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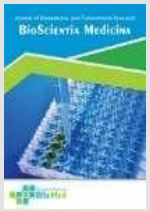

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Correlation between Troponin I Levels and Location of Acute Myocardial Infarction in ST-Elevation Myocardial Infarction Patients

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A B S T R A C T

Background: Acute myocardial infarction (AMI) remains a leading cause of mortality in Indonesia. Cardiac troponin I (cTnI) is a highly sensitive and specific biomarker for AMI, and its levels may correlate with infarct location and prognosis. This study investigated the relationship between cTnI levels and the location of myocardial infarction in ST-elevation myocardial infarction (STEMI) patients. **Methods:** A cross-sectional study was conducted on 57 STEMI patients admitted to H. Abdul Manap Regional General Hospital in Jambi City from April 2022 to 2024. Patients were consecutively enrolled based on inclusion and exclusion criteria. cTnI levels were measured using a point-of-care testing (POCT) device. The correlation between cTnI levels and infarct location was analyzed using the Pearson correlation test. **Results:** The majority of patients were male (78.49%). The highest incidence of AMI was observed in the anterolateral location, with an average cTnI level of 12.16 ng/ml. A strong positive correlation was found between cTnI levels and the location of infarction ($r = 0.891$, $p < 0.05$). **Conclusion:** Higher cTnI levels are associated with specific AMI locations, particularly anterolateral infarctions. This finding highlights the importance of cTnI measurement in assessing AMI severity and potential prognosis.

1. Introduction

Acute myocardial infarction (AMI), commonly known as a heart attack, is a leading cause of mortality and morbidity worldwide, including in Indonesia. AMI occurs when blood flow to the heart muscle is abruptly cut off, causing irreversible damage to the cardiac tissue. This blockage is typically caused by a blood clot forming in a coronary artery that has been narrowed by atherosclerosis, a buildup of plaque on the artery walls. The severity and prognosis of AMI are influenced by various factors, including the location and extent of the infarction, the time elapsed before treatment, and the presence of comorbidities. Early diagnosis and risk stratification are crucial for effective management and improved outcomes. The prevalence of AMI varies across different regions and populations. Developed countries have historically had higher rates of AMI, but the incidence is increasing in developing countries due to lifestyle changes, urbanization, and aging populations. In Indonesia, AMI is a significant public health problem, with a rising trend in recent years. Data from the Indonesian Ministry of Health indicate that heart disease is the leading cause of death in the country, with AMI being a major contributor. AMI is primarily caused by the rupture or erosion of an atherosclerotic plaque in a coronary artery. Atherosclerosis is a chronic inflammatory process characterized by the accumulation of lipids, cholesterol, and cellular debris within the arterial wall, forming plaques that can

obstruct blood flow. When a plaque ruptures or erodes, it triggers the formation of a blood clot (thrombus) at the site of injury. This thrombus can partially or completely block the coronary artery, leading to reduced blood flow (ischemia) and subsequent myocardial damage. The extent of myocardial damage depends on the severity and duration of ischemia, as well as the presence of collateral circulation. If blood flow is not restored promptly, the affected myocardial tissue undergoes irreversible necrosis (cell death), resulting in an infarction. The location and size of the infarction determine the clinical presentation and prognosis of AMI.[1-3]

Patients with AMI typically present with a constellation of symptoms, the most prominent being chest pain. This pain is often described as crushing, squeezing, or tightness, and it may radiate to the left arm, jaw, neck, or back. Other symptoms may include shortness of breath, sweating, nausea, vomiting, and lightheadedness. However, it is important to note that not all patients present with the classic symptoms, and some individuals, particularly those with diabetes or elderly patients, may have atypical presentations, such as shortness of breath or fatigue. The diagnosis of AMI is based on a combination of clinical presentation, electrocardiographic (ECG) findings, and cardiac biomarkers. The ECG is a crucial tool in the diagnosis of AMI. It can detect changes in the electrical activity of the heart caused by ischemia and infarction. ST-segment elevation myocardial infarction (STEMI) is characterized by ST-segment elevation on the ECG, indicating transmural (full-thickness) infarction. Non-ST-segment elevation myocardial infarction (NSTEMI) is characterized by ST-segment depression or T-wave inversion, indicating subendocardial (partial-thickness) infarction. Cardiac biomarkers play a pivotal role in the diagnosis of AMI. These biomarkers are substances released into the bloodstream when the heart muscle is damaged. Cardiac troponins, specifically troponin I (cTnI) and troponin T (cTnT), are the preferred biomarkers for diagnosing AMI. These proteins are released into the bloodstream following myocardial injury and are highly specific for cardiac muscle damage. High-sensitivity cardiac troponin (hs-cTn) assays can detect even minor elevations in troponin levels, allowing for earlier diagnosis and risk stratification.[4,5]

