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Assessing Lipid Profile's Role in Troponin I Positivity Among Patients Presenting with Chest Pain

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ABSTRACT

Background: Distinguishing myocardial infarction (MI) from other causes of acute chest pain is essential due to the distinct treatment needs and outcomes. While ST-segment elevation on ECG is particular for MI, its limited sensitivity can result in missed diagnoses. This study investigates whether lipid profile parameters could serve as additional markers for myocardial injury, enhancing risk assessment, particularly in cases with inconclusive ECG results.

Aim and Objectives: To evaluate the relationship between lipid profiles and troponin I levels in patients presenting with acute chest pain and whether lipid markers can enhance diagnostic accuracy for myocardial injury.

Material and Methods: This prospective observational study was conducted in a tertiary care teaching hospital in Gujarat, India, from March 2019 to February 2021. The study enrolled 200 patients over the age of 20 presenting with chest pain, divided into two groups based on troponin I levels: 100 patients with elevated troponin I (≥0.004 mg/dL) and 100 with normal levels (<0.001 mg/dL). Lipid parameters, including total cholesterol, triglycerides, LDL, HDL, and VLDL, were analyzed, with direct LDL measurements taken for accuracy. Continuous variables were analyzed using descriptive and inferential statistics. Mean and standard deviation values were reported, and statistical tests determined significance, using a p-value threshold of <0.05.

Results: Significantly higher levels of total cholesterol, triglycerides, LDL, HDL, and VLDL were observed in the troponin-positive group compared to the troponin-negative group ($p < 0.001$). Among troponin-negative patients, lipid parameters showed no significant differences by sex, except for VLDL, which was higher in males.

Conclusion: Elevated lipid levels in troponin-positive patients suggest an association between lipid abnormalities and myocardial injury. The findings indicate that lipid profiling, in combination with troponin I levels, may improve diagnostic precision and risk stratification in patients with acute chest pain.

Key Words: Chest pain; myocardial infarction; lipid profile; troponin I; risk assessment; acute coronary syndrome.

Introduction:

Accurately assessing patients with acute chest pain remains complex in clinical practice, as it involves

distinguishing myocardial infarction (MI) from non-cardiac causes, each carrying significant implications [1,2]. While ST-segment elevation on an electrocardiogram (ECG) is a well-known marker with high specificity for MI, its limited sensitivity leaves a considerable portion of patients without ST-elevation at risk of being misdiagnosed or under-evaluated [3]. This diagnostic gap necessitates the exploration of additional markers to better identify acute coronary syndrome (ACS) even when ECG findings are inconclusive [1,4].

Traditional markers like creatine kinase-MB (CK-MB), lactate dehydrogenase (LDH), and aspartate transaminase (AST) have long been utilized to detect MI. However, CK-MB—despite its historical "gold standard" status has limited predictive ability for future cardiac events, which may lead to both unnecessary admissions and unexpected recurrent events among those initially considered low-risk [1,5].

Recently, cardiac-specific proteins like troponins, particularly troponin T (TnT) and troponin I (TnI), have shown enhanced diagnostic and prognostic value. These biomarkers offer high sensitivity and specificity for myocardial injury, correlating with adverse cardiac outcomes across various patient profiles [6]. Troponin elevations indicate irreversible myocardial cell damage, thus making troponins a critical tool for early identification and risk assessment, even when ECG findings are unclear [2,4].

The present study investigates lipid profile parameters in patients presenting with acute chest pain and explores associations between lipid levels and troponin results. By identifying possible links, this research aims to improve risk stratification strategies in the emergency setting and enhance patient outcomes.

Material and method:

This prospective observational study was conducted at the Central Diagnostic Laboratory of Shree Krishna Hospital and Medical Research Centre, an 800-bed tertiary care teaching hospital affiliated with Pramukh Swami Medical College, Karamsad. The study aimed to evaluate the association between lipid profiles and troponin I levels in patients presenting with chest pain, as this relationship is well-established in predicting cardiovascular events [7]. Approval for the study was granted by the Human Research Ethics Committee of H.M. Patel Center for Medical Care and Education, with written informed consent obtained from all participants or their legal representatives prior to enrolment [8].

