Study of Risk Factors and Heart Performance on Atherosclerosis Potential

Rohayati[1*], I Ketut Adnyana[2], Neng Fisheri Kurniati [2]
[1]Department of Medical Laboratory Technology, Poltekkes Kemenkes Bandung
[2]Institut Teknologi Bandung
[*]Email correspondence: rohayati.tlm@staff.poltekkesbandung.ac.id

ABSTRACT

Atherosclerosis is a condition where the potential for myocardial infarction is very large. Several risk factors have an important role as a precursor to myocardial infarction, such as smoking, obesity, degenerative diseases, diabetes mellitus, heart disease, and several other factors that have a role in the occurrence of this attack. Early detection of this condition is necessary because it can prevent and reduce the potential for atherosclerosis.

The purpose of this study was to analyze how much the atherosclerosis risk factors correlated with the performance of cardiac enzymes such as HS-CRP and Troponin I. used 23 samples that met the inclusion criteria and the Acute Coronary Syndrome (ACS) category and 20 control group patients with at least one risk factor.

The test results showed that the levels of Troponin I and Hs-CRP in the control and ACS groups were significantly different. From the risk factors studied, such as BMI, blood pressure systole, blood pressure diastole, age, total cholesterol, and random blood sugar test, the most dominant positive correlation was RBS with a Pearson correlation number of 0.667 with

Keywords: *Atherosclerosis, myocardial infraction, risk factors, acute coronary syndrome*

INTRODUCTION

Myocardial infarction or heart attack is a case that has an ever-increasing number every year and causes death both in Indonesia and in the world. The causes are very complex, and the symptoms can suddenly make this disease relatively difficult to prevent and control. Several risk factors have an important role as a precursor to myocardial infarction, such as smoking, obesity, degenerative diseases, diabetes mellitus, heart disease, and several other factors that have a role in the occurrence of this attack.

Atherosclerosis is a condition where the potential for myocardial infarction is very large. This condition occurs in chronic and progressive. With the current lifestyle, it is very difficult to avoid atherosclerosis. The risks that often arise in this condition such as a body mass index that exceeds normal, smoking, hypertension, high blood sugar, and high cholesterol have the potential to support the occurrence of atherosclerosis.

Early detection of this condition is necessary because it can prevent and reduce the potential for atherosclerosis. By paying attention to these risk factors and conducting periodic checks of cardiac performance parameters, such as troponin I and Hs-CRP, it is hoped that it can monitor heart function and performance so as to minimize the risk of atherosclerosis.

RESEARCH METHOD

This study was an experimental study, which used 23 samples that met the inclusion criteria and the Acute Coronary Syndrome (ACS) category and 20 control group patients with at least one risk factor. Inclusion criteria: >18 years, in the ICU, one day after the doctor's diagnosis, Exclusion criteria: bypass, chronic kidney failure, terminal illness, steroid therapy, liver disorders, malignant cancer, septicemia. This study conducted HS-CRP and Troponin I examinations as biomarkers that have been used as markers of myocardial infarction. Examination using the ELISA Kit, HS-CRP (Biochem) sensitivity 10 ng/mL, Troponin I (Fine test) sensitivity 7.5 pg/mL.

Physical examination and interview

Physical examination and interviews with respondents were carried out in person, with structured questions and direct interaction so that they could assess whether the prospective respondent met the inclusion criteria or not. Then asked whether they are willing or not if they are willing to continue as respondents and sign informed consent.

Weight examination

Weight checks are carried out using digital scales that have previously been calibrated. Then it is placed in a flat place, where the respondent's weight is measured in kg and recorded. Only respondents who meet the inclusion criteria will proceed to the next stage.

Height examination

First, find a perfectly level floor and walls. Then remove the respondent's shoes, and also remove accessories that can add weight if possible. Stand straight with your heels on the wall and floor, and make sure your head, shoulders, and buttocks are against the wall. Head straight with eyes straight ahead. Pull out the height meter and see how tall the respondent is and then record it.

Examination of Body Mass Index

With the metric system, the BMI formula is body weight (kg) divided by height (m2). Since height is usually measured in centimeters, an alternative calculation formula is dividing weight in kilograms by height in centimeters squared and then multiplying by 10,000.

