients and vice versa. An altered lipid profile can be a risk factor for the development of atherosclerosis disease in anemic patients. Hence clinicians should be aware of the prevalence of anemia and altered lipid profiles in patients.

SUMMARY

- A female predominance was reported in the current study with 82 females (56.2%) and males were 64 (43.8%). In the case group, 42 females were seen and 40 males were included, followed by 31 females and 33 males in the control group.
- Out of 73 patients in the case group, 41 patients presented with moderate anemia (56.2%), followed by severe anemia in 29 patients (39.7%).
- All participants in the current study were observed with normal levels of HDL in both groups.
- The study reports a significant difference in the LDL levels in patients in the case and control groups (p-value = 0.001). Elevated levels of LDL were reported in 7 patients in the case group and 23patientss in the control group.
- The current study reports that severe anemia patients were seen with increased triglyceride levels (24.1%), followed by increased VLDL levels in 24.1% of the patients. Patients with moderate anemic conditions were

seen with elevated levels of triglycerides (14.6%). An increased level of LDL was seen in patients with moderate anemia (14.6%).

- The study finds that the control group significantly had elevated levels of cholesterol levels when compared with the case group and reports a statistical difference in HDL and LDL levels for the case and control group respectively (p-value <0.0001).
- The study reports a significant difference concerning the total cholesterol levels among patients with mild, moderate, and severe anemia (p-value <0.0001). In addition, the study also reports a significant difference in the HDL and LDL levels in the case group with a p-value of 0.001.
- A 3-month follow-up revealed mild anemia in 32.9% of the patients, and 50.7% of patients were not anemia anymore. However, a loss to follow-up was reported in 12 patients (16.4%).
- 50.7% of patients after a 3-month follow-up were reported with no amenia, however, the prevalence of mild anemia was seen with a mean of 120.54 (SD = 14.44) in 32.9% of patients. The study reports a significant

difference in total cholesterol levels with the severity of anemia after 3 months.

CONCLUSION

Anemia and lipid profile pose a direct relationship that adversely affects the clinical condition of patients. The current study was conducted among case and control groups with a mean age of 39.52 (SD = 17.08) and 37.26 (SD = 15.75) respectively with a female predominance of 56.2%.

The severity of anemic condition differed in the participants out of which 56.2% were moderately anemic and 39.7% were severely anemic. The comparison of LDL levels in the case and control group revealed a significant difference which revealed that patients with anemia are more prone to a decreased level of lipid profile. A significant difference was reported in the comparison of case and control groups concerning the differences in HDL and LDL levels among patients (p-value <0.0001). In addition, the severity of anemia and the decreased levels of total cholesterol in the case group with a significant difference (p-value <0.0001) which demonstrates the correlation of anemic condition with lipid profile.

To assess further, the study also conducted a 3-month follow-up for which reported 50.7% of normalized patients and 32.9% with the mild anemic

condition. The study finds a significant correlation between total cholesterol levels and a 3-month severity assessment of anemia (p-value <0.0001).

To conclude, the current study reports a direct relationship between anemia and the prevalence of altered lipid profiles among patients affected majorly with decreased levels of total cholesterol, HDL, and LDL levels. Hence, patients with anemic conditions can be tested for a lipid profile to prevent further at-risk complications. Similarly, low lipid levels in patients with unknown etiology can be correlated with the incidence of anemia.

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CONSENT FORM – SERUM LIPID PROFILE AND ITS CORRELATION WITH IRON DEFICIENCY ANEMIA

Patient Name :
Patient Age :
OP number :

Patient may check in ( )these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ☐

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ☐

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well-being or any unexpected or unusual symptoms. ☐

I hereby consent to participate in this study. ☐

I hereby give permission to undergo complete clinical examination and diagnostic tests including haematological, biochemical, radiological tests.

