THE RELATIONSHIP BETWEEN TROPONIN I (TNI) AND HIGH-SENSITIVE TROPONIN I (HS-TNI) LEVELS AND LIPID PROFILE LEVELS IN ACUTE MYOCARDIAL INFARCTION PATIENTS

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ABSTRACT

Acute myocardial infarction (AMI) is the leading cause of death in Indonesia, with Approximately 94.56 deaths per 100,000 patients. One of the important indicators in the diagnosis of AMI is increased levels of troponin I (TnI) or high sensitive troponin I (hs-TnI). Approximately 70% of fatal AMI cases are attributed to occlusion caused by atherosclerosis. Atherosclerosis is characterized by lipid accumulation. The study aimed to determine the relationship between TnI and hs-TnI levels with lipid profile levels in AMI patients. The comparative analytical study design used primary data from 35 samples at RSMC in February – May 2024, and used spearman-rho correlation test analysis. Most AMI patients were in the age group of 56-65 (40%) and the percentage of male AMI patients (68.6%) is higher compared to females (31.4%). High-sensitivity troponin assays detect troponins (100%) but at a much lower concentration than classical assays. Low HDL levels (74.3%) indicate that the patient is at higher risk of detecting AMI incidences. We emphasize no significant relationship between TnI and hs-TnI with lipid profiles on AMI incidences because of several factors; time examination, complexity in cardiovascular disease, individual variability in response to atherosclerosis, and myocardial infarction.

Keywords: AMI, Tropinin I (TnI), high sensitivity troponin I (hs-TnI), lipid profile.

INTRODUCTION

Acute myocardial infarction is caused by a complete cessation of blood flow to the part of the myocardium. Myocardial cell death and necrosis occur due to the oxygen decrease in the myocardium over a long period (1). According to a study conducted by WHO, around 100 million people worldwide suffer from AMI every year, and around 9 million people died. in 2019, representing 16% of the total world deaths (2).

Elevated troponin I (TnI) levels are important indicators in the diagnostic accuracy of AMI. TnI is highly sensitive and released into the blood after cardiac damage is much greater than other biomarkers (3). The peak release occurs about 12 hours after cardiac injury and can remain detectable for seven days(3).

Several TnI diagnostic methods developed high-sensitive TnI (hs-TnI). The high-sensitive troponin I (hs-TnI) test detected low concentrations of troponin in the blood, thus identifying smaller heart muscle damage that may not be detected by conventional troponin I (TnI) tests (3). The high-sensitivity cardiac troponin tests have been approved for clinical use in the United States since 2017 for diagnosis and exclusion of AMI (4). A study by Ahmed et al., (2020) (5) showed that early use of a sensitive troponin I test significantly improved the early diagnosis of AMI, with excellent sensitivity and specificity at higher troponin I values ≥ 0.5 ng/dL.

Approximately 70% of AMI patients with fatal cases found atherosclerotic plaque blockage characterized by lipid accumulation, inflammatory reactions, cell death, and fibrosis in the artery wall, triggering significant changes in the coronary blood vessels. Sudden complete blockage can occur when a non-stenotic plaque ruptures, causing thrombosis to form, thus disrupting the oxygenated blood supply to the heart (6). A study by Aswania and Yasmin (7) showed that dyslipidemia is a strong predictive factor for serious cardiovascular events in hospitals for patients suffering from AMI.

In preventing an increase in the incidence of AMI, patients with cardiovascular disease are routinely recommended to undergo lipid profile tests, including total cholesterol, Low-Density Lipoprotein (LDL), High-Density Lipoprotein (HDL), and triglyceride levels (8). LDL level test is needed as an early predictor of the risk of AMI caused by atherogenic in the arteries, triggering the process of atherosclerotic plaque formation (7). Darussalam and Nofiyanto (8) found a statistically significant relationship between AMI patient mortality and total cholesterol, LDL, HDL, and triglyceride levels. Seeing the importance of troponin I (TnI), high sensitivity troponin I (hs-TnI), and lipid profile tests in AMI patients, researchers are interested in knowing the relationship between troponin test using the flow immunoassay (FIA) method and the high sensitivity troponin I (hs-TnI) method (12) (13) to lipid profile levels in AMI patients.

