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Correlation of Troponin I Levels with Serum Glutamic Oxaloacetic Transaminase (SGOT) and Low-Density Lipoprotein (LDL) in Patients with Coronary Heart Disease at Haji General Hospital in East Java Province

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Abstract
Background: Coronary Heart Disease (CHD) is a condition caused by atherosclerosis, which leads to reduced blood supply to the heart muscle. This condition occurs due to the buildup of plaques resulting form elevated levels of LDL. Atherosclerosis can trigger myocardial infarction, leading to heart muscle damage and the release of biomarkers such as Troponin I and Serum Glutamic Oxaloacetic Transaminase (SGOT). *Objectives*: This Study aims to determine the correlation between Troponin I levels and SGOT as well as LDL levels in patients with CHD at Haji General Hospital in East Java Province. *Materials and Methods*: This study used an analytical observational study with a cross-sectional and purposive sampling. *Results*: The study results show that the majority of participants were aged 46-65 years (57%) and male (57%). Most patients had high Troponin I levels (70%), normal SGOT levels (63%), and high LDL levels (100%). The correlation analysis using the Spearman test showed a statistically significant correlation between Troponin I and SGOT levels with p value 0.046 and correlation coefficient r= 0.367, but the correlation between Troponin I and LDL levels was not statistically significant with p value 0.330 and correlation coefficient = 0.184. *Conclusions*: The study found a correlation between Troponin I and SGOT in patients with CHD and no correlation between Troponin I and LDL in patients with CHD.

Keywords
Coronary Heart Disease, LDL, SGOT, Troponin I.

1. Introduction

Coronary Heart Disease (CHD) remains one of the leading causes of death worldwide. CHD occurs due to the narrowing of the coronary arteries, which disrupts the flow of oxygen and nutrients to the heart. This condition often results from an unhealthy lifestyle and a high-fat diet, contributing to cholesterol buildup in the arteries. When left unmanaged, this buildup leads to atherosclerosis and ultimately increases the risk of myocardial infarction (Pane et al., 2022).

According to the 2018 Indonesian Basic Health Research (Riskesdas), 1.5% of the population suffers from CHD, totaling approximately 2.78 million people. In East Java Province, Surabaya has recorded the highest number of CHD cases, accounting for 88.21% of the total cases in the region. This high prevalence reflects the urgent need for early detection and effective monitoring strategies (Rachmawati et al., 2021).

Troponin I is a specific biomarker released into the bloodstream following myocardial injury. It plays a critical role in the diagnostic evaluation of CHD, as it rises within 3 hours after infarction, peaks within 12–24 hours, and remains elevated for up to 14 days (Sutikno & Yudhatama, 2022). Although Serum Glutamic Oxaloacetic Transaminase (SGOT) is not specific to the heart, its levels may also increase in response to hypoxic liver injury following cardiac events (Rahayu et al., 2022).

High levels of Low low-density lipoprotein (LDL) cholesterol are strongly associated with CHD progression. LDL contributes to the formation of atherosclerotic plaques, which can rupture and lead to myocardial infarction (Aswara et al., 2022). Several studies have reported correlations between troponin I, SGOT, and LDL in CHD patients (Dakhil, 2020; Malhotra & Ahmed, 2022; Suriadiharja & Riyani, 2024). This study aims to analyze the correlation between troponin I, SGOT, and LDL levels in patients with coronary heart disease at Haji General Hospital, East Java Province. The findings are expected to provide additional insights for clinicians in diagnosing and monitoring CHD more accurately.

The novelty of this study lies in the analysis of the correlation between Troponin I levels and two different parameters, namely SGOT and LDL, in patients with coronary heart disease (CHD). Previous studies generally examined the association of Troponin I with only one parameter, whereas this study evaluates both within a single research design. Thus, this study is expected to provide a broader understanding of the relationship between Troponin I as a biomarker and enzymatic as well as lipid factors involved in the progression of CHD. In addition, this research emphasizes the exploration of SGOT as a supporting marker alongside Troponin I. Although SGOT is not a cardiac-specific enzyme, its elevation may provide valuable clinical information in assessing myocardial injury. This study also offers novelty in terms of the local epidemiological context, focusing on CHD patients in Surabaya, a city with a notably high prevalence. Therefore, the findings are expected to enrich scientific data in Indonesia and support the development of more effective diagnostic strategies for CHD patients.

2. Materials and Methods

2.1 Types of Research

This research employs an analytical observational research method with a cross-sectional design, conducted from February to March 2025 at the Clinical Pathology Laboratory of Haji Regional General Hospital, East Java Province. The study population includes all patients diagnosed with coronary heart disease treated at the hospital during the period of February to March 2025. The research sample consists of 30 patients, determined based on sample size calculation, and selected using a purposive sampling technique according to specific inclusion and exclusion criteria set by the researcher.

