

Frequency of Patients with NSTEMI Electrocardiographic Changes that Have Potential to Become STEMI

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Abstract-

Acute Coronary Syndrome (ACS) can be divided into a subgroup of ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), and Unstable Angina (UA). ACS carries significant morbidity and mortality and the prompt diagnosis, and appropriate treatment is essential. STEMI diagnosis and management are discussed elsewhere. NSTEMI and unstable angina are very similar, with NSTEMI having positive cardiac biomarkers. While the cause of this mismatch in STEMI is nearly always coronary plaque rupture resulting in thrombosis formation occluding a coronary artery, there are several potential causes of this mismatch in NSTEMI. There may be a flow-limiting condition such as a stable plaque, vasospasm as in Prinzmetal angina, coronary embolism, or coronary arteritis. The "typical" presentation of NSTEMI is a pressure-like substernal pain, occurring at rest or with minimal exertion. The pain generally lasts more than 10 minutes and may radiate to either arm, the neck, or the jaw. History, ECG, and cardiac biomarkers are the mainstays in the evaluation. An ECG should be performed as soon as possible in patients presenting with chest pain or those with a concern for ACS. A normal ECG does not exclude ACS and NSTEMI. ST-elevation or anterior ST depression should be considered a STEMI until proven otherwise and treated as such. Findings suggestive of NSTEMI include transient ST elevation, ST depression, or new T wave inversions. ECG should be repeated at predetermined intervals or if symptoms return.

Keywords: classic symptoms of ACS, evolution, NSTEMI, ST-elevation, ST-depression, fibrinolytic, and percutaneous intervention (PCI)

INTRODUCTION

Data from the World Health Organization (WHO) in 2012 showed that 17.5 million people died from cardiovascular disease, or 31% of 56.5 million deaths worldwide. More than 3/4 of deaths from cardiovascular disease occur in developing countries with low to moderate-income [1;2]. Based on 2013 *RISKESDAS* data, it is seen that the highest prevalence for cardiovascular disease in Indonesia is Coronary Heart Disease (CHD), which is 1.5%. Of these, the highest rate was in East Nusa Tenggara Province (4.4%) and the lowest in Riau Province (0.3%). According to WHO data in 2015, the incidence of CHD also increases with age, smoking habits, excessive bodyweight, etc., are important risk factors.

The high mortality due to CHD is one sign that this disease requires early diagnosis and proper handling [3;4;5]. At present the American College of Cardiology/American Heart Association (AHA) has published a 12-lead electrocardiogram reading guide that must be applied when dealing with patients with typical complaints of chest pain, approximately 10 minutes after the complaint appears [6;7]. Interpretation of electrocardiographic results is a step in diagnostic evaluation and therapeutic planning. Because of the importance of ECG images in the determination of diagnosis and therapy, it is necessary to conduct research aimed at knowing the ECG features and risk factors for myocardial infarction.

METHOD

This research is a descriptive-observational study conducted at the Berkat Agung Clinic since 12 February 2018 - 26 September 2019. The study population was all patient medical records at the Berkat Agung Clinic, while the affordable population was patients with myocardial infarction. The Slovin formula calculates the sample size. For the precision level set in determining the sample is 10% because the total population is less than 1000, so Slovin formula is applied:

$$n = \frac{N}{1 + Ne^2}$$

Information:

n = number of samples

N = total population

e = looseness of inaccuracy due to tolerable sampling errors

Based on the formula above, a sample size of 50 is obtained, with inclusion criteria: 40 - 80 years old, having stable and/or unstable angina pectoris episodes, a history of heart disease, risk factors for hypertension, hypercholesterolemia, or both. An ECG examination was performed, and there were troponin enzyme data. Medical record data is not used if the data is incomplete. Data collected from medical records in the form of age, STEMI and NSTEMI ECG images and troponin enzymes. Besides, note the risk factors in the