Troponin I is a contractile protein found in cardiac muscle cells. It plays a crucial role in regulating muscle contraction and relaxation. In healthy individuals, troponin I levels in the blood are very low or undetectable. However, when myocardial injury occurs, such as during an AMI, troponin I is released into the bloodstream in proportion to the extent of damage. Troponin I is considered the gold standard biomarker for diagnosing AMI due to its high sensitivity and specificity. It can detect even minor myocardial injury, which may not be evident on an ECG. Troponin I levels typically rise within 3-4 hours after the onset of chest pain, peak at 12-24 hours, and remain elevated for 5-10 days. The magnitude of troponin elevation can also provide prognostic information, with higher levels indicating a greater extent of myocardial damage and a worse prognosis. The location of myocardial infarction, determined by the affected coronary artery, significantly impacts the clinical presentation and prognosis of AMI. Anterior MI, often associated with LAD coronary artery occlusion, is typically more extensive and carries a higher risk of complications. It can lead to left ventricular dysfunction, heart failure, and life-threatening arrhythmias. Inferior MI, related to RCA occlusion, may present with bradycardia and hypotension. It can also lead to right ventricular infarction and conduction abnormalities. Lateral MI, involving the LCx coronary artery, can lead to conduction abnormalities and ventricular arrhythmias. The location of infarction also influences the choice of treatment strategies. For example, patients with anterior MI may benefit from early reperfusion therapy, such as percutaneous coronary intervention (PCI) or fibrinolytic therapy, to restore blood flow to the affected area. Patients with inferior MI may require treatment for bradycardia or hypotension.[6-8]

Several studies have investigated the relationship between troponin levels and infarct location. Baheti et al. (2002) observed variations in troponin levels based on the time of measurement and infarct location, with higher levels in anterior MI. Sagala et al. (2016) reported a correlation between troponin T levels and infarct location in AMI patients, but their study focused on NSTEMI. Research on the correlation between cTnI levels and infarct location in STEMI patients remains limited, particularly in the Indonesian context.[9,10] This study aimed to address this gap by investigating the relationship between cTnI levels and the location of myocardial infarction in STEMI patients at H. Abdul Manap Regional General Hospital in Jambi City.

2. Methods

This research employed a cross-sectional design to investigate the relationship between troponin I levels and the location of acute myocardial infarction (AMI) in ST-elevation myocardial infarction (STEMI) patients. The study was conducted at H. Abdul Manap Regional General Hospital in Jambi City, Indonesia, a tertiary care hospital serving a diverse population. The study period spanned from April 2022 to 2024, allowing for the recruitment of a representative sample of STEMI patients admitted to the hospital during this timeframe.

The study population comprised 57 patients diagnosed with acute STEMI who were admitted to the cardiology department of H. Abdul Manap Regional General Hospital. STEMI, characterized by the complete occlusion of a coronary artery, is a severe form of AMI that requires prompt medical intervention. The inclusion of only STEMI patients ensured homogeneity in the study population and minimized the confounding effects of other types of AMI. A consecutive sampling technique was employed to recruit patients into the study. This non-probability sampling method involves selecting every consecutive patient who meets the predefined inclusion and exclusion criteria until the desired sample size is reached. Consecutive sampling is often preferred in clinical research as it is relatively easy to implement and minimizes selection bias compared to other non-probability sampling methods.

To ensure the validity and reliability of the study findings, specific inclusion and exclusion criteria were established. Patients were included in the study if they met the following criteria; Age > 25 years: This criterion ensured that the study participants were adults, as AMI is relatively rare in younger individuals; Clinical presentation of chest pain consistent with AMI: Patients were required to have symptoms suggestive of AMI, such as chest pain, discomfort, or pressure that may radiate to the left arm, jaw, neck, or back; Electrocardiogram (ECG) findings indicative of STEMI: The ECG is a crucial diagnostic tool for AMI. Patients were included if their ECG showed ST-segment elevation, a hallmark of STEMI. Patients were excluded from the study if they met any of the following criteria; Elevated levels of creatine kinase-MB (CK-MB): CK-MB is another cardiac biomarker that can be elevated in AMI. However, it is less specific for cardiac muscle damage than troponin I. Patients with elevated CK-MB levels were excluded to ensure that the study focused specifically on the relationship between troponin I levels and infarct location; Elevated levels of troponin T: Troponin T is another cardiac-specific troponin that can be used to diagnose AMI. However, it has a slightly lower specificity for cardiac muscle damage compared to troponin I. Patients with elevated troponin T levels were excluded to maintain consistency in the study and focus solely on troponin I.

