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Relationship Mean Platelet Volume (Move) With Number of Coronary Artery lesions On Acute Coronary Syndrome

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Abstract:
Introduction: *Platelet activation and aggregation play a role in the pathophysiology of coronary heart disease. Platelet plays an important role in contributing to thrombus formation after coronary plaque rupture. In addition, platelets also play a role in the formation of atherosclerotic plaques. Mean Platelet Volume (MPV) is an index of platelet size that can be correlated with the functional status of platelets. In patients with acute coronary syndrome (ACS), increased MPV is associated with a degree of coronary artery disease lesion.**Aim:**To know the association of MPV with degree of coronary artery lesion so that MPV can be used as a marker to estimate the severity of coronary artery lesions in ACS patients.**Methods:** A cross sectional study of 50 patients with SKA. MPV examination of routine blood and vessel score assessment of angiography was performed. The study was conducted in RSUP. H. Adam Malik Medan. Analysis using Chi Square.**Results:** Mean of MPV content in this study is 10.15 ± 0.798 fl. Chi-square statistical test between MPV and vessel score (p = 0,068).**Conclusion:** There was no significant association between MPV levels and the number of coronary artery lesions described by Vessel score.*

Keyword: *Acute Coronary Syndrome, Mean Platelet Volume, Vessel score*

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I. Introduction

Background

Cardiovascular disease is one of the non-communicable diseases that cause death in the world. The disease shows an increase every year. Cardiovascular disease kills 17.5 million people in 2012. Based on WHO data in 2012, Coronary Heart Disease (CHD) is the leading cause of death by reaching 7.4 million deaths annually worldwide (WHO, 2016).

According to the results of Basic Health Research (RISKESDAS) in 2013, the prevalence of CHD is based on a diagnosed physician or symptom that reaches 1.5%, and is included in 10 major causes of death according to (Ministry of Health Research and Development, Ministry of Health, 2013).

CHD is a collective term for a disease that occurs when the coronary artery wall narrows gradually due to a fatty deposit called ateroma (Ireland Public Health, 2012). CHD is defined as a stenosis that is more than 50% the diameter of the blood vessels. The blockage in these coronary arteries can be partial or total of one or more coronary arteries and / or branches. The degree of stenosis in the coronary artery can be seen by coronary angiography and is usually measured by a visual evaluation of the percentage diameter reduction relative to the adjacent normal segment (Scanlon PJ et al, 1999).

CHD often presents with various clinical features of the asymptomatic, stable angina, acute coronary syndrome until sudden cardiac death. Acute Coronary Syndrome (ACS) is a clinical condition of acute ischemic myocardial level and depending on the degree of occlusion occurring, may be unstable angina pectoris (APTS),acute non-elevated myocardial infarction (IMA non STE), or acute elevated myocardial infarction ST elevation (IMA STE). ACS is an emergency case that must be quickly diagnosed and addressed, to avoid morbidity and mortality (Achyar et al, 2008)

Atherosclerosis is the main cause of CHD. Atherosclerotic processes are initially characterized by damage to the endothelial lining, formation of foam cells and fatty streaks, fibrousplaque (connective tissue lesions) and unstable atherosclerotic plaque rupture processes.Atherosclerosis is a chronic inflammatory process in which inflammation plays an important role in every stage of atherosclerosis from the beginning of plaque development to plaque rupture that can cause thrombosis (Hanson GK, 2005).

Platelet activation and aggregation has long been noted in the pathophysiology of coronary heart disease, because platelets play an important role in contributing to thrombus formation after coronary plaque rupture. Mean Platelet Volume (MPV) is a simple and reliable thrombocyte size index, which correlates with

platelet functional status, as a marker of the risk of atherothrombosis. In addition, previous evidence suggests that MPV may be a factor of independent risk for recurrent myocardial infarction of existing risk factors such as hypertension, dyslipidemia, increased fibrinogen, white blood cell count, or plasma viscosity (Lippi G et al, 2009). Metabolic and enzymatic platelets are more active than small platelets, so platelet activation can be measured by MPV (Yaghoubi et al, 2013).

Research has concluded that MPV as an indicator of platelet activation, suggests that it is important in patients with acute coronary syndromes.The results of a study in Iran confirmed that MPV at admission is not only predicted reperfusion disorders and attacks on IMA patients with ST segment treated and percutaneous coronary intervention (IKP), but also could be considered as a practical way of identifying high-risk patients (Yaghoubi et al, 2013).

De Luca et al (2009) measured MPV levels in 1411 patients undergoing coronary angiography, and showed that MPV was not related to the levels.

II. Literature Review

2.1. Atherosclerosis

Atherosclerosis is a major cause of CHD. The pathophysiological knowledge of atherosclerosis is growing. Former atherosclerosis is considered a degenerative process, but today, atherosclerosis is an inflammatory journey in arterial blood vessels (Verhamme P and Paul Holvoet, 2002).

