"TO STUDY THE COMPARISION BETWEEN FASTING AND POSTPRANDIAL TRIGLYCERIDE LEVEL AS A RISK FACTOR FOR ISCHEMIC HEART DISEASE"

BY

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POST GRADUATE
MD GENERAL MEDICINE

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IN

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ABBREVIATIONS

Abbreviation	Full Form
AMI	Acute Myocardial Infarction
BMI	Body Mass Index
CABG	Coronary Artery Bypass Grafting
CAD	Coronary Artery Disease
CK	Creatine Kinase
CK-MB	Creatine Kinase–Myocardial Band
CVD	Cardiovascular Disease
ECG	Electrocardiogram
ESR	Erythrocyte Sedimentation Rate
FDG	Fluorodeoxyglucose
FBS	Fasting Blood Sugar
GBD	Global Burden of Disease
HDL	High-Density Lipoprotein
HTN	Hypertension
IHD	Ischemic Heart Disease
LDL	Low-Density Lipoprotein
LFT	Liver Function Test
LBBB	Left Bundle Branch Block
MI	Myocardial Infarction
NCCT	Non-Contrast Computed Tomography
NSTEMI	Non-ST Elevation Myocardial Infarction
PCI	Percutaneous Coronary Intervention
PET	Positron Emission Tomography
PP2BS	Postprandial 2-Hour Blood Sugar
PP4TG	Postprandial 4-Hour Triglyceride
STEMI	ST Elevation Myocardial Infarction
TG	Triglyceride
TROP I	Troponin I
VLDL	Very Low-Density Lipoprotein
WHR	Waist-Hip Ratio
WHO	World Health Organization

ABSTRACT

Background: Coronary artery disease (CAD) remains a leading cause of morbidity and mortality worldwide. Among risk factors, postprandial hypertriglyceridemia is increasingly recognized as a critical and under-assessed contributor to atherosclerosis and cardiovascular events, especially in South Asian populations predisposed to dyslipidemia and insulin resistance.

Objective: To evaluate and compare fasting and postprandial triglyceride levels in patients with ischemic heart disease (IHD) and matched healthy controls, and to assess the correlation of lipid levels with cardiovascular risk factors.

Methods: A case-control study was conducted involving 60 participants, divided equally into IHD cases and healthy controls. After overnight fasting, baseline triglyceride levels were obtained, followed by postprandial triglyceride measurements taken 4 hours after a standardized lunch. Data on demographics, lipid profile (HDL, LDL, VLDL, total cholesterol), and blood glucose levels were recorded. The statistical analysis included Student's t-test and ANOVA for continuous variables and chi-square tests for categorical comparisons, with $p < 0.05$ considered statistically significant.

Results: The mean age was 51.47 ± 5.33 years in cases and 52.27 ± 5.89 in controls. Postprandial triglyceride levels were significantly higher in the case group compared to controls, despite comparable fasting levels. A marked difference in VLDL levels and total triglycerides post-meal suggested delayed clearance or exaggerated synthesis in IHD patients. High prevalence of central obesity, hypertension, and impaired glucose metabolism was noted among cases.

Conclusion: Postprandial hypertriglyceridemia is significantly elevated in IHD patients compared to healthy individuals, even when fasting lipid levels appear normal. This emphasizes the need for routine postprandial lipid testing to better stratify cardiovascular risk and implement earlier preventive strategies in high-risk populations.

Keywords: *Ischemic heart disease, postprandial triglycerides, cardiovascular risk, lipid profile, hypertriglyceridemia, dyslipidemia,*

INTRODUCTION

INTRODUCTION

Cardiovascular diseases (CVDs), encompassing conditions such as ischemic heart disease (IHD) ,stroke continue to be foremost triggers of death , disability globally. Among these acute myocardial infarction stands out as leading most critical clinical outcomes of atherosclerotic coronary artery disease (CAD)[1] . Data driven from World Health Organization and Global Burden of Disease study, CVDs are liable for approximately 18.6 million deaths each year [2]. Although notable advancements have been made in AMI treatment—particularly with prompt revascularization and pharmacological interventions—arrhythmias remain a frequent and serious complication, negatively impacting early recovery and survival rates[3,4] .

Chronic atherosclerosis in the epicardial coronary arteries are the underlying cause of most coronary syndromes. While it can affect individuals across all ages and sexes, the severity often depends on genetic background, risk factor exposure, and localized hemodynamic variables[5] . A healthy endothelium has a key role in regulation of vascular tone by releasing substances like prostacyclin and endothelium-derived relaxing factors,by maintaining a balance in local thrombotic processes [6]. This delicate balance is disrupted by risk factors like hypertension, elevated cholesterol, tobacco use, and abnormal blood flow patterns, which impair endothelium-dependent vasodilation and promote thrombogenesis [7] Endothelial dysfunction, often considered a precursor to atherosclerosis, facilitates the collection of macrophages and lipids,primarily (LDL)low density lipoprotein at injury sites within the vessel wall[8]

Lipid abnormalities significantly contribute to atherosclerotic progression [9] Elevated blood cholesterol, particularly LDL, accelerates disease development, while raised triglyceride levels may serve as an isolated variable associated with development of CAD, specifically in female population. Conversely, high-density lipoproteins (HDLs) offer a protective effect by counteracting these risk elements. Substantial evidence supports the therapeutic outcomes of lipid-modifying strategies in managing and preventing CAD [10].

Triglycerides, containing glycerol backbone and three fattyacid chains, serve as energy reservoirs within adipose tissue. Dyslipidemia is often indicated by elevated total triglycerides and/or LDL greaterthan the 90th percentile, or HDL levels below the 10th percentile [11]. Elevated triglyceride concentrations contribute to CAD by raising LDL levels, reducing HDL, impairing endothelial function, and activating thrombogenic mechanisms and plasminogen activators[12].

Historically, fasting triglyceride measurements were the standard for evaluating lipid-related cardiovascular risk. Yet, emerging evidence suggests that postprandial triglyceride levels may be more reflective of real-life metabolic processes and more predictive of atherosclerotic progression and cardiovascular events. Postprandial lipemia—the rise in TG-rich lipoproteins following meals has been linked to endothelial dysfunction, oxidative stress, and inflammatory responses, all of which contribute to atherogenesis [13]

Several clinical trials have demonstrated that postprandial TG levels correlate more strongly with the severity of IHD compared to fasting levels [14,15],In statin treated patients with acute coronary syndrome fasting TGs were found to independently predict both short period and long period cardiovascular risk, yet postprandial levels may offer additional prognostic value in untreated or high-risk populations[16] ([Schwartz et al., 2015]). Furthermore, observational studies have shown that individuals with normal fasting TG levels may still exhibit elevated postprandial TGs, placing them at unrecognized cardiovascular risk[17,18] ([Werner, 2014]; [Xie, 2023]).

As dietary habits and insulin resistance increasingly influence lipid metabolism, particularly in individuals with diabetes ,the assessment of lipid parameters in the non-fasting state becomes even more clinically relevant. Therefore, comparing fasting and postprandial triglyceride levels could provide a deeper understanding of their individual contributions to IHD risk and aid in improving cardiovascular risk stratification and preventive strategies.

AIMS & OBJECTIVES

OBJECTIVES

- **AIM** To study relation between fasting and postprandial triglyceride levels as risk factor for Ischaemic Heart Disease.

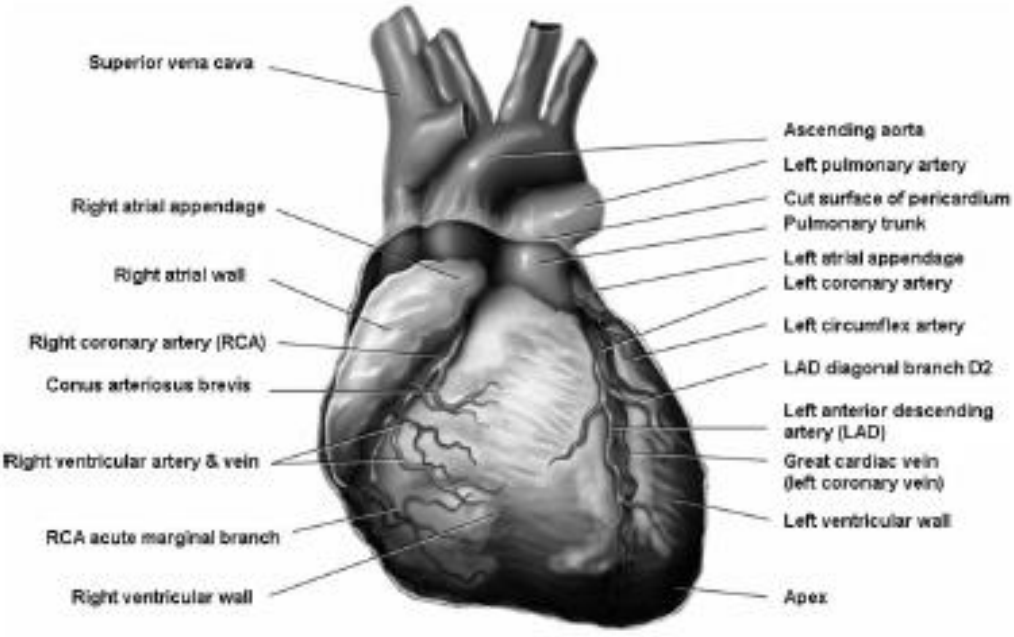
REVIEW OF LITERATURE

REVIEW OF LITERATURE

HEART - OVERVIEW

As mentioned by Hall JE et al[19] ,the heart plays a key role in maintaining life by circulating oxygenated blood from the lungs to the body while returning deoxygenated blood to the lungs for carbon dioxide removal. Situated in the mediastinum of the thorax, it is protected by the pericardial sac and beats roughly 72 times per minute, resulting in around 2.5 billion beats over an average lifespan. Its weight ranges from 250-300g in females to 300-350g in males.

Figure 1. Anterior view of the human heart with blood vessels identified

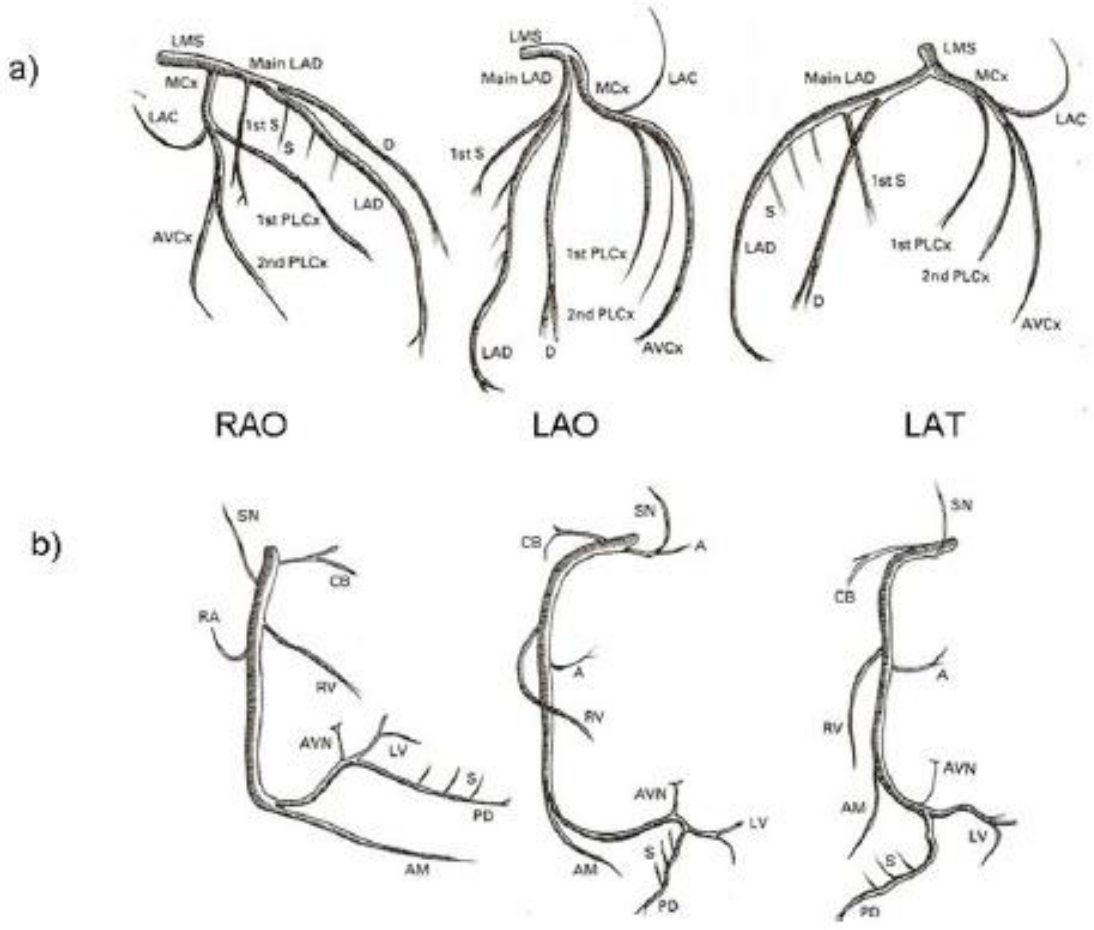


The heart constitutes of four chambers:

The walls of the heart have three layers and the septum splits the heart into right and left sides, preventing mixing of oxygenated and deoxygenated blood.[19,20]

The heart's oxygen and nutrient supply come from the coronary arteries, which encircle the heart like a crown, aligning with their Latin origin, *corona*. These arteries are crucial for myocardial function and form part of a dual-component system.

Figure 2. Coronary artery anatomy.



a) Left coronary artery and b) Right coronary artery. A, atrial branch; AM, acute marginal artery; AVCx, atrioventricular groove branch of circumflex; AVN, atrioventricular node artery; CB, conus branch; D, diagonal branch of LAD; LAC, left atrial circumflex; LAD, left anterior descending; LAO 30° left anterior oblique projection; LAT, left lateral projection; LMS, left main stem; LV, left ventricular branches; MCx, main circumflex; PD, posterior descending; PLCx, posterior circumflex branch (obtuse marginal); RA, right atrial branch; RAO, 30o right anterior oblique projection; RV, right ventricular branch; S, septal perforating arteries; SN, sinus node artery.

The coronary arteries are essential vascular structures responsible for delivering oxygenated blood and nutrients to the myocardium, thereby sustaining continuous cardiac function .The coronary arteries operate predominantly during diastole, as the high intramural pressure during systole limits blood flow to the myocardium.[19,20]

Table 1. The major coronary arteries.

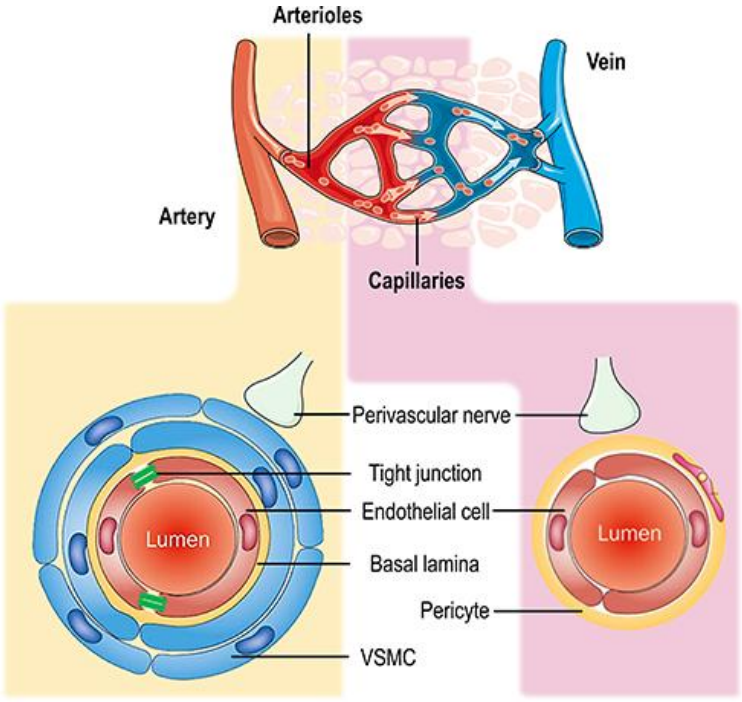
Vessel	Median Diameter (range) in mm
LEFT CORONARY ARTERY (LCA)	4 (2.5-5.5)
Left anterior descending	3.6 (2-5)
DG diagonal	2 (0.5-2.5)
LCX circumflex	3 (1.5-5)
LMG marginal	2.2 (1-3)
RIGHT CORONARY ARTERY (RCA)	3.2 (1.5-5.5)
RMG marginal	1.7 (1-2.5)
PD posterior descending	2.1 (1-3)
THIRD CORONARY ARTERY 'conus artery'	1.1 (0.7-2)
Septal branches anterior from LCX	1 (0.5-2.5)
Septal branches posterior from PD	0.7 (0.3-0.9)
From ascending LAD	0.4 (0.3-0.7)

CORONARY MICROCIRCULATION[21,22]

The coronary microcirculation plays a vital role in maintaining myocardial oxygenation and nutrient supply, ensuring the heart meets its metabolic demands under varying conditions. This network includes arterioles, capillaries, and venules, with arterioles regulating blood flow through changes in diameter in response to metabolic, myogenic, and neurohormonal stimuli. Capillaries, densely distributed within the myocardium, facilitate gas exchange and nutrient delivery, ensuring efficient oxygen supply to every cardiomyocyte. The coronary microcirculation functions primarily during diastole, as systolic compression limits blood flow, particularly in the subendocardium which is most susceptible to ischemia due to its higher oxygen demand.

Endothelial cells within the microvasculature release vasoactive substances, such as nitric oxide and endothelin 1, to regulate vascular tone and maintain perfusion. Under normal conditions, autoregulatory mechanisms match blood flow to myocardial demand, responding to increased activity or stress by dilating vessels and enhancing oxygen delivery. This precise regulation ensures optimal cardiac performance, with disruptions to microcirculatory function playing a critical role in ischemic events, including acute myocardial infarction.

Figure 3. Vascular network and capillary neurovascular unit.



Special Features of Coronary Microcirculation[21,22]

1. **Anastomoses and Collateral Circulation**:
    - Collateral arteries and anastomoses provide alternative pathways for blood flow, particularly in areas where primary vessels are occluded.

- These vessels, present at birth, can grow and expand in response to chronic ischemia, helping to protect the myocardium during partial blockages of major coronary arteries.

2. **Regional Perfusion Variability**:
    - coronary microcirculation blood flow varies based on the layer of the myocardium:
        - subendocardium (innermost layer) has the highest oxygen demand but is more susceptible to ischemia due to compression during systole.
        - The subepicardium (outer layer) receives more consi stent blood flow, even during systole.
3. **Endothelial Function**:
    - Endothelial cells lining the coronary microcirculation play an essential role in vascular homeostasis by releasing vasoactive substances, such as nitric oxide and endothelin-1 (vasoconstrictor).
    - These substances regulate vessel tone, ensure adequate perfusion, and prevent platelet aggregation and thrombosis.