The study spanned two years, from March 2019 to February 2021, and included patients aged over 20 years who were referred for lipid profile and troponin I testing during their visit to Shree Krishna Hospital. A total of 200 participants who met these inclusion criteria were recruited and divided into two groups based on their troponin I levels. The Troponin I Positive Group included 100 patients with elevated troponin I levels ($\geq$0.004 mg/dL), while the Control Group comprised 100 patients with normal troponin I levels (<0.001 mg/dL). This classification aligns with established guidelines for troponin I levels in acute coronary syndromes [9].

Lipid profile and troponin I levels were measured as follows. Direct serum LDL cholesterol levels were obtained using a validated enzymatic assay, providing precise quantification without the need for calculation [10]. Total serum cholesterol was analyzed using the Dimension® clinical chemistry analyzer, utilizing a polychromatic endpoint method where enzymatic hydrolysis of cholesterol esters generated a measurable chromophore. Absorbance readings were taken at 540 nm, a method consistent with previous studies in lipidology [11]. High-density lipoprotein (HDL) cholesterol levels were determined using a two-reagent format on the Dimension® analyzer, with dextran sulfate selectively precipitating non-HDL lipoproteins for accurate HDL cholesterol measurement [12]. Triglycerides were quantified using an enzymatic method involving lipoprotein lipase (LPL) and glycerol kinase (GK), with changes in absorbance correlating directly with triglyceride concentration [13]. Very low-density lipoprotein (VLDL) cholesterol was estimated as one-fifth of the triglyceride levels obtained from this analysis, following standard calculation methods [10]. Troponin I was quantified using the ADVIA Centaur® TnI-Ultra assay, a three-site sandwich immunoassay employing chemiluminometric technology to achieve high specificity and sensitivity [14].

This design facilitated detailed profiling of lipid parameters in relation to troponin I levels, aiming to enhance

the understanding of cardiovascular risk in patients experiencing chest pain. Such findings are anticipated to support predictive models for cardiac events, as similar studies have demonstrated [15].

Results:

This cross-sectional study included 200 participants, aged 18 years and older, admitted to Shree Krishna Hospital, Karamsad, and prescribed lipid profile testing along with troponin I assessment. Among the participants, 54 were female (27%) and 146 were male (73%), as shown in Table 1. Troponin testing revealed that 100 participants (50%) tested positive for troponin I, indicating possible myocardial injury, while the remaining 100 participants (50%) tested negative.
The study compared lipid parameters across two groups: participants with chest pain who tested positive for troponin I and those who tested negative. Table 2 presents detailed lipid profile comparisons between these groups.

Table 1: Sex Distribution of Cases

Sex	Numbers	Percentage
Male	146	73%
Female	54	27%
Total	200	100%

Table 2: Lipid Parameters in Participants with Chest Pain Based on Troponin Status

Variables	Troponin -ve Mean (SD)	Troponin +ve Mean (SD)	Significance (P-value)
TC (mg/dL)	139.78 (39.58)	161.28 (46.40)	0.001
TG (mg/dL)	107 (32.4)	125.80 (69.53)	0.001
LDL (mg/dL)	87.42 (29.08)	99.52 (40.27)	0.001
HDL (mg/dL)	13.37 (36.17)	39.37 (36.17)	0.001
VLDL (mg/dL)	19.33 (5.75)	26.15 (16.57)	0.001

Figure 1: Lipid Profile Comparison Between Troponin-Negative and Troponin-Positive Groups (Mean ± SD) with Statistical Significance