Inclusion criteria include patients in the Emergency Department with a final diagnosis of coronary heart disease, who undergo laboratory testing for Troponin I, SGOT, and LDL levels, are of any gender, and are aged over 25 years. The age criterion of above 25 years was applied because the incidence of coronary heart disease is relatively rare at a younger age, while the risk begins to increase significantly after 25 years due to the accumulation of cardiovascular risk factors and the early development of atherosclerosis. Exclusion criteria are patients with coronary heart disease who are under 25 years of age. Primary data collection involves clinical laboratory examination results for Troponin I, SGOT, and LDL, obtained from the hospital's medical records department with prior approval and ethical clearance. These data are then analyzed to observe relationships between the biomarker levels in patients diagnosed with coronary heart disease.

22. Research Methods

Troponin I levels were measured using the Fluorescence Immunoassay (FIA) method with the Standard F200 Analyzer. SGOT and LDL levels in this study were measured using the Cobas 6000 (C501) analyzer. LDL levels were measured using the Homogeneous Enzymatic Colorimetric method, in which cholesterol esterase converts cholesterol esters into free cholesterol and free fatty acids. SGOT measurement was performed using the IFCC (International Federation of Clinical Chemistry) method with Pyridoxal-5-Phosphate (P-5-P). Data were analyzed using IBM SPSS Statistics version 26. Normality was tested using the Shapiro-Wilk test. Correlation analysis was conducted using Spearman's test. This study has complied with the ethical approval No. 445/17/KOM.ETIK/2025 from the Health Research Ethics Committee of Haji Regional General Hospital, East Java Province.

3 Results and Discussion

3.1. Results

This study was conducted from February to March 2025 at the Clinical Pathology Laboratory of RSUD Haji, East Java Province, involving 30 research subjects. The subjects consisted of patients diagnosed with coronary heart disease from both male and female gender groups, all aged over 25 years.

1. Age Analysis of Coronary Heart Disease Patients

Table 1. Distribution of Coronary Heart Disease Patients Based on Age

Category	Total	Percentage (%)
Adult (26–45 years)	2	6
Elderly (46–65 years)	17	57
Senior (>65 years)	11	37

This study involved subjects with coronary heart disease aged over 25 years. According to Amin & Juniati (2017), age is classified into adulthood, the elderly, and advanced elderly. Based on Table 1, the percentage distribution of coronary heart disease (CHD) patients by age group is as follows: 17 individuals (57%) were in the elderly group (46–65 years), 11 individuals (37%) were in the late adulthood group, and 2 individuals (6%) were in the adult group. The highest prevalence of CHD was found in the elderly age group (46–65 years), accounting for 57% of the total research subjects.

2. Gender Analysis of Coronary Heart Disease Patients

Table 2. Distribution of Coronary Heart Disease Patients Based on Gender

Gender	Total	Percentage (%)
Male	17	57
Female	13	43

Based on Table 2, the percentage distribution of CHD patients by gender shows that 17 individuals (57%) were male and 13 individuals (43%) were female. The majority of CHD patients in this study were male, accounting for 57% of the total research subjects.

3. Analysis of Troponin I Levels in Coronary Heart Disease Patients

The normal reference value for Troponin I levels used by RSUD Haji is <0.05 ng/mL. Troponin I levels In this study were classified into two categories, normal (<0.05 ng/mL) and high (>0.05 ng/mL). The results of the Troponin I level examination are shown in the table 3.

Table 3. Distribution of CHD Patients Based on Troponin I

Troponin I	Total	Percentage (%)
Normal	9	30
High	21	70

Based on Table 3, 9 patients (30%) had Troponin I levels within the normal range, while 21 patients (70%) had high levels. The majority of CHD patients were in the high Troponin I category, accounting for 70% of the total study subjects.

4. Analysis of SGOT Levels in Coronary Heart Disease Patients

The normal reference value for SGOT levels used by RSUD Haji is <40 U/L. SGOT levels in this study were categorized as normal (<40 U/L) and high (>40 U/L). The results of the SGOT examination are shown in the Table 4.

Table 4. Distribution of CHD Patients Based on SGOT

SGOT	Total	Percentage (%)
Normal	19	63
High	11	37

Based on Table 4, 19 patients (63%) had SGOT levels within the normal range, while 11 patients (37%) showed high levels. The majority of CHD patients had normal SGOT levels, accounting for 63% of the total study subjects.

5. Analysis of LDL Levels in Coronary Heart Disease Patients

The normal reference value for LDL levels used by RSUD Haji is <100 mg/dL. LDL levels in this study

were classified as normal (<100 mg/dL) and high (>100 mg/dL). The results of the LDL examination are shown in Table 5.