METHOD

This study used a comparative analytical research method and spearmans-rho correlation test. A total of 35 patients based on lemeshow formula met the inclusion criteria of AMI for this analysis. Primary data from 35 people was collected by directly examining Troponin I (TnI),

high sensitivity troponin I (hs-TnI) and lipid profile levels in patients diagnosed with AMI at RSMC in February - May 2024. This study received a research ethics test certificate from the Rumah Sakit Marinir Cilandak (04 / IV / 2024 / RSMC).

RESULTS AND DISCUSSION

This study used primary data obtained from AMI patients in the RSMC Emergency Department in 2024 with a sample of 35 based on inclusion and exclusion criteria. The research data included age, gender, Troponin I (TnI) levels, high sensitivity troponin I (hs-TnI) levels, and lipid profile levels which can be seen in the following table:

Table 1. Distribution of AMI Patients by Age

Age	Number of AMI Patients	
	n	%
26 – 35	1	2,9
36 – 45	3	8,6
46 – 55	6	17,1
56 – 65	14	**40,0**
>65	11	31,4
Total	35	100

Table 1 showed the percentage incidence of AMI was dominated by the age group of 56-65 years (40%). This is in line with research by Ahmed SA et al (5) that the age range with the highest prevalence of AMI was an average age of 56.6 years. Kumar N et al (11) also stated that the prevalence of AMI was highest in patients aged 51-60 years (32.8%). Age is a strong risk factor that cannot be modified in cardiovascular diseases. On the other hand, arterial aging is identical to arteriosclerosis, which is a degenerative process related to age that increases stiffness in the lumen of the arteries.

Table 2. Distribution of AMI Patients Based on Troponin I (TnI) and high sensitivity troponin I (hs-TnI) Test Results

Results	Number of AMI Patients	
	n	%
Troponin		
Normal (< 0,05 ng/mL)	11	31,4
Abnormal (≥ 0,05 ng/mL)	24	68,6
hs - TnI		
Normal (< 29 ng/mL)	0	0
Abnormal (≥ 29 ng/mL)	35	100

The precentage of troponin I (TnI) levels in the number of patients consisted of a normal group of 31.4% people and an abnormal group of 70.6% people (table 2). The test of troponin I (TnI) uses a Biosensor device that is able to read troponin I (TnI) concentrations between 0.05 - 20 ng/mL. Fluorescence-based biosensors are present as a valuable alternative to conventional colorimetric sensing devices, which although accessible in terms of cost, simplicity, and fast visual reading, have shortcomings related to their sensitivity and accuracy (12), (13).

The precentage high sensitivity troponin I (hs-TnI) levels in AMI patients resulted in 100% of patients being included in the abnormal group. The high sensitivity troponin I (hs-TnI) levels were measured using the Pathfast device with the chemiluminescent enzyme immunoassay

(CLEIA) method with a concentration reading range of 2.33 - 50000. High-sensitivity troponin tests detect troponin but at much lower concentrations than classical tests. This test offers several advantages with highly sensitive providing more rapid recognition of AMI (3)(9).

Table 3. Distribution of AMI Patients Based on Troponin and high sensitivity troponin I (hs-TnI) Test Results

Results	Number of AMI Patients	
	n	%
Cholesterol		
Normal (< 200 mg/dL)	28	80
Abnormal (≥ 200 mg/dL)	7	20
Triglyceride		
Normal (< 150 mg/dL)	26	74,3
Abnormal (≥ 150 mg/dL)	9	25,7
LDL		
Normal (< 150 mg/dL)	32	91,4
Abnormal (≥ 150 mg/dL)	3	8,6
HDL		
Rendah (< 40 mg/dL)	26	74,3
Normal (40-59 mg/dL)	7	20
Abnormal (60 mg/dL)	2	5,7

The highest cholesterol levels in the normal category of 80% of people (table 2). Cholesterol is very important for all cells in the body and is widely used as a major structural component of cell membranes and as a substrate for the synthesis of other steroids such as bile acids, vitamin D, and sex hormones such as estradiol, progesterone, androsterone, and testosterone. Accumulation of total cholesterol and LDL levels in plasma is an important risk factor for cardiovascular disease (14). Although AMI patients have normal total cholesterol values, they will still be at high risk if HDL levels are low, because the ratio between total cholesterol and HDL increases (15).