Signature/thumb impression:

Patient Name and Address: Place: Date:

Signature of investigator :

Study investigator's Name : Place: Date :

ந ோயோளி ஒப்புதல் படிவம்

ஆய்வு விவரம்: "வளர்சிததமோற்ற ந ோய்க்குறிகள் மற்றும் இதய ோள அபோயக் கோரணிகள் கோண்ட கழுத்துப் பகுதியில் உள்ள சங்கத்தத மதிப்பிடுவதற்கு-ஒரு குறுக்கு கவட்டு ஆய்வு"

ஆய்வு தமயம்: அரசு கீழ்ப்போக்கம் மருத்துவக் கல்லூரி, கசன்தை

ந ோயோளி கபயர்:

ந ோயோளி வயது

புறந ோயோளி எண்:

ந ோயோளி இந்தப் கபட்டிகள் உள்நள கசய்யலோ

இந்நல உள்ள ஆய்வின் தடமுதறயின் நி ோக்கத்தத ோள் புரிந்துகோண்நடன் ளை உறுதிப்படுத்துகிநறன். நகள்வி நகட்பதற்கு ளைக்கு வோய்ப்பு உள்ளது, ளைது முழு திருப்திக்கு ளைது நகள்விகளும் சந்நதகங்களும் பதிலளிக்கப்பட்டை.

இந்த ஆய்வில் ோள் பங்நகற்பது தன்ளைோர்வத்திலோைது மற்றும் ளைது சட்டபூர்வ உரிதமகள் போதிக்கப்படோமல் எந்த ந ரத்திலும் எந்தக் கோரணமும் கூறோமல் விலகிக்கோள்வதற்கு ோள் சுதந்திரமோக உள்நளன் என்பதத ோள் புரிந்துகோண்நடன்.

சிகிச்தசயக ஆய்நவ வழங்கும் பிறவம், ஆய்நவ வழங்கும் பிறவத்தின் சோர்போக பணிபுரியும் மற்றவர்கள், க றிமுதறக் குழு மற்றும் ஒழுங்குமுதற அதமப்புகள், தற்நபோததய ஆய்வு மற்றும் அவற்றில் இருக்கக் கூடிய எந்த ஒரு ஆநரோய்ச்சியிலும் ளைது உடல் லப் பதிநவடுகதளப் போர்ப்பதற்கு ளைது அனுமதி நததவயில்தல என்று

ோள் புரிந்துகோண்நடன். அது கதோடர்போக டத்தப்படும், ோள் ஆய்விலிருந்து விலகிைோலும் கூட இந்த அனுகதல ஒப்புக்கோள்கிநறன். ளைலும், சட்டத்தின் கீழ்

நததவப்பட்நடோல் ஒழிய, மூன்நறோம் தரப்பிைருக்கு கவளியிடப்படும் அல்லது பிரசுரிக்கப்படும் எந்தத் தகவல்களில் ளைது அதடயோளம் கவளிப்படுத்தப்படோது என்பதத ோள் புரிந்துகோண்நடன். இந்த ஆய்விலிருந்து எழும் தரவுகள் அல்லது முடிவுகளின் பயன்போட்தட கட்டுப்படுத்தோமல் இருக்க ோள் ஒப்புக்கோள்கிநறன்

நமநல உள்ள ஆய்வில் பங்நகற்க ோள் ஒப்புக்கோள்கிநறன். ஆய்வின் நபோது அளிக்கப்பட்ட அறிவுதரகளுக்கு இணங்க நவண்டும். ஆய்வுக் குழுவுடன் விகவோசத்துடன் ஒத்துதழக்கவும், ளைது உடல் லத்தில் அல்லது ல்வோழ்வுத் தில் ஏநதனும் சர்நகடு ஏற்பட்டோல் அல்லது எதிர்போரோத அல்லது வழக்கத்திற்கு மோறோை அறிகுறிகள்

இந்த ஆய்வில் பங்நகற்பதற்கு ோள் ஒப்புதல் அளிக்கிநறன்.