Data were collected from patients' medical records, ensuring patient confidentiality and adherence to ethical guidelines. The following information was systematically recorded for each patient; Demographic data: Age and gender were recorded as basic demographic information; Clinical presentation: The type, location, and duration of chest pain were documented to assess the clinical presentation of AMI; ECG findings: The location of ST-segment elevation on the ECG was noted to determine the specific area of the heart affected by the infarction; Laboratory data:

Troponin I levels were measured using a point-of-care testing (POCT) device (YHLO UNICELL-S) based on fluorescence immunochromatography. This method provides rapid and quantitative results, facilitating timely clinical decision-making.

Troponin I levels were measured using a point-of-care testing (POCT) device (YHLO UNICELL-S) based on fluorescence immunochromatography. This method provides rapid and quantitative results, facilitating timely clinical decision-making. The POCT device used in this study has been shown to have high sensitivity and specificity for detecting troponin I, comparable to laboratory-based assays. Data were analyzed using SPSS version 21, a statistical software package widely used in medical research. Descriptive statistics were used to summarize patient characteristics and troponin I levels. The correlation between troponin I levels and infarct location was assessed using the Pearson correlation test. A p-value < 0.05 was considered statistically significant.

3. Results

Table 1 provides a breakdown of the characteristics of the 57 participants enrolled in the study investigating the correlation between troponin I levels and the location of acute myocardial infarction (AMI) in ST-elevation myocardial infarction (STEMI) patients. The majority of participants were male (45 participants, 78.9%). This is consistent with the general trend of AMI being more prevalent in men than women, particularly in the age groups included in this study. Most participants fell within the 51-70 year age range (38 participants, 66.7%). This highlights that AMI is most common in middle-aged and older adults. A smaller proportion of participants were younger (25-50 years, 28.1%) or older (>70 years, 5.3%). The most frequent AMI location was anterior (20 participants, 35.1%), followed by anteroseptal (15 participants, 26.3%). These locations are commonly associated with occlusion of the left anterior descending (LAD) coronary artery, which supplies a large portion of the left ventricle. Other AMI locations observed were inferior (8 participants, 14.0%), lateral (6 participants, 10.5%), and anterolateral (8 participants, 14.0%). The mean troponin I level was 7.17 ± 3.75 ng/ml. The median troponin I level was 5.8 ng/ml (interquartile range: 1.2-10.5 ng/ml). Troponin I levels ranged from 0.1 to 12.16 ng/ml. These values reflect the variability in troponin I levels among the participants, which could be influenced by factors such as the extent of myocardial damage, time elapsed since the onset of AMI, and individual patient characteristics.

Table 1. Participants characteristics.

Characteristic	Category	Number	Percentage (%)
Gender	Male	45	78.9
	Female	12	21.1
Age (years)			
	25-50	16	28.1
	51-70	38	66.7
	>70	3	5.3
AMI location			
	Anterior	20	35.1
	Anteroseptal	15	26.3
	Inferior	8	14.0
	Lateral	6	10.5
	Anterolateral	8	14.0
Troponin I level (ng/ml)			
Mean ± SD	7.17 ± 3.75	-	-
Median (IQR)	5.8 (1.2-10.5)	-	-
Minimum-maximum	0.1-12.16	-	-

Table 2 presents the average troponin I levels across different locations of acute myocardial infarction (AMI) in the study participants. It also provides the standard deviation (SD) and the number of patients for each AMI location. Patients with anterolateral AMI had the highest average troponin I level (12.16 ng/ml), significantly higher than those with other AMI locations. This suggests a potentially larger extent of myocardial damage in this group. Conversely, patients with lateral AMI had the lowest average troponin I level (0.10 ng/ml). The table clearly shows a variation in average troponin I levels across different AMI locations. This supports the hypothesis that troponin I levels may be related to the location of infarction. The Pearson correlation coefficient (r) of 0.891 indicates a strong positive correlation between troponin I levels and the location of AMI. This means that higher troponin I levels tend to be associated with specific AMI locations, particularly anterolateral. The p-value of 0.000 indicates that the observed correlation is statistically significant. This means that the relationship between troponin I level and AMI location is unlikely to be due to chance.

Table 2. Average troponin I level.