Atherosclerosis is the main cause of CHD. Atherosclerosis is a multifactorial process with interrelated mechanisms. Atherosclerotic processes are initially characterized by damage to the endothelial lining, formation of foam cells and fatty streaks, fibrouscap formation (connective tissue lesions) and unstable atherosclerotic plaque rupture processes. Inflammation plays an important role in every stage of atherosclerosis from plaque development to plaque rupture that can lead to thrombosis. Atherosclerosis is considered an inflammatory disease because cells that act like macrophages derived from monocytes and lymphocytes are the result of inflammatory processes (Hanson GK, 2005; Verhamme P and Paul Holvoet, 2002).

The pathogenesis of atherosclerosis (atherogenesis) begins when there is damage (due to various risk factors in different intensities and lengths of exposure) in the arterial endothelium, thus it is causing endothelial dysfunction. Damage to the endothelium will trigger a variety of mechanisms that induce and promote atherosclerotic lesions. Endothelial dysfunction is caused by traditional risk factors such as dyslipidemia, hypertension, DM, obesity, smoking and other risk factors such as homocysteine and hemostatic abnormalities (Gawaz Meinrad et al, 2005).

2.2. Acute Coronary Syndrome

Definition of Acute Coronary Syndrome (ACS) refers to a set of complaints and clinical signs appropriate to acute myocardial ischaemia.ACS is a spectrum in the course of patients with coronary heart disease (coronary atherosclerosis) which may be unstable angina pectoris such as myocardial infarction, or sudden cardiac death (Achyar et al, 2008).

2.2.2. Epidemiology

Acute myocardial infarction is one of the most common inpatient diagnoses in developed countries. Data from GRACE 2001, it was found that of all patients who came to the hospital with chest pain complaint was the most cause was IMA-STE (34%), IMA non STE (31%) and APTS (29%) (Budaj et al, 2003).

The mortality rate in hospital care at IMA-STE is 7% whereas IMA non STE is 4%, but in the long term (4 years), the mortality rate of IMA non STE patients is 2 times higher than IMA-STE patients (Budaj et al, 2003).

2.2.3. Risk factors

The susceptibility of the narrowing of the lumen to the blood vessel is increased due to several factors known as risk factors. The risk factor of **ACS** is divided into two major parts, ie, non-modifiable factors and modifiable factors. Risk factors that can be modified include: smoking, hypertension, hyperlipidemia, diabetes mellitus, stress, high fat diet, and lack of physical activity. These risk factors can still be altered, thus potentially slowing the astherogenic process. While unmodified risk factors such as age, sex, tribe / race, and family illness history (Bender JR et al, 2011).

2.2.4. Pathophysiology

Most **ACS**are acute manifestations of coronary artery atheroma plaque that are torn or broken. This is related to changes in plaque composition and thinning of the fibrus hood covering the plaque. This event will be followed by the process of platelet aggregation and the activation of the coagulation pathway. Formed platelet is rich thrombus (white thrombus). This thrombus will block the coronary artery vessels, either totally or partially,

or become micro emboli that clog the more distal coronary vessels. In addition, the releases of vasoactive substances that cause vasoconstriction to exacerbate the disruption of coronary blood flow. Reduced coronary blood flow causes myocardial ischemia. An oxygen supply that stops for approximately 20 minutes causes myocardium to have necrosis (myocardial infarction) (PERKI, 2015).

2.2.5. Diagnosis of Acute Coronary Syndrome

The diagnosis of acute coronary syndromes is based on a typical clinical presentation of chest pain, ECG changes and elevated cardiac enzymes (PERKI 2015).

The patient's complaints with myocardial ischemia may be typical chest pain (typical angina) or atypical (angina equivalent). Typical anginal complaints are depressed / severe retrosternal regions, radiating to the left arm, neck, jaw, interscapular area, shoulders, or epigastrium. These complaints may be intermittent / several minutes or persistent (> 20 minutes). Typical angina complaints are often accompanied by comorbid complaints such as diaphoresis, nausea / vomiting, abdominal pain, shortness of breath, and syncope. Chest pain is also usually accompanied by other systemic symptoms of nausea, vomiting and cold sweat (PERKI, 2015).

2.3. Mean Platelet Volume

Mean Platelet the average platelet volume or volume is the most commonly used marker for assessing platelet count. MPV is often used as a potential marker in assessing platelet reactivity. MPV is routinely examined in outpatients and inpatients, and the cost is relatively cheap. The average platelet volume (MPV) comes from the platelet impedance size distribution curve. Large platelets are hemostasis more active than smaller and more functional platelets than smaller platelets. Increased MPV has been observed in patients at risk and after myocardial infarction and cerebral infarction (Chu SG et al, 2010; Briggs Carol et al., 2011).

2.4. The Role of Platelet Volume Mean on Atherosclerosis and Acute Coronary Syndrome

Platelets are important in primary hemostasis and endothelium repair and are considered to play an important role in the development of acute coronary syndromes and contribute to cerebrovascular events. In addition, platelets also play a role in the formation of atherosclerotic plaques (Verhamme P and Paul Holvoet, 2002).

2.5. Coronary angiography

To find out the coronary vein image, in 1959 it found an invasive method of investigation known as coronary angiography. Coronary angiography was first performed by Sones by inserting a catheter followed by injecting a contrast agent into the coronary artery and recording it with a radiographic photograph. The growing development of perioperative techniques and management makes the outcome better and reduces complications. Coronary angiography is helpful in determining diagnosis, prognosis and management of cardiovascular therapy (Jomansyah MUA 2013).