இரத்தவியல், உயிர்நவதியியல், நரடிநயோலோஜிக்கல் பரிநசோததகள் உட்பட முழுதமயோை மருத்துவ பரிநசோததை மற்றும் பகுப்போய்வு பரிநசோததைகதள நமற்கோள்ள ோள் அனுமதி கோடுக்கிநறன்.

ஆய்வாளர்/மருத்துவரின் கபயர் மற்றும் முகவரி:

ந ோயோளியின் கபயர் மற்றும் முகவரி: ஆய்வோளரின் நததி தகயோப்பம்:

SL.No	Group	AGE	GENDER	HB	ANEMIA SEVERITY	Heart rate	Systolic BP	Diastolic BP	TC	DC	RBC	PCT	RANDOM BLOOD SUG.	UREA	CREATIN INE	TOTAL CHOLES TERO	TOTAL CHOLES TERO	TRIGLYC ERIDES	TRIGLYC ERIDES	HDL	LDL	LDL	VLDL	VLDL
1	1	70	M	3.8	Severe	72	136	72	5.300	592,50,111	1.5	12	94	38	0.8	131	Normal	175	Increased	28	Normal	38	Normal	35	Increased
2	1	76	M	6.8	Severe	70	157	94	6.500	76,20,120	2.4	16	84	16	0.6	112	Normal	95	Normal	24	Normal	69	Normal	19	Normal
3	1	32	M	5	Severe	84	100	73	7.300	69,30,102	2	14	90	16	1	132	Normal	110	Normal	38	Normal	22	Normal	22	Normal
4	1	62	M	8.6	Severe	88	121	92	6.100	70,20,102	2.9	22	86	18	0.9	141	Normal	130	Normal	19	Normal	43	Normal	26	Normal
5	1	65	M	9.4	Moderate	97	128	82	7.100	60,20,104	3.4	28	110	31	0.9	141	Normal	145	Normal	25	Normal	89	Normal	29	Normal
6	1	65	M	9.6	Moderate	61	130	90	5.800	47,50,65	3.6	30	83	39	0.8	135	Normal	115	Normal	24	Normal	88	Normal	23	Normal
7	1	35	M	6.0	Severe	88	118	75	6.900	62,36,0	2.2	18	92	36	0.6	105	Normal	100	Normal	24	Normal	61	Normal	20	Normal
8	1	60	M	9.4	Moderate	91	122	82	6.100	69,30,102	3.3	26	93	42	1.2	142	Normal	145	Normal	21	Normal	92	Normal	29	Normal
9	1	65	M	7.6	Severe	77	112	95	6.700	60,30,102	2.9	20	76	42	0.9	105	Normal	140	Normal	25	Normal	55	Normal	28	Normal
10	1	38	F	6.4	Severe	87	129	80	6.400	68,30,102	2.4	20	88	18	0.7	116	Normal	110	Normal	26	Normal	68	Normal	22	Normal
11	1	40	F	9.2	Moderate	60	142	83	7.800	68,32,0	1.2	10	99	31	0.8	104	Normal	140	Normal	16	Normal	60	Normal	28	Normal
12	1	16	F	9.5	Moderate	97	139	78	7.300	63,32,0	3.6	10	76	35	0.9	110	Normal	125	Normal	36	Normal	40	Normal	25	Normal
13	1	38	F	9.4	Moderate	111	119	90	4.600	40,36,94	3	10	111	29	1	121	Normal	155	Normal	24	Normal	70	Normal	27	Normal
14	1	30	F	4.3	Severe	64	110	71	4.900	54,36,0	1.2	10	96	27	0.6	102	Normal	90	Normal	18	Normal	65	Normal	18	Normal
15	1	40	F	4.6	Severe	87	137	97	4.400	70,20,0	1.3	10	99	35	0.6	110	Normal	100	Normal	24	Normal	66	Normal	20	Normal
16	1	37	F	9.4	Moderate	62	120	98	11.000	60,30,0	3.4	24	86	28	0.5	133	Normal	145	Normal	28	Normal	76	Normal	29	Normal
17	1	53	F	6.8	Severe	90	134	90	7.800	72,30,0	2.6	20	95	28	0.5	133	Normal	115	Normal	33	Normal	97	Normal	23	Normal