Location of AMI	**Average troponin I level (ng/ml)**	**SD**	**Number of patients**
Anteroseptal	6.58	1.25	15
Anterior	5.32	0.95	20
Anterolateral	12.16	1.85	8
Inferior	4.15	0.75	8
Lateral	0.10	0.25	6
Total	**7.17**	**3.75**	**57**
P-value	**0.000**	-	-
Pearson correlation (r)	**0.891**	-	-

Figure 1 visually represents the relationship between Troponin I levels and the location of acute myocardial infarction (AMI) in the 57 patients studied. The type of Graph is a scatter plot, a common way to display the relationship between two variables. Each dot represents a single patient in the study. The x-axis shows Troponin I levels (ng/ml), which is a measure of heart muscle damage. The y-axis represents the "numerical location" of the AMI. While the exact method of assigning numerical values to AMI location isn't specified in the figure, it's likely a system where higher numbers indicate a more lateral location in the heart. The figure shows a clear upward trend. As Troponin I levels increase, the numerical location of the AMI tends to be higher (more lateral). This is confirmed by the fitted line on the graph, which slopes upwards. The strong positive correlation (r = 0.891) statistically confirms this visual trend. This value of 'r' close to 1 indicates a strong linear relationship between the two variables. In simpler terms, higher Troponin I levels are strongly associated with AMI occurring in a more lateral location in the heart. A possible correlation suggests that the location of the AMI within the heart may influence the extent of myocardial damage, as reflected by Troponin I levels. It's possible that AMIs in certain locations (lateral, in this case) may result in more extensive damage to the heart muscle, leading to higher Troponin I release.

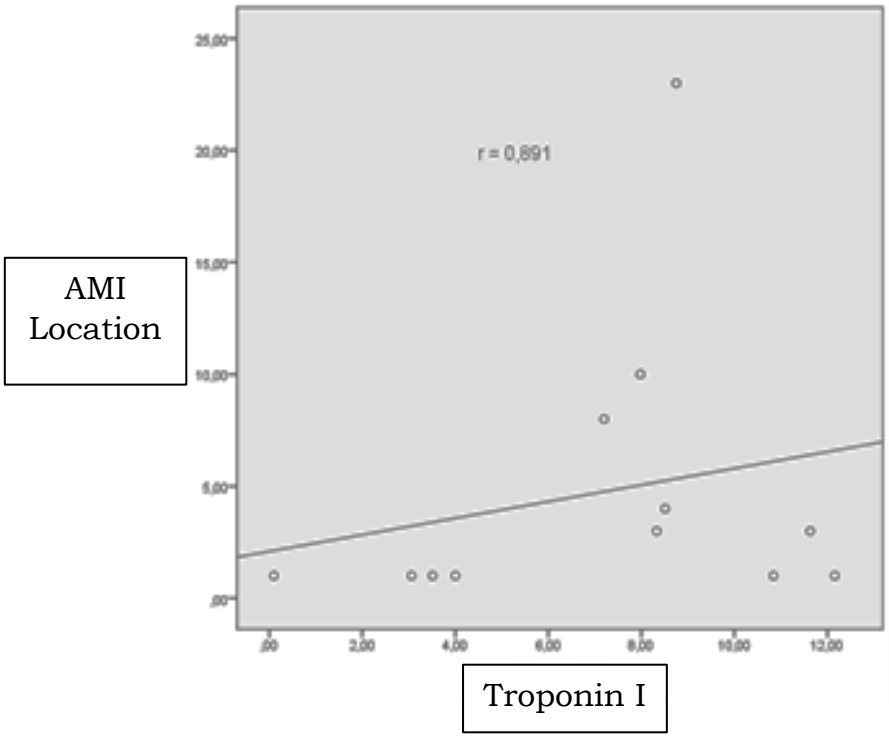

Figure 1. Correlation between Troponin I Levels and AMI Location. Scatter plot demonstrating the relationship between Troponin I levels (ng/ml) and the numerical location of acute myocardial infarction (AMI) in 57 patients. A strong positive correlation (r = 0.891) was observed, indicating that higher Troponin I levels are associated with a more lateral AMI location.