Blood Pressure Check

How measure blood pressure with a digital blood pressure device is relatively more practical than a manual blood pressure device. How: Wash your hands with soap and running water or use a hand sanitizer correctly. Adjust the position of the person whose blood pressure is to be measured so that the body is relaxed and can be sitting in a comfortable position and leaning back. Place the arm to be measured in a supine position on the table, can be the right arm or left. Then, roll up the sleeves to be measured. Place the cuff on the arm about three centimeters above the fold of the arm, not too tight or loose. Turn on the power button of the digital blood pressure monitor, after that the cuff will inflate automatically. so as not to forget the two numbers of this digital blood pressure meter, for example, 120/80 mmHg.

Examination of Total Cholesterol and blood sugar while

Prepare tools and materials to be used. The blood sample is frozen for 15-30 minutes. The frozen blood sample was centrifuged at 3000 rpm for 5 minutes until serum was obtained. Separated serum can be used as an examination sample. Pipette the serum into a test tube. Homogenized, incubate for 10 minutes. Read at a wavelength of 546 nm.

Table 1: table of total cholesterol glucose examination

	Blank	Standard	Control	Sample
Aquadest	10 µL			
Standard		10 µL		
Control			10 µL	
Serum				10 µL
Reagent	1000 µL	1000 µL	1000 µL	1000 µL

Troponin I and Hs-CRP Examination with Elisa Kit

Ten wells on the microplate were prepared for standard. In wells 1 and 2, add 100 ul standard and 50 ul standard diluent, then mix. In wells 3 and 4, add 100 ul of liquid from wells 1 and 2 and 50 ul of standard diluent, then mix. In wells 5 and 6, add 100 ul of liquid from wells 3 and 4 and 50 ul of standard diluent, then mix. In wells 7 and 8, add 100 ul of liquid from wells 5 and 6 and 50 ul of standard diluent, then mix. In wells 9 and 10, add 100 ul of liquid from wells 7 and 8 and 50 ul of standard diluent, then mix. Added Capture antibody to each well. Then incubate for 30 minutes at 37oC or overnight at 4oC. Wash solution preparation: dissolve 30x wash solution with aquadest (1 ml wash solution is added to 29 ml aquadest). Next, drain the liquid from the wells and wash the wells 5 times with the washing solution that has been prepared in step. Added blocking buffer, to make the antigen in the sample stick to the plate. Incubate the plate for 60 minutes, at 37oC or overnight at 4oC. Sample preparation. Add 10 ul sample and 40 ul sample diluent to each well. The sample should be put directly into the bottom of the well. Next, mixing is carried out so that the sample and the diluent sample are well mixed. Incubate the plates for 120 minutes at room temperature. Add 100 ul biotinylated antibody to each well. Incubate the plate for 60 minutes, at 37oC or overnight at 4oC. Next, drain the liquid from the wells and wash the wells 5 times with the washing solution prepared in the previous stage. Add 100 ul ABC solution to each well. Incubate for 30 minutes at 37oC. Next, drain the liquid from the wells and wash the wells 5 times with the washing solution prepared in the previous stage. Then 90 ul HRP-conjugate and 90 ul TMB were added to each well. Incubate the plate for 30 minutes, at 37oC. Furthermore, 100 ul stop solution was added to each well, so that the color change from blue to yellow would occur. Next, read the OD value at a wavelength of 450 nm on an ELISA reader. Next, the OD value of the sample will be obtained.

Data analysis

The data is then processed statistically using SPSSS including descriptive analysis and pearson correlation.

RESULTS AND DISCUSSION

Sample Frequency Distribution

All data on the results of the respondents are exposed in table 2.