III. Research Methods

3.1. Research design

This research is an observational research with cross sectional data collection method

3.2. Place and time

The study was conducted at Adam Malik Hospital Medan. Sampling is done from January 2017 - April 2017 or until the number of samples is fulfilled after obtaining permission from the USU / RSUP H. Adam Malik Medan Ethics Committee.

3.3. Research subject

Patients with acute coronary syndrome treated at Adam Malik Hospital Medan in accordance with the criteria of inclusion and exclusion.

3.4. Inclusion and Exclusion Criteria

3.4.1. Inclusion Criteria

a. Patients with acute coronary syndrome are enforced by anamnesis, physical examination, ECG, and cardiac enzyme examination.
b. Age≥ 18 year old.
c. Willing to follow the research.

3.4.2. Exclusion Criteria

a. Patients with renal impairment and liver disorders.

b. Patients with infections.
c. Patients with malignancy.
d. Patients with blood disorders.

3.5. Population and Sample
- **Population:** ACS patient at Haji Adam Malik Hospital Medan.
- **Sample:** ACS sufferer according to criteria of sample size.
- **Sample size:** Based on the hypothesis test on a single proportion

$$n \geq \frac{\left(Z_{(1-\alpha/2)}\sqrt{P_o(1-P_o)} + Z_{(1-\beta)}\sqrt{P_a(1-P_a)}\right)^2}{\left(P_a - P_o\right)^2}$$

where :

$Z_{(1-\alpha/2)}$ = standard deviation of alpha, for α = 0,05then the default value is normal = 1,96

$Z_{(1-\beta)}$ = standard deviation betha, for β = 0,10then the default value is normal = 1,282

P_0 = proportion of patients with ACS=0,015 (1,5%)

P_a = estimated proportion of ACS patients studied, is = 0,115 (11,5%)

$P_a - P_o$ = significant difference in proportion is set at 0,10

So a minimum sample for this study is 42 people.

3.6. How to do Research
- All subjects are asked for approval to follow the study.
- In all subjects, anamnesis was performed, physical examination, 12 lead eye, routine laboratory, and cardiac enzyme.
- Enforcement of ACS diagnoses based on diagnostic criteria.
- MPV is checked at full blood count. MPV examination was done at the installation of Clinical Pathology Laboratory of RSUP H. Adam Malik Medan
- ACS patients underwent angiographic examination. Action and assessment of angiographic results are performed by the same cardiologist
- Coronary artery stenosis lesions are evaluated and classified based on the number of coronary arteries with> 50% stenosis.
- Further coronary artery lesions are associated with patient MPV levels

IV. Results and Discussion

4.1. Research result

During the study period (January 2017 to October 2017) at Cardiovascular Unit Care (CVCU) Department of Cardiology RSUP H. Adam Malik Medan obtained 50 research subjects with Acute Coronary Syndrome in accordance with inclusion and exclusion criteria in this study. All subjects were willing to take coronary angiography (Table 4.1)

Table 4.1 The basic characteristics of research subjects

Variable	Frequency
Sex(n); (%)	
- Male	45 (90%)
- Famale	5 (10%)
Age(± SD) (year)	53.68 ± 8.93
Body Mass Index (kg)	25,39 ± 3,50
Risk factor (n); (%)	
- Hypertension	28 (56%)
- DM	13 (26%)
- Smoking	36 (72%)
- Dyslipidemia	9 (18%)
Laboratory	
- Hb (gr/dl)	13.95 ± 1.89
- Leukocytes (/mm^3)	10343 ± 2462,72
- Platelets (/mm^3)	254000 (145000-779000)
- MPV (fL)	10.15 ± 0.7985
- KGD (mg/dl)	120 (81-368)
- Creatinine (mg/dl)	0.85 ± 0.21
- Total Cholesterol (mg/dl)	162,5 (110-340)

- LDL (mg/dl)	114,5 (38-305)
- HDL (mg/dl)	38 (20-83)
- Triglycerides (mg/dl)	134 (59-320)
Characteristics of ACS (n); (%)	
- APTS	8 (16%)
- NSTEMI	8 (16%)
- STEMI	34 (68%)
MPV levels	
- ≥ 9,8	32 (64%)
- < 9,8	18 (36%)
Vessel score	
- 0	6 (12%)
- 1	15 (30%)
- 2	14 (28%)
- 3	15 (30%)

Subjects of male gender are 45 patients (90%), and female sex are 5 patients (10%), and the age range 28-78 years with the mean is 53,68 ± 8,93. The mean Body Mass Index in this study 25,39 ± 3,50 kg.

Table 4.2. Characteristics of ACS risk factors for multiple lesions

Risk Factor	Vessel score		p
	0+1	2+3	
Hypertension	12 (24%)	16 (32%)	1,000
DM	4 (8%)	9 (18%)	0,531
Smoking	15 (30%)	21 (42%)	1,000
Dyslipidemia	3 (6%)	6 (12%)	0,835

Chi-Square Analysis

From the above table we found ACS risk factor with multiple lesions more than non-lesions, although there was no statistically significant relationship.