4. Discussion

The strong positive correlation between troponin I levels and the location of acute myocardial infarction (AMI) is a crucial finding in this study. It suggests that the location of an AMI within the heart significantly influences the extent of myocardial damage, as reflected by the levels of troponin I released into the bloodstream. This observation has important implications for understanding the pathophysiology of AMI, risk stratification, and treatment strategies. The correlation between troponin I levels and AMI location can be attributed to several factors, primarily related to the variations in the distribution of myocardial blood supply and the specific characteristics of the affected coronary arteries. The heart muscle receives its blood supply from the coronary arteries, which branch out into a complex network of smaller vessels. The distribution of blood flow within the myocardium is not uniform, with some regions receiving a richer blood supply than others. This variation in blood supply can influence the extent of myocardial damage in the event of an AMI. Infarctions in regions with a richer blood supply may result in less damage due to the presence of collateral circulation, which can provide alternative routes for blood flow. Conversely, infarctions in regions with a poorer blood supply may lead to more extensive damage due to the limited availability of collateral circulation. The coronary arteries exhibit anatomical variations among individuals, particularly concerning coronary dominance. Coronary dominance refers to the artery that supplies the posterior descending artery (PDA), which provides blood to the inferior wall of the left ventricle and the posterior portion of the interventricular septum. In right dominant hearts (approximately 85% of individuals), the PDA is supplied by the right coronary artery (RCA). In left dominant hearts (approximately 8% of individuals),

the PDA is supplied by the left circumflex artery (LCx). In co-dominant hearts (approximately 7% of individuals), the PDA is supplied by both the RCA and the LCx. Coronary dominance can influence the extent of myocardial damage in AMI. For instance, in a right dominant heart, occlusion of the RCA can lead to a larger infarction involving both the inferior and posterior walls of the left ventricle, potentially resulting in higher troponin I levels. In addition to the macrovascular circulation (coronary arteries), the microvasculature (smaller vessels within the heart muscle) also plays a crucial role in myocardial perfusion. Microvascular dysfunction, characterized by impaired blood flow through the microvasculature, can occur in AMI and contribute to myocardial damage. The extent of microvascular dysfunction may vary depending on the location of the infarction, influencing the release of troponin I. The study found that patients with anterolateral AMI had the highest average troponin I levels, while those with lateral AMI had the lowest. These observations can be explained by the specific characteristics of the coronary arteries supplying these regions. The anterolateral wall of the left ventricle is supplied by the LAD coronary artery, which also supplies a significant portion of the interventricular septum. Occlusion of the LAD can lead to a large area of infarction, potentially causing more myocardial damage and higher troponin I release compared to infarctions in other locations. The LAD artery is often referred to as the "widow maker" due to its association with high-risk infarctions and sudden cardiac death. The lateral wall of the left ventricle is supplied by the LCx coronary artery, which typically supplies a smaller area of the myocardium compared to the LAD artery. Therefore, occlusion of the LCx artery may result in less extensive myocardial damage and lower troponin I levels. However, it is important to note that lateral AMI can still have significant clinical implications, as it can lead to complications such as mitral regurgitation and ventricular arrhythmias. The correlation between troponin I levels and AMI location has important clinical implications for risk stratification, treatment decision-making, and prognostication. Troponin I levels are already used as a risk stratification tool in AMI patients, with higher levels indicating a greater risk of adverse outcomes. The findings of this study suggest that the location of the infarction should also be considered in risk stratification. Patients with high troponin I levels and infarctions in certain locations (such as anterolateral) may be at even higher risk and may require more intensive monitoring and treatment. The location of the infarction can also influence treatment decisions. For example, patients with anterior AMI may benefit from early reperfusion therapy, such as percutaneous coronary intervention (PCI) or fibrinolytic therapy, to restore blood flow to the affected area. Patients with inferior AMI may require treatment for bradycardia or hypotension, which are common complications of RCA occlusion. Troponin I levels have been shown to be a strong predictor of prognosis in AMI patients. The findings of this study suggest that the location of the infarction may also play a role in prognosis. Patients with infarctions in certain locations may have a worse prognosis, even after adjusting for troponin I levels.[11,12]

The observation that patients with anterolateral AMI had the highest average troponin I levels is a crucial finding in this study and aligns with previous research in the field. This observation underscores the severity of anterolateral AMIs and their potential for substantial myocardial damage, leading to a worse prognosis for patients. The anterolateral wall of the left ventricle receives its blood supply primarily from the left anterior descending (LAD) coronary artery. This artery is often referred to as the "widow maker" due to its critical role in supplying a large portion of the left ventricle, the heart's main pumping chamber. Occlusion of the LAD artery can have devastating consequences, leading to extensive myocardial damage and a cascade of complications. The LAD coronary artery originates from the left main coronary artery and courses down the anterior interventricular groove, giving off branches that supply the anterior wall of the left ventricle, a portion of the lateral wall, and the anterior two-thirds of the interventricular