Normal triglyceride levels in this study (74.3%) were the highest. Normal triglyceride levels in AMI patients (74.3%) are caused by nutritional status which is generally normal, does not consume alcohol, and has started to reduce foods high in sugar and fat (16). The results of the study on HDL levels were dominated by low HDL levels (74.3%). HDL has a biological function as a cardioprotective, which is a process involved in the removal of excess cholesterol accumulated in peripheral tissues, transporting it to the liver to be excreted into the feces through bile. This shows that HDL plays an important role in protecting the development of atherosclerosis (14). HDL levels can decrease due to several factors such as genetic factors, type 2 diabetes mellitus, smoking, obesity, and poor lifestyle (8).

The results of this study showed that normal LDL levels were the highest (91.4%). This finding is in line with the research of Darussalam and Nofiyanto (8) (16) which also found that most patients had normal total cholesterol, LDL, and triglyceride levels. This may be due to the fact that acute myocardial infarction is influenced by many factors, not only dyslipidemia but also smoking, hypertension, obesity, diabetes, and hereditary factors. Another factor that plays a role

is the atherosclerosis process which can form long before an attack occurs. In addition, patients may have reduced their consumption of high-fat foods and received medication so that their lipid levels are no longer high (8).

Table 4. Correlation Test Results of Troponin I (TnI), high sensitivity troponin I (hs-TnI) and Lipid Profile Test

		Cholesterol	Triglyceride	HDL	LDL
Troponin I (TnI)	Coefficient Correlation	.214	.105	.029	.056
	Sig. (2-tailed)	.218	.548	.869	.750
	N	35	35	35	35
high sensitivity troponin I (hs-TnI)	Coefficient Correlation	.053	.110	.071	.112
	Sig. (2-tailed)	.764	.531	.686	.521
	N	35	35	35	35

Based on Table 4., The results of the correlation test of troponin I (TnI) levels with lipid profile levels in AMI patients did not show any correlation (relationship). Likewise, high sensitivity troponin I (hs-TnI) levels and lipid profile levels in AMI patients at the Cilandak Marine Hospital did not show any correlation (relationship). Several factors that play a role in the absence of correlation between troponin I (TnI) levels, high sensitivity troponin I (hs-TnI) to lipid profile levels in AMI patients include: a). The time factor is troponin I (TnI) levels increase immediately after heart muscle damage occurs and peak within hours to days after a heart attack. Meanwhile, the lipid profile reflects the long-term blood lipid status and does not change significantly immediately after an AMI event. b.) Complexity of cardiovascular disease, Cardiovascular disease is influenced by various risk factors and complex pathophysiological mechanisms. c). Individual variability, in response to atherosclerosis, inflammation, and myocardial damage can affect TnI levels and lipid profiles differently, thus blurring the correlation (17) (18).

Through this research, it is expected that health service institutions and medical personnel will become educators and facilitators in preventing AMI and regarding AMI risk factors. It is also expected that further research can be conducted on factors related to IMA, in order to reduce the prevalence of IMA patients.

CONCLUSION

The incidence of AMI is dominated in the age group of 56-65 years with male gender. There is no relationship between troponin I (TnI) and high sensitivity troponin I (hs-TnI) with lipid profiles on the incidence of AMI. Based on the results of the study, it is expected to be a reference for hospitals in providing appropriate supporting tests for AMI patients.

REFERENCES

1. Ojha, N. (1996) ‘Myocardial infarction’, *Reviews in Contemporary Pharmacotherapy*, 7(1), pp. 23–38.

2. Ferrero, L. (2023) ‘In-Hospital Mortality of Acute Myocardial Infarction’, *Epidemic Preparedness and Control [Working Title]*.

