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Original Research Paper

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LEVELS OF TROPONIN I AND CK-MB AS PREDICTORS OF SEVERITY OF ACUTE MYOCARDIAL INFARCT ASSOCIATED WITH GENSINI SCORE

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ABSTRACT Background. Coronary Heart Disease (CHD) or cardiovascular disease is currently one of the main and first causes of death in developed and developing countries, including Indonesia. This makes laboratory markers of this disease very important, especially markers that can be used to determine the severity of acute myocardial infarction. Troponin and CK-MB have been known as a cardiac marker in CHD, but whether CK-MB and Troponin can also describe the severity of acute myocardial infarction, this still needs to be studied.

Purpose: Seeing the relationship between troponin I and CK-MB levels with the severity of acute myocardial infarction. Method: A total of 48 patients with acute myocardial infarction who underwent percutaneous coronary intervention were

examined.

Results: From the 48 patients with acute myocardial infarction who underwent percutaneous coronary intervention, the median grade of Troponin I: 3.3 u / L (0.16 - 32), the median value of CK-MB: 59.5 (15 - 673), and the mean value of Gensini Score : 61.16 ± 21.43 . By using spearman correlation, the correlation between Troponin I and the gensini score was p = 0.53 with a value of r = 0.092, the correlation between CK-MB and Gensini score p = 0.62 with a value of r = 0.74. Conclusion: From the results of the study it was concluded that there was no significant relationship between troponin I and CK-MB levels with the severity of acute myocardial infarction associated with gensini scores.

KEYWORDS : Acute myocardial infarction, Troponin I, CK-MB, Gensini score

INTRODUCTION

Cardiovascular disease is the leading cause of death in the world in 2011. According to the data from world health organization (who), cardiovascular disease caused 1.7 million deaths in 2011, this shows that 3 out of 10 deaths in the world are caused by cardiovascular disease. According to the american heart association (aha) in 2015, the dearth rate from cardiovascular disease in the united states was 31.1%. According to the reports from the indonesian directorate general of health services (ditjen yanmed) during 2005, circulatory system diseases, including cardiovascular disease and stroke were the main causes of death.¹²

Myocardial infarction is defined as myocardial cell death due to prolonged ischemia.³ myocardial cell death does not occur immediately after the onset of myocardial ischemia, but occurs ≥ 6 hours. Atherosclerosis is by far the most common cause of myocardial infarction. The main risk factors for atherosclerosis are hyperlipidemia, diabetes, smoking, hypertension, gender, and age. Endothelial dysfunction and inflammation have a major role in the initiation of atherosclerotic plaque formation.^{4,5} atherosclerosis is characterized by the accumulation of lipids in the vessel walls leading to the formation of atherosclerotic plaques consisting of a central lipid core surrounded by foamy macrophages and smooth muscle cells covered by a fibrous cap. Rupture of the fibrous cap causes communication between the lipid content of the plaque and blood flowing through the artery lumen. Tissue factors expressed by macrophages activate platelets which ultimately lead to intraluminal thrombus formation. Finally blockage of the coronary arteries by thrombus reduces blood supply to the myocardial tissue leading to ischemia and necrosis, ultimately leading to myocardial infarction.⁶

Myocardial infarction consists of myocardial infarction with st segment elevation (stemi) and myocardial infarction without st segment elevation (nstemi). The diagnosis of acute myocardial infarction (ami) requires confirmation and identification because it is a basic requirement and relates to proper patient care. The main reason for an accurate and prompt diagnosis is to immediately provide the patient with ami therapy and appropriate interventions.⁷

Diagnosis of acute myocardial infarction using who criteria, where there are two or more criteria, namely chest pain, electrocardiogram changes (ekg) and biochemical markers. The diagnostic criteria have limitations because only a few patients with ami have characteristic symptoms.⁷

The biochemical markers that were examined were myoglobin, total creatinine kinase enzyme (ck-total), lactate dehydrogenase (ldh), creatinine kinase isoenzyme (ck-mb), cardiac troponin i and t (ctnl & ctnt). Myoglobin, total ck and ldh are not specific for cardiac muscle, whereas ck-mb is unable to detect small lesions and is only slightly elevated in muscle trauma.⁸⁹

Cardiac troponin i and t (ctnl & ctnt) are specific for cardiac muscle with high sensitivity and increased small myocardial necrosis (microscopic zone). Quantitative ctnl and ctnt can be used to stratify the risk of death from heart attack and follow the course of the disease. Cardiac troponin t has been standardized by who so that the variation in sensitivity and specificity between products is relatively small and is affected by renal failure and muscle failure.^{2,3} this study aims to assess whether the markers of acute myocardial infarction, namely troponin i and ck-mb can be used as predictors of the severity of a cute my ocardial infarction.

METHODS

This study is an analytical study with a cross sectional design. The study was conducted from april to june 2019. The population was patients suffering from coronary heart disease who were treated in the inpatient room of the adam malik general hospital in medan and underwent angiography procedures. The sample in this study was 48 people. The instrument used to measure ck-mb levels and troponin i level was ramp. The severity of coronary lesions using gensini score. Data analysis used statistical computer applications, with a confidence level of 95% (α =0,05).