septum. The LAD's extensive distribution makes it a critical determinant of cardiac function. When the LAD artery is occluded, the blood supply to the anterolateral wall of the left ventricle is compromised, leading to ischemia (lack of oxygen) and subsequent infarction (tissue death). The extent of myocardial damage depends on several factors, including the location and severity of the blockage, the duration of ischemia, and the presence of collateral circulation. In the case of anterolateral AMI, the occlusion of the LAD artery often results in a large area of infarction due to its extensive distribution. This extensive damage leads to a significant release of troponin I, a protein found in heart muscle cells that is released into the bloodstream when the heart muscle is injured. The higher the troponin I level, the greater the extent of myocardial damage. Patients with anterolateral AMI and high troponin I levels are at increased risk of complications such as heart failure, cardiogenic shock, and life-threatening arrhythmias. This information can help clinicians identify high-risk patients who may require more aggressive treatment and closer monitoring. The findings underscore the importance of prompt reperfusion therapy in patients with anterolateral AMI. Reperfusion therapy aims to restore blood flow to the affected area of the heart, either through percutaneous coronary intervention (PCI) or fibrinolytic therapy. Early reperfusion can limit the extent of myocardial damage and improve patient outcomes. Higher troponin I levels in patients with anterolateral AMI are associated with a worse prognosis. This information can help clinicians counsel patients and their families about the potential outcomes of the condition. The observation that patients with lateral AMI had the lowest average troponin I levels in this study aligns with the understanding of coronary artery anatomy and its influence on the extent of myocardial damage during an AMI. While lateral AMI may generally result in lower troponin I levels compared to other AMI locations, it's crucial to emphasize that it remains a serious condition with potential for significant complications. The lateral wall of the left ventricle is primarily supplied by the left circumflex (LCx) coronary artery. Unlike the left anterior descending (LAD) artery, which supplies a larger portion of the left ventricle, the LCx typically perfuses a smaller area of the myocardium. This anatomical distinction plays a key role in the extent of myocardial damage observed in lateral AMI. The LCx coronary artery arises from the left main coronary artery and courses along the atrioventricular groove, giving off branches that supply the lateral wall of the left ventricle and, in some individuals, the posterior wall. The LCx's territory of supply is generally smaller compared to the LAD, influencing the potential extent of damage in a lateral AMI. When the LCx artery is occluded, the blood supply to the lateral wall of the left ventricle is compromised, leading to ischemia and subsequent infarction. However, due to the smaller area typically supplied by the LCx, the extent of myocardial damage in lateral AMI may be less extensive compared to AMI involving the LAD. Troponin I, a protein specific to heart muscle, is released into the bloodstream in response to myocardial injury. The magnitude of troponin I release correlates with the extent of damage. In lateral AMI, the relatively smaller area of infarction often results in lower troponin I levels compared to other AMI locations, particularly those involving the LAD. Even with lower troponin I levels, lateral AMI can still lead to significant complications. Therefore, clinicians should not solely rely on troponin I levels for risk stratification in lateral AMI. Other factors, such as ECG changes, clinical presentation, and echocardiographic findings, should be considered to assess the severity and risk associated with lateral AMI. Prompt reperfusion therapy remains crucial in lateral AMI, even with lower troponin I levels. Restoring blood flow to the affected area can limit the extent of myocardial damage and prevent complications. Treatment strategies should also address specific complications associated with lateral AMI, such as mitral regurgitation and ventricular arrhythmias. While lower troponin I levels in lateral AMI may generally suggest a less extensive infarction, it's important to consider individual patient factors

and potential complications when assessing prognosis. Close monitoring and appropriate management of complications are essential for optimizing outcomes in lateral AMI. The lateral wall of the left ventricle supports the mitral valve apparatus. Infarction in this region can disrupt the valve's function, leading to mitral regurgitation, a condition where blood leaks back from the left ventricle to the left atrium during systole. Mitral regurgitation can cause heart failure symptoms and may require medical or surgical intervention. Lateral AMI can disrupt the electrical conduction system of the heart, increasing the risk of ventricular arrhythmias, including life-threatening ones like ventricular tachycardia and ventricular fibrillation. Close monitoring and prompt treatment of arrhythmias are crucial in lateral AMI.[13,14]