The results of MPV levels were divided into 2 groups: MPV ≥ 9,8fL (32 patients) and MPV < 9,8 fL (18 patients).

Table 4.3. Patient Characteristics Based on MPV levels

Variable	MPV < 9,8	MPV ≥ 9,8
Sex(n); (%)		
- Male	17 (94,4 %)	28 (87,5 %)
- Female	1 (5,6 %)	4 (12,5 %)
Age(± SD) (year)	55,39 ± 11,19	52,72 ± 7,41
Body Mass Index	24,98 ± 3,64	25,62 ± 3,46
Risk factor		
- Hypertension	10 (55,6 %)	18 (56,3 %)
- DM	4 (22,2 %)	9 (28,1 %)
- Smoking	15 (83,3%)	21 (65,6 %)
- Dyslipidemia	4 (22,2 %)	5 (15,6 %)
Laboratory		
- Hb	13,91 ± 1,79	13,97 ± 1,98
- Leukocytes	10.320 ± 1900,39	10.355,94 ± 2.757,83
- Platelets	272.555,5±59.680,74	278.500 ± 113.591,79
- MPV	9,35 ± 0,30	10,60 ± 0,60
- KGD	149,06 ± 86,68	154,13 ± 70,94
- Creatinine	0,80 ± 0,21	0,88 ± 0,19
- Total cholesterol	184,56 ± 45,12	165,97 ± 47,34
- LDL	133,44 ± 39,49	118,97 ± 48,70
- HDL	43,33 ± 10,45	37,78 ± 12,07
- Triglycerides	140,22 ± 30,41	141, 53 ± 55,31

From the results of angiography examination the number of lesions of coronary artery stenosis was assessed from the assessment of a vessel score consisting of a score of 0-3 based on the number of major coronary arteries undergoing stenosis ≥ 50%. In this study, there were 6 patients' vessel score, 1 vessel score of 15 patients, 2 vessel score of 14 patients, and vessel 3 score of 15 patients (Table 4.4).

Table 4.4. Patient Characteristics Based on the Vessel score

Variable	Vessel score			
	0	1	2	3
Sex (N); (%)				
- Male				
- Female	6 (100 %)	13 (86,7 %)	13 (92,9%)	13 (86,7 %)

	0 (0 %)	2 (13,3%)	1 (7,1 %)	2 (13,3 %)
Age(± SD) (year)	47,5 ± 6,71	52,13 ± 7,48	53,07 ± 10,00	52,87 ± 8,53
Body mass index	24,98 ± 4,47	25,67 ± 3,67	25,72 ± 3,72	24,97 ± 3,00
Risk factor				
- Hyperteson	2 (33,3 %)	10 (66,7 %)	6 (42,9 %)	10 (66,7 %)
- DM	0 (0 %)	4 (26,7 %)	2 (14,3 %)	7 (46,7 %)
- Smoking	5 (83,3%)	10 (66,7 %)	10 (71,4 %)	11 (73,3 %)
- Dyslipidemia	1 (16,7 %)	2 (13,3%)	4 (28,6 %)	2 (13,3 %)
Laboratoy				
- Hb	14,21 ± 1,65	13,88 ± 1,55	14,76 ± 1,79	13,16 ± 2,19
- Leukocytes	9.508,33 ± 1.238,18	10.799,33 ± 3.106,89	10.591,43 ± 2.209,26	9.988,67 ± 2403,53
- Platelets	246.333,33 ± 32.836, 97	315.933,33 ± 147.415,09	257.071,43 ± 48.623,30	266.800 ± 177.969,95
- MPV	10,55 ± 1,09	10,36 ± 0,53	9,97 ± 0,99	9,96 ± 0,63
- KGD	99,17± 10,34	165,33±80,18	135,71±56,22	176 ± 92,83
- Creatinine	0,72 ± 0,25	0,89 ± 0,22	0,81 ± 0,16	0,91 ± 0,192
- TtlCholesterol	140,33 ± 23,56	175,22 ± 45,97	173,93 ± 35,00	181,87 ± 61,01
- LDL	96,67 ± 37,47	121,93 ± 36,95	126,43 ± 36,07	135,33 ± 61,44
- HDL	40,83 ± 9,53	44,87 ± 13,13	37,86 ± 13,53	36,07 ± 7,70
- Tryglicerides	138,83 ± 32,99	141,67 ± 43,55	138,14 ± 43,91	144,07 ± 61,65

4.1.2. MPV Relation and Vessel Score

To know the relationship of MPV with Vessel score is done hypothesis test of unpaired categorical variable using Chi-Square test.

Table 4.5. Patient Distribution based on MPV and Vessel Scores

MPV levels	Vessel score				Total
	0 (n)	1 (n)	2 (n)	3 (n)	
< 9,8 fl	2 (4%)	2 (4%)	7 (14%)	7 (14%)	18 (36%)
≥ 9,8 fl	4 (8%)	13 (26%)	7 (14%)	8 (16%)	32 (64%)
Total	6 (12%)	15 (30%)	14 (28%)	15 (30%)	50 (100%)

From the distribution table above shows the largest number of patients which is found in the group Vessel Score 1 with MPV levels ≥ 9,8fl, which is 13 patients (26%).