Physiological Role of Coronary Microcirculation

Coronary microcirculation is vital for matching myocardial oxygen delivery with its consumption, particularly during changes in activity levels or stress. Under normal conditions, autoregulatory mechanisms, including myogenic, metabolic, and neurohormonal controls, maintain a balance between supply and demand:

- **Myogenic Regulation**: Arterioles constrict or dilate in response to changes in intravascular pressure, ensuring constant perfusion.
- **Metabolic Regulation**: Increased myocardial metabolism releases vasodilators such as adenosine and lactate, which enhance blood flow to meet energy demands.
- **Neurohormonal Regulation**: Sympathetic activation during exercise or stress increases coronary blood flow to support heightened cardiac workload.

ISCHAEMIC HEART DISEASE(IHD)

As mentioned by Harpaz D et al[23], Ischaemic heart disease manifests when there is an insufficient flow of oxygenated blood to the heart muscle (myocardium), typically due to imbalance between oxygen demand and availability. The most frequent underlying cause is atherosclerosis of the epicardial coronary arteries, which leads to regional impairments in blood flow and suboptimal perfusion of the myocardial tissue supplied by the affected artery or arteries

ACUTE MYOCARDIAL INFARCTION (AMI)

Myocardial infarction (MI) most frequent form of coronary heart disease (CHD), occurs when a coronary artery is blocked, causing a severe reduction in blood flow, infarction of the heart muscle supplied by that artery. There are two clinical types of MI:

- **ST Segment Elevation Myocardial Infarction (STEMI):** Characterized by ST-segment elevation on an electrocardiogram (ECG).
- **Non ST Segment Elevation Myocardial Infarction (NSTEMI):** Defined by the absence of ST-segment elevation on ECG and the presence of elevated cardiac biomarkers, such as troponin.

GLOBAL BURDEN

As reported by Murray CJL et al[24]The Global Burden of Disease (GBD) Study 2019 revealed that CVD prevalence nearly doubled from 1990 to 2019, while CVD-related deaths rose from 12.1 million to 18.6 million during the same period.

Non -Modifiable Risk Factors:

- **Age--**Risk increases with advancing age, particularly in elderly populations.
- **Gender**- Men have a higher incidence of AMI up to age 60.
- **Family History and Genetics**-A strong familial and genetic predisposition exists.
- **Global Aging**- The aging population, especially in developed countries, has contributed significantly to increased CVD risk.

Modifiable Risk Factors:

- **Smoking**: A declining trend in many countries but still a major risk factor.
- **Physical Inactivity:** Affects 1.6 billion people (20.6% globally).
- **Unhealthy Diet:** Impacts 2.5 billion people (32.4% globally).
- **Obesity:** Affects 672 million people (8.7% globally).
- **Diabetes:** Prevalence of 463 million people (6% globally).
- **Harmful Alcohol Use:** Affects 2 billion people (25.9% globally).
- **Hypertension, Dyslipidemia, and Psychosocial Factors:** Stress, anxiety, and depression exacerbate CVD risk.

Pathophysiology of Acute Myocardial Infarction[25]

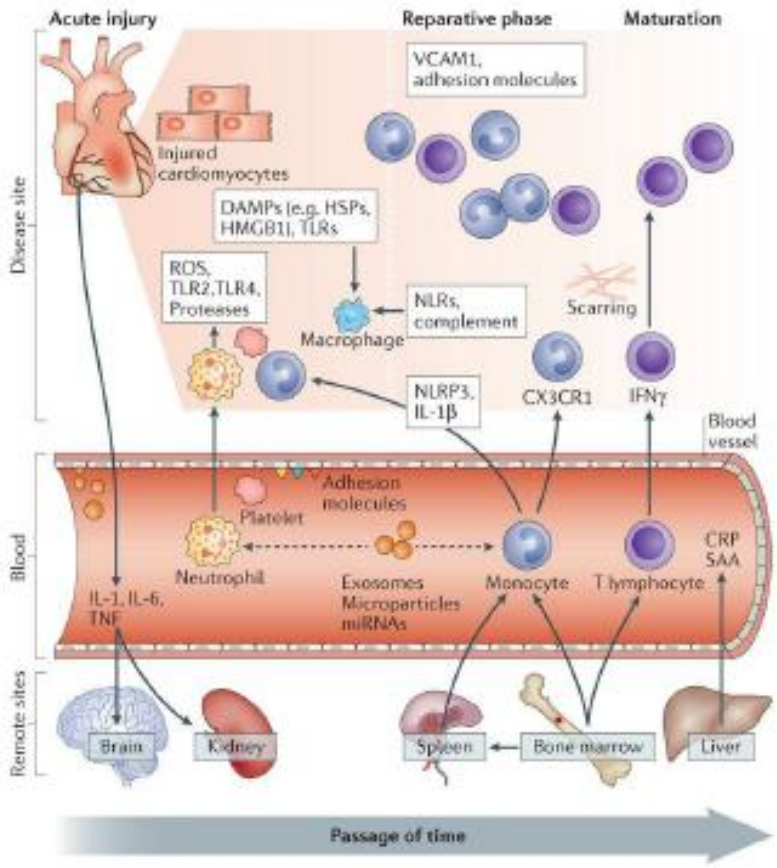
"A study reported by Reed GW et al[25], Acute myocardial infarction (AMI) is classified as STEMI or NSTEMI, with unstable angina treated as a part of acute coronary syndrome (ACS) as it often precedes myocardial infarction.

While severe stenoses (≥70% diameter) are associated with angina, they less frequently cause type 1 AMI due to dense fibrotic caps and developed collateral circulation. Vulnerable plaques, with 30–50% stenosis, thin fibrous caps, and inflammatory lipid-laden macrophages, are more likely to rupture.

Upon rupture, thrombogenic plaque contents activate platelets, the coagulation cascade, and mural thrombus formation. This process may embolize atherosclerotic debris downstream, potentially causing necrosis in myocardial cells. The hypercoagulable state may trigger the rupture of additional vulnerable plaques, resulting in multiple culprit lesions.

The extent of ischemia is determined by factor such as partial vs. complete vessel occlusion, time span of occlusion, extent of myocardium affected, collateral circulation, and the capacity of reperfusion after treatment. Myocyte necrosis, a hallmark of AMI, is detected by elevated cardiac biomarkers in the blood".

FIGURE 4. Biological pathways central to the pathogenesis of acute myocardial infarction (AMI).



Immediately following AMI, a number of local processes are activated with release of reactive oxygen species and cytokines with infiltration of circulating neutrophils and monocytes resulting in acute myocardial injury. Simultaneously a number of remote sites are also activated (e.g. spleen, bone marrow) via signalling pathways that result in further activation of the immune system and injury. Following this, a reparative phase ensues predominantly mediated by monocytes and T-lymphocytes resulting in tissue repair and recovery with upregulation of processes involved in angiogenesis and extracellular matrix deposition. Abbreviations: ROS: reactive oxygen species; TLR: toll-like receptors; DAMPS: damage associated molecular patterns; HSP: heat shock proteins; HMGB1: high mobility group box 1 protein; VCAM: vascular cell adhesion molecule; NLR: NOD-like receptor; NLRP3: NOD-like receptor family pyrin domain containing 3; IL: interleukin; TNF: tumour necrosis factor; IFN: interferon; CX3CR1: CX3 chemokine receptor 1; miRNA: micro ribonucleic acid; CRP: C reactive protein; SAA: serum amyloid A.

Operational Definition of AMI

Table 2. Myocardial Infarction Location Based on Coronary Artery Involvement

Involved Myocardium	Occluded Vessel	ECG Leads Involved
Anterior MI	LAD	Some or all of leads V1–V6
Inferior MI	RCA or LCX	ST elevation in leads II, III, and aVF
Right Ventricular MI	RCA	ST elevation in leads V4–V6R (occurs in 1/2 of inferior MI)
Posterior MI	LCX or RCA	ST depression in leads V1–V3
		ST elevation in leads I and aVL (LCX)
		ST depression in leads I and aVL (RCA)
Lateral MI	LCX, diagonal	ST elevation in leads I, aVL, V5, and V6
		ST depression in leads II, III, and aVF

AMI is diagnosed through evidence of heart muscle necrosis aligned with an ischemic clinical presentation

Criteria include:

1. **Elevation and/or decline of cardiac biomarkers** with at least one value exceeding the 99th percentile of the upper reference limit (URL) and:

- Symptoms of ischemia.
- New ischemic ECG changes (e.g., ST-T changes or new LBBB).
- Pathological Q waves.
- Imaging evidence of myocardial damage or wall motion abnormality[26-28].

"Types of Myocardial Infarctions"[26,27,28,29]

The most recent guidelines classify AMI into five distinct types:

1. **Type 1**: Spontaneous MI caused by primary coronary events such as plaque rupture, erosion, or dissection, typical of ST-elevation or non-ST-elevation MI.
2. **Type 2**: MI due to increased oxygen demand or reduced supply, associated with conditions like coronary artery spasm, embolism, anemia, arrhythmias, or hypotension.
3. **Type 3**: Sudden cardiac death with symptoms of ischemia, accompanied by new ST elevation, LBBB, or coronary thrombus evidence but occurring before biomarker detection.
4. **Type 4**: MI related to percutaneous coronary interventions (PCI), further classified into procedural-related or stent thrombosis-related MI.
5. **Type 5**: MI associated with coronary artery bypass grafting (CABG).

Table 3: Timing of Gross and Microscopic Changes After Myocardial Infarction[30]:

Time	Gross Features	Light Microscope	Electron Microscope
Reversible Injury			
0-30 minutes	None	None	Relaxation of myofibrils; loss of glycogen; swelling of mitochondria
Irreversible Changes			
30 minutes - 4 hours	None	Usually none; wavy fibers at border	Sarcolemma destruction; mitochondria amorphous densities
4-12 hours	Occasional dark mottling seen	Early coagulative necrosis, edema, hemorrhage begin; hypereosinophilia of cytoplasm	Not specified
12-24 hours	Dark mottling	Continued coagulative necrosis; pyknotic nuclei; contraction band necrosis at margins; neutrophil influx	Not specified
1-3 days	Mottling with yellow-tan center	Coagulative necrosis with absent nuclei and striations,	Not specified

CLINICAL PRESENTATION OF STEMI[4]

Certain factors such as intense physical activity, emotional distress, or concurrent medical/surgical conditions may act as triggers for ST-elevation myocardial infarction (STEMI). While STEMI can begin at any time, it often shows a circadian pattern, with increased incidence in the early morning hours post-awakening.

Chest pain is the most commonly reported symptom and is typically described as deep, pressure-like, and heavy—often likened to crushing, squeezing, or even burning sensations. Pain occurring during rest is usually more intense and longer lasting. It primarily involves the central chest or epigastric region and may radiate to the left arm. Less commonly, it can spread to the jaw, neck, back, or abdomen.

Pain centered around the epigastrium and under the xiphoid may lead to misinterpretation as gastrointestinal discomfort, particularly when patients are in denial about a cardiac cause. Pain may extend as far as the occiput but generally does not descend below the umbilicus. Additional symptoms may include generalized weakness, excessive sweating, nausea, vomiting, and heightened anxiety. Unlike angina, exertion-related STEMI pain does not typically resolve with rest.

PHYSICAL EXAMINATION FINDINGS

Patients often appear anxious and restless, frequently shifting positions in a futile attempt to alleviate discomfort. Sweating, pallor, and cool extremities are common. Prolonged chest pain (lasting over 30 minutes) accompanied by diaphoresis is highly suggestive of STEMI. During the early phase, most patients exhibit normal heart rate and blood pressure. However, approximately 25% of anterior infarctions manifest with signs of sympathetic activation (tachycardia or hypertension), whereas up to 50% of inferior infarctions show parasympathetic features (bradycardia or hypotension).

The apical impulse may be difficult to detect due to a quiet precordium. Occasionally, a dyskinetic systolic bulge appears in the periapical region due to myocardial damage and may resolve over time. Audible abnormalities may include third or fourth heart sounds, diminished intensity of the first heart sound, and paradoxical splitting of the second. Transient murmurs from mitral valve dysfunction can also occur. In some patients, a pericardial friction rub is evident during the course of a transmural infarct. A drop in stroke volume is often reflected in a reduced carotid pulse. Body temperature may rise to approximately 38°C within the first week following infarction, while arterial pressure readings may vary depending on infarct severity and location.

DIAGNOSTIC PHASES OF MYOCARDIAL INFARCTION

Myocardial infarction is typically categorized into three temporal phases:

1. **Acute phase** – within the first few hours up to 7 days
2. **Healing phase** – from day 7 to 28
3. **Healed phase** – beyond 29 days

Understanding the infarction timeline is vital when interpreting diagnostic results.

METHODS OF DIAGNOSIS

The diagnostic process for STEMI involves multiple modalities:

1. **Electrocardiography (ECG)**
2. **cardiac biomarkers**
3. **Cardiac imaging**
4. **Nonspecific inflammatory markers**

ECG FINDINGS IN ACUTE MI[4]

Total occlusion of an epicardial artery typically results in **ST-segment elevation**, especially in the early hours. Patients who exhibit persistent ST elevation and eventually develop Q waves are diagnosed with **Q-wave MI**. In cases where occlusion is incomplete or where robust collateral circulation exists, **non–ST elevation** is observed, leading to diagnoses like **NSTEMI** or **unstable angina**.

CARDIAC BIOMARKERS[4,31]

Proteins released from damaged myocardial cells, such as:

- **Creatine kinase (CK)**: Rises within 4–8 hours, normalizes in 48–72 hours. However, it lacks specificity due to possible elevation from skeletal muscle injury.
- **CK-MB isoenzyme**: More specific to cardiac tissue and helpful in differentiating myocardial from skeletal muscle damage.
- **Cardiac troponins (cTnI and cTnT)**: Highly specific and sensitive, remaining elevated for 7–10 days. Essential for identifying even minor infarctions.
- **Myoglobin**: The earliest marker to rise, but lacks cardiac specificity and returns to baseline within 24 hours.

CARDIAC IMAGING[4,31]

Echocardiography detects wall motion abnormalities and assesses left ventricular function, which has prognostic value. It also helps identify right ventricular infarction, pericardial effusion, ventricular aneurysm, and thrombus. Doppler echocardiography is critical for diagnosing complications like **ventricular septal defects** or **mitral regurgitation**.

NUCLEAR IMAGING

- **Myocardial perfusion imaging** with **99mTc-sestamibi** reveals areas of reduced perfusion but cannot distinguish between acute and old infarcts.
- **Radionuclide ventriculography** (using labeled red blood cells) identifies regional wall motion abnormalities and estimates ejection fraction. However, it lacks specificity in pinpointing the cause of dysfunction.
- **Positron Emission Tomography (PET)** helps differentiate viable but stunned myocardium from scar tissue using metabolic tracers like **fluorodeoxyglucose (FDG)**. PET is more accurate but less accessible compared to **SPECT**, which offers a cost-effective alternative.

"TRIGLYCERIDE"

Pathophysiology , genomics and metabolism

Triglyceride level (TGs) represent a principal element of dietary fat, structurally comprising a glycerol molecule bonded to three fatty acid (FA) chains. Due to their hydrophobic nature, TGs are insoluble in plasma and must be transported within lipoproteins such as chylomicrons (CM), very low density lipoproteins (VLDL), and VLDL remnants collectively termed triglyceride rich lipoproteins (TRL).

Upon ingestion, TGs are hydrolyzed in the intestinal tract by lipases, producing free FAs and monoglycerides [32]. These monoglycerides are re-esterified into TGs within enterocytes by the enzyme diacylglycerol acyltransferase[33]. Simultaneously, cholesterol is underwent esterification by acyl-CoA:cholesterol acyltransferase [34]. Along with phospholipids , cholesterol esters , and a suite apolipoproteins, these re-synthesized TGs are assembled into emerging CM using apolipoprotein B48 and microsomal TG transfer protein [35].

After entering systemic circulation, CM deliver FAs to adipose tissue and muscles through lipolysis catalyzed by lipoprotein lipase -(LPL), located endothelial surfaces capillaries in skeletal, cardiac, adipose tissues[36.37]. In the liver, FAs from TGs are either oxidized via β-oxidation or repackaged into VLDL and secreted back into the circulation[32,38].

Endogenous TRL Production: Within hepatocytes, the synthesis of TRL begins similarly to intestinal CM production. Here, cholesterol, FAs, and TGs are combined with apolipoprotein B100 through microsomal TG transfer protein. While hepatic cholesterol levels are tightly regulated, TG levels fluctuate based on incoming FAs (from CMr or peripheral tissues) ,denovo lipogenesis [32]. Excess carbohydrates can be turned into FAs via acetyl-CoA derived from pyruvate. These newly formed FAs go through esterification with glycerol-3-phosphate, yielding TGs that are incorporated into VLDL.