3. Lazar, D.R., Lazar, F.L., Homorodean, C., Cainap, C., Focsan, M., Cainap, S., et al. (2022) ‘High-Sensitivity Troponin: A Review on Characteristics, Assessment, and Clinical Implications’, *Disease Markers*, 2022, pp. 1–10.

4. Stark, M., Cc, K., Troponin, S.S., In, A., Internet, S., Island, T., et al. (2024) *Troponin Perkenalan Patofisiologi*.

5. Ahmed, S.A., Ismail, J., Nafees, T., Faraz, A., Tahir, M. and Rehman, M.U. (2020) ‘Sensitivity and Specificity of Bedside Qualitative Troponin I Test Kit as Compared with the Standardized Quantitative Lab Test for Troponin I’, *Cureus*, 134, pp. 65–67.

6. Iriana, D., Nurulita, A. and Rauf, D. (2019) ‘Hubungan kadar troponin I dan high-sensitivity troponin I dengan angiografi koroner pada pasien suspek coronary artery disease: studi di Rumah Sakit Umum Pusat dr.Wahidin Sudirohusodo Makassar-Indonesia tahun 2017’, *Intisari Sains Medis*, 10(2), pp. 420–425.

7. Aswania, G.M. and Yasmin, A.D.A. (2020) ‘Dislipidemia sebagai prediktor kejadian kardiovaskular mayor pada pasien infark miokard akut’, *Jurnal Medika Udayana* [Online], 9(11), pp. 91–100. Available at: https://ocs.unud.ac.id/index.php/eum/article/view/71028 (Accessed: 19 November 2024).

8. Darussalam, M. and Nofiyanto, M. (2017) ‘Profil Lipid Dan Mortalitas Pasien Infark Miokard Akut Di Rsud Panembahan Senopati Tahun 2015’, *Media Ilmu Kesehatan*, 6(2), pp. 83–90.

9. Card, M.C.E. and Sheet, C.D. (2022) < *REAGENT FOR PATHFAST* >, pp. 3–5.

10. Penelitian, K.K. and Tinggi, P. (2017) *Panduan Operator*, pp. 1–142.

11. Kumar, A. and Sathian, B. (2013) ‘Correlation between lipid profile and troponin I test results in patients with chest pain in Nepal’, *Asian Pacific Journal of Tropical Biomedicine*, 3(6), pp. 487–491.

12. Campu, A., Muresan, I., Craciun, A.M., Cainap, S., Astilean, S. and Focsan, M. (2022) ‘Cardiac Troponin Biosensor Designs: Current Developments and Remaining Challenges’, *International Journal of Molecular Sciences*, 23(14), pp. 1–15.

13. SD Biosensor Inc. (2017) *Buku Panduan STANDARDTM F TnI FIA*, pp. 4–5.

14. Wang, H.H., Garruti, G., Liu, M., Portincasa, P. and Wang, D.Q.H. (2017) ‘Cholesterol and lipoprotein metabolism and atherosclerosis: Recent advances in reverse cholesterol transport’, *Annals of Hepatology*, 16, pp. s27–s42.

15. Ahmad, N.H.D.F., Irwan, A., Astuty, E., Zulkarnain, K., Kusadhiani, I. and Ikhsan, I. (2021) ‘Hasil Penelitian Hubungan Rasio Kolesterol Total terhadap High Density Lipoprotein dengan’, *Jurnal Penelitian Kesehatan*, 3, pp. 42–54.

16. Purba, K.J., Tjiptaningrum, A. and Mustofa, S. (2023) ‘Gambaran Profil Lipid Pasien Infark Miokardium Akut di RSUD DR. H. Abdul Moeloek Lampung tahun 2021’, *Medical Profession Journal of Lampung*, 13(1), pp. 151–157.

17. Libby, P. (2021) ‘The changing landscape of atherosclerosis’, *Nature*, 592, pp. 524–533.

18. Ridker, P.M., Revkin, J., Amarenco, P., Brunell, R., Curto, M., Civeira, F., et al. (2017) ‘Cardiovascular Efficacy and Safety of Bococizumab in High-Risk Patients’, *New England Journal of Medicine*, 376(16), pp. 1527–1539.