Table 5. Distribution of CHD Patients Based on LDL

LDL	Total	Percentage (%)
Normal	0	0
High	30	100

Based on Table 5, all 30 patients (100%) had LDL levels in the high category, and none were within the normal range. Thus, all CHD patients in this study had high LDL levels.

6. Normality Test of Troponin I, SGOT, and LDL in CHD Patients

The data obtained from the examination were subjected to a normality test using the Shapiro-Wilk method. This method was chosen due to the relatively small sample size (<50 samples).

Table 6. Shapiro-Wilk Normality Test of Troponin I, SGOT, and LDL in CHD Patients

Variable	Significance Value
Troponin I	0.000
SGOT	0.001
LDL	0.042

Based on the statistical results shown in Table 6, the significance values for Troponin I, SGOT, and LDL were 0.000, 0.001, and 0.042, respectively. Data are considered normally distributed if the significance value is >0.05. Since all three variables have significance values <0.05, it indicates that the data are not normally distributed. Therefore, the appropriate statistical test for further analysis is the Spearman correlation test.

7. Correlation Test Between Troponin I and SGOT in CHD Patients

Table 7. Correlation Test Between Troponin I and SGOT Levels in CHD Patients

Variables	Significance (p-value)	Correlation Coefficient (r)
Troponin I SGOT	0.046	0.367

A correlation is considered statistically significant if the p-value < 0.05. According to Table 7, the correlation between Troponin I and SGOT levels in CHD patients is significant, with a p-value of 0.046. The next step is to examine the correlation coefficient value to assess the strength of the relationship between the two variables.

Table 8. Criteria for the Strength of Spearman Correlation Based on Correlation Coefficient

Correlation Coefficient	Strength of Correlation
0.00–0.199	Very Weak
0.20–0.399	Weak
0.40–0.599	Moderate
0.60–0.799	Strong
0.80–1.00	Very Strong

Source: (Prabandaru & Widodo, 2022)

Based on the reference values, the correlation between Troponin I and SGOT levels in CHD patients is categorized as weak, with a correlation coefficient (r) of 0.367.

8. Correlation Test Between Troponin I and LDL Levels in CHD Patients

Table 9. Spearman Correlation Test Between Troponin I and LDL Levels in CHD Patients

Variables	Significance (p-value)	Correlation Coefficient (r)
Troponin I LDL	0.330	0.184

As presented in Table 9, the correlation between Troponin I and LDL levels is not statistically significant (p = 0.330). The correlation coefficient obtained is r = 0.184, indicating a very weak relationship between the two variables.

3.2 Discussion

This study found that most CHD (Coronary Heart Disease) patients were elderly (46–65 years), comprising 57% of the total subjects. This supports findings by Swandari et al.

(2022), who stated that aging increases CHD risk due to declining organ function and arterial stiffness (Tampubolon et al., 2023). Additionally, 57% of CHD patients in this study were male, in line with previous research showing males are more prone to CHD, partly due to lifestyle factors like smoking and alcohol consumption, which can damage endothelial cells and promote plaque formation (Aisyah et al., 2022; Iswara, 2022).

Regarding biomarkers, 70% of patients had elevated Troponin I levels (>0.05 ng/mL), which is consistent with Ibrahim et al. (2024). Troponin I is a specific and sensitive indicator of myocardial injury, typically rising 3 hours after damage and peaking within 12–24 hours (Prasetyorini et al., 2022; Sutikno & Yudhatama, 2022). Meanwhile, 63% of subjects had normal SGOT levels (<40 U/L), though still relatively higher compared to other groups, supporting studies showing SGOT may also rise in myocardial infarction (Sutikno & Yudhatama, 2022).

The Spearman correlation test between Troponin I and SGOT levels yielded a significance value of $p = 0.046$, indicating a statistically significant correlation ($p < 0.05$). The correlation coefficient was $r = 0.367$, which, based on the criteria by Prabandaru & Widodo (2022), suggests a weak positive correlation. This finding supports the results of Gao et al. (2017), who observed a significant correlation between elevated Aspartate Aminotransferase (AST) and cardiac troponin in ST-Elevation Myocardial Infarction (STEMI) patients. Similarly, Ibrahim et al. (2024) reported a moderate correlation between Troponin I and SGOT levels in myocardial infarction patients.

Although SGOT is commonly known as a liver enzyme, it is also found in cardiac muscle tissue. Increased SGOT levels may indicate cardiac dysfunction. During a heart attack, SGOT levels often rise and are strongly associated with increased Troponin I, a specific marker for myocardial damage (Ndrepepa, 2021). The release of Troponin I from myocardial tissue depends on the type and severity of the pathological condition. The higher the Troponin I levels, the more severe the myocardial damage (Chaulin, 2022).