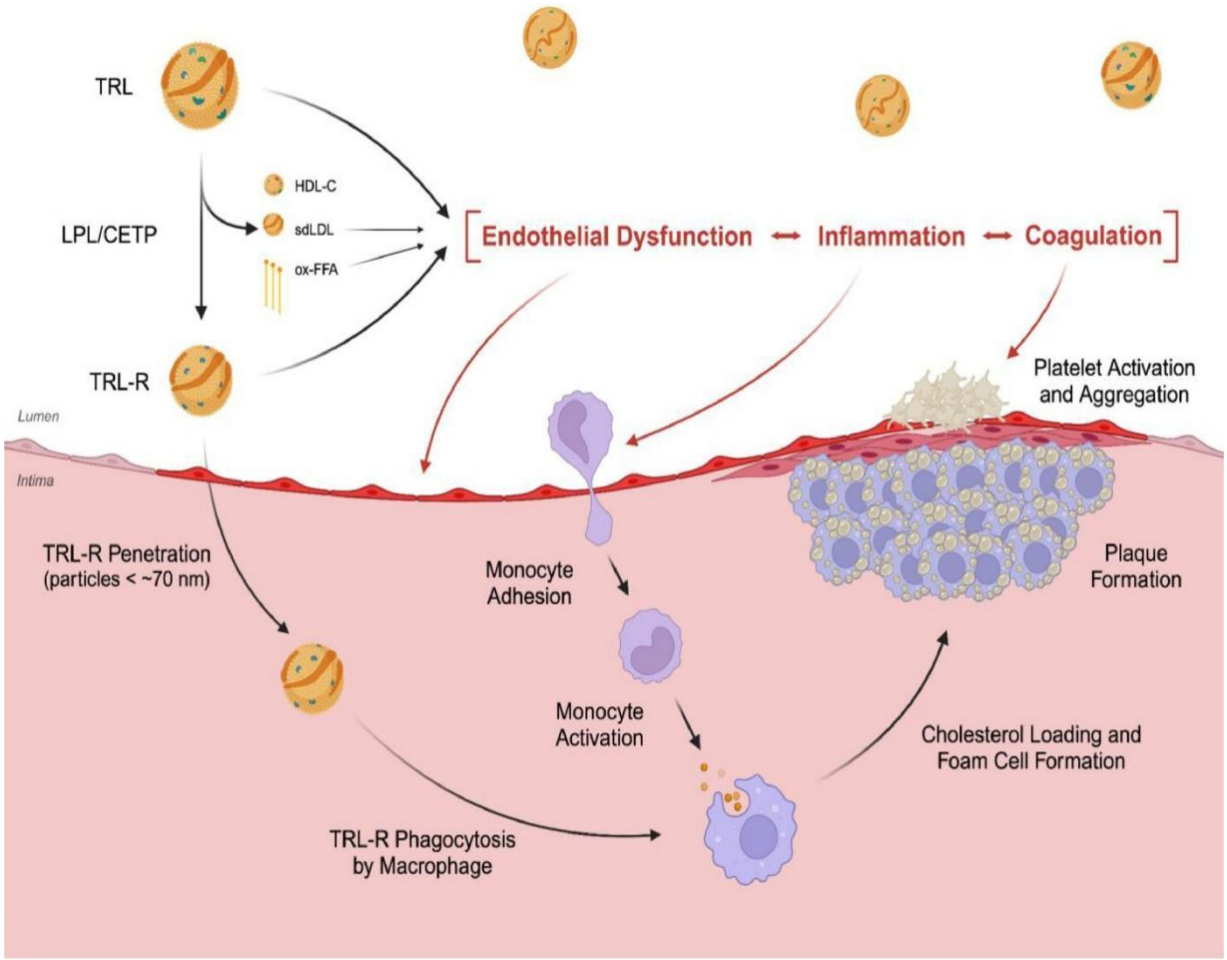
VLDLs are hydrolyzed by LPL, releasing FAs to peripheral tissues [39]. As TGs are removed, the VLDL particles shrink and increase in density, acquiring cholesterol and evolving into intermediate-density lipoproteins (IDLs), precursors to low-density lipoproteins (LDL) [40]. The cholesterol content carried within TRL particles is referred to as **remnant cholesterol (RC)** [41]. VLDL and its remnants can be cleared by VLDL receptors, LRP1, or heparan sulfate proteoglycans, while IDL particles are removed via LDL receptors and LRP1 [36,37].

Atherogenic Potential of TRLs

Similar to LDL, TRL particles can become trapped in the intimal layer of arterial walls (Fig. 2). Once retained, they are taken up by macrophages, forming foam cells and triggering inflammatory responses [42]. Free FAs released within subendothelial space exhibit pro-inflammatory effects ,contribute to atherogenesis[42,43] These remnants also promote pro-inflammatory cytokine release and may damage endothelial cells directly [44,45], cultivating a pro-oxidant and pro-atherogenic environment. Due to their high heterogeneity, TRL and remnants vary

significantly between individuals, making it difficult to define precise features responsible for their atherogenicity [46].

Fig. 5. Pathophysiology of triglyceride proatherogenic effects.



Genomics of Hypertriglyceridemia

Genetic investigations particularly genome-wide association studies have identified variants contributing to hypertriglyceridemia (HTG) [47]. While monogenic forms of HTG (e.g., familial chylomicronemia syndrome) are rare (~2%), most cases arise from interactions between multiple genes and environmental factors [47]. This genetic diversity leads to a wide spectrum of TG levels, with familial and multifactorial chylomicronemia syndromes representing the most severe phenotypes.

Definition of Hypertriglyceridemia

Triglyceride (TG) levels below 150 mg/dL are considered within the normal fasting range. The American College of Cardiology , American Heart Association (AHA) classify hypertriglyceridemia as moderate when TG levels range from 175–499 mg/dL (2.5–5.6 mmol/L), and severe when fasting levels exceed 500 mg/dL (5.65 mmol/L) [48,49]. In contrast, the European Society of Cardiology (ESC) categorizes TG levels range between 175–885 mg/dL (2.0–9.9 mmol/L) as mild to moderate, above 886 mg/dL (10 mmol/L) as severe, over 1,770 mg/dL (20 mmol/L) as very severe [50,51].

HTG and Coronary Plaque Formation

TG-rich lipoproteins contribute to vascular pathology primarily through **endothelial dysfunction**. Although large CM and VLDL particles do not cross the endothelium, their remnants, VLDL remnants and chylomicron remnants (CMr)[52].Once internalized, they contribute to **foam cell formation**, which initiates **fatty streak development**, a hallmark of early atherosclerosis [45].

TRL particles are significantly larger and carry substantially more cholesterol than LDL, amplifying their atherogenic potential. **Free fatty acids (FFAs)**, liberated from these lipoproteins, further exacerbate vascular injury by increasing **oxidative stress**, disrupting **insulin signaling**, and activating the **renin-angiotensin system**, which leads to **endothelial cell death** via apoptosis [46] These particles also provoke inflammation by boosting **cytokine** and **interleukin** production [47]. Despite these mechanisms, large cohort studies like **MESA** and **PESA** suggest inconsistent relationships between TG levels and **coronary artery calcium (CAC)**. For instance, in the PESA study, TG ≥150 mg/dL were associated with non-coronary subclinical atherosclerosis (OR 1.35; 95% CI 1.08–1.68; p = 0.008) and arterial inflammation (OR 2.09; 95% CI 1.29–3.40; p = 0.003) [53,54]

In symptomatic populations, studies using **optical coherence tomography (OCT)**, **intravascular ultrasound (IVUS)**, and **coronary CT angiography (CCTA)** have shown a correlation between higher TG or RC (remnant cholesterol) levels and **plaque vulnerability**, including necrotic cores, positive remodeling, and spotty calcifications[55-57] The **TG/HDL-C ratio** has emerged as a reliable indicator of **metabolic syndrome** and **remnant cholesterol** burden[58]. A retrospective study of 587 patients further confirmed the link between RC and total plaque volume, particularly in patients with optimally controlled LDL-C (OR 3.87 / 1 mmol/L RC increase- p = 0.004)[59]. These findings reinforce the notion that **TG levels act as a surrogate** for elevated RC, the true mediator of atherosclerotic risk.

HTG and ASCVD Risk

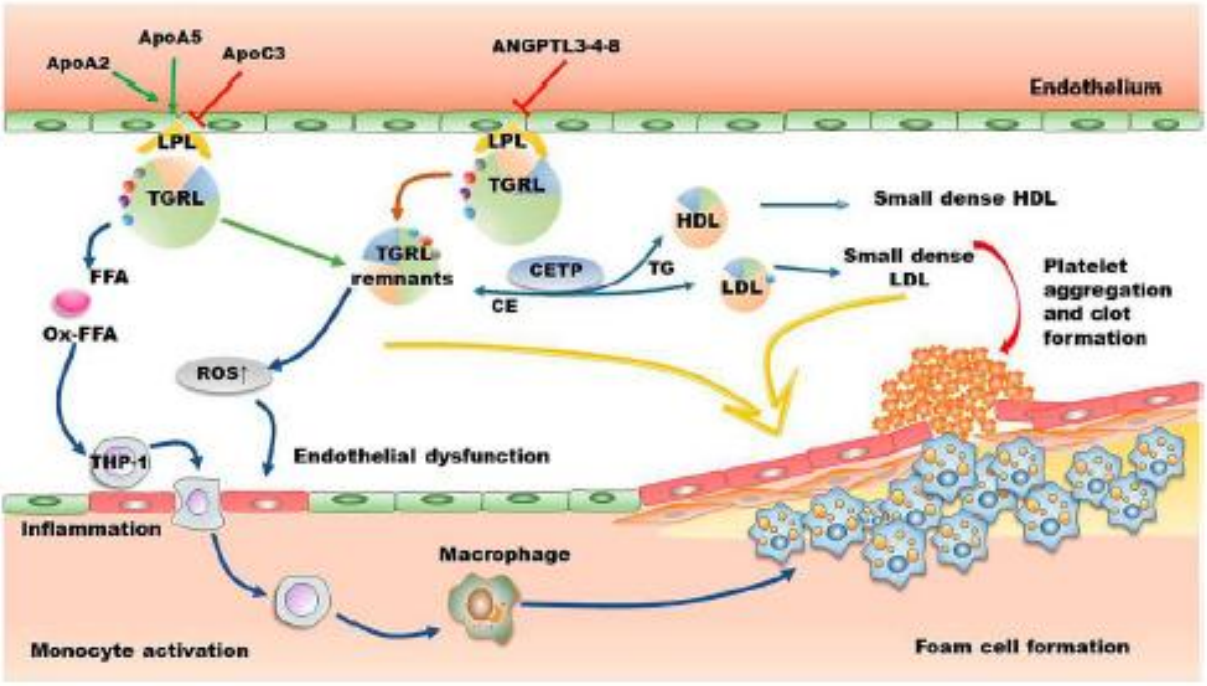
While LDL-C remains the cornerstone of ASCVD prevention, recent decades have expanded the focus to include TG as a contributor to cardiovascular risk [60]. Even within ranges previously deemed normal (<150 mg/dL), elevated TG levels were linked to higher cardiovascular risk in analyses of the **ARIC** and **Framingham Offspring studies** [61]

As per the study done by madsen CM et al [62] ,In the **Copenhagen General Population Study** (58,547 individuals), those with TGs between 352–439 mg/dL, {4.0–4.99 mmol/L} had markedly higher rates of **myocardial infarction (MI)** ,**major adverse cardiovascular events (MACE)** than those with TG <88 mg/dL (<1.0 mmol/L). Similar outcomes were observed in the **PREDIMED trial**, where both TG and RC (but not LDL-C) were independent predictors of ASCVD events [63]. Reducing TG to <150 mg/dL among statin users significantly lowered the risk of recurrent events such as cardiovascular death or MI, even when LDL-C levels were already controlled [64,65,66] In another massive dataset of 15.6 million Korean adults, each doubling of TG was associated with a log-linear increase in CVD, MI, and stroke mortality—even in those with LDL-C <100 mg/dL[67,68]

Genetically reduced TG levels, whether via **Mendelian variants** or GWAS signals, have been connected to lower risks of ischemic heart disease {HR 0.40; 95% CI 0.31–0.52} [69] These associations remain robust even when accounting for LDL-C and other risk factors [70]. In large population studies, RC levels ≥30 mg/dL were associated with significantly increased ASCVD risk and even **all-cause mortality**, underscoring RC's pathogenic role [65,65] HTG is also implicated in **peripheral artery disease (PAD)**.

Ischemic stroke risk also appears to correlate with TG levels. Two prospective Chinese studies found that each 1 mmol/L TG increase led to a 6–7% rise in stroke risk, even after adjustment for common risk factors [71,72,73]. Evidence from **genetic and Mendelian randomization studies** further supports a causal link. One large meta-analysis (62,199 individuals; 12,099 CHD events) found that TG-related SNPs were associated with higher CHD risk (OR 1.61–1.62 per log-unit increase) [74,75] Variants in **APOA5** and **LPL** genes were also associated with increased MI risk and lower TG levels, respectively. Notably, carriers of more **TG-lowering LPL alleles** had 31% lower TG, 23% lower RC, and 15% higher HDL-C levels—with improved survival outcomes [76,77.]

FIGURE 6. The mechanisms of TGRL on promoting atherosclerosis.



In circulation, **apoAII** and **apoAV** enhance LPL activity, facilitating TGRL lipolysis, while **apoAIII** and **ANGPTL3/4/8** inhibit it—though some studies suggest apoAIII has no direct effect. Lipolysis releases **free fatty acids (FFAs)**, triggering **ROS production**, **inflammation**, and **endothelial dysfunction**, leading to **foam cell formation**. **CETP-mediated lipid exchange** promotes formation of **small dense LDL and HDL**, accelerating atherosclerosis. Additionally, **lipolytic byproducts** activate **platelets**, increasing **thrombosis risk**.

ANGPTL, angiopoietin-like protein; Apo, apolipoprotein; CE, cholesteryl ester; CETP, cholesteryl ester transport protein; FFA, free fatty acid; HDL, high density lipoprotein; LDL, low-density lipoprotein; LPL, lipoprotein lipase; Ox-FFA, oxidized FFA; ROS, reactive oxygen species; TG, triglycerides; TGRL, triglyceride-rich lipoprotein.

Methods and Pathophysiological importance of Elevated Fasting Triglycerides

Methods resulting in Elevated Fasting Triglycerides

Understanding the metabolic origins and clinical implications of elevated triglycerides—both fasting and postprandial—is essential, especially given their established link to atherosclerosis. High fasting triglyceride levels typically arise due to increased secretion of very low-density lipoprotein triglycerides (VLDL-TG), often associated with hepatic steatosis, and/or diminished clearance of triglycerides via lipoprotein lipase (LPL) activity [(64)].

Although visceral fat is more insulin resistant than subcutaneous tissue, the liver primarily receives FFAs from secondary fat depots [130,131]. Additionally, hepatic denovo lipogenesis, especially in obese individuals with hyperinsulinemia, contributes to liver fat and further VLDL production [(65)]. On the clearance side, insulin resistance and type 2 diabetes impair LPL activity and expression in adipose tissue, hampering VLDL-TG hydrolysis and prolonging the presence of these particles in circulation [(31–33,132)]. Elevated levels of apolipoprotein C-III (apoC-III) further interfere with triglyceride clearance by restricting both LPL and receptor-mediated uptake of apoB-containing lipoproteins [80,81].

Pathophysiological Significance of High Fasting Triglycerides

Even though ongoing debate over triglycerides as an independent cardiovascular risk factor, several mechanistic insights support their pathological role. In insulin-resistant individuals, VLDL particles are overproduced and persist longer in the circulation [82][(10–12,31–33)]. This prolonged residence time, coupled with CETP-mediated lipid exchange, leads to the transfer of triglycerides from VLDL to LDL and HDL particles. Once hydrolyzed by hepatic lipase, these lipoproteins transform into smaller, denser forms—small dense LDL and HDL—which are linked

to increased atherogenicity due to reduced LDL receptor clearance, increased subendothelial retention, and greater HDL catabolism [83][(1)].

In addition to these changes, VLDL remnants—particles smaller than 70 nm—can penetrate the arterial wall and engulfed by macrophages, promoting foam cell formation even in the absence of oxidation, mimicking the behavior of LDL particles [(36,133,134)]. TRL remnants and their hydrolysis products exert pro-inflammatory effects in vitro. They increase monocyte adhesion to endothelial cells and upregulate adhesion molecules (VCAM-1, ICAM-1, E-selectin), elevate endothelial cytokine production (TNF-α, IL-1β), and stimulate platelet activation [(135–137)]. ApoC-III enrichment in TRLs further promotes these effects via NF-κB activation [(138–140)], while LPL-generated lipolysis products can induce endothelial cell apoptosis and inflammation [84][(141–144)].

While the cholesterol content of TRL remnants may primarily drive their atherogenicity, hepatic overproduction of VLDL—due to increased FFA influx, de novo lipogenesis, or pre-existing hepatic triglyceride stores—remains the core mechanism sustaining elevated triglycerides and their harmful effects [85][(130)].

Methods and Pathophysiological importance of Elevated Postprandial Triglycerides

Functions Behind Elevated Postprandial Triglycerides

Mechanisms Behind Elevated Postprandial Triglycerides

The terms "non-fasting" and "postprandial" triglycerides are often used exchangable, as they represent the body's lipid response to feeding. In the fed state, competition between newly secreted chylomicrons and existing VLDL particles for LPL-mediated hydrolysis becomes a central issue [(1,31–33)]. This effect is magnified in insulin-resistant individuals, where both VLDL production is unchecked and LPL function is compromised[86] [(31–33,67,68,101)].

Insulin resistance also promotes the overproduction of chylomicrons due to enhanced stability of apoB48 and microsomal transfer protein, alongside increased enterocyte lipogenesis [(28–30)]. This raises chylomicron remnant levels and contributes to liver fat, fueling further dyslipidemia [(130)]. Studies show that hepatic de novo lipogenesis is 14–18% higher postprandially compared to the fasted state following mixed meals, enriching the circulating triglyceride pool [87][(145)]. Consequently, fasting TG levels predict, but do not fully explain, postprandial lipid spikes[88] [(23,146)].

Pathophysiological Relevance of Postprandial Hypertriglyceridemia

In the postprandial period, a surge in TRL remnants, stemming from both dietary (chylomicron-derived) and hepatic (VLDL-derived) lipids, increases atherogenic risk. These remnants are more numerous and persist longer due to LPL saturation, especially after high-fat meals[89] [(147)]. Their longer residence increases subendothelial infiltration and plaque development.

High-fat meals also trigger systemic inflammation, marked by elevated serum IL-6, IL-8, TNF-α, and soluble adhesion molecules like sICAM-1 and sVCAM-1 [(148–150)]. Nappo et al. demonstrated that these markers significantly correlate with postprandial TG levels [(150)]. In vitro, TRLs from hypertriglyceridemic individuals upregulate pro-inflammatory and adhesion-related genes in endothelial cells, with greater effects seen postprandially[90] [(151,152)]. Additionally, both fasting and postprandial TRLs boost NF-κB activity, intensifying inflammatory responses.

Further, postprandial inflammation is linked to innate immune activation. Studies show increases in neutrophils, lipopolysaccharides (LPS), and TLR-4 activity following high-fat intake[91] [(153–155)]. However, newer findings suggest that LPL-mediated hydrolysis of saturated FFAs

may drive this inflammatory response more than LPS itself [(156)]. Lauric acid and other lipolysis products activate TLR-2 and TLR-4 signaling in macrophages [(141–143)].

Complement activation also occurs postprandially, with increased levels of C3 observed in both healthy individuals and CAD patients [(157)]. This may be stimulated by chylomicron–adipocyte interactions [(158)], though the significance is debated due to concurrent rises in C4b-binding protein, a complement inhibitor [92][(159)].

Postprandial triglycerides also impair vascular function. Flow-mediated dilation (FMD), a predictor of cardiovascular events, is consistently reduced after high-fat meals due to oxidative stress, which limits nitric oxide availability [(160–162)]. This state is also associated with increased oxidized LDL, a key driver of foam cell formation [(163,164)]. Additionally, VEGF-A and VEGF-C levels rise postprandially, although their implications in plaque instability or vascular remodelling remain uncertain[93,94] [(149,165,166)].

RELATED STUDIES:

"In a study done by **Brown[95]** (**1966**) to assess the relationship between serum lipid levels and ischemic heart disease (IHD), 470 middle-aged men underwent fasting and 9-hour postprandial cholesterol and triglyceride testing following a 70 g fat meal labeled with radioactive triolein. They found that individuals with clinical signs of IHD had significantly elevated fasting and postprandial cholesterol and triglyceride levels, but no significant difference in lipid-bound radioactivity. The strong correlation between fasting and 9-hour triglyceride levels suggested that the fat tolerance test offered no diagnostic advantage over fasting triglyceride measurements. They concluded that while IHD was more common in hypertriglyceridemic individuals, cholesterol was also elevated, making it difficult to identify a single causal lipid. Notably, the highest IHD prevalence was seen in those with elevated cholesterol but normal triglycerides, indicating that various forms of hypertriglyceridemia may still play a role in IHD development".