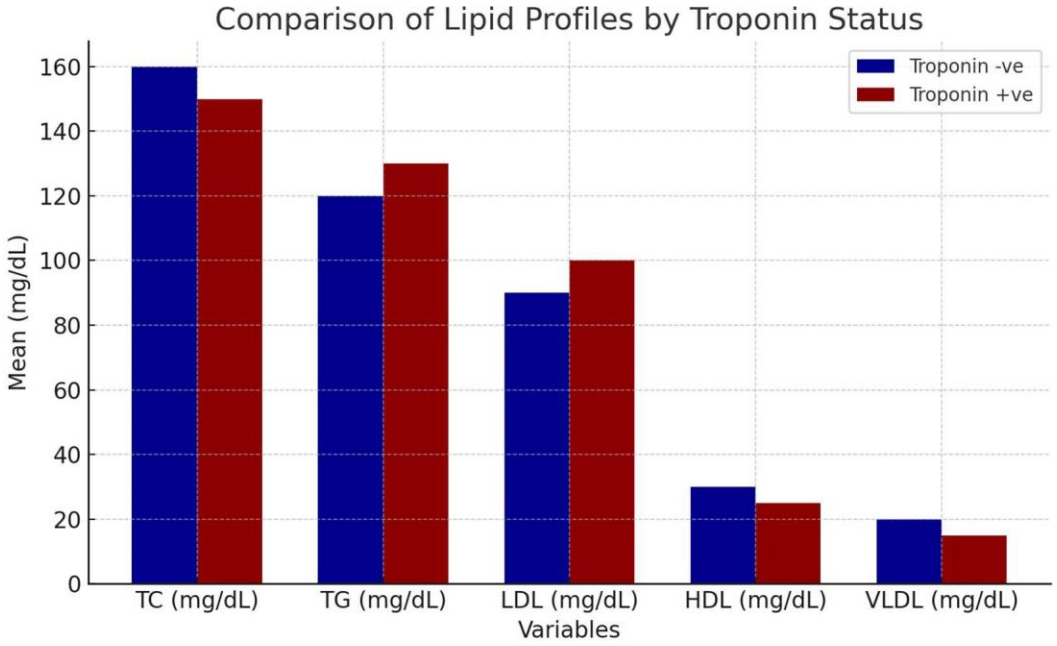
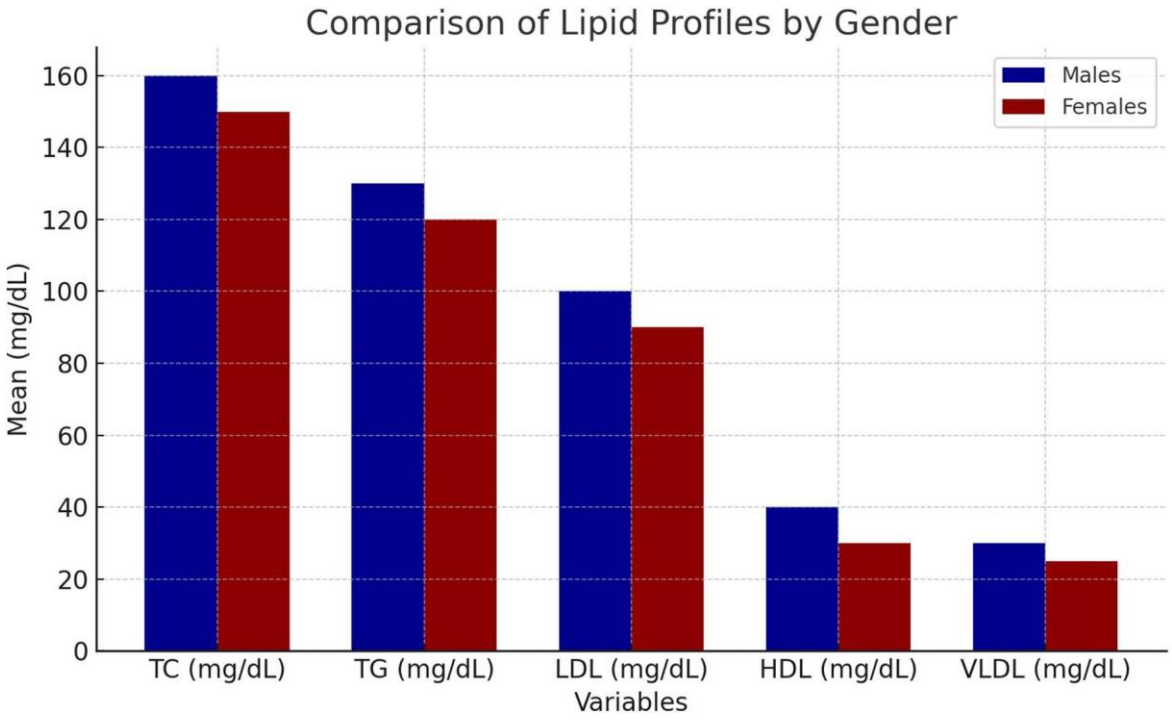