18	1	37	F	7.8	Moderate	81	124	90	6.400	60,32,0	3	20	89	27	0.7	148	Normal	140	Normal	34	Normal	86	Normal	28	Normal
19	1	45	F	7.4	Moderate	73	133	76	5.600	60,36,94	3	22	152	21	0.8	136	Normal	125	Normal	29	Normal	102	Increased	25	Normal
20	1	13	F	9.4	Moderate	107	122	79	9.300	58,30,94	3.5	26	101	29	0.9	144	Normal	165	Increased	36	Normal	75	Normal	33	Increased
21	1	35	F	9.8	Moderate	86	139	89	5.600	60,30,12	3.2	28	82	34	1.3	151	Normal	130	Normal	33	Normal	92	Normal	26	Normal
22	1	16	M	8.4	Severe	75	116	75	7.100	67,20,15	1.5	12	90	18	0.8	124	Normal	100	Normal	26	Normal	78	Normal	20	Normal
23	1	65	M	9.0	Severe	75	126	78	7.600	45,30,23	2.2	24	89	41	1.1	124	Normal	130	Normal	25	Normal	75	Normal	26	Normal
24	1	39	M	7.2	Severe	92	137	88	4.200	54,40,06	2.6	18	76	29	0.6	128	Normal	139	Normal	24	Normal	81	Normal	26	Normal
25	1	37	M	5.6	Severe	80	98	96	7.200	58,30,94	2	14	70	21	0.8	119	Normal	130	Normal	23	Normal	70	Normal	26	Normal
26	1	45	M	9.6	Moderate	89	126	67	8.000	64,20,08	3.2	24	95	42	1.2	144	Normal	130	Normal	26	Normal	92	Normal	26	Normal
27	1	80	M	10.8	Moderate	94	133	80	7.000	62,16,0	3.6	26	120	28	0.9	160	Normal	140	Normal	31	Normal	101	Increased	28	Normal
28	1	29	F	9	Moderate	97	118	85	6.100	38,60,0	3.2	24	89	22	0.9	140	Normal	110	Normal	31	Normal	87	Normal	22	Normal
29	1	35	F	7.4	Moderate	75	144	86	7.000	44,50,06	2.8	20	78	19	1	132	Normal	120	Normal	25	Normal	85	Normal	24	Normal
30	1	16	F	8.6	Moderate	99	147	90	6.400	52,30,0	3	14	86	24	0.8	123	Normal	130	Normal	34	Normal	63	Normal	26	Normal
31	1	45	F	9.8	Moderate	65	95	94	9.800	56,40,04	3.4	26	89	33	0.6	155	Normal	130	Normal	29	Normal	100	Normal	26	Normal
32	1	45	F	8.6	Moderate	103	122	83	7.200	56,40,04	3	24	202	21	1.1	164	Normal	200	Increased	37	Normal	87	Normal	40	Increased
33	1	45	F	8.2	Moderate	72	102	104	9.800	58,40,04	3.1	24	89	33	0.6	155	Normal	130	Normal	29	Normal	100	Normal	26	Normal
34	1	40	M	6.0	Severe	91	95	76	6.600	56,40,04	2	12	121	22	0.7	136	Normal	130	Normal	19	Normal	91	Normal	26	Normal
35	1	70	M	9.2	Moderate	70	143	93	6.100	80,14,06	3	22	150	17	0.9	156	Normal	130	Normal	22	Normal	108	Increased	26	Normal
36	1	38	M	9.6	Moderate	85	127	79	6.300	60,30,12	3.5	26	124	20	0.8	165	Normal	130	Normal	16	Normal	61	Normal	17	Normal
37	1	40	M	10.2	Moderate	70	117	75	7.100	70,10,04	3.6	26	112	26	1.2	120	Normal	125	Normal	21	Normal	74	Normal	25	Normal