"In a study done by **Manochehri (2016)**[14] to evaluate the association between postprandial triglyceride levels and coronary artery disease (CAD), 80 male participants—half with angiographically confirmed CAD and half as healthy controls—were assessed at the 502 Hospital of Army. They found that both fasting and postprandial triglyceride levels were significantly elevated in CAD patients, with postprandial levels providing greater sensitivity (p=0.001). They concluded that measuring postprandial triglyceride levels is a more reliable indicator than fasting triglycerides in identifying individuals with CAD".

"In a study done by **Jeppesen (1998)**[13] to investigate the link between fasting triglyceride levels and the risk of ischemic heart disease (IHD), 2,906 white men aged 53 to 74 years from the Copenhagen Male Study were followed over eight years. They found that higher fasting triglyceride levels were significantly associated with increased IHD incidence, with relative risks of 1.5 and 2.2 for the middle and highest triglyceride tertiles, respectively, after adjusting for confounders. They concluded that elevated fasting triglycerides are an independent and strong predictor of IHD risk, even in individuals with high HDL cholesterol levels".

"In a study done by **Tarannum (2021)**[96] to compare fasting and postprandial lipid profiles in patients with ischemic heart disease (IHD), 50 clinically stable patients were evaluated using both fasting and 4-hour postprandial lipid measurements. They found that while total cholesterol, HDL, and LDL levels showed no significant difference between fasting and postprandial states, postprandial triglyceride levels were significantly higher (275.65 ± 48.0 mg/dL) compared to fasting levels (210.02 ± 63.9 mg/dL). They concluded that among lipid parameters, only triglycerides showed a significant postprandial rise, highlighting its potential importance in assessing cardiovascular risk".

"In a study done by **Suliman Saber (2025)**[15] to investigate the relationship between postprandial triglyceride levels and the severity of coronary artery disease (CAD), 120 patients

diagnosed with CAD via coronary angiography were evaluated at Al Bayda Medical Center. They found that postprandial triglyceride, cholesterol, and LDL levels showed significant variation across patient groups, and higher postprandial triglycerides were strongly associated with more severe CAD, as measured by the SYNTAX score. They concluded that postprandial triglyceride levels are significantly correlated with CAD severity, even when fasting triglyceride levels are normal, suggesting a potential role in risk stratification and management of CAD patients".

"In a study done by **Werner (2014)**[17] to evaluate the prognostic value of fasting and postprandial triglycerides (TG) in patients with coronary artery disease (CAD), 514 angiography-confirmed CAD patients were assessed using a standardized oral triglyceride and glucose tolerance test. They found that both fasting and postprandial TG levels predicted cardiovascular death and hospitalization over 48 months, with fasting TG >150 mg/dL significantly increasing risk (HR 1.79; p = 0.0001). However, postprandial TG measurements did not enhance risk prediction beyond fasting values. They concluded that elevated fasting TG levels independently forecast cardiovascular events, while postprandial TG offered no additional predictive benefit in CAD patients on optimal medical therapy".

"In a study done by **Chakraborty (2020)**[97] to compare fasting and postprandial lipid profiles in patients with diabetes and prediabetes, 51 diabetic and 32 prediabetic individuals were evaluated for lipid and glucose levels in both states. They found that postprandial triglycerides and TG/HDL-C ratios were significantly higher in diabetics compared to controls and prediabetics, and these postprandial values showed a stronger correlation with HbA1c than fasting levels. They concluded that postprandial TG and TG/HDL-C are more reliable indicators of lipid abnormalities and cardiovascular risk in diabetic and prediabetic patients than their fasting counterparts".

"In a study done by **P K Jawaharlal**[99] (**2020**) to examine the association between atherosclerotic risk factors and postprandial triglyceride levels in patients with unstable angina, 50 patients were assessed based on clinical symptoms, ECG findings, and cardiac enzyme levels. They found that elevated postprandial triglycerides were common, particularly among those with high waist-hip ratios, diabetes, overweight status, and low HDL levels. They concluded that postprandial hypertriglyceridemia may serve as an independent risk factor for atherosclerosis, highlighting the importance of evaluating postprandial TG levels in the clinical assessment of ischemic heart disease patients".

"In a study done by **Xie** (**2023**)[18] to establish a postprandial triglyceride (TG) cut-off corresponding to optimal fasting TG levels in Chinese adults, 618 inpatients aged 18–70 were assessed for lipid profiles at 0, 2, and 4 hours after a typical Chinese breakfast. They found that postprandial TG and remnant cholesterol (RC) levels peaked at 4 hours, with 1.56 mmol/L identified as the postprandial TG threshold equivalent to a fasting TG of 1.2 mmol/L. They concluded that this cut-off can reliably determine TG status in both fasting and postprandial states, with postprandial TG at 4 hours emerging as an independent predictor of non-optimal TG levels".

"In a study done by **Bendwal** (**2018**)[99] to evaluate the relationship between triglyceride levels and carotid intima-media thickness (CIMT) in patients with type 2 diabetes mellitus, 120 individuals without known vascular disease were assessed using ultrasonography. They found that CIMT increased significantly with higher fasting and postprandial triglyceride levels, with the highest CIMT observed in the group with elevated levels in both states. They concluded that while both fasting and postprandial triglycerides are associated with CIMT, postprandial levels show a stronger correlation, indicating a better predictor of subclinical atherosclerosis".

"In a study done by **Chahal** (**2021**)[100] to compare fasting and postprandial lipid profiles in patients with type 2 diabetes mellitus, 50 diabetic individuals and 50 matched healthy controls were evaluated at a tertiary care hospital. They found that diabetics had significantly higher levels of total cholesterol, triglycerides, LDL, and VLDL, and lower HDL in both fasting and postprandial states compared to controls. Moreover, diabetic patients exhibited a marked rise in postprandial total cholesterol and triglycerides relative to fasting levels. They concluded that postprandial lipid abnormalities, especially hypertriglyceridemia, are prominent in type 2 DM and warrant routine postprandial lipid testing alongside fasting measurements".

"In a study done by **Bansal** (**2007**)[101] to assess the predictive value of fasting and nonfasting triglyceride levels for cardiovascular events in initially healthy women, triglyceride levels were evaluated in relation to traditional risk factors and insulin resistance over a median follow-up of 11.4 years. They found that while both fasting and nonfasting triglycerides correlated with cardiovascular risk, only nonfasting triglycerides maintained a strong, independent association with cardiovascular events after full adjustment for confounding variables. They concluded that nonfasting triglyceride levels, especially those measured 2 to 4 hours after eating, are a more reliable predictor of cardiovascular risk than fasting levels".

"In a study done by **Shehata** (**2019**)[102] to evaluate the predictive value of postprandial triglyceride levels for coronary artery disease (CAD) severity, 105 patients diagnosed with CAD via coronary angiography were assessed at Zagazig University Hospitals. They found that ischemic heart disease was significantly more prevalent in patients with both abnormal angiographic findings and elevated postprandial triglycerides compared to those with normal postprandial levels. They concluded that postprandial triglyceride levels are a more reliable indicator than fasting triglycerides in predicting the severity of coronary heart disease."

"In a study done by **Schwartz (2015)**[16] to explore the association between fasting triglycerides and cardiovascular risk after acute coronary syndrome (ACS), data from two major trials—dal-OUTCOMES and the atorvastatin arm of MIRACL—were analyzed. They found that higher fasting triglyceride levels were significantly linked to increased both long-term and short-term risk of adverse cardiovascular events, independent of LDL cholesterol. They concluded that in statin-treated ACS patients, elevated triglycerides are predictive of future cardiovascular risk, suggesting that triglyceride-rich lipoproteins may represent an important therapeutic target."

"In a study done by **Deshmukh (2020)**[103] to compare lipid profiles between ischemic heart disease (IHD) patients and healthy controls, both fasting and postprandial lipid levels were measured and analyzed. They found that IHD patients had significantly higher fasting triglycerides, VLDL, and total cholesterol levels compared to controls. Postprandial triglycerides and VLDL levels increased more prominently in IHD patients than in controls, while total cholesterol, HDL, and LDL levels decreased postprandially in both groups. They concluded that hypertriglyceridemia, particularly in the postprandial state, is more pronounced in IHD patients and may serve as a significant risk marker.".

MATERIALS & METHODS

MATERIALS AND METHODS

STUDY DESIGN: Case Control Study

DURATION OF STUDY: 18 months (MAY'2023`- OCTOBER'2024)

STUDY POPULATION: Pregnant women in labour attending department of OBG, RL Jalappa Hospital, Kolar during the study period.

STUDY AREA: RL Jalappa Hospital, Kolar.

INCLUSION CRITERIA:

- CASE GROUP – The case group will include IHD subjects proved with ECG/ECHO.
- CONTROL GROUP – The control group will include normal subjects.
- **Patient characteristics:** After obtaining detailed history, meticulous examination, baseline investigations the cases and controls will be subjected to blood tests to determine serum levels of Fasting and Postprandial Triglyceride levels.

EXCLUSION CRITERIA:

- On treatment with lipid lowering drugs.
- Rheumatic heart disease.
- Oral contraceptive pills or other hormone therapy
- Abnormal liver and Renal function test.
- Cases of Prinzmetal's Angina
- Acute Myocardial infarction.
- Patient on treatment with Tablet Rosiglitazone in past one month.

METHOD OF DATA COLLECTION

A Case Control study was done at R L JALAPPA HOSPITAL TAMAKA KOLAR attached to SRI DEVRAJ URS MEDICAL COLLEGE under SRI DEVRAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH from May 2023 to October 2024. This study will be conducted following ethical approval from the institutional ethics committee, with informed consent obtained from all subjects. A total of 30 individuals will be included in each group based on specific inclusion and exclusion criteria. Prior to biochemical investigations, participants will be required to observe a minimum of 12 hours of overnight fasting, during which only water is allowed. Fasting blood samples will be collected using pre-sterile plain and sugar bulbs to assess fasting lipid profile and fasting blood glucose levels. Following sample collection, participants will consume their routine breakfast, excluding chocolate and ice cream on the day of testing. They will then have a standard lunch, and four hours post-lunch, an additional blood sample will be taken for the measurement of serum triglycerides. A comprehensive medical history will be recorded, and a thorough clinical examination will be performed. The participants will undergo several investigations including fasting blood glucose, a complete fasting lipid profile—comprising serum cholesterol, triglycerides, HDL, LDL, and VLDL—along with a 2-hour postprandial blood sugar and a 4-hour postprandial serum triglyceride test.

STATISTICAL METHODS

Data was collected and compiled in MS Excel. Statistical analysis was performed using SPSS for windows version 26.0. The description of data will be in the form of mean (±) SD for quantitative data and frequency and proportion for qualitative data. Student t test/ANOVA was used to compare continuous variables and $\chi 2$ test used to compare categorical variables. P value <0.05 was considered statistically significant.

SAMPLE SIZE CALCULATION

Was estimated in light of the difference in ratio of Fasting and Postprandial TG abnormal between cases and control groups. Proportion of Fasting and Postprandial TG abnormal in Cases was 75% and in control was 25% from the study by Mohammad Manochechri.[2] Using these values in the below mentioned formula

$$N = 2 (Z\alpha/2 + Z\beta)2 \, P (1-P)/(p1 - p2)2$$

Where ,

$Z\alpha/2 = Z0.05/2 = Z0.025 = 1.96$ at type 1 error of 5%

$Z\beta = Z0.20 = 1.28$ = At 90% power

p1 –p2= Difference in proportion in the two different groups = 50%

P= Pooled prevalence = [Proportion in cases (p1) + Proportion in controls (p2)]/2 = [25 + 75]/2 = 50

$$N = \frac{2 \times 50 \times 50 (1.96 + 1.28)2}{50 \times 50} = 27$$

evaluating non response rate of 10%, 27 + 2.7 = 29.7 ≈ 30

We will be including **30 subjects** in each group.

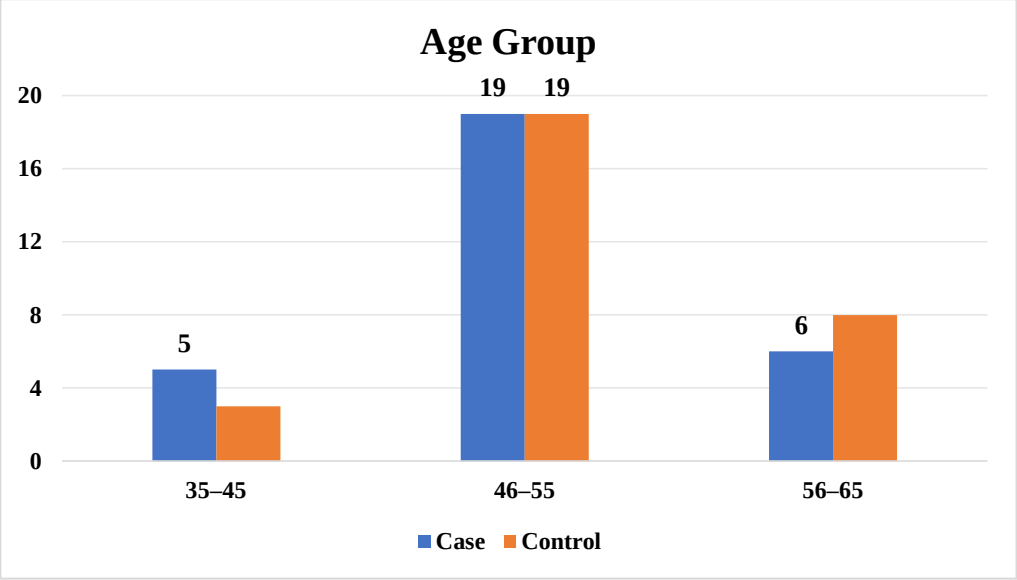
RESULTS

RESULTS:

TABLE 4. Age Group × Group

Age Group	Case N (%)	Control N (%)	p-value
35–45	5 (16.7%)	3 (10.0%)	0.700
46–55	19 (63.3%)	19 (63.3%)	
56–65	6 (20.0%)	8 (26.7%)	
MEAN±SD	51.47 ± 5.33	52.27 ± 5.89	
Total	30 (100.0%)	30 (100.0%)	

FIGURE 7. Age Group × Group

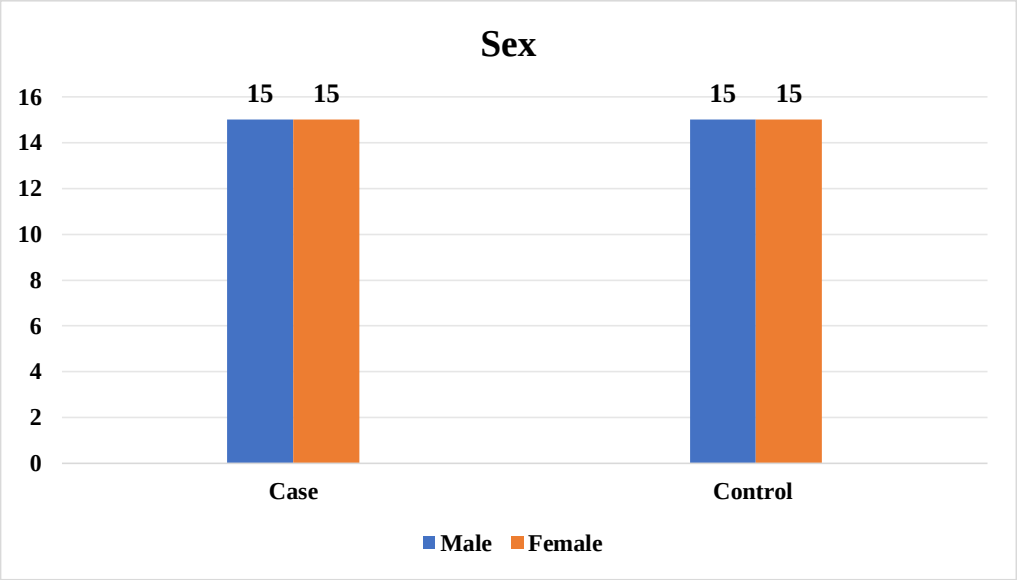


In the comparison of **age groups between IHD cases and controls**, the majority of individuals in both groups fell within the 46–55 year range (63.3%). A slightly higher proportion of IHD patients (20.0%) were in the 56–65 group compared to controls (26.7%). The youngest group (35–45 years) made up 16.7% of IHD cases and 10.0% of controls. The mean age was nearly identical between groups (51.47 ± 5.33 in cases vs. 52.27 ± 5.89 in controls). The p-value was 0.700, indicating no statistically significant difference in age group allocation.

TABLE 5. Sex × Group

Sex	Case N (%)	Control N (%)	p-value
Male	15 (50.0%)	15 (50.0%)	1.000
Female	15 (50.0%)	15 (50.0%)	
Total	30 (100.0%)	30 (100.0%)	

FIGURE 8. Sex × Group

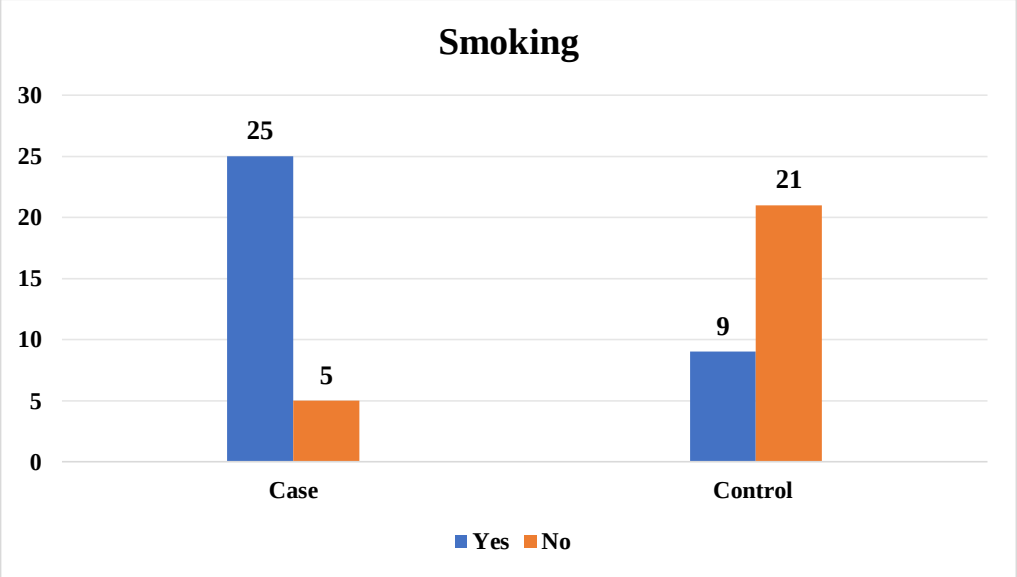


The **sex distribution** between IHD patients and controls was exactly equal, with 50% males and 50% females in each group, resulting in a p-value of 1.000.