The predominance of male patients in this study aligns with the well-established trend of acute myocardial infarction (AMI) being more prevalent in men than women, especially within the age ranges examined in this research. This gender disparity in AMI risk is a complex phenomenon influenced by a multitude of factors, including hormonal differences, lifestyle choices, and variations in the onset and progression of atherosclerosis. Estrogen, the primary female sex hormone, is believed to exert cardioprotective effects. Estrogen promotes a more favorable lipid profile by increasing high-density lipoprotein (HDL) cholesterol (often referred to as "good cholesterol") and decreasing low-density lipoprotein (LDL) cholesterol (often referred to as "bad cholesterol"). This helps prevent the buildup of atherosclerotic plaque in the coronary arteries. Estrogen promotes vasodilation, the widening of blood vessels, which improves blood flow and reduces the risk of ischemia (lack of oxygen) to the heart muscle. Estrogen has antioxidant properties, protecting the cells lining the blood vessels (endothelial cells) from damage caused by free radicals. This helps maintain the health of blood vessels and prevents the formation of blood clots. Estrogen also exhibits anti-inflammatory effects, reducing inflammation in the blood vessels, which is a key contributor to atherosclerosis. The cardioprotective effects of estrogen are most prominent in premenopausal women. After menopause, estrogen levels decline significantly, leading to an increased risk of AMI in women. This risk continues to rise with age, approaching that of men in later years. Lifestyle choices play a significant role in the development of AMI, and some gender-specific patterns contribute to the observed disparity in AMI prevalence. Smoking is a major risk factor for AMI, and men are generally more likely to smoke than women. Smoking damages blood vessels increases the risk of blood clot formation, and contributes to the development of atherosclerosis. Men tend to consume diets higher in saturated fat and cholesterol, which can contribute to the development of atherosclerosis. Women are generally more likely to follow healthier dietary patterns. Men are often less physically active than women, and physical inactivity is a risk factor for AMI. Regular physical activity helps maintain a healthy weight, lowers blood pressure, and improves lipid profile, all of which contribute to cardiovascular health. Men and women may experience and cope with stress differently. Chronic stress can contribute to the development of AMI by increasing blood pressure, heart rate, and inflammation. Atherosclerosis, the underlying cause of most AMIs, is a chronic process that begins in early adulthood and progresses gradually over time. Men tend to develop atherosclerosis earlier than women, which may contribute to the higher prevalence of AMI in men at younger ages. This earlier onset may be related to hormonal differences, as well as lifestyle factors. In addition to hormonal differences, lifestyle factors, and atherosclerosis onset, other factors may contribute to the gender disparity in AMI prevalence. Men may have a genetic predisposition to developing AMI. Certain genes have been linked to an increased risk of AMI, and these genes may be more prevalent in men. Men are more likely to have certain comorbidities that increase the risk of AMI, such as diabetes and hypertension. Women may be less likely to seek

medical attention for symptoms of AMI, which can delay diagnosis and treatment, leading to worse outcomes. Healthcare providers should be aware of the gender differences in AMI risk and educate patients accordingly. Preventive measures, such as lifestyle modifications and risk factor management, should be emphasized for both men and women. Clinicians should be vigilant in diagnosing AMI in women, as they may present with atypical symptoms. Prompt and appropriate treatment is crucial for both men and women to improve outcomes.[15,16]

The observation that the majority of participants in this study fell within the 51-70-year age range aligns with the well-established epidemiological trend of increased acute myocardial infarction (AMI) risk with advancing age. This age-related vulnerability to AMI is primarily attributed to the progressive nature of atherosclerosis, the underlying cause of most AMIs, and the cumulative impact of various cardiovascular risk factors that tend to accumulate over time. Atherosclerosis is a chronic inflammatory process characterized by the gradual buildup of plaque within the walls of arteries. This plaque is composed of lipids, cholesterol, cellular debris, and calcium deposits. Over time, the plaque can harden and narrow the arteries, reducing blood flow to vital organs, including the heart. The endothelium, the inner lining of blood vessels, plays a crucial role in maintaining vascular health. Endothelial dysfunction, characterized by impaired function of the endothelium, is an early step in atherosclerosis development. It can be triggered by various factors, including high blood pressure, smoking, high cholesterol, and diabetes. When the endothelium is dysfunctional, LDL cholesterol can infiltrate the arterial wall. This LDL cholesterol can undergo oxidation, triggering an inflammatory response. The inflammatory response attracts white blood cells, particularly macrophages, to the site of LDL cholesterol accumulation. Macrophages engulf the oxidized LDL cholesterol, becoming foam cells. The accumulation of foam cells, along with other cellular debris and calcium deposits, forms atherosclerotic plaque. This plaque can grow and protrude into the artery lumen, narrowing the vessel and reducing blood flow. In advanced stages of atherosclerosis, the plaque can become unstable and rupture. This rupture triggers the formation of a blood clot (thrombus) at the site of injury. The thrombus can further obstruct or completely block the artery, leading to ischemia (lack of oxygen) and subsequent infarction (tissue death) in the affected organ. As people age, the atherosclerotic process continues, leading to a greater accumulation of plaque in the coronary arteries. This increased plaque burden increases the likelihood of plaque rupture and thrombosis, triggering an AMI. With age, the arteries become less elastic and more rigid. This reduced elasticity makes the arteries more susceptible to damage from high blood pressure and other stressors, further contributing to atherosclerosis progression. Endothelial dysfunction tends to worsen with age, making the arteries more vulnerable to inflammation and plaque formation. Older adults are more likely to have comorbidities that increase the risk of AMI, such as diabetes, hypertension, and chronic kidney disease. These comorbidities can accelerate atherosclerosis progression and contribute to other cardiovascular complications. In addition to age-related changes, the accumulation of cardiovascular risk factors over time contributes to the increased risk of AMI. Smoking damages blood vessels, increases the risk of blood clot formation, and accelerates atherosclerosis progression. High blood pressure puts extra strain on the arteries, increasing the risk of damage and plaque formation. High levels of LDL cholesterol contribute to plaque buildup in the arteries. Diabetes damages blood vessels and increases the risk of atherosclerosis and other cardiovascular complications. Obesity is associated with several cardiovascular risk factors, including high blood pressure, high cholesterol, and diabetes. Physical inactivity increases the risk of AMI by contributing to obesity, high blood pressure, and other risk factors. A family history of AMI increases an individual's risk, likely due to genetic factors and shared lifestyle habits. The increased risk of AMI with advancing age has important clinical implications.