79	78	2	50	F	12.2	Normal		103	117	80	6,200	49,44,07	4.2	36	146	27	0.6	113	Normal	145	Normal	22	Normal	62	Normal	29	Normal
80	79	2	18	F	12.4	Normal		104	111	86	6,300	72,26,02	4.2	36	95	28	0.5	153	Normal	115	Normal	33	Normal	97	Normal	23	Normal
81	80	2	29	F	12	Normal		75	123	93	8,500	59,37,04	4	34	99	54	1.6	187	Normal	125	Normal	41	Normal	121	Increased	25	Normal
82	81	2	28	F	14.1	Normal		84	112	81	6,900	64,33,03	4	32	89	18	1	262	Increased	385	Increased	34	Normal	151	Increased	77	Increased
83	82	2	38	F	12.8	Normal		63	155	109	3,800	78,20,02	4.5	38	85	16	1	108	Normal	155	Increased	19	Normal	58	Normal	31	Increased
84	83	2	25	F	12	Normal		83	133	101	8,000	64,28,08	4.4	38	95	42	1.2	144	Normal	130	Normal	26	Normal	92	Normal	26	Normal
85	84	2	29	F	15.1	Normal		71	108	74	4,800	59,40,01	4.1	34	122	25	0.9	98	Normal	130	Normal	18	Normal	54	Normal	26	Normal
86	85	2	20	F	12	Normal		67	118	91	3,800	78,20,02	4	32	85	16	1	108	Normal	155	Increased	19	Normal	58	Normal	31	Increased
87	86	2	45	F	12.4	Normal		95	135	85	8,000	64,28,08	4.2	36	95	42	1.2	144	Normal	130	Normal	26	Normal	92	Normal	26	Normal
88	87	2	40	F	12.2	Normal		85	119	76	5,600	60,28,12	4.2	38	82	34	1.3	151	Normal	130	Normal	33	Normal	92	Normal	26	Normal
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90	89	2	70	F	12.6	Normal		105	144	76	6,800	70,28,02	4.4	34	88	14	1	170	Normal	120	Normal	39	Normal	107	Increased	24	Normal
91	90	2	40	F	12.4	Normal		94	136	96	6,100	48,50,02	4.3	36	152	21	0.8	156	Normal	125	Normal	29	Normal	102	Increased	25	Normal
92	91	2	58	F	12	Normal		86	124	87	6,900	52,46,02	4.1	32	202	21	1.1	164	Normal	200	Increased	37	Normal	87	Normal	40	Increased