Table 4.6. MPV Relation with Vessel Score

Vessel Score	MPV levels (n)		p
	< 9,8 fl	≥ 9,8 fl	
0	2 (4%)	4 (8%)	1,000
1	2 (4%)	13 (26%)	0,062
2	7 (14%)	7 (14%)	0,338
3	7 (14%)	8 (16%)	0,479

In the table above got the value p > 0,05for each vessel score on MPV levels based on Chi-Square analysis.

Table 4.7. MPV relationship with multiple lesions

		Vessel Score		p
		0+1	2+3	
MPV levels	MPV < 9,8 fL	4 (8%)	14 (28%)	0,068
	MPV ≥ 9,8 fL	17 (34%)	15 (30%)	
Total		21 (42%)	29 (58%)	

In the table above found that the value of p > 0,05for MPV relation to multi-vessel lesions based on Chi-Square analysis.

From the results of Chi-Square analysis, obtained p value > 0,05, so it was concluded that there was no relationship between MPV levels and the number of coronary artery lesions measured by a vessel score.

4.2. Discussion

From all sample of research, there were 45 male patients (90%), and female (5%). The mean age of the study sample was 53.68 ± 8.93 a year.

The proportion of subjects with hypertension risk factors (56%) was greater than those without hypertension (44%). The proportion of subjects with smoking risk factors (72%) was greater than that of non-smokers (28%). This is in accordance with studies that prove that hypertension or smoking is a risk factor for the occurrence of ACS.

The proportion of subjects with diabetes mellitus risk factor (26%) was lower than those without diabetes mellitus (74%). The proportion of subjects with risk factor for dyslipidemia (18%) was lower than that of subjects without dyslipidemia (82%). This is not in line with previous research which states that diabetes mellitus or dyslipidemia is a risk factor for ACS.

The difference in outcomes seen in the proportion of risk factors for diabetes mellitus, and dyslipidemia in this study did not deny the results of previous studies that have proven that these variables are risk factors for ACS. This happens because the purpose of this study is to look at the frequency distribution of risk factors without looking at the relationship between risk factors and ACS which require more comprehensive research design with large sample calculations that can facilitate the goal.

Research conducted by Pal Randheer et al (2014) reported that the diagnostic test results showed that MPV examination was diagnostic of ACS with sensitivity of 89.42% and specificity of 46.84%.Positive predictive value of 61.18% for MPV levels> 9.8 fl and negative predictive value of 82.53% is for MPV levels < 9,8 fl. In this study obtained MPV levels ≥ 9,8fl on the ACS is of 32 patients (64%).

Chi-Square comparative categorical hypothesis test in this study showed no correlation between MPV levels with Vessel score and multi-vessel lesion (p> 0,05), so it was concluded that MPV could not be used as marker in coronary artery lesion.

This study is in line with research by De Luca et al (2009) measuring MPV levels in 1411 patients undergoing coronary angiography, and showed that MPV was not associated with coronary artery disease levels. De Luca et al (2009) compares MPV levels based on quartiles I (≤10,5 fl), quartiles II (10,5-11,3 fl), quartiles III (≥11,3) to Vessel score with result p=0,48.

Pizzulli L et al (1998) studied 981 patients with coronary heart disease undergoing angiography, and obtained MPV results on a vessel score 0 = 8,2 ± 0.95 fl, vessel score 1 = 8,7 ± 1,19 fl, vessel score2 = 8,7 ±1,12 fl, vessel score3 = 8,8 ± 1,18 fl. The results concluded that there was no significant difference between MPV levels and the degree of atherosclerosis at a vessel score of 1 to 3.

Based on the literature review wchich is concluded that MPV plays a role in the process of atherosclerosis. In some literature such as Murat SaniNamik et al (2013) who studied 395 patients with ACS, there was an increase in the number of coronary artery lesions along with increased MPV levels (p = 0.002). The study also showed a positive correlation between MPV and Gensini score (r = 0.304, p <0.001) and Syntax score (r = 0.314, p = 0.001).

The study of 435 patients with coronary artery disease by EkiciBerkay et al (2013) concluded that MPV is a marker of platelet activity and as a risk factor for coronary artery disease. MPV increased according to the degree of severity of coronary artery disease, evidenced by MPV that correlated positively to Gensini score (r = 0.290, p <0.001) and Syntax score (r = 0,504, p <0.001), where both scores were used as an assessment of the severity of atherosclerosis.

In this study, MPV cannot be used as a marker of coronary artery lesions. The coronary artery lesion described as a process of atherosclerosis is a chronic process, whereas the presence of acute coronary syndrome is an acute process. Atherosclerosis is a chronic inflammatory process that is an important component of acute coronary syndromes. The association between chronic and acute vascular inflammation is unclear (Davi G and Carlo P, 2007).

The change in platelet size is determined by the process of thrombopoiesis.Platelet age ± 10 days,and the possibility of large platelets circulating in blood circulation during initiation of symptoms (Pizzulli L et al, 1998). Increased MPV is suspected of being responsible for pretrombotic state and ultimately leading to thrombus formation (Endler Georg et al., 2002).