Healthcare providers should assess AMI risk in older adults, considering age, atherosclerotic burden, and other cardiovascular risk factors. Preventive measures, such as lifestyle modifications (healthy diet, regular exercise, smoking cessation) and risk factor management (controlling blood pressure, cholesterol, and diabetes), should be emphasized for older adults to reduce AMI risk. Clinicians should be vigilant in diagnosing AMI in older adults, as they may present with atypical symptoms. Prompt and appropriate treatment is crucial to improve outcomes in this population. Specialized geriatric cardiology care may be beneficial for older adults with complex health conditions to optimize AMI management and prevent complications.[17,18]

The findings of this study contribute significantly to the existing body of knowledge on the relationship between troponin levels and infarct location in acute myocardial infarction (AMI) patients. By specifically investigating this correlation in ST-elevation myocardial infarction (STEMI) patients, this research adds a new dimension to our understanding of AMI pathophysiology and its clinical implications. Several previous studies have explored the relationship between troponin levels and infarct location, providing valuable insights into this complex interplay. Baheti et al. (2002) observed variations in troponin levels based on the time of measurement and infarct location, with higher levels in anterior MI. This finding aligns with the current study's observation that patients with anterolateral AMI, which often involves the LAD coronary artery supplying the anterior wall, had the highest average troponin I levels. Sagala et al. (2016) reported a correlation between troponin T levels and infarct location in AMI patients. However, their study focused on non-ST elevation MI (NSTEMI), which differs from STEMI in terms of the extent of coronary artery occlusion and myocardial damage. This study expands on their findings by specifically investigating the correlation between troponin I levels and infarct location in STEMI patients, providing a more comprehensive understanding of this relationship in a specific AMI subtype. By specifically investigating STEMI patients, this study provides a more focused analysis of the correlation between troponin I levels and infarct location in a homogenous patient population. STEMI is a severe form of AMI characterized by complete coronary artery occlusion and transmural myocardial infarction. This focus allows for a more precise assessment of the relationship between troponin I levels and infarct location without the confounding effects of other AMI subtypes. This study provides a detailed analysis of AMI location, considering various locations such as anterior, anteroseptal, inferior, lateral, and anterolateral. This comprehensive approach allows for a more nuanced understanding of how different infarct locations may influence troponin I levels. The findings of this study have important clinical implications for risk stratification, treatment decision-making, and prognostication in STEMI patients. By highlighting the correlation between troponin I level and infarct location, this research emphasizes the need to consider both factors when assessing the severity and risk associated with STEMI.[19,20]

5. Conclusion

This study investigated the relationship between troponin I levels and the location of acute myocardial infarction (AMI) in ST-elevation myocardial infarction (STEMI) patients. The findings revealed a strong positive correlation between troponin I levels and the location of AMI, with higher troponin I levels associated with a more lateral AMI location. This suggests that the location of the infarction may influence the extent of myocardial damage, as reflected by troponin I levels. This study has certain limitations, including the relatively small sample size, the cross-sectional design, and the single-center setting. Future research should address these limitations and explore the relationship between troponin I levels, AMI location, and other factors that may influence patient outcomes. Despite these limitations, this study provides valuable insights into the relationship between troponin I levels and AMI location in STEMI patients. The findings have

important clinical implications for risk stratification, treatment decision-making, and prognostication.

6. References

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