Table 2 Sample Frequency Distribution

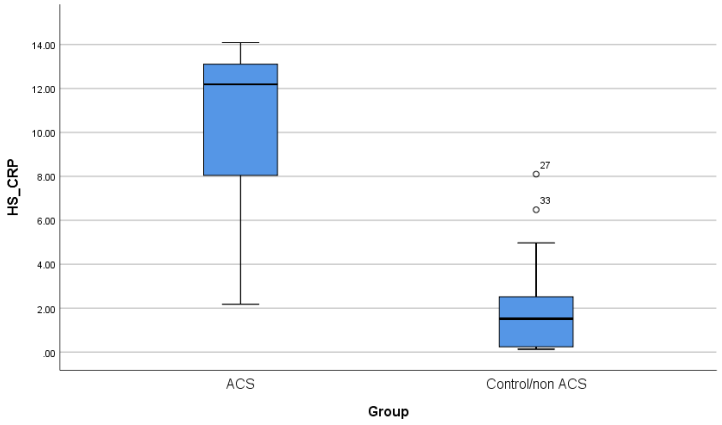
Frequency Table	Control Group		ACS patients	
	f(n)	%	f(n)	%
Systolic Blood Pressure				
>120 mmHg	5	25	17	74
<120 mmHg	15	75	6	26
Diastolic Blood Pressure				
>80 mmHg	1	5	14	61
<80 mmHg	19	95	9	39
Random Blood Sugar				
>180 mg/dL	0	0	4	17
<180 mg/dL	20	100	19	83
Gender				
L	10	50	17	73.9
P	10	50	6	26.1
Age				
Young adults	6	30	0	0
Adult	14	70	19	82.6
elderly	0	0	4	17.4
BMI				
<25	16	80	11	48
> 25	4	20	12	52
T Chol				
<200 mg/dL	11	55	20	87
> 200mg/dL	9	45	3	13

Based on samples from respondents, biomarkers were examined to determine heart performance. Obtained in table 3 below:

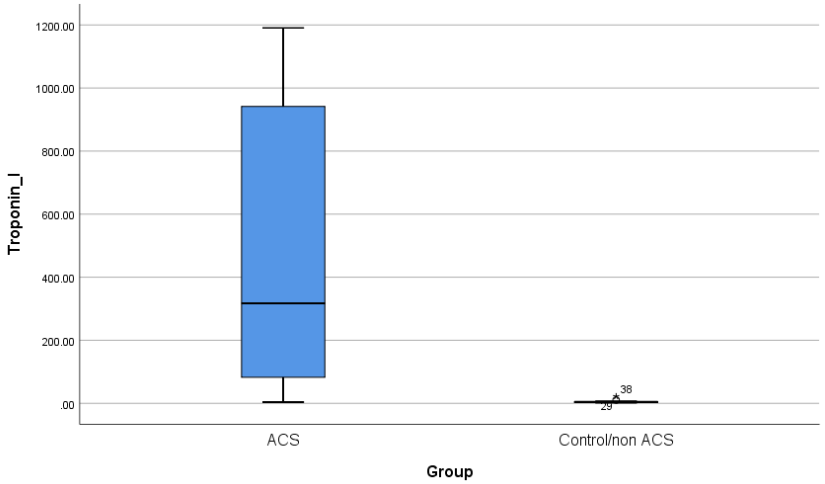
Table 3. Biomarker of cardiac performance

Biomarker	Control		ACS		P
	Mean±SD	Median	Mean±SD	Median	
HS-CRP (mg/dL)	2.056±2.18	1.513	10.241±3.94	12.19	0
Troponin I (pg/mL)	5.04±4.47	4.2	488.22±448.22	317	0

Table 3 shows that the average Hs-CRP level of the control group was 2.056 ± 2.18 mg/dL, and Troponin I levels were 5.04 ± 4.47 pg/mL. While the ACS group obtained an average Hs-CRP level of 10.241 ± 3.94 mg/dL, and Troponin I level of 488.22 ± 448.22 pg/mL. Where there was a significant difference between the control group and the ACS group. Graphically the plot is shown in the image below:



Pigure 1: level of HS-CRP between ACS and Non ACS Sample



Pigure 2 level of Troponin I between ACS and Non ACS Sample

Of the 43 samples, we looked at the relationship between risk factors and tested whether there was a relationship between these risk factors Hs-CRP and also Troponin I levels.