The study of De Luca et al (2009) states that there is no MPV relationship with the number of coronary artery lesions and does not exclude anti-platelet use in the study. Although there was no significant difference of mean MPV content on clopidogrel (p = 0,94) or aspirin (p = 0,55).

In some studies say the use of anti-platelets can affect MPV levels. Antiplatelet can modify platelet aggregation by interfering with the aggregation process. For example, aspirin inhibits cyclooxygenase in platelets thereby decreasing the aggregated aggregation amplification agent (especially thromboxane A2) from the arachidonic acid pathway. ADP receptor antagonists, such as ticlopidine or clopidogrel, interfere with ADP binding to their receptors and prevent changes in platelets in the aggregation process, including the amplification process resulting from the release of stored ADP. GP IIb / IIIa antagonists, such as abciximab, tirofiban and integrelin, inhibit platelet aggregation by intervening in ligand bonds with activated GP IIb / IIIa complex (Kamath S et al, 2001).

Research conducted by Haungsaithong R et al (2015) in 21 patients with acute ischemic stroke, concluded MPV levels decreased after antiplatelet use such as aspirin, dipyridamole, clopidogrel, cilostazol. Where MPV before antiplatelet use (MPV = 8.40 fl) and decreased after antiplatelet use (MPV = 8.22 fl).

Shah Binita et al (2014) studied the effects of aspirin 81 mg / day on MPV in 48 healthy volunteers. Aspirin is given for 7 days. In the study, MPV levels were obtained prior to aspirin (10.6 fL [9.8-11]) versus MPV after aspirin (10.5 fL [9.9–11]; *p*=0.81).

Prolonged MPV examination of blood taking time, may also affect MPV levels. Blood sampling in this study used EDTA as anticoagulant. From some literatures, EDTA and citrate as anticoagulants to blood sampling can cause platelet swelling, which may change the interpretation of MPV (RL Adrian et al., 2002; Dastjerdi MS et al., 2006).

Dastjerdi MS et al (2006) reserached the comparison of MPV levels by using EDTA and citrate in 61 subjects. The time between blood sampling and MPV examination (with a hematology analyzer) was performed within 1 hour. Results of average MPV with EDTA is 7.860 ± 0.8924 fL, average MPV with citrate 7.200 ± 0.7901 fL. The statistical comparison of both methods is significant (p = 0.000).

Shah Binita et al (2014) examined the ratio of MPV levels by the time of venous puncture with MPV measurements (with a hematology analyzer) in 10 of 48 healthy volunteers. The subject of the study underwent a repetition of MPV examination from one blood sampling in time 15, 30, 60, 90, 120, 180, 240, 300, and 360 minutes after*vena puncture*. MPV levels increased significantly with the Spearman test, along with increasing the time interval between the puncture vein and the MPV measurement (r = 0.94, *p* = 0.001).

In addition, MPV levels have many confounding factors such as age, hypertension, DM, smoking, LDL and HDL, hiperhomocysteine, antioxidant use, and anti-platelet use (Verhamme P and Paul Holvoet, 2002).For example cross-sectional studies by Zuberi et al (2008) in 204 patients treated for diabetes mellitus, impaired fasting glucose (IFG), and non-diabetes mellitus, showed an average MPV in DMis 9,34 fl, at IFG is 8,98 fl, and at non DM of 8,63 fl. Comparison of MPV in all three groups shows a significant relationship with p < 0.00.

Tavil Yusuf et al (2007) conducted a study of association of MPV levels in 205 subjects with metabolic syndrome compared with 140 patients without metabolic syndrome as control. The study showed high MPV levels in metabolic syndrome patients who had a risk of coronary artery disease. In the study, MPV levels did not have a significant difference in patients with coronary artery disease. However, there was a significant difference between MPV levels in patients with metabolic syndrome accompanied by coronary artery disease compared to patients with metabolic syndrome without coronary artery disease (p < 0,05). Therefore, MPV is considered to be a follow-up marker in patients with metabolic syndrome as an alarm of coronary artery disease.

Research conducted by Endler Georg et al (2002), which compared 188 patients with coronary heart disease with a history of myocardial infarction with 185 stable heart disease patients (not myocardial infarction) undergoing angiography, proved a significant difference in MPV levels in both groups (p = 0,04). Patients with coronary artery disease with an increasePatients with coronary artery disease with an increase MPV (≥11,6 fl) have a significant increase in the risk of myocardial infarction (OR = 2,6; 95% CI 1,5 – 5,0; p = 0,01). The study concluded patients with coronary artery disease with increased MPV ≥11,6fl is a major risk of myocardial infarction, which can be identified through routine blood tests.

V. Conclusion and Suggestion

5.1. Conclution

From the results of this study can be concluded as follows:

1. Mean of MPV content in this research is 10.15 ± 0.798 fl. Number of patients with levels of MPV ≥ 9,8 fl in this study is more (32 people) compared with levels MPV < 9,8 fl (18 people). The highest Vessel Score Distribution is found on Vessel 1 score with content MPV ≥ 9,8flwhich amounted to 13 patients (26 %).
2. Comparative Chi-Square hypothesis test results in this study shows that there is no significant relationship between MPV levels with the number of coronary artery lesions described by Vessel score (p = 0,068).