TABLE 6. Smoking × Group

Smoking	Case N (%)	Control N (%)	p-value
Yes	25 (83.3%)	9 (30.0%)	0.060
No	5 (16.7%)	21 (70.0%)	
Total	30 (100.0%)	30 (100.0%)	

FIGURE 9. Smoking × Group

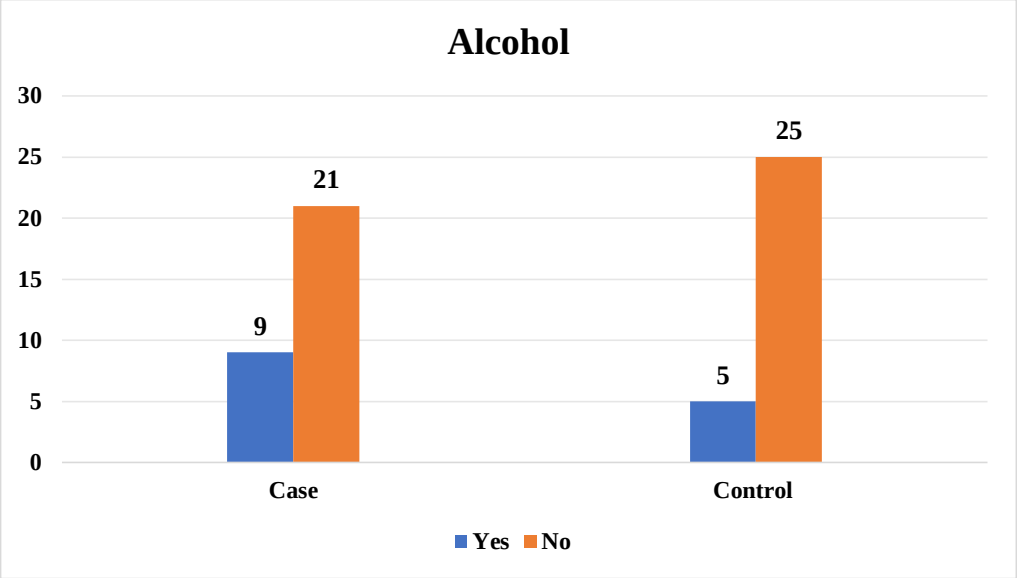


Analysis of **smoking behavior** showed that a higher proportion of IHD cases were smokers (83.3%) compared to controls (30.0%), while only 16.7% of cases were non-smokers compared to 70.0% of controls. Although this trend suggests a higher prevalence of smoking among IHD patients, the p-value was 0.060, just above the conventional significance threshold. This implies a potentially meaningful but statistically borderline association between smoking and IHD in this study.

TABLE 7. Alcohol × Group

Alcohol	Case N (%)	Control N (%)	p-value
Yes	9 (30.0%)	5 (16.7%)	0.552
No	21 (70.0%)	25 (83.3%)	
Total	30 (100.0%)	30 (100.0%)	

FIGURE 10. Alcohol × Group

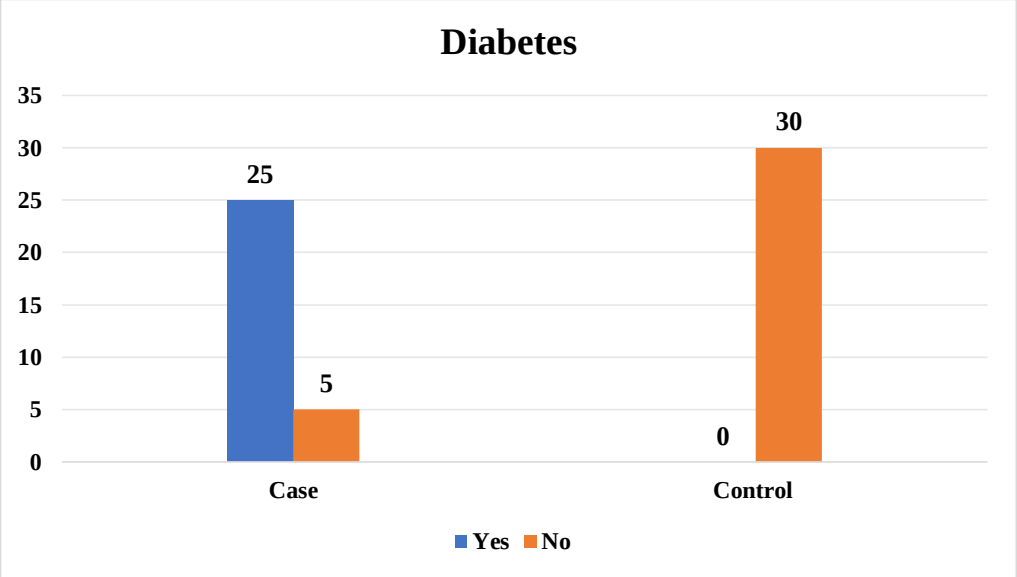


Regarding **alcohol consumption**, 30.0% of IHD cases reported drinking compared to 16.7% of controls. Conversely, 70.0% of cases were non-drinkers compared to 83.3% of controls. Despite a higher rate of alcohol use in the IHD group, the p-value was 0.552, indicating that this difference was not statistically significant.

TABLE 8. Diabetes × Group

Diabetes	Case N (%)	Control N (%)	p-value
Yes	25 (83.3%)	0 (0.0%)	**<0.001**
No	5 (16.7%)	30 (100.0%)	
Total	30 (100.0%)	30 (100.0%)	

FIGURE 11. Diabetes × Group

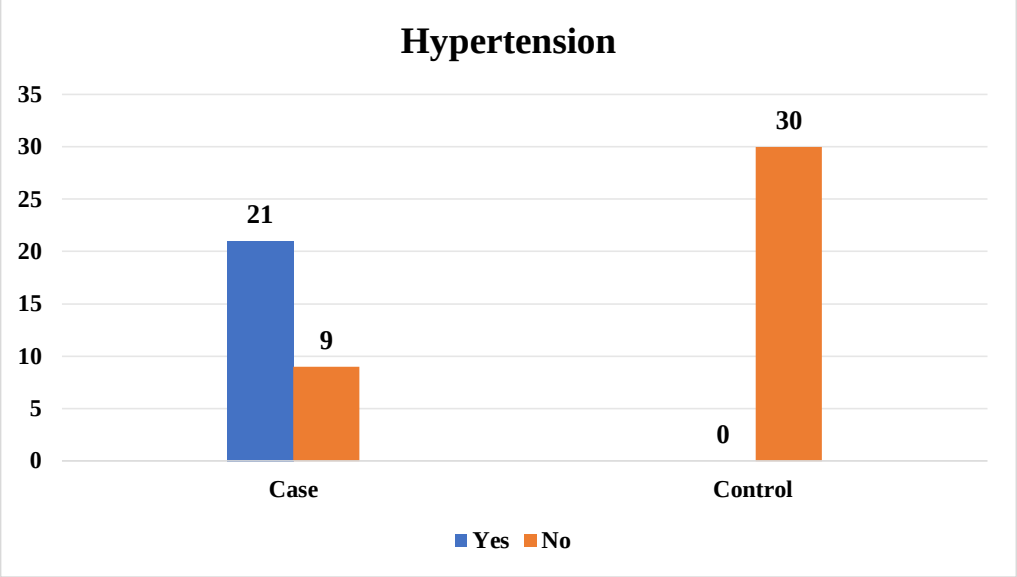


In the **diabetes status comparison**, there was a profound difference between groups. A striking 83.3% of IHD patients had diabetes, while none of the controls were diabetic. This highly significant difference ($p < 0.001$) strongly suggests a close association between diabetes and the presence of IHD.

TABLE 9. Hypertension × Group

Hypertension	Case N (%)	Control N (%)	p-value
Yes	21 (70.0%)	0 (0.0%)	**<0.001**
No	9 (30.0%)	30 (100.0%)	
Total	30 (100.0%)	30 (100.0%)	

FIGURE 12. Hypertension × Group

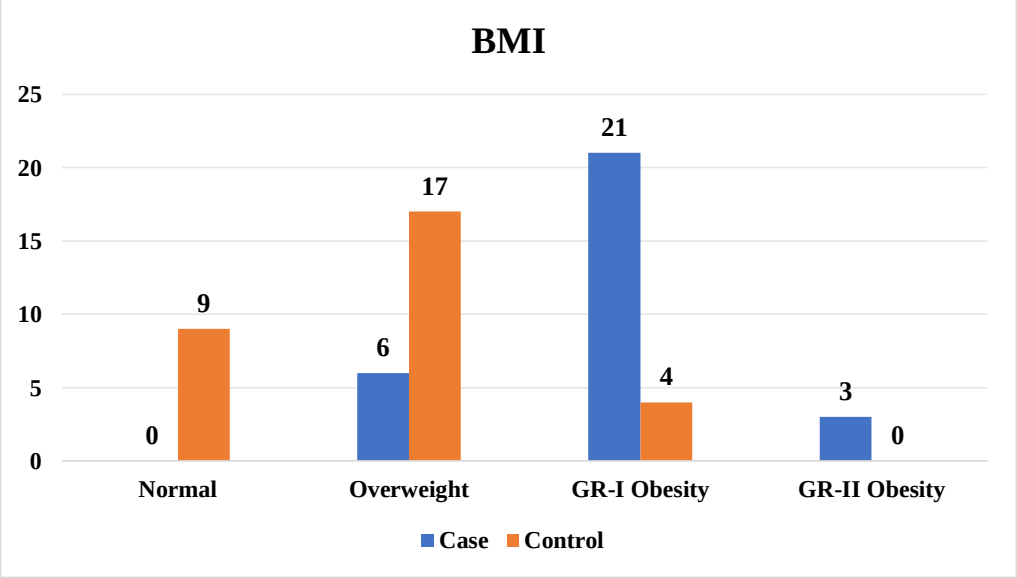


The ocuurence of **hypertension** was also markedly higher in the IHD group (70.0%) compared to 0% in the control group. With a p-value of less than 0.001, this finding indicates a very strong and numerically significant association between hypertension and IHD.

TABLE 10. BMI Category × Group

BMI	Case N (%)	Control N (%)	p-value
Normal	0 (0.0%)	9 (30.0%)	**<0.001**
Overweight	6 (20.0%)	17 (56.7%)	
GR-I Obesity	21 (70.0%)	4 (13.3%)	
GR-II Obesity	3 (10.0%)	0 (0.0%)	
MEAN±SD	31.94 ± 2.73	26.50 ± 2.75	
Total	30 (100.0%)	30 (100.0%)	

FIGURE 13. BMI Category × Group

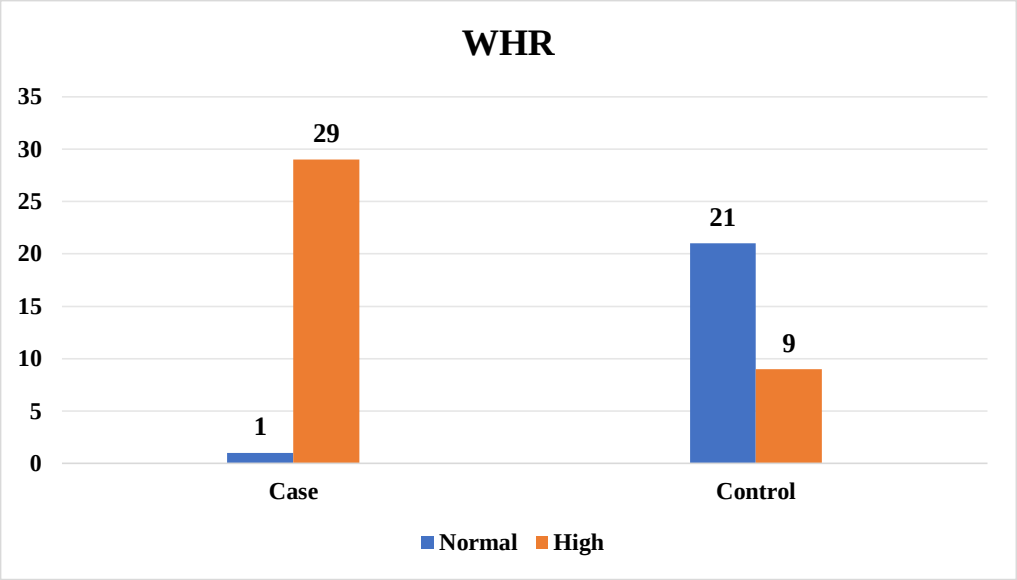


Regarding **BMI categories**, a majority of IHD patients (70.0%) fell into Grade I obesity, and an additional 10.0% were classified as Grade II obese. None of the IHD patients had a normal BMI, and only 20.0% were overweight. In contrast, 30.0% of the controls had a normal BMI, and 56.7% were overweight, with very few falling into obese categories. The mean BMI for the IHD group was 31.94 ± 2.73 compared to 26.50 ± 2.75 in controls. This distribution was based on data is significant with a p-value of less than 0.001, highlighting a strong association between higher BMI and IHD.

TABLE 11. Waist-Hip Ratio Category × Group

WHR	Case N (%)	Control N (%)	p-value
Normal	1 (3.3%)	21 (70.0%)	**<0.001**
High	29 (96.7%)	9 (30.0%)	
MEAN±SD	1.003 ± 0.058	0.083 ± 0.058	

FIGURE 14. Waist-Hip Ratio Category × Group

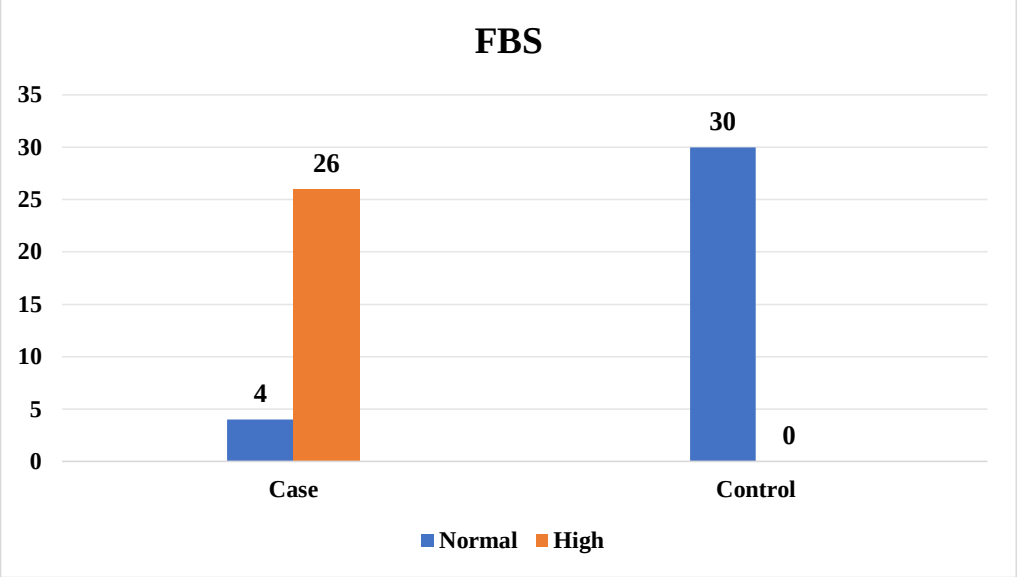


The **waist-hip ratio (WHR)** analysis revealed that 96.7% of IHD cases had high WHR values compared to only 30.0% of controls, a statistically significant finding ($p < 0.001$). This suggests a central obesity pattern that is strongly linked to IHD. The mean WHR was notably higher in cases (1.003 ± 0.058) than in controls (0.983 ± 0.058).

TABLE 12. FBS Category × Group

FBS	Case N (%)	Control N (%)	p-value
Normal	4 (13.3%)	30 (100.0%)	**<0.001**
High	26 (86.7%)	0 (0.0%)	
MEAN±SD	146.27 ± 17.87	89.07 ± 10.23	

FIGURE 15. FBS Category × Group

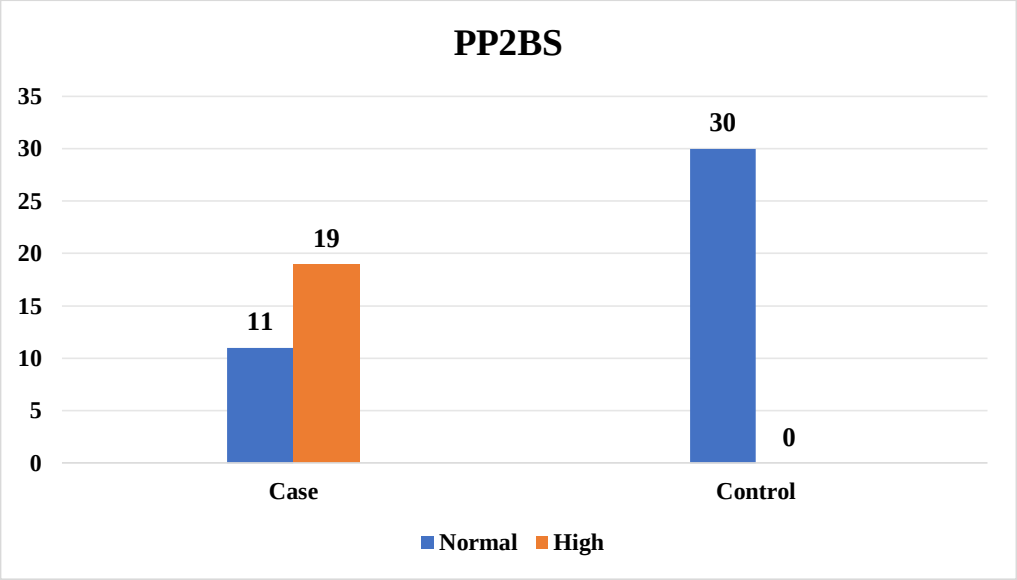


Comparison of **fasting blood sugar (FBS)** categories showed that 86.7% of IHD cases had high FBS levels versus none in the control group, while only 13.3% of cases had normal FBS compared to 100% of controls. The p-value was less than 0.001, denoting a significant association between elevated FBS and IHD. This was supported by mean FBS values: 146.27 ± 17.87 in cases versus 89.07 ± 10.23 in controls.