93	92	2	18	F	12	Normal		81	130	98	5,900	64,30,06	4	32	96	23	0.8	159	Normal	145	Normal	29	Normal	101	Increased	29	Normal
94	93	2	70	F	14.2	Normal		94	136	57	8,200	44,48,08	3.9	30	95	18	0.9	118	Normal	130	Normal	20	Normal	72	Normal	26	Normal
95	94	2	59	F	12.8	Normal		78	141	81	6,100	48,50,02	4.6	38	152	21	0.8	156	Normal	125	Normal	29	Normal	102	Increased	25	Normal
96	95	2	48	F	12.2	Normal		82	140	72	6,500	64,33,03	4.2	34	194	33	1.2	143	Normal	155	Increased	24	Normal	88	Normal	31	Increased
97	96	2	20	F	12.8	Normal		72	128	79	8,600	56,40,04	4.7	40	89	33	0.6	155	Normal	130	Normal	29	Normal	100	Normal	26	Normal
98	97	2	27	F	12.8	Normal		86	125	89	7,700	58,38,04	4.8	40	101	29	0.9	144	Normal	165	Increased	36	Normal	75	Normal	33	Increased
99	98	2	36	F	12.6	Normal		103	124	95	7,200	56,40,04	4.4	38	115	25	0.8	152	Normal	185	Increased	29	Normal	86	Normal	37	Increased
100	99	2	50	F	12.6	Normal		81	127	81	8,400	65,30,05	4.4	38	202	21	1.1	164	Normal	200	Increased	37	Normal	87	Normal	40	Increased
101	100	2	23	F	12.6	Normal		76	122	76	7,200,	54,40,06	4.2	34	130	28	0.9	160	Normal	140	Normal	31	Normal	101	Increased	28	Normal
102	101	2	35	F	13.6	Normal		70	121	80	6,100	48,50,02	3.9	32	152	21	0.8	156	Normal	125	Normal	29	Normal	102	Increased	25	Normal
103	102	2	14	M	13.6	Normal		76	119	85	6,100	48,50,02	4.6	40	152	21	0.8	156	Normal	125	Normal	29	Normal	102	Increased	25	Normal
104	103	2	60	M	13.8	Normal		74	100	84	6,900	52,46,02	4.5	38	202	21	1.1	164	Normal	200	Increased	37	Normal	87	Normal	40	Increased
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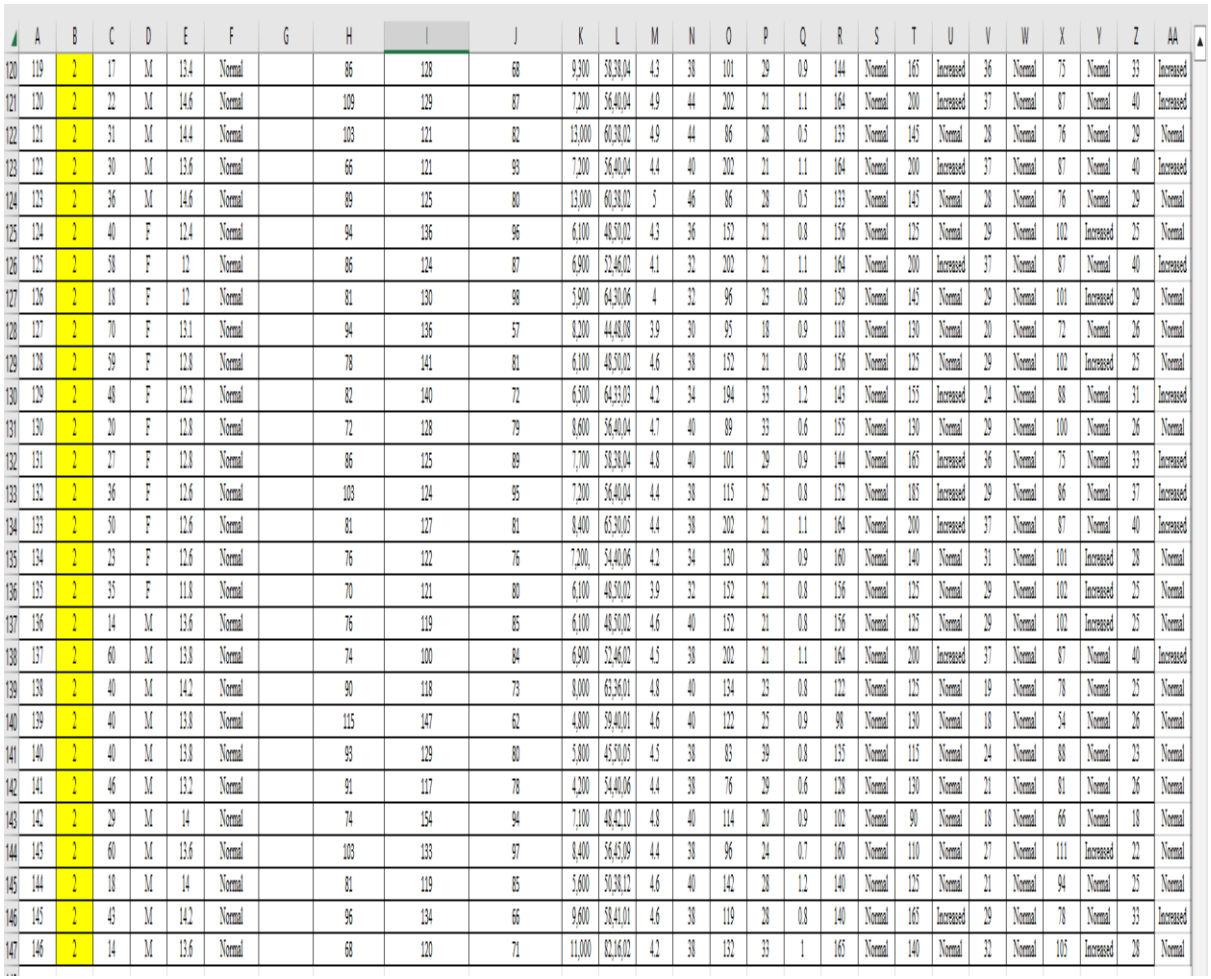