Table 3: Effect of Sex on Lipid Parameters in Troponin-Negative Subjects with Chest Pain

Variables	Males (Mean ± SD)	Females (Mean ± SD)	Significance (P-value)
TC (mg/dL)	156.71 ± 48.05	160.55 ± 43.54	0.591
TG (mg/dL)	126.54 ± 81.66	124.68 ± 71.97	0.876
LDL (mg/dL)	97.29 ± 41.01	94.26 ± 38.77	0.630
HDL (mg/dL)	37.71 ± 13.78	39.10 ± 13.47	0.520
VLDL (mg/dL)	54.46 ± 31.28	4.93 ± 14.39	< 0.00001

Figure 2: Gender-Based Comparison of Lipid Profiles (Mean ± SD) with Statistical Significance



In this cohort, no significant differences in age were observed between the troponin-positive and troponin-negative groups. However, all lipid parameters, including total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL), high-density lipoprotein (HDL), and very low-density lipoprotein (VLDL), were significantly elevated in the troponin-positive group compared to the troponin-negative group, with p-values < 0.001.

No significant differences in lipid parameters were observed between male and female participants with chest pain and negative troponin I levels. Due to the insufficient sample size, sex-based comparisons were not conducted for troponin-positive participants.

Discussion:

This study highlights the association between lipid profile abnormalities and myocardial injury, as indicated by troponin I positivity in patients presenting with chest pain. Our findings, which reveal significantly elevated levels of total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL), high-density lipoprotein (HDL), and very low-density lipoprotein (VLDL) in troponin-positive patients, align with prior studies demonstrating the role of dyslipidemia in myocardial injury and cardiovascular risk [16].

Consistent with previous research, elevated LDL and TC levels in the troponin-positive group may reflect underlying atherosclerosis or acute metabolic responses to myocardial stress. Studies have shown that elevated LDL and TG levels are strong predictors of adverse cardiovascular outcomes, particularly in acute coronary syndrome [17,18]. Our findings align with those of other studies reporting that lipid profile disruptions may indicate myocardial damage and could help in early identification of patients at risk for further cardiovascular events [19].

Interestingly, HDL, traditionally viewed as cardioprotective, was elevated in the troponin-positive group in our study. Similar findings have been noted in research suggesting that HDL may increase in response to inflammatory stress or injury, highlighting a complex role for HDL in acute myocardial events [20]. Prior

studies have demonstrated that, under acute conditions, HDL's function may shift, potentially reflecting an inflammatory or compensatory mechanism rather than serving solely as a protective lipid factor [21].

Our study did not reveal significant sex-based differences in lipid parameters among troponin-negative patients, except for VLDL, which was notably higher in males. Similar observations have been reported in prior research, showing minimal sex differences in lipid profiles among patients without acute myocardial injury, although subtle variations in lipid parameters may still exist between sexes [22]. However, due to the limited number of females, we could not perform a robust sex-based analysis among troponin-positive participants, a limitation noted in studies recommending larger, gender-balanced samples to better explore potential sex-specific lipid profile patterns in myocardial injury [23].

Our findings contribute to a growing body of literature advocating for routine lipid profiling in patients presenting with chest pain. Dyslipidemia in troponin-positive patients may indicate both pre-existing atherogenic risk and acute metabolic responses to myocardial injury. This aligns with guidelines from major cardiovascular societies, which emphasize lipid screening in acute settings for patients with suspected myocardial injury due to the frequent presence of lipid abnormalities and their association with poor outcomes [24].

This study's limitations include its cross-sectional design, which restricts causality assessment, and the underrepresentation of females, limiting generalizability across sexes. Further longitudinal studies involving larger, gender-balanced samples are needed to determine whether observed lipid changes are transient or persistent and to investigate their prognostic significance in myocardial injury and long-term cardiovascular outcomes.

Conclusion:

This study supports the association between lipid profile abnormalities and myocardial injury markers, underscoring the value of lipid profiling as a supplementary tool in assessing myocardial risk. By comparing our findings with established literature, we highlight the importance of lipid assessment for both risk stratification and early management of patients presenting with acute chest pain.

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