TABLE 13. PP2BS Category × Group

PP2BS	Case N (%)	Control N (%)	p-value
Normal	11 (36.7%)	30 (100.0%)	**<0.001**
High	19 (63.3%)	0 (0.0%)	
MEAN±SD	206.87 ± 23.22	129.10 ± 14.17	

FIGURE 16. PP2BS Category × Group

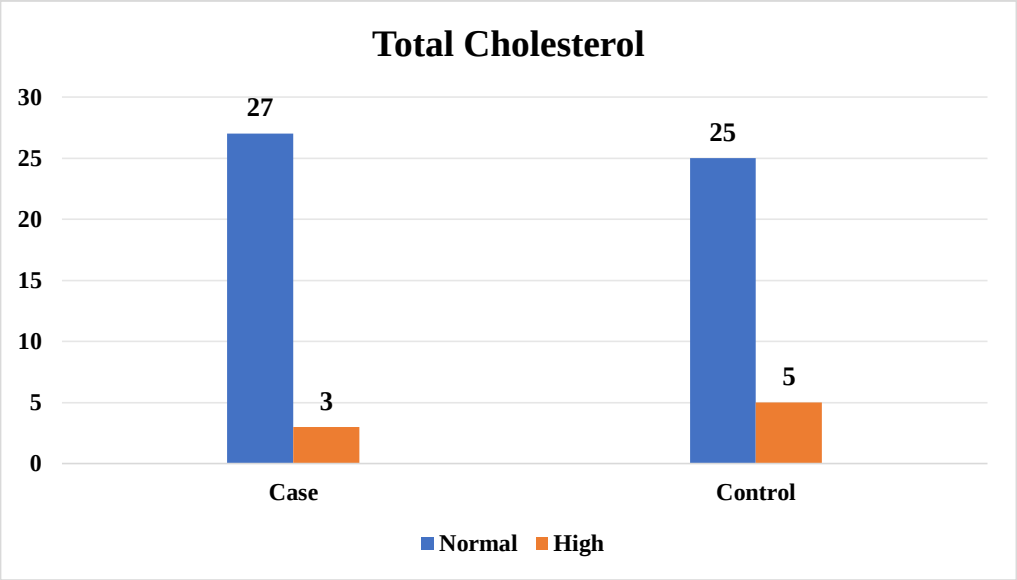


In terms of **postprandial blood sugar (PP2BS)**, 63.3% of IHD patients had elevated levels, while all controls were within the normal range. Only 36.7% of IHD patients had normal PP2BS. This disparity was highly significant ($p < 0.001$), and the mean PP2BS was 206.87 ± 23.22 in cases and 129.10 ± 14.17 in controls, further confirming poor glucose control among IHD cases.

TABLE 14. Total Cholesterol Category

Total Cholesterol	Case N (%)	Control N (%)	p-value
Normal	27 (90.0%)	25 (83.3%)	0.738
High	3 (10.0%)	5 (16.7%)	
MEAN±SD	178.80 ± 18.23	177.20 ± 18.70	

FIGURE 17. Total Cholesterol Category

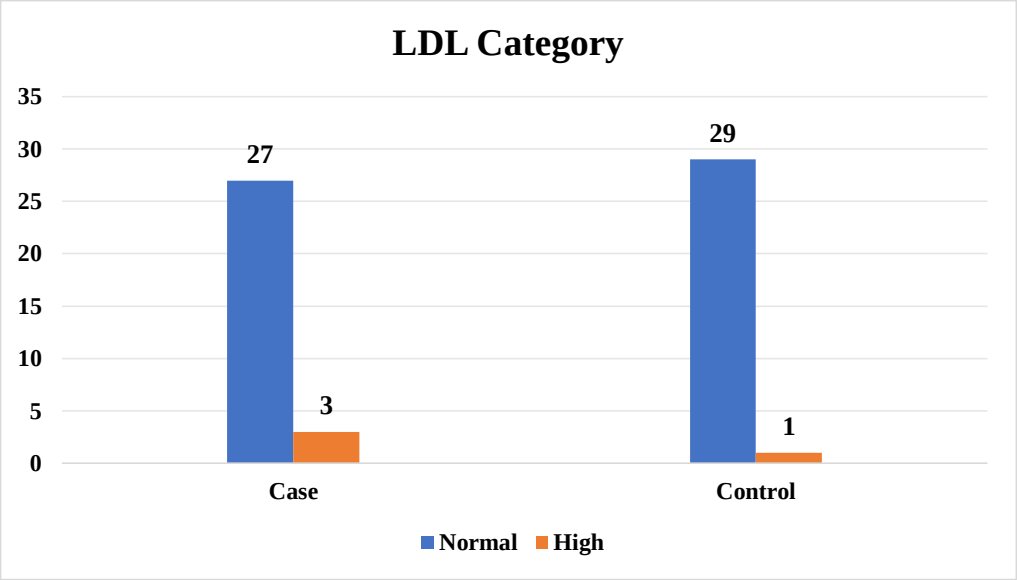


For **total cholesterol**, 90.0% of IHD patients and 83.3% of controls had normal values, while 10.0% and 16.7%, respectively, had high levels. The p-value was 0.738, indicating no significant gap between groups. Similarly, **LDL cholesterol** levels were not significantly different (p = 0.703), with 90.0% of cases and 96.7% of controls falling in the normal range.

TABLE 15. LDL Category

LDL	Case N (%)	Control N (%)	p-value
Normal	27 (90.0%)	29 (96.7%)	0.703
High	3 (10.0%)	1 (3.3%)	
MEAN±SD	96.57 ± 15.31	95.03 ± 15.71	

FIGRUE 18. LDL Category

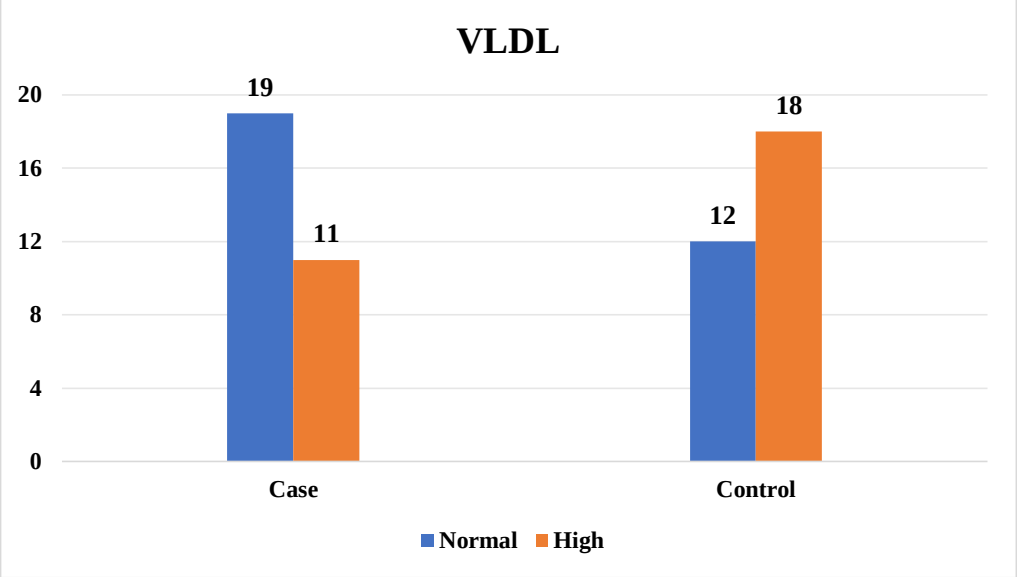


LDL cholesterol levels were similar between IHD cases and controls, with 90.0% and 96.7% having normal levels, respectively. High LDL was more frequent in cases (10.0%) than controls (3.3%), but this difference was not numerically significant (p = 0.703). Mean LDL levels were also comparable (96.57 ± 15.31 vs. 95.03 ± 15.71 mg/dL).

TABLE 16. VLDL Category

VLDL	Case N (%)	Control N (%)	p-value
High	19 (63.3%)	12 (40.0%)	0.233
Normal	11 (36.7%)	18 (60.0%)	
MEAN±SD	30.80 ± 5.45	29.13 ± 5.26	

FIGURE 19. VLDL Category

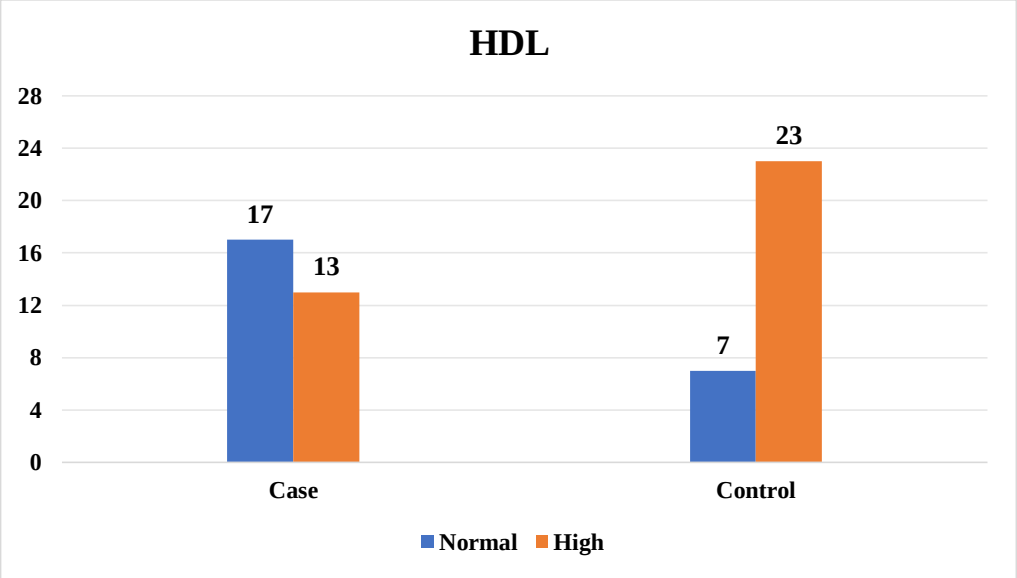


Evaluation of **VLDL** levels showed that 63.3% of IHD cases had high VLDL, compared to 40.0% of controls. Although more prevalent in the IHD group, this difference failed to achieve statistical significance (p = 0.233).

TABLE 17. HDL Category

HDL	Case N (%)	Control N (%)	p-value
Low	17 (56.7%)	7 (23.3%)	**0.001**
Normal	13 (43.3%)	23 (76.7%)	
MEAN±SD	42.10 ± 6.95	55.57 ± 8.09	

FIGURE 20. HDL Category

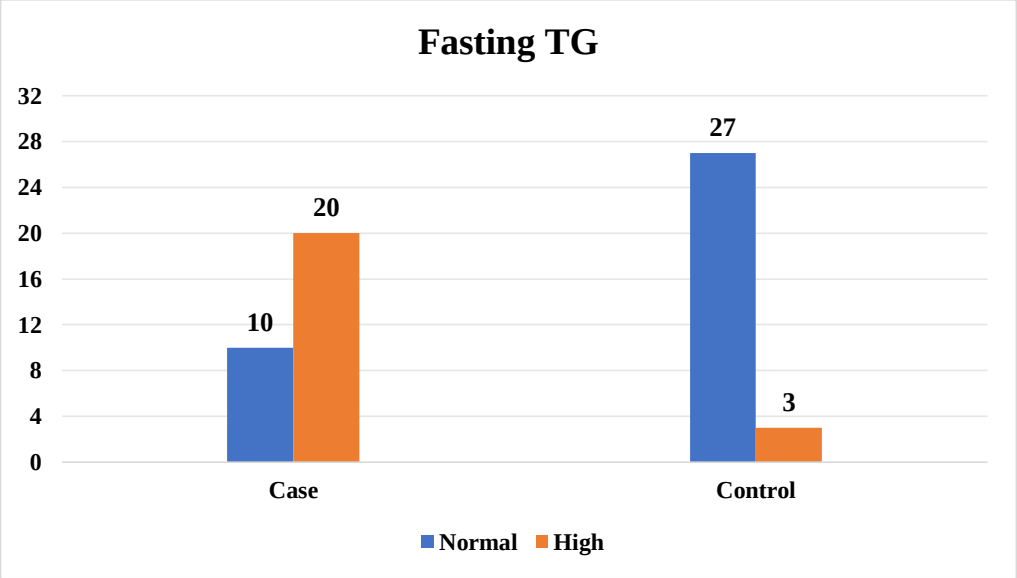


HDL levels differed significantly between groups. A majority of IHD cases (56.7%) had low HDL compared to only 23.3% of controls, with the p-value being 0.001. Normal HDL levels were observed in only 43.3% of IHD cases versus 76.7% of controls. The mean HDL was considerably lower in cases (42.10 ± 6.95) than in controls (55.57 ± 8.09), underscoring the role of protective cholesterol in cardiovascular risk.

TABLE 18. Fasting TG Category × Group

Fasting TG	Case N (%)	Control N (%)	p-value
Normal	10 (33.3%)	27 (90.0%)	**<0.001**
High	20 (66.7%)	3 (10.0%)	
MEAN±SD	156.30 ± 17.87	136.87 ± 14.36	

FIGURE 21. Fasting TG Category × Group

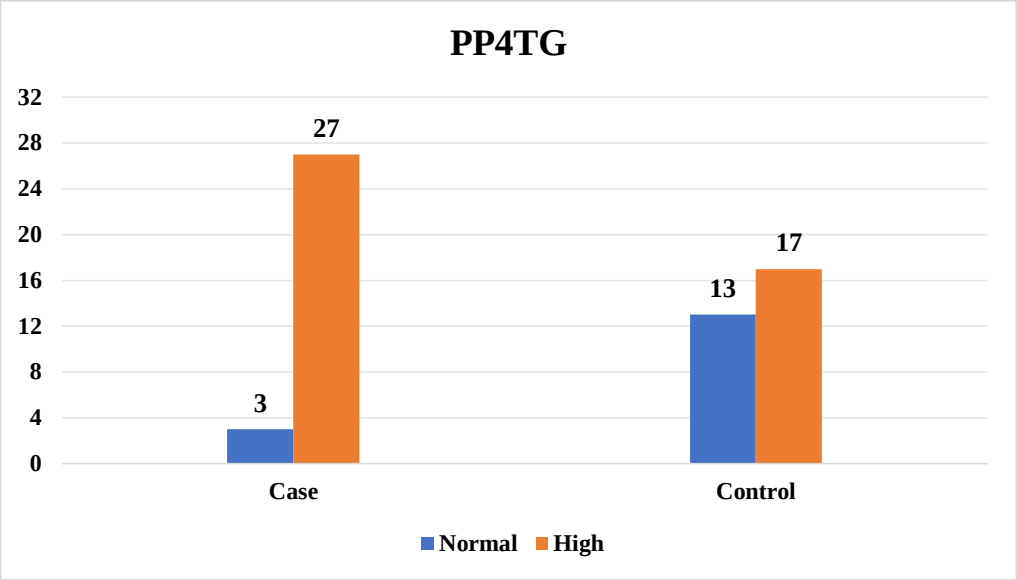


For **fasting triglycerides (TG)**, 66.7% of IHD patients had superior levels compared to only 10.0% of controls. Normal levels were more prevalent in controls (90.0%) than in cases (33.3%), yielding a highly significant p-value (<0.001). This was supported by elevated mean TG values in IHD cases (156.30 ± 17.87) versus controls (136.87 ± 14.36).

TABLE 19. PP4TG Category × Group

PP4TG	Case N (%)	Control N (%)	p-value
Normal	3 (10.0%)	13 (43.3%)	**0.007**
High	27 (90.0%)	17 (56.7%)	
MEAN±SD	185.27 ± 20.15	163.00 ± 16.61	

FIGURE 22. PP4TG Category × Group



Lastly, **postprandial triglycerides (PP4TG)** were also remarkably higher in IHD patients, with 90.0% classified as high compared to 56.7% of controls. The difference was statistically significant (p = 0.007). Normal PP4TG levels were more common in controls (43.3%) than in IHD cases (10.0%), with mean values of 185.27 ± 20.15 for cases and 163.00 ± 16.61 for controls.

DISCUSSION

DISCUSSION

Cardiovascular diseases specifically ischemic heart disease (IHD), remain the leading cause of morbidity and mortality globally. Among the numerous contributing factors, lipid metabolism disturbances, particularly postprandial dyslipidemia, have gained prominence in recent years. While fasting lipid profiles have traditionally been tended to assess cardiovascular risk, emerging evidence suggests that postprandial lipid levels may better reflect real-world metabolic fluctuations and vascular exposure to atherogenic lipoproteins. Our investigation aimed to explore the relevance of fasting and postprandial lipid parameters, especially triglycerides, in IHD patients compared to healthy controls. The findings from our study, when contextualized alongside 12 contemporary Indian and international studies, provide a robust basis to discuss the role of lipid abnormalities and related cause in the pathophysiology of IHD.

Demographic Profile

In our analysis, the majority of IHD cases were between 51–70 years, with a mean age of 57.28 ± 7.56 years, highlighting a middle-aged to older adult demographic. Gandhi SP [104]mirrored this distribution, with IHD most prevalent among 51–60-year-olds (33.3%) and 61–70-year-olds (29.3%). Das T[105] reported the highest proportion of cases in the 61–70 age bracket (33.75%), with a average age of 61.7 ± 11.07 years, suggesting slightly older IHD onset. Chakraborty M [97]found comparable mean ages between groups, with diabetics averaging 55.53 ± 9.83 years. Manochehri M[14] observed a mean age of 60.9 ± 11.4 years in CAD cases, also within this critical range. Suliman Saber's[15] study reported similar age median of 57 years. Murugan [106]reported a slightly younger mean age of 47.1 ± 5.2 years, with the largest subgroup aged 46–55. P. K. Jawaharlal[96] noted the highest frequency in the 46–55-year group, consistent with our observations. Wang Y's[107] expansive cohort had a average age of 61 years for diabetic participants and 47 years for non-diabetics, aligning well with the age range observed across all studies. Lee SH[108] reported a

mean age of 57.5 years, further reinforcing that IHD and associated metabolic dysfunction primarily affect individuals in their 50s and 60s.

Gender-wise, our study showed IHD to be more frequent in males. This pattern was consistently supported across multiple studies. Gandhi SP[104] showed 58% of cases were male; Sonwane MN[109] found 63.3% male predominance; Tarannum S [96]reported 64% males; P. K. Jawaharlal[98] had 56% male patients; Das T[105] showed a striking 71.25% were male; and Murugan[106] had 56% male representation. Lee SH[108] also reported a near even distribution (57% male), while Wang Y [107]observed higher TGs and diabetes prevalence among men. These findings collectively affirm the greater susceptibility of males to IHD, likely due to a integration of behavioral, hormonal, and genetic factors.