107	106	2	40	M	13.8	Normal		93	129	80	5,800	45,50,05	4.5	38	83	39	0.8	135	Normal	115	Normal	24	Normal	88	Normal	23	Normal
108	107	2	46	M	13.2	Normal		91	117	78	4,200	54,40,06	4.4	38	76	29	0.6	128	Normal	130	Normal	21	Normal	81	Normal	26	Normal
109	108	2	29	M	14	Normal		74	154	94	7,100	48,42,10	4.8	40	114	20	0.9	102	Normal	90	Normal	18	Normal	66	Normal	18	Normal
110	109	2	60	M	13.6	Normal		103	133	97	8,400	56,45,09	4.4	38	96	24	0.7	160	Normal	110	Normal	27	Normal	111	Increased	22	Normal
111	110	2	18	M	14	Normal		81	119	85	5,600	50,38,12	4.6	40	142	28	1.2	140	Normal	125	Normal	21	Normal	94	Normal	25	Normal
112	111	2	43	M	14.2	Normal		96	134	66	9,600	58,41,01	4.6	38	119	28	0.8	140	Normal	165	Increased	29	Normal	78	Normal	33	Increased
113	112	2	14	M	13.6	Normal		68	120	71	11,000	82,16,02	4.2	38	132	33	1	165	Normal	140	Normal	32	Normal	105	Increased	28	Normal
114	113	2	28	M	14.2	Normal		104	136	88	8,300	68,30,02	4.9	42	199	38	1.5	147	Normal	135	Normal	27	Normal	93	Normal	27	Normal
115	114	2	43	M	13	Normal		77	157	92	6,300	60,28,12	4.1	42	124	20	0.8	94	Normal	85	Normal	16	Normal	61	Normal	17	Normal
116	115	2	15	M	13.8	Normal		80	167	87	6,100	80,14,06	4.5	40	150	17	0.9	156	Normal	130	Normal	22	Normal	108	Increased	26	Normal
117	116	2	15	M	13.4	Normal		112	134	78	7,100	48,42,10	4.4	40	114	20	0.9	102	Normal	90	Normal	18	Normal	66	Normal	18	Normal
118	117	2	50	M	13.8	Normal		80	106	85	8,400	56,45,09	4.5	40	96	24	0.7	160	Normal	110	Normal	27	Normal	111	Increased	22	Normal
119	118	2	15	M	13.6	Normal		102	120	90	9,800	56,40,04	4.5	38	89	33	0.6	155	Normal	130	Normal	29	Normal	100	Normal	26	Normal