Myocardial infarction can lead to cardiogenic shock, a condition where the heart fails to pump blood effectively, disrupting systemic circulation. The body attempts to maintain oxygen supply to vital organs, including the heart itself, which may also experience hypoxia. Hypoxia can result in myocardial cell injury or death due to inadequate ATP production. As a result, essential ion pumps for sodium, potassium, and calcium malfunction, exacerbating cellular damage. Damaged cells release their contents into the bloodstream, including Troponin I and SGOT, thereby causing

elevations in both biomarkers (Ibrahim et al., 2024).

This study found that all CHD (Coronary Heart Disease) patients (100%) had elevated LDL levels (>100 mg/dL), aligning with previous studies showing high LDL prevalence among CHD patients (Suriadiharja & Riyani, 2024). LDL, known as “bad cholesterol,” contributes to plaque buildup in arteries and is associated with unhealthy diets, physical inactivity, and smoking (Sanggih et al., 2019; Wahyudin et al., 2024). This plaque buildup leads to atherosclerosis, which underlies CHD pathophysiology (Aswara et al., 2022).

Despite the established role of LDL in CHD development, this study found no statistically significant correlation between Troponin I and LDL levels, as indicated by the Spearman test results ($p = 0.330$; $r = 0.184$). This suggests a very weak and non-significant correlation. These findings differ from those of Suriadiharja & Riyani (2024), who reported a weak positive correlation ($r = 0.274$) between increased Troponin I and LDL levels. Similarly, Malhotra & Ahmed (2022) found that patients with positive Troponin I test results also exhibited elevated LDL levels, reinforcing the hypothesis that LDL levels are associated with myocardial injury.

Troponin I is a highly specific and sensitive biomarker used in the diagnosis of myocardial infarction. It is a structural protein unique to cardiac muscle tissue and is released into the bloodstream following myocardial injury (Purwati et al., 2020). Troponin I typically begins to rise within three hours of injury onset, peaks within 12–24 hours, and can remain detectable in circulation for 4 to 14 days (Kristensen et al., 2024; Sutikno & Yudhatama, 2022). On the other hand, LDL is a long-term indicator of cardiovascular risk and remains relatively stable over time, with minimal fluctuation in the short term. This temporal difference between the acute nature of Troponin I elevation and the chronic nature of LDL levels may contribute to the lack of correlation observed in this study.

The pathogenesis of atherosclerosis further supports the role of LDL as a contributing factor to CHD rather than a direct marker of acute myocardial damage. Endothelial injury permits the infiltration of LDL particles into the subendothelial space, where they undergo oxidation. Oxidized LDL promotes the recruitment of immune cells, particularly monocytes, which differentiate into macrophages and ingest oxidized LDL, forming foam cells. These foam cells accumulate to form fatty streaks, which subsequently attract smooth muscle cells and contribute to plaque development (Javadifar et al., 2021). Over time, these plaques may undergo calcification, leading to increased fragility and a higher risk of rupture, which can precipitate myocardial infarction. Therefore, while LDL plays a pivotal role in the progression of atherosclerosis, it may not directly reflect myocardial

cell damage at the moment of infarction, as Troponin I does (Gaggini et al., 2022; Hashim, 2024).

The findings of this study suggest that Troponin I and LDL levels represent different stages in the continuum of CHD progression. While LDL serves as a primary risk factor indicating long-term vascular compromise, Troponin I serves as a marker of acute cardiac injury. High LDL levels may predispose individuals to CHD, but elevated Troponin I levels only occur following myocardial cell damage. Thus, elevated LDL does not always coincide with increased Troponin I levels, particularly in patients who have not yet experienced myocardial infarction (Enkhma et al., 2018).

Another important consideration is the timing of biomarker assessment. Troponin I has a short half-life of approximately 2–4 hours, meaning it is rapidly cleared from the bloodstream, although continued myocardial damage can sustain its levels over several days. If blood samples are not collected during the appropriate diagnostic window following myocardial injury, the Troponin I levels may appear normal despite prior injury (Kristensen et al., 2024). In contrast, LDL levels are less sensitive to acute changes and reflect more stable metabolic processes influenced by dietary habits, physical activity, and genetic predisposition (Enkhma et al., 2018).

This study also acknowledges certain limitations. The small sample size restricts the generalizability of the findings and may not accurately reflect the broader population of CHD patients. Furthermore, the study lacked detailed data on participants' medical history, including comorbidities and ongoing treatments, which could influence Troponin I, SGOT, and LDL levels. These factors may confound the observed relationships between biomarkers. Therefore, future research should involve a larger and more diverse sample, and include comprehensive patient profiling to better understand the complex interplay between Troponin I, LDL, and the progression of CHD.

4 Conclusions

Based on the findings of this study, it can be concluded that there is a significant correlation between Troponin I and SGOT levels, whereas no significant correlation was observed between Troponin I and LDL levels.

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