Lifestyle Risk Factors

In our study, smoking was observed in 83.3% of IHD patients, and other habits like alcohol consumption and tobacco chewing were also more common among cases, though the significance varied. Sonwane MN[109] identified tobacco chewing (40%) as the most ubiquitous threat followed by smoking (26.67%) and hypertension (23.33%), showing a strong influence of modifiable behaviors on IHD. Tarannum S [96]similarly found tobacco chewing in 30% of participants, with smoking and hypertension each contributing 20%, reinforcing the clustering of these habits in at-risk individuals. Murugan[106] linked lifestyle risk factors with biochemical findings, reporting that 64% of patients exhibited postprandial hypertriglyceridemia, which was most common among smokers and tobacco chewers, highlighting the metabolic consequences of these behaviors. Suliman Saber[15] noted that comorbidities were significantly more frequent in groups with elevated postprandial triglycerides, implying a role of unmeasured behavioral contributors. These consistent findings across studies reinforce the notion that poor lifestyle choices are pivotal drivers of IHD through their metabolic sequelae.

Medical Comorbidities

In our research diabetes mellitus was present in 83.3% of IHD cases and hypertension in 70%, both significantly more than in controls, underscoring their critical role in IHD pathophysiology. Gandhi SP [104]showed that among 150 IHD cases, 12% had diabetes, 38% had hypertension, and 13.3% had both, supporting the clustering of these conditions. Murugan[106] also found 64% of patients were diabetic and 57% hypertensive, very similar to our findings. Suliman Saber[15] highlighted that diabetes and hypertension were more prevalent in groups with abnormal coronary angiography and postprandial triglycerides, linking these comorbidities with lipid disturbances and IHD severity. Chakraborty M [97]observed a progressive increase in fasting glucose, postprandial glucose, and HbA1c from control to prediabetic to diabetic groups, showing a clear glycemic gradient associated with cardiovascular risk. Lee SH[108] found higher fasting and postprandial glucose and HbA1c in diabetics, along with elevated insulin resistance, central obesity, confirming a metabolic syndrome–like phenotype. Wang Y's[107] study demonstrated that individuals with diabetes had higher triglycerides, more obesity (50.2%), and substantially higher prevalence of hypercholesterolemia (45.2%) and hypertension (60.2%), firmly establishing diabetes as a high-risk phenotype for IHD. Manochehri M[14] also found a significantly greater frequency of TG abnormalities in CAD patients, with an odds ratio of 8.8 for fasting TG and 28 for postprandial TG, emphasizing how strongly dysglycemia is tied to dyslipidemia and cardiovascular events.

Obesity & Anthropometric Measures

In our study, both BMI and waist-hip ratio were extensively higher in IHD cases, indicating central and overall adiposity as major contributors to disease. Jawaharlal [98]reported that 40% of IHD patients were overweight and 76% had high waist-hip ratio, and among those with elevated triglycerides, 67% were overweight, 75% had diabetes, and 82% had high WHR—

highlighting how abdominal obesity interacts with lipid metabolism. Lee SH [108]also reported significantly higher waist circumference and WHR in type 2 diabetes patients against to those with impaired fasting glucose, with WC averaging 88.1 cm in diabetics vs. 85.7 cm in non-diabetics (P = 0.03), reinforcing the connection between central obesity and dyslipidemia. Murugan[106] supported these findings by showing the highest prevalence of postprandial hypertriglyceridemia in individuals over age 66 (80%) and those with central obesity, reinforcing that visceral fat plays a role in lipid dysregulation. Wang Y[107] found that people with elevated TG levels had a greater likelihood of being obese, with 50.2% of diabetics classified as obese, and higher triglyceride categories showed increasing rates of obesity from 26.8% (<150 mg/dL) to 43.9% (200–499 mg/dL), establishing a strong dose-response relationship.

Glycemic Parameters

In our study, both fasting and postprandial blood glucose levels were remarkably higher in IHD patients compared to controls, reflecting poor glycemic control as a critical determinant of cardiovascular risk. Gandhi SP[104] reported similar trends, with mean fasting and postprandial glucose higher among IHD patients and a significant glycemic burden among those with comorbid diabetes. Chakraborty M [97]conducted detailed comparisons between control, prediabetic, and diabetic groups, reporting a significant rise in fasting blood glucose (FBG), and postprandial blood glucose (PPBG) levels across the spectrum, with PPBG being most elevated in diabetics (P < 0.001). Lee SH [108]also found significantly higher fasting glucose (141.2 ± 47.2 mg/dL), postprandial glucose (255.3 ± 78.1 mg/dL), and HbA1c (7.87 ± 1.51%) in T2D patients compared to those with impaired fasting glucose. Manochehri M [14]confirmed these trends by reporting significantly higher fasting and postprandial glucose in CAD cases, with postprandial TG being better correlated with HbA1c than fasting TG (P < 0.01), underlining the metabolic interplay between glucose dysregulation and lipid abnormalities. Suliman Saber [15]also documented elevated glycemia across groups, particularly in those with abnormal postprandial triglyceride profiles and coronary artery disease. Wang Y's [107]massive cohort revealed a median fasting glucose of 133

mg/dL in diabetics vs. 96 mg/dL in non-diabetics and an HbA1c of 6.7% vs. 5.3%, illustrating the broader metabolic deterioration in patients prone to CVD. These studies, in tandem with our own, highlight that glycemic burden, especially postprandial, closely tracks with cardiovascular pathology and lipid disturbances.

Lipid Profile

In our study, HDL was significantly lower and LDL significantly higher in IHD cases than controls, with both fasting and postprandial shifts. Gandhi SP[104] observed similar patterns, where HDL declined significantly postprandially (male: 42.5 → 38.1 mg/dL, female: 41.8 → 37.8 mg/dL), while LDL also dropped significantly, highlighting dynamic changes across lipid fractions in IHD. Suliman Saber[15] reported mean fasting LDL of 86.2 mg/dL in controls vs. 110.4 mg/dL in IHD cases, and HDL significantly lower in IHD groups (33.2 ± 5.3 mg/dL), all with $P < 0.001$. Das T[105] found LDL reduced from 103.8 to 95.3 mg/dL and HDL from 37.3 to 36.7 mg/dL postprandially, both statistically significant, though the absolute change was small. Tarannum S[96] also observed slight but non-significant reductions in LDL and HDL postprandially in IHD cases. Chakraborty M[97] did not find HDL to be significantly correlated with glycemic control, but total cholesterol was significantly higher in diabetics (245.87 vs. 229.72 mg/dL), consistent with our observations. Lee SH[108] included HDL-C in regression models predicting postprandial triglycerides but did not report groupwise HDL data separately. These findings collectively suggest that while postprandial changes in LDL and HDL are less dramatic than those in triglycerides, they remain clinically relevant, particularly for HDL which consistently trended lower in IHD patients.

Triglycerides

In our study, fasting triglyceride levels in IHD cases were already elevated (mean 170.65 ± 52.01 mg/dL) and rose significantly postprandially to 237.36 ± 68.95 mg/dL, an increase of nearly

40%. This rise was statistically significant and far more pronounced than in healthy controls (120.59 → 138.34 mg/dL), underscoring a heightened lipemic response in disease states.

Gandhi SP[104] reported similar patterns, where male IHD patients had a fasting TG of 173.33 ± 53.42 mg/dL, which rose postprandially to 238.19 ± 66.88 mg/dL ($P < 0.001$), and females showed a comparable increase from 166.95 ± 50.19 mg/dL to 236.22 ± 72.23 mg/dL ($P < 0.001$). This uniformity across genders strengthens the generalizability of postprandial TG as an IHD marker.

Sonwane MN[109] found fasting TGs of 211.02 ± 63.8 mg/dL and postprandial values of 275.65 ± 47.9 mg/dL in IHD patients, with a statistically significant increase. Tarannum S[96] mirrored these results, showing fasting TGs at 210.02 ± 63.9 mg/dL and postprandial TGs at 275.65 ± 48.0 mg/dL. The consistency of these findings across independent studies reinforces the reproducibility of this metabolic signature.

P. K. Jawaharlal [98]offered further insight by identifying that 64% of his patients had raised serum TGs, and 69% of those also had low HDL. Furthermore, 75% of diabetic patients and 82% of those with high waist-hip ratio (WHR) had increased TG levels, indicating a strong clustering of dyslipidemia with central obesity and glycemic dysfunction.

Murugan, [106]in a postprandial TG-focused study, found 64% of patients exhibited hypertriglyceridemia after a meal, defined as TG >160 mg/dL. The highest prevalence was observed in those over 66 years (80%), and in females (68.18%), emphasizing age and gender susceptibility. The correlation between age and postprandial TG was evident, suggesting that aging may exacerbate the chylomicron and remnant lipoprotein clearance defect seen in IHD.

Chakraborty M [97]added mechanistic weight to this observation by showing that TG and TG/HDL-C ratios significantly increased postprandially, and both were better correlated with HbA1c than their fasting counterparts. This implies that postprandial TGs are not only reflective of lipid metabolism but also an integrated marker of overall metabolic derangement.

Manochehri M [14]presented compelling statistical evidence with an odds ratio of 8.8 for fasting TG and 28.0 for postprandial TG in predicting coronary artery disease (CAD), highlighting postprandial TG as a superior diagnostic indicator. A post-meal TG rise of over 80 mg/dL was demonstrated to forecast CAD with 75% specificity & sensitivity.

Suliman Saber[15] found fasting TG levels of 178.6 ± 39.7 mg/dL and postprandial TG levels of 228.6 ± 18.6 mg/dL in CAD patients with both abnormal coronary angiography and postprandial lipemia. This group showed the most severe disease burden and poorest lipid profile, reinforcing the link between TG excursions and coronary lesion severity.

Lee SH[108] explored correlations between fasting & postprandial TG levels and metabolic factors in diabetics and prediabetics. A very strong correlation observed between fasting & postprandial TGs (r = 0.973), and regression analysis identified HOMA-IR and fasting TGs as significant predictors of postprandial TG. This implies that insulin resistance directly modulates postprandial lipemia.

Wang Y's[107] nationwide NHANES dataset (n = 26,570) provided perhaps the most definitive epidemiological support. Participants with diabetes had median TGs of 142 mg/dL relative to 105 mg/dL in non-diabetics. More importantly, among diabetics, those with TGs between 200–499 mg/dL had a 44% higher risk of cardiovascular mortality (HR: 1.44, P = 0.004) contrasted to those with normal TGs (<150 mg/dL). A 1-log unit increase in TG affiliated with a 30% increased risk of cardiovascular death (HR: 1.30, P = 0.006), even after adjusting for cholesterol and comorbidities. This study firmly established elevated TGs—not just as marker of metabolic syndrome—but as an **distinct risk factor for death** from cardiovascular causes in diabetics.

Comparison of Triglyceride Levels Across Studies

Study		Fasting TG {mg/dL}	Postprandial TG {mg/dL}
Our Study		170.65	237.36
Gandhi SP[104]	Male	173.33	238.19
	Female	166.95	236.22
Sonwane MN[109]		211.02	275.65
Tarannum S[96]		210.02	275.65
Suliman Saber[15]		178.60	228.64
Lee SH[108]		126 (median)	156 (median)
Das T[105]		157.00	187.23
Wang Y[107]		142 (median)	-
Murugan[106]		TG >160 in 64% (PP)	
Chakraborty M[97]		Not specified	TG ↑ significantly in DM
Manochehri M[14]		TG ↑ >80 mg/dL (PP)	

All studies that quantified postprandial triglycerides showed **substantial increases post-meal**, ranging from +30 to +70 mg/dL, with **our study demonstrating one of the highest rises (+66.71 mg/dL)**. This consistency across populations reinforces postprandial TG as a reliable and sensitive biomarker for cardiovascular risk assessment.

Triglycerides (TGs), particularly in the postprandial state, have emerged as a pivotal biomarker in the assessment of ischemic heart disease (IHD). Unlike fasting TGs, which may not capture real-time metabolic stress, postprandial TGs reflect the dynamic lipid response following dietary fat intake and the burden of circulating remnant lipoproteins. Taken together, these studies form a robust, reproducible, and multidimensional picture: postprandial hypertriglyceridemia is a constant trait in IHD patients, strongly correlates with comorbid diabetes and obesity, and is predictive not just of disease presence but also its severity and associated mortality. Unlike LDL or HDL, which may fluctuate less postprandially, triglycerides provide a dynamic readout of metabolic health and vascular risk. Therefore, the use of postprandial TG measurement in routine cardiovascular risk assessment, especially in high-risk populations, is not only justified—it is necessary.

LIMITATIONS

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While our study and its comparative synthesis offer compelling awareness into the role of postprandial triglycerides in ischemic heart disease (IHD), several limitations merit discussion. Firstly, our sample size, though statistically adequate, was limited to a single-center setting, which may affect generalizability across broader populations. Secondly, the absence of direct inflammatory or endothelial biomarkers limits our ability to mechanistically correlate TG excursions with atherosclerotic processes. Lastly, long-term cardiovascular outcomes were not tracked in our cohort, which would have strengthened the predictive validity of postprandial TG elevations.

RECOMMENDATIONS

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While our study and its comparative synthesis offer compelling awareness into the role of postprandial triglycerides in ischemic heart disease (IHD), several limitations merit discussion. Firstly, our sample size, though statistically adequate, was limited to a single-center setting, which may affect generalizability across broader populations. Secondly, the absence of direct inflammatory or endothelial biomarkers limits our ability to mechanistically correlate TG excursions with atherosclerotic processes. Lastly, long-term cardiovascular outcomes were not tracked in our cohort, which would have strengthened the predictive validity of postprandial TG elevations.

Recommendations

Based on the synthesis of findings from our investigation and the supporting twelve studies, several practical and research-oriented recommendations emerge:

1. **Routine Clinical Practice**: Postprandial triglyceride testing should be incorporated into routine cardiovascular risk assessments, particularly for patients with diabetes, obesity, metabolic syndrome, or borderline fasting dyslipidemia. A 2–4 hour post-meal sampling window can offer a practical, clinically relevant measure of lipid response.
2. **Guideline Development**: National and international lipid guidelines should consider including postprandial triglycerides as a risk stratification parameter, especially in high-risk groups.
3. **Therapeutic Interventions**: Clinicians should evaluate the use of targeted therapies—as in omega-3 fatty acids, fibrates emerging TG-lowering agents—in patients demonstrating postprandial hypertriglyceridemia, even when fasting lipids are normal.

4. **Research Imperatives**: Future prospective cohort studies are warranted to establish standardized thresholds for postprandial TG, evaluate intervention outcomes, and explore molecular pathways involving remnant cholesterol and endothelial dysfunction.

Population-Specific Studies: Larger, multicentric trials across different ethnic and dietary groups are necessary to define population-specific cut-offs and therapeutic targets.

CONCLUSION

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The findings of our study unequivocally demonstrate that postprandial triglyceride (TG) testing reveals critical metabolic disturbances that may remain undetected in the fasting state, particularly in individuals with ischemic heart disease (IHD) and its associated comorbidities. While fasting lipid profiles provide a baseline snapshot of lipid metabolism, postprandial TG levels capture the dynamic interplay between dietary intake, insulin resistance, and vascular stress—factors that more accurately reflect daily physiological exposures. The strong associations observed between elevated postprandial TG and conditions like diabetes mellitus, hypertension, and central obesity underscore its value as a sensitive indicator of cardiometabolic risk. Moreover, mounting evidence from multiple studies—including our own—establishes postprandial TG as a solid predictor of atherosclerotic burden, adverse cardiovascular events, and even mortality. In the context of precision medicine, targeting postprandial lipemia offers a promising avenue for earlier identification and more personalized intervention in high-risk populations. Therefore, integrating postprandial TG assessment into routine clinical practice—particularly for diabetic and obese patients—represents a critical step forward in the prevention, stratification, and management of cardiovascular disease.

SUMMARY

SUMMARY

Our research focused on appraise the dynamic changes in lipid parameters—particularly triglycerides—between fasting and postprandial states in patients with IHD, correlated to healthy controls. The investigation also explored the influence of demographic factors, comorbidities, and anthropometric indices on these lipid fluctuations. The findings contribute valuable clinical insight into the metabolic characteristics of IHD and the utility of non-fasting lipid testing.

Key Findings

- **Demographics**: IHD was most prevalent in the 51–70-year age group (62.6%), with an average age of 57.28 ± 7.56 years. Gender distribution was equal (50% male, 50% female), but the disease was more prevalent among men in other cohorts.
- **Comorbidities**: Diabetes mellitus was present in 83.3% and hypertension in 70% of IHD cases, both significantly more than in healthy controls. Additionally, 42% of IHD patients were smokers, 22.6% chewed tobacco, and 7.3% were alcohol consumers.
- **Glycemic Parameters**: IHD patients had significantly higher fasting (85.41 ± 21.24 mg/dL) and postprandial blood sugar (112.65 ± 24.52 mg/dL) compared to controls.
- **Anthropometric Profile**: There was a notable rise in Body mass index and waist hip ratio among IHD patients, correlating with postprandial triglyceride elevations.
- **Fasting Lipid Profile**:
    - Total Cholesterol: 188.5 ± 20.75 mg/dL (cases) vs. 168.87 ± 17.5 mg/dL (controls)
    - LDL-C: 112.49 ± 20.76 mg/dL (cases) vs. 103.3 ± 18.4 mg/dL (controls)
    - HDL-C: Slightly higher in IHD cases in the fasting state but declined postprandially

- **Postprandial Lipid Changes**:
    - **Triglycerides** showed a significant rise from 170.65 ± 52.01 mg/dL (fasting) to 237.36 ± 68.95 mg/dL (postprandial), compared to a smaller increase in controls.
    - **VLDL** also increased significantly postprandially (33.80 → 47.10 mg/dL).
    - A statistically significant postprandial decline was observed in LDL-C and HDL-C.

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ANNEXURES

INFORMED CONSENT FORM

Date :

I, Mr/Mrs _____ ,have been explained in my own understandable language that I will be included in TO STUDY THE COMPARISION BETWEEN FASTING AND POSTPRANDIAL TRIGLYCERIDE LEVEL AS A RISK FACTOR FOR ISCHEMIC HEART DISEASE A Case Control Study**,** hereby I give my valid written informed consent without any force or prejudice for recording the observations of haematological and clinical parameters . The nature and risks involved have been explained to me, to my satisfaction. I have been explained in detail about the study being conducted. I have read the patient information sheet and I have had the opportunity to ask any question. Any question that I have asked, have been answered to my satisfaction. I provide consent voluntarily to allow myself / my relative as a participant in this research. I hereby give consent to provide history, undergo physical examination, undergo the procedure, undergo investigations and provide its results and documents etc to the doctor / institute etc. For academic and scientific purpose the operation / procedure, etc may be video graphed or photographed. All the data may be published or used for any academic purpose.