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121	120	2	22	M	14.6	Normal		109	129	87	7,200	56,40,04	4.9	44	202	21	1.1	164	Normal	200	Increased	37	Normal	87	Normal	40	Increased
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123	122	2	30	M	13.6	Normal		66	121	93	7,200	56,40,04	4.4	40	202	21	1.1	164	Normal	200	Increased	37	Normal	87	Normal	40	Increased
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125	124	2	40	F	12.4	Normal		94	136	96	6,100	48,50,02	4.3	36	152	21	0.8	156	Normal	125	Normal	29	Normal	102	Increased	25	Normal
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127	126	2	18	F	12	Normal		81	130	98	5,900	64,30,06	4	32	96	23	0.8	129	Normal	145	Normal	29	Normal	101	Increased	29	Normal
128	127	2	70	F	15.1	Normal		94	136	57	8,200	44,48,08	3.9	30	95	18	0.9	118	Normal	130	Normal	20	Normal	72	Normal	26	Normal
129	128	2	59	F	12.8	Normal		78	141	81	6,100	48,50,02	4.6	38	152	21	0.8	156	Normal	125	Normal	29	Normal	102	Increased	25	Normal
130	129	2	48	F	12.2	Normal		82	140	72	6,500	64,33,03	4.2	34	194	33	1.2	145	Normal	155	Increased	24	Normal	88	Normal	31	Increased
131	130	2	20	F	12.8	Normal		72	128	79	8,600	56,40,04	4.7	40	89	33	0.6	155	Normal	130	Normal	29	Normal	100	Normal	26	Normal
132	131	2	27	F	12.8	Normal		86	125	89	7,700	58,30,04	4.8	40	101	29	0.9	144	Normal	165	Increased	36	Normal	75	Normal	33	Increased
133	132	2	36	F	12.6	Normal		103	124	95	7,200	56,40,04	4.4	38	115	25	0.8	152	Normal	185	Increased	29	Normal	86	Normal	37	Increased
134	133	2	50	F	12.6	Normal		81	127	81	8,400	65,30,05	4.4	38	202	21	1.1	164	Normal	200	Increased	37	Normal	87	Normal	40	Increased
135	134	2	23	F	12.6	Normal		76	122	76	7,200,	54,40,06	4.2	34	150	28	0.9	160	Normal	140	Normal	31	Normal	101	Increased	28	Normal
136	135	2	25	F	11.8	Normal		70	121	80	6,100	48,50,02	3.9	32	152	21	0.8	156	Normal	125	Normal	29	Normal	102	Increased	25	Normal
137	136	2	14	M	13.6	Normal		76	119	85	6,100	48,50,02	4.6	40	152	21	0.8	156	Normal	125	Normal	29	Normal	102	Increased	25	Normal
138	137	2	60	M	13.8	Normal		74	100	84	6,900	52,46,02	4.5	38	202	21	1.1	164	Normal	200	Increased	37	Normal	87	Normal	40	Increased
139	138	2	40	M	14.2	Normal		90	118	73	8,000	62,36,01	4.8	40	134	23	0.8	122	Normal	125	Normal	19	Normal	78	Normal	25	Normal
140	139	2	40	M	13.8	Normal		115	147	62	4,800	59,40,01	4.6	40	122	25	0.9	98	Normal	130	Normal	18	Normal	54	Normal	26	Normal
141	140	2	40	M	13.8	Normal		93	129	80	5,800	45,50,05	4.5	38	83	39	0.8	155	Normal	115	Normal	24	Normal	88	Normal	23	Normal
142	141	2	46	M	13.2	Normal		91	117	78	4,200	54,40,06	4.4	38	76	29	0.6	128	Normal	130	Normal	21	Normal	81	Normal	26	Normal
143	142	2	29	M	14	Normal		74	154	94	7,100	48,42,10	4.8	40	114	20	0.9	102	Normal	90	Normal	18	Normal	66	Normal	18	Normal
144	143	2	60	M	13.6	Normal		103	133	97	8,400	56,45,09	4.4	38	96	24	0.7	160	Normal	110	Normal	27	Normal	111	Increased	22	Normal
145	144	2	18	M	14	Normal		81	119	85	5,600	50,38,12	4.6	40	142	28	1.2	140	Normal	125	Normal	21	Normal	94	Normal	25	Normal
146	145	2	43	M	14.2	Normal		96	134	66	9,600	58,41,01	4.6	38	119	28	0.8	140	Normal	165	Increased	29	Normal	78	Normal	33	Increased
147	146	2	14	M	13.6	Normal		68	120	71	11,000	52,16,02	4.2	38	152	33	1	165	Normal	140	Normal	32	Normal	105	Increased	28	Normal