Correlations

		HS-CRp	BMI	Age	T CoL	Systole	Diastole	RBS
HS-CRP	Pearson Correlation	1	.277	.576**	-.024	.395**	.490**	.667*
	Sig. (2-tailed)		.072	.000	.876	.009	.001	.000
	N	43	43	43	43	43	43	43

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

From the several risk factors studied, such as body mass index, age, total cholesterol levels, systolic and diastolic blood pressure, and temporary blood sugar, it appears that a positive correlation with the Pearson value which is quite large is the factor of temporal blood sugar which shows a correlation with heart performance, in this case, is the hs-CRP level which is equal to 0.667 Pearson correlation.

Correlations

		Troponin I	BMI	Age	T CoL	Sistole	Diastole	RBS
Troponin I	Pearson Correlation	1	.306*	.506**	-.409**	.449**	.509**	.324*
	Sig. (2-tailed)		.046	.000	.006	.003	.001	.034
	N	43	43	43	43	43	43	43

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

From the several risk factors studied, such as body mass index, age, total cholesterol levels, systolic and diastolic blood pressure, and temporary blood sugar, it appears that a positive correlation with the Pearson value which is quite large is the factor of diastole blood pressure which shows a correlation with heart performance, in this case, is the Troponin I level which is equal to 0.509 Pearson correlation.

Conclusion

From the study, it can be concluded that all risk factors are good for BMI. Age. Blood pressure, cholesterol levels, and blood sugar levels while having a correlation with the increase in cardiac biomarkers. Where blood sugar hs-CRP levels when it has a Parson correlation of 0.667, while Troponin I levels are shown by diastolic blood pressure levels it has a Pearson correlation number of 0.59.

Acknowledgments

This study was founded by Poltekkes Kemenkes Bandung and also Institut Technology Bandung. The authors thank to head of Poltekkes Kemenkes Bandung and all staff di Institut Teknologi Bandung.

Bibliography

1. Anderson JL, Morrow DA. Acute myocardial infarction. New England Journal of Medicine. 2017 May 25;376(21):2053-64.
2. Badan Penelitian dan Pengembangan Kesehatan. Laporan Nasional Riset Kesehatan Dasar 2018. Kementerian Kesehatan Republik Indonesia. 2018.
3. Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, Caforio AL, Crea F, Goudevenos JA, Halvorsen S, Hindricks G. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC).

European heart journal. 2018 Jan 7;39(2):119-77.

4. Laura C O'Brien, 1. E. M. B. W. V. T. C. M., 2014. Interleukin-18 as a Therapeutic Target in Acute Myocardial. Molecular Medicine, pp. 221-229.
5. Mechanic, J., Gavin2, M. & Grossman3, S. A., 2021. Acute Myocardial Infarction. s.l.:NCBI Bookshelf. StatPearls Publishing.
6. Quratt Ul ain. Multi Maker Study To Evaluate The Prognostic Values Of IL-6, il-18, IL-1B, And Il-10 in Acute Coronary Syndrome Patients, Thesis ITB, 2022.
7. Rathore V, Singh N, Mahat RK, Kocak MZ, Fidan K, Ayazoglu TA, Aydin Karahan YG, Onk D, Akar E, Yolcu A. Risk factors for acute myocardial infarction: A review. EJMI. 2018;2(1):1-7.
8. Van Tassell BW, et al. (2012) Enhanced interleukin-1 activity contributes to exercise intolerance in patients with systolic heart failure. PloS One. 7:e33438.
9. Go AS, et al. (2013) Heart disease and stroke statistics—2013 update: a report from the American Heart Association. Circulation. 127:e6–e245.
10. Seropian IM, Toldo S, Van Tassell BW, Abbate A. (2014) Anti-inflammatory strategies for ventricular remodeling following ST-segment elevation acute myocardial infarction. J. Amer. Coll. Cardiol. 63:1593–603.
11. Gullestad L, et al. (2012) Inflammatory cytokines in heart failure: mediators and markers. Cardiology. 122:23–35.
12. Kalogeropoulos AP, Georgiopoulou VV, Butler J.(2012) From risk factors to structural heart disease: the role of inflammation. Heart Fail. Clin. 8:113–23.
13. Dinarello CA, Pomerantz BJ. (2001) Proinflammatory cytokines in heart disease. Blood Purif. 19:314–21.