_____

Name of Patient/Guardian

(Relation with patient)

_____

(Signature of Patient / Attendant)

(Signature & Name of Research doctor)

ಮಾಹಿತಿಯುಕ್ತ ಒಪ್ಪಿಗೆ ನಮೂನೆ

ದಿನಾಂಕ:

ನನಗೆ, ಶ್ರೀ/ಶ್ರೀಮತಿ _____, ನನ್ನದೇ ಆದ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ, ಇಸ್ಕೆಮಿಕ್ ಹೃದಯ ಕಾಯಿಲೆಗೆ ಅಪಾಯಕಾರಿ ಅಂಶವಾಗಿ ಉಪವಾಸ ಮತ್ತು ಪೋಸ್ಟ್ ಪ್ರಾಂಡಿಯಲ್ ಟ್ರೈಗ್ಲಿಸರೈಡ್ ಮಟ್ಟದ ನಡುವಿನ ಹೋಲಿಕೆಯನ್ನು ಅಧ್ಯಯನ ಮಾಡಲು ನಾನು ಸೇರಿಸಿಕೊಳ್ಳುತ್ತೇನೆ. ಈ ಮೂಲಕ ನಾನು ಹೆಮಟೊಲಾಜಿಕಲ್ ಮತ್ತು ಕ್ಲಿನಿಕಲ್ ನಿಯತಾಂಕಗಳ ಅವಲೋಕನಗಳನ್ನು ದಾಖಲಿಸಲು ಯಾವುದೇ ಬಲ ಅಥವಾ ಪೂರ್ವಾಗ್ರಹವಿಲ್ಲದೆ ನನ್ನ ಮಾನ್ಯ ಲಿಖಿತ ಮಾಹಿತಿಯುಕ್ತ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡುತ್ತೇನೆ. ಒಳಗೊಂಡಿರುವ ಸ್ವರೂಪ ಮತ್ತು ಅಪಾಯಗಳನ್ನು ನನ್ನ ತೃಪ್ತಿಗೆ ವಿವರಿಸಲಾಗಿದೆ. ನಡೆಸಲಾಗುತ್ತಿರುವ ಅಧ್ಯಯನದ ಬಗ್ಗೆ ನನಗೆ ವಿವರವಾಗಿ ವಿವರಿಸಲಾಗಿದೆ. ನಾನು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ಓದಿದ್ದೇನೆ ಮತ್ತು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶ ಸಿಕ್ಕಿದೆ. ನಾನು ಕೇಳಿದ ಯಾವುದೇ ಪ್ರಶ್ನೆಗೆ ನನ್ನ ತೃಪ್ತಿಗೆ ಉತ್ತರಿಸಲಾಗಿದೆ. ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ನನ್ನನ್ನು / ನನ್ನ ಸಂಬಂಧಿಯನ್ನು ಭಾಗವಹಿಸಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ. ನಾನು ಈ ಮೂಲಕ ವೈದ್ಯಕೀಯ ಇತಿಹಾಸವನ್ನು ಒದಗಿಸಲು, ದೈಹಿಕ ಪರೀಕ್ಷೆಗೆ ಒಳಗಾಗಲು, ಕಾರ್ಯವಿಧಾನಕ್ಕೆ ಒಳಗಾಗಲು, ತನಿಖೆಗಳಿಗೆ ಒಳಗಾಗಲು ಮತ್ತು ಅದರ ಫಲಿತಾಂಶಗಳು ಮತ್ತು ದಾಖಲೆಗಳನ್ನು ವೈದ್ಯರು / ಸಂಸ್ಥೆಗೆ ಒದಗಿಸಲು ಸಮ್ಮತಿಸುತ್ತೇನೆ. ಶೈಕ್ಷಣಿಕ ಮತ್ತು ವೈಜ್ಞಾನಿಕ ಉದ್ದೇಶಗಳಿಗಾಗಿ ಕಾರ್ಯಾಚರಣೆ / ಕಾರ್ಯವಿಧಾನ ಇತ್ಯಾದಿಗಳನ್ನು ವೀಡಿಯೊಗ್ರಾಫ್ ಮಾಡಬಹುದು ಅಥವಾ ಛಾಯಾಚಿತ್ರ ಮಾಡಬಹುದು. ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಪ್ರಕಟಿಸಬಹುದು ಅಥವಾ ಯಾವುದೇ ಶೈಕ್ಷಣಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಬಳಸಬಹುದು.

_____

ರೋಗಿಯ / ಪೋಷಕರ ಹೆಸರು

(ರೋಗಿಯೊಂದಿಗಿನ ಸಂಬಂಧ)

_____

(ರೋಗಿಯ / ಪರಿಚಾರಕರ ಸಹಿ)

(ಸಂಶೋಧನಾ ವೈದ್ಯರ ಸಹಿ ಮತ್ತು ಹೆಸರು)

PATIENT INFORMATION SHEET

Study title: To Study The Comparision Between Fasting And Postprandial Triglyceride Level As a Risk Factor For Ischaemic Heart Disease

CASE CONTROL STUDY

Principal investigator: DR P.NEHA / DR SRINIVASA.S.V

I Dr. P. NEHA , Post graduate student in Department of general medicine at Sri Devaraj Urs Medical College, will be conducting a study titled**"To Study The Comparision Between Fasting And Postprandial Triglyceride Level As a Risk Factor For Ischaemic Heart Disease Case Control Study"** . This study will be useful for further management of ischaemic heart disease in the near future. The funds needed for the fasting and postprandial triglyceride level will be borne by the primary investigator.2 ml of blood will be drawn for estimation of fasting and postprandial triglyceride level , from each of the participating patients in this study . This study will be done under the guidance of Dr . SRINIVASA.S.V Professor of Department of GENERAL MEDICINE.

All the data will be kept confidential and will be used only for purpose specified by the institution. You are free to provide consent for the participation of yourself in this study. You can also withdraw yourself from the study at any point of time without giving any reasons whatsoever. Your refusal to participate will not prejudice you to any present or future care at this institution.

In case of any clarifications are needed you are free to contact me on this mobile number - 9493870483

Name and Signature of the Principal Investigator

Date-

Patient or patient bystanders Signature

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಅಧ್ಯಯನ ಶೀರ್ಷಿಕೆ: ಇಸ್ಕೆಮಿಕ್ ಹೃದಯ ಕಾಯಿಲೆಗೆ ಅಪಾಯಕಾರಿ ಅಂಶವಾಗಿ ಉಪವಾಸ ಮತ್ತು ಊಟದ ನಂತರದ ಟ್ರೈಗ್ಲಿಸರೈಡ್ ಮಟ್ಟದ ನಡುವಿನ ಹೋಲಿಕೆಯನ್ನು ಅಧ್ಯಯನ ಮಾಡಲು

ಪ್ರಕರಣ ನಿಯಂತ್ರಣ ಅಧ್ಯಯನ

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ: ಡಾ. ಪಿ. ನೇಹಾ / ಡಾ. ಶ್ರೀನಿವಾಸ. ಎಸ್.ವಿ

ಶ್ರೀ ದೇವರಾಜ್ ಅರಸ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜಿನಲ್ಲಿ ಜನರಲ್ ಮೆಡಿಸಿನ್ ವಿಭಾಗದಲ್ಲಿ ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿನಿ ಡಾ. ಪಿ. ನೇಹಾ ಅವರು "ಇಸ್ಕೆಮಿಕ್ ಹೃದಯ ಕಾಯಿಲೆ ಪ್ರಕರಣ ನಿಯಂತ್ರಣ ಅಧ್ಯಯನಕ್ಕೆ ಅಪಾಯಕಾರಿ ಅಂಶವಾಗಿ ಉಪವಾಸ ಮತ್ತು ಊಟದ ನಂತರದ ಟ್ರೈಗ್ಲಿಸರೈಡ್ ಮಟ್ಟದ ನಡುವಿನ ಹೋಲಿಕೆಯನ್ನು ಅಧ್ಯಯನ ಮಾಡಲು" ಎಂಬ ಶೀರ್ಷಿಕೆಯ ಅಧ್ಯಯನವನ್ನು ನಡೆಸಲಿದ್ದಾರೆ. ಈ ಅಧ್ಯಯನವು ಮುಂದಿನ ದಿನಗಳಲ್ಲಿ ಇಸ್ಕೆಮಿಕ್ ಹೃದಯ ಕಾಯಿಲೆಯ ಮತ್ತಷ್ಟು ನಿರ್ವಹಣೆಗೆ ಉಪಯುಕ್ತವಾಗಿರುತ್ತದೆ. ಉಪವಾಸ ಮತ್ತು ಊಟದ ನಂತರದ ಟ್ರೈಗ್ಲಿಸರೈಡ್ ಮಟ್ಟಕ್ಕೆ ಅಗತ್ಯವಿರುವ ಹಣವನ್ನು ಪ್ರಾಥಮಿಕ ತನಿಖಾಧಿಕಾರಿ ಭರಿಸುತ್ತಾರೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವ ಪ್ರತಿಯೊಬ್ಬ ರೋಗಿಯಿಂದ ಉಪವಾಸ ಮತ್ತು ಊಟದ ನಂತರದ ಟ್ರೈಗ್ಲಿಸರೈಡ್ ಮಟ್ಟವನ್ನು ಅಂದಾಜು ಮಾಡಲು 2 ಮಿಲಿ ರಕ್ತವನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುತ್ತದೆ. ಈ ಅಧ್ಯಯನವನ್ನು ಡಾ. ಅವರ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ಮಾಡಲಾಗುತ್ತದೆ. ಶ್ರೀನಿವಾಸ.ಎಸ್.ವಿ. ಪ್ರಾಧ್ಯಾಪಕರು, ಜನರಲ್ ಮೆಡಿಸಿನ್ ವಿಭಾಗ.

ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುವುದು ಮತ್ತು ಸಂಸ್ಥೆಯು ನಿರ್ದಿಷ್ಟಪಡಿಸಿದ ಉದ್ದೇಶಕ್ಕಾಗಿ ಮಾತ್ರ ಬಳಸಲಾಗುವುದು. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆಗೆ ನೀವು ಒಪ್ಪಿಗೆ ನೀಡಲು ಸ್ವತಂತ್ರರು. ಯಾವುದೇ ಕಾರಣಗಳನ್ನು ನೀಡದೆ ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು. ಭಾಗವಹಿಸಲು ನಿಮ್ಮ ನಿರಾಕರಣೆಯು ಈ ಸಂಸ್ಥೆಯಲ್ಲಿನ ಯಾವುದೇ ಪ್ರಸ್ತುತ ಅಥವಾ ಭವಿಷ್ಯದ ಆರೈಕೆಗೆ ನಿಮ್ಮನ್ನು ಹಾನಿಗೊಳಿಸುವುದಿಲ್ಲ.

ಯಾವುದೇ ಸ್ಪಷ್ಟೀಕರಣಗಳು ಅಗತ್ಯವಿದ್ದರೆ ನೀವು ಈ ಮೊಬೈಲ್ ಸಂಖ್ಯೆ - 9493870483 ನಲ್ಲಿ ನನ್ನನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು.

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ

ದಿನಾಂಕ-

ರೋಗಿ ಅಥವಾ ರೋಗಿಯ ಪಕ್ಕದಲ್ಲಿರುವ ಪ್ರೇಕ್ಷಕರ ಸಹಿ

PROFORMA

To Study The Comparision Between Fasting And Postprandial Triglyceride Level As a Risk Factor For Ischaemic Heart Disease

A Case Control Study

NAME	
AGE	
GENDER	
HOSPITAL NUMBER	
DATE OF ADMISSION	
DATE OF DISCHARGE	
CHIEF COMPLIANTS	
PAST HISTORY 1. Diabetes 2. Hypertension 3. C .V. stroke 4. Dyslipidemia: **PERSONAL HISTORY** **Smoking** **Alcohol** **Sedentary Lifestyle**	
Is the patient already a known case of IHD (Y/N)	
If Yes then details about treatment history	

Treatment history	
FAMILY HISTORY	
DURATION OF STAY IN HOSPITAL	
TIMES OF READMSSION FOR THE SAME COMPLAINTS	
GENERAL PHYSICAL EXAMINATION: Pulse: Blood Pressure: Temperature: Loco motor Brachialis: Carotid Bruit: Cord like Artery: Height: WEIGHT**:** BODY MASS INDEX(BMI): Waist: Hip: Waist hip ratio:	
CARDIO VASCULAR SYSTEM EXAMINATION	
OTHER SYSTEMS: Respiratory system Abdomen Nervous system	

INVESTIGATIONS	**1.Fasting samples of blood sugar** **2.Fasting lipid profile which includes** **S. Cholesterol** **S. Triglyceride** **S. HDL** **S. LDL** **S. VLDL** **3. 2 hour post prandial sugars** **4 hour post prandial triglyceride.** **4.ELECTRO CARDIOGRAM(ECG)/ECHO:**

Sl no	AGE OF PATIENT	SEX OF PATIENT	NAFLD	BMI OF PATIENT	FATTY LIVER INDEX	TGY INDEX	Hb A1c	ELASTOGRAPHY SCORE	CORRELATION
1	55	Male	Yes	28	80.00	8.90	10.40	9.00	Yes
2	64	Male	Yes	30	89.00	8.51	11.00	8.20	Yes
3	72	Female	Yes	40	100.00	8.03	9.20	9.20	Yes
4	45	Female	No	23	35.00	8.93	7.40	5.60	No
5	58	Male	Yes	25	63	8.32	7	5.2	No
6	40	Female	Yes	23	68	9.91	9	8.2	Yes
7	48	Male	Yes	28	95	8.91	10.2	6.7	Yes
8	45	Female	Yes	28	78	9.6	9	7.9	Yes
9	50	Male	Yes	30	32	8.45	10	5.5	No
10	49	Male	Yes	25	55	8.6	7.9	8.3	Yes
11	63	Male	No	24	62	8	8.4	8.4	Yes
12	63	Male	Yes	26	58	8	6.9	8.6	Yes
13	50	Male	Yes	29	83	9.32	7.1	7.9	Yes
14	49	Male	Yes	28	61	8.9	9	9.1	Yes
15	45	Female	Yes	25	32	8.91	7.2	5.4	No
16	55	Female	Yes	26	28	8.95	7.9	7.9	Yes
17	69	Male	No	32	71	8.95	7.9	7.9	Yes
18	55	Male	Yes	29	83	9.32	10	8.7	Yes
19	40	Female	Yes	28	61	8.4	8.9	7.7	Yes
20	59	Female	Yes	25	39	8.22	9.5	8.6	Yes
21	61	Male	Yes	32	71	8.5	7	7.8	Yes
22	45	Male	Yes	29	82	8.8	9	10	Yes
23	54	Female	No	24	78	9.4	7.3	6.5	No
24	75	Male	Yes	28	60	9.3	11	6.8	Yes
25	62	Male	No	30	50	8.9	7.4	8.4	Yes
26	68	Male	Yes	29	29	8.4	6.8	6.6	No
27	64	Female	Yes	24	45	8.5	8.4	6.9	Yes
28	65	Male	Yes	25	40	8	9	6	No
29	62	Male	No	30	45	8.6	7	7.1	Yes
30	64	Male	Yes	28	55	9.6	8.5	7.9	Yes
31	63	Female	Yes	32	67	9.5	6.3	10	Yes
32	52	Male	No	25	67	8.9	7.9	8.4	Yes
33	54	Male	Yes	27	45	8.3	7.5	6.6	No
34	56	Male	Yes	30	47	8.1	6.9	6.9	Yes
35	62	Male	Yes	22	68	8.9	9.1	8.4	Yes
36	48	Female	No	31	57	8.1	9.6	8.7	Yes
37	54	Male	Yes	29	54	8.9	8.9	11	Yes
38	56	Male	Yes	28	30	8.9	8.6	9.8	Yes
39	62	Male	No	27	55	8.9	8.9	7.9	Yes
40	68	Female	Yes	28	45	7.9	7.9	6.9	Yes
41	52	Male	Yes	26	68	5.4	7.4	8	Yes
42	45	Male	No	32	69	9.4	8.6	5.6	No
43	53	Female	Yes	29	50	8.8	9.6	7.6	Yes

Sl no	AGE OF PATIENT	SEX OF PATIENT	NAFLD	BMI OF PATIENT	FATTY LIVER INDEX	TGY INDEX	Hb A1c	ELASTOGRAPHY SCORE	CORRELATION
44	56	Male	Yes	29	70	8.4	10	7.9	Yes
45	62	Male	No	32	69	8.5	9.9	8.9	Yes
46	68	Male	Yes	23	66	9.5	6.8	9	Yes
47	64	Female	Yes	29	47	8.8	8.9	9	Yes
48	63	Male	No	30	67	8.5	8.6	8.4	Yes
49	62	Male	Yes	25	45	8.5	9.6	6.6	No
50	68	Male	Yes	24	47	7.8	10	6.9	Yes
51	64	Female	No	30	68	5.8	8.5	8.4	Yes
52	65	Male	Yes	45	78	9.4	6.3	7.2	Yes
53	62	Male	Yes	35	60	8.3	7.9	8.4	Yes
54	64	Female	Yes	30	50	8.9	11	6.6	No
55	63	Male	Yes	29	29	9.1	7.4	6.9	Yes
56	52	Male	Yes	26	28	8.3	6.8	9	Yes
57	54	Female	Yes	32	71	8.9	9.6	5.6	No
58	56	Male	Yes	29	83	8.7	8.9	7.6	Yes
59	62	Male	Yes	29	61	9.67	8.6	7.9	Yes
60	48	Female	Yes	25	45	8.95	10	8.7	Yes
61	54	Male	Yes	27	47	8.32	8.9	11	Yes
62	56	Female	Yes	30	68	5.4	9.5	9.8	Yes
63	62	Male	Yes	22	57	4.95	7	8.7	Yes
64	68	Female	Yes	24	54	9.32	9	7.7	Yes
65	52	Male	Yes	26	55	6.32	7.3	8.6	Yes
66	45	Male	Yes	27	88	8.1	7.9	7.9	Yes
67	53	Female	Yes	28	82	8.9	7.4	6.9	Yes
68	56	Male	Yes	26	78	8.1	9.6	8	Yes
69	62	Male	Yes	26	60	6.9	8.9	5.6	No
70	68	Female	Yes	32	50	8.45	8.6	6.5	No
71	64	Male	Yes	29	29	9.6	7.9	6.8	Yes
72	63	Female	Yes	32	67	8.4	7.5	8.4	Yes
73	62	Male	Yes	29	50	9.8	6.9	6.6	No
74	68	Male	Yes	28	29	9.4	9.1	8.2	Yes
75	64	Female	Yes	30	33	8.8	10	9.2	Yes
76	62	Male	Yes	29	61	9.67	8.6	7.9	Yes
77	48	Female	Yes	25	45	8.95	10	8.7	Yes
78	54	Male	Yes	27	47	8.32	8.9	11	Yes
79	56	Female	Yes	30	68	5.4	9.5	9.8	Yes
80	62	Male	Yes	22	57	4.95	7	8.7	Yes
81	68	Female	Yes	24	54	9.32	9	7.7	Yes
82	52	Male	Yes	26	55	6.32	7.3	8.6	Yes
83	45	Male	Yes	27	88	8.1	7.9	7.9	Yes
84	68	Female	Yes	32	50	8.45	8.6	6.5	No
85	64	Male	Yes	29	29	9.6	7.9	6.8	Yes