5.2. Suggestion

1. Large-scale prospective studies and randomized clinical trials are required to see the association of MPV with the number of coronary artery lesions
2. The results of this study may be additional new science that encourages clinicians to be able to make conclusions from the mean role of platelet volume as a marker of atherosclerosis

References

[1]. Achyardkk. 2008. Buku Panduan Kursus Bantuan Hidup Jantung Langsung (Advanced Cardiac Life Support/ ACLS). Perhimpunan Dokter Spesialis Kardiovaskular Indonesia (PERKI).

[2]. Antman E, Anbe D, Armstrong P, dkk. 2004. ACC/AHA guidelines for themanagement of patients with ST elevation myocardial infartion. A report ofthe American College of cardiology/ American Heart Assosiation task force inpractice guidelines). Am Coll Cardiol J. 44(suppl I):1-212.

[3]. Aryanto Dodo. 2015. Uji Diagnostik Mean Platelet Volume (MPV) terhadap Troponin T pada Kasus Sindroma Koroner Akut. Tesis. Departemen Ilmu Penyakit DalamFakultas KedokteranUniversitas SumateraUtara Medan.

[4]. Badan Penelitian dan Pengembangan Kesehatan Kementerian Kesehatan RI. 2013. Riset Kesehatan Dasar (Riskesdas) tahun 2013. Jakarta.

[5]. Badimon L, J J Badimon, Valentin Fuster. 2002. Pathophysiology of arterial thrombosis. In: Platelets in Thrombotic and non Thrombotic Disorders: Pathophysiology, Pharmacology, and Therapeutics. New York: Cambridge University Press. 727-737.

[6]. Bender JR, Russell KS, Rosenfeld LE, Chaundry S. 2011. Risk factors for coronary artery disease: Preventive cardiology. In: Oxford American Handbook of Cardiology. New York : Oxford. 256-260.

[7]. Bessman JD dkk. 1981. Mean Platelet Volume. American Society of Clinical Pathologist. 76 (3):289-293.

[8]. Briggs Carol, Barbara J bain. Chapter 3: Basic Haematological Techniques. In Dacie and Lewis Practical Haematology. 11th edition. Churchil Livingstone. 2011.

[9]. Budaj A, Brieger D, Steg G, Goodman SG, Dabbous OH, Fox KAA, dkk. 2003. Global patterns of use of antithrombotic and antiplatelet therapies in patients with acute coronary syndromes: Insights from the Global Registry of Acute Coronary Events (GRACE). American Heart Journal. 146(6):999-1006.

[10]. Chu SG, Becker RC, Berger PB, Bhatt DL, Eikelboom JW, Konkle B, dkk. 2010. Mean platelet volume as a predictor of cardiovascular risk : a systematic review and meta-analysis. J Thromb Haemost. 8:148-56.

[11]. Dastjerdi MS dkk. 2006. Mean platelet volume measurement, EDTA or citrate?. Hematology. 11(5/6): 317–319

[12]. Davi G. Carlo P. 2007. Platelet Activation and Atherothrombosis. New England Journal Medicine. 357:2482-2494.

[13]. De Luca G, Santagostino M, Secco GG, dkk. 2009. Mean platelet volume and the extent of coronary artery disease: results from a large prospective study. Atherosclresosis. 206(1):292-297.

[14]. Demirin H dkk. 2011. Normal Range of Mean Platelet Volumein Healthy subjects: insight from a large epidemiologic study. Thrombosis Research. 128(4):358-360.

[15]. Ekici Berkay dkk. 2013. Is Mean Platelet Volume Associated with the Angiographic Severity of Coronary Artery Disease. Kardiologia Polska. 71(8):832-838.

[16]. Endler georg dkk. 2002. Mean Platelet Volume is an independent risk factor for myocardial infarction but not for coronary artery disease. British Journal of Haematology. 117:399-404.

[17]. Gawaz Meinrad, Herald Langer, and Andreas E M. 2005. Platelets in Inflammation and Atherogenesis. The Journal of Clinical Investigation. 115(12): 3378-3384.

[18]. Hansson GK. 2005. Mechanism of Disease Inflammation, Atherosclerosis, and Coronary Artery Disease. New England Journal Medicine. 352(16):1685-1695.

[19]. Haungsaithong R dkk. 2015. Change in Mean Platelet Volume after using of antiplatelets drugs in acute ischemic stroke: a randomizedcontrolled trial. J Med Assoc Thai. 98(9):852-856.

[20]. Huang G, Zhao JL, Du H, Lan XB, Yin YH. 2010. Coronary score adds prognostic information for patients with acute coronary syntrome. Circulation Journal. 74(3):490-495.

[21]. Ireland Public Health. 2012. Coronary Hearts Disease Briefing. http://www.publichealth.ie/sites/default/files/documents/files/CHD_Briefing_26_Jun_2012.pdf. 16 Mei 2016.

[22]. Jomansyah MUA. 2013. Angiografi Koroner. Cermin Dunia Kedokteran-207. 40(8):626-628.