RESULTS AND DISCUSSION

The result showed that most coronary heart disease sufferers were male, namely 36 people (75%) with a minimum age of 35 years and a maximum age of 80 years with an average age of 57.81 years (± 10.25). The largest age group is 50-59 years, as many as 19 people (60.4%). This is consistent with the results of the study sahin et al. Where the characteristic data on patient with heart disease in their study were 89% men with a mean age of 57.6 ±9.710. A similar result was obtained by cetin et al, where there were more male (70%) male patients with coronary heart disease with an average age of 61 ± 1111 . This is because the risk of coronary atherosclerosis is greater in men than women. Women are relatively more resistant to the disease until menopause, and then become just as vulnerable as men. The protective effect of estrogen is thought to explain the presence of immunity in women before menopause, namely protecting blood vessels from damage. Age is the most important risk factor for the occurrence of chd, getting older, the risk of coronary heart disease is getting higher and generally starts at the age of 40 years and over. Individual susceptibility to coronary atherosclerosis increases with age, age 40-60 years the incidence of acute myocardial infarction increases by fivefold.¹⁴

The results of the spearman correlation between serum ck-mb levels and gensini score in the angiography procedure showed a value of p = 0.61 with a value of r = 0.07. This indicates that there is no significant correlation between serum ckmb levels and gensini scores with very weak strength levels (table 1).

Table 1. Correlation Between Ck-mb Level With Gensini Score

	CK-MB	Gensini Score	p Value	r Value
Median (Min -	59,50 (15 –	61,00 (24,00 -	0,61	0,07
Max)	673)	112,00)		

This result is different from a study conducted by now et al, where they found that CK-MB levels were higher in myocardial infarction patients with a higher number of coronary lesions (p = 0.03).¹²

The results of the spearman correlation between the Troponin I level and the Gensini score in the angiography procedure showed a value of p = 0.53 with a value of r = -0.09. This shows that there is no significant correlation between the RDW value and the Gensini score with a very weak strength level (Table 2).

Table 2. Correlation Between Troponin I Levels And Gensini Score

	Troponin I	Gensini Score	p Value	r Value	
Median (Min	3,31 (0,16 -	52,00 (6,00-	0,53	0,09	
- Max)	32,00)	112,00)			

This result is not in line with a study conducted by Riska et al, where Troponin I levels were higher in acute myocardial infarction patients with significant coronary lesions compared to patients with acute myocardial infarction with insignificant coronary lesions.

This can be caused by increased oxygen demand and insufficient oxygen supply to the heart muscles, leading to increased troponin I and CK-MB in patients with normal coronary arteries⁶.

This can also be caused because myocardial infarction does not only occur in the atherosclerosis process but also in the coronary artery spasm¹³.

CONCLUSION

From this study it can be concluded that there is no significant relationship between CK-MB and Troponin I levels with the

severity of coronary heart disease associated with this Gensini score.

Suggestion

Further studies with more samples are needed so that the results are more representative of the parameters examined.

REFERENCES

- 1. Organization WH. The top 10 causes of death. 2015.
- Association AH. ACCF/AHA focused update incorporated into to the ACCF/AHA 2007 guidelines for the management of patients with unstable angina/non-ST elevation myocardial infarction: A report of the American College of Cardiology Foundation/American Heart Association task. Circulation 2013; 127: 663.
- Jennings R, Reimer K. Factors involved in salvaging ischemic myocardium: Effect of reperfusion of arterial blood. Circ J 1983; 68: 25–36.
- Cunningham K, Gotlieb A. The role of shear stress in the pathogenesis of atherosclerosis. Lab Invest 2005; 85: 9–23.
- Libby P, Ridker P, Maseri A. Inflammation and atherosclerosis. Circ J 2002; 105: 1135–1143.
 Mythili S, Malathi N. Diagnostic markers of acute myocardial infarction (
- Review D. Biomed REPORTS 2015; 3: 743–748.
 Janet M, Alison E, Burke M. Acute Coronary Syndromes. J Am Med Assoc 2010;
- John M, Jinson J, Barke M. Actie Corollary Syncholies, J Am Med Assoc 2010, 303:90.
 Fatonah S, Widijanti A, Hernowati T. Nilai diagnostik uji Troponin I kuantitatif
- metode Immuno kromatografi. Indones J Clin Pathol Med Lab 2007; 14: 20–23. 9. Samsu N, Sargowo D. Sensitivitas dan spesifitas Troponin T dan I pada
- diagnosis infark miokard akut. Majalah Kedokteran Indonesia, 2007, pp. 364–372.
- Şahin I, Karabulut A, Kaya A, et al. Increased level of red cell distribution width is associated with poor coronary collateral circulation in patients with stable coronary artery disease. Turk Kardiyol Dem Ars 2015; 43: 123–130.
- Çetin M, Kocaman SA, Bostan M, et al. Red Blood Cell Distribution Width (RDW) and its Association with Coronary Atherosclerotic Burden in Patients with Stable Angina Pectoris. Eur J Gen Med 2018; 9:7–13.
- Kini A, Marmur JD, Kini S, et al. Creatine Kinase-MB Elevation After Coronary Intervention Correlates With Diffuse Atherosclerosis, and Low-to-Medium Level Elevation Has a Benign Clinical Course Implications for Early Discharge After Coronary Intervention. J Am Coll Cardiol 1999; 34:664–669.
 Ong P, Athanasiadis A, Dominik M, et al. Coronary artery spasm as a cause
- Ong P. Athanasiadis A, Dominik M, et al. Coronary artery spasm as a cause for myocardial infarction in patients with systemic in fl ammatory disease. Int J Cardiol 2011; 151:e32–e34.
- Zahara F, Syafri M, Yerizel E. Gambaran Profil Lipid pada Penderita Sindrom Koroner Akut di Rumah Sakit Khusus Jantung Sumatera Barat Tahun 20112012. Jurnal Kesehatan Andalas. 2013; 3(2): 167-1.