[23]. Kamath S, A D Blann, G Y H Lip. 2001. Platelet activation: assessment and quantification. European Heart Journal. 22: 1561-1571.

[24]. Knudtson Merril. Coronary Scoring Systems A Historical Perspective. 2014. http://www.approach.org/pdfs/tutorials/History_of_Coronary_Scoring_March2014.pdf. 16 Mei 2016.

[25]. Kumar A, Cannon P. 2009. Acute Coronary Syndromes: Diagnosis and Management Part I. Mayo Clin Proc. 84(10):917-93.

[26]. Libby P. 2013. Mechanisms of Disease Mechanisms of Acute Coronary Syndromes and Their Implications for Therapy. New England Journal Medicine. 368(21):2004-2013.

[27]. Lippi G dkk. 2009. Increased Mean Platelet Volume in Patients With Acute Coronary Syndrome. Arch Pathol Lab Med. 133:1441-1443.

[28]. Mercan R, Demir C, Dilek I, Asker M, Atmaca M. 2010. Mean Platelet Volume In Acute Coronary Syndrome. Van Tip Dergisi. 17(3):89-95.

[29]. Michelson Alan D. 2004. Platelet function testing in cardiovascular disease. Circulation. 110:489-493.

[30]. Mirzaie AZ, Abolhasani M, Ahmadinejad B, Panahi M. 2012. Platelet count and MPV, routinely measured but ignored parameters used in conjunction with the diagnosis of acute coronary syndrome: single study center in Iranian population 2010. Medical Journal of Islamic Republic of Iran. 26(1):17-21

[31]. Murat Sani Namik dkk. 2013. Relation Between Mean Platelet Volume and Severity of Atherosclerosis in Patients With Acute Coronary Syndromes. Angiology. 64(2):131-136.

[32]. Packard R.R.S, Libby P. 2008. Inflammation in Atherosclerosis: From Vascular Biology To Biomarker Discovery And Risk Prediction. Clinical Chemistry. 54(1):24-38.

[33]. Pal Randheer dkk. 2014. Mean Platelet Volume in Patients with Acute Coronary Syndromes: A supportive diagnostic predictor. Journal of Clinical and Diagnostic Research. 8(8): 01-04.

[34]. Perhimpunan Dokter Spesialis Kardiovaskular Indonesia (PERKI). 2015. Pedoman Tatalaksana Sindroma Koroner Akut. Edisi ke 3. Centra Communications.

[35]. Pizzulli L dkk. 1998. Changes in platelet size and count in unstable angina compared to stable angina or non cardiac chest pain. European Heart Journal. 19:80-84

[36]. Rahajuningsih D S. 2009. Patofisiologi trombosis dalam hemostasis dan trombosis. Edisi ke 4. Fakultas Kedokteran Universitas Indonesia. Jakarta

[37]. RL Adrian dkk. 2002. The platelet shape change. In: Platelets in Thrombotic and non Thrombotic Disorders: Pathophysiology, Pharmacology, and Therapeutics. New York: Cambridge University Press. 319-337.

[38]. Scanlon PJ, dkk. 1999. ACC/AHA guidelines for coronary angiography. A report of the american college of cardiology/ american heart association Task Force on Practice Guidelines (comittee on coronary angiography). Circulation 99: 2345-2357.

[39]. Shah Binita dkk. 2014. Mean platelet volume reproducibility and association with platelet activity and anti-platelet therapy. Platelets. 25(3): 188–192.

[40]. Tavil Yusuf dkk. 2007. Mean Platelet Volume in Patients with Metabolic Syndrome and its relationship with coronary artery disease. Thrombosis Research. 120:245-250.

[41]. Verhamme P, Paul Holvoet. 2002. Platelets and Atherosclerosis. In: Platelets in Thrombotic and non Thrombotic Disorders: Pathophysiology, Pharmacology, and Therapeutics. New York: Cambridge University Press. 738-752.

[42]. World Health Organization. 2016. The top 10 causes of death. http://www.who.int/mediacentre/factsheets/fs310/en/index2.html. 16 Mei 2016.

[43]. Wright RS, Cynthia DA, Jeffrey LA, Adams CD, Bridges CR, Casey DE, dkk. 2011. ACCF/AHA Focused Update of the Guidelines for the Management of Patients With Unstable Angina/Non-ST-Elevation Myocardial Infarction (Updating the 2007 Guideline). Circulation. 123:2022-60.

[44]. Yaghoubi A, Golmohamadi Z, Alizadehasl A, Azarfarin R. 2013. Role of platelet parameters and haematological indices in myocardial infarction and unstable angina. J Park Med Assoc. 63(9):1133-1137.
[45]. Yang Yi-Ning, Hong Mei Lai, dkk.2015. Usefulness of Mean Platelet Volume as a biomarker for angiographic thrombus burden and short-term mortality in patients with ST segment elevation myocardial infarction undergoing primary percutaneous coronary intervention. J Am Coll Cardiol. 65(10S).
[46]. Zuberi BF, Akhtar N, Afsar S. 2008. Comparison of Mean Platelet Volume in patients with diabetes mellitus, impaired fasting glucose, and non diabetic subjects. Singapore Med J. 49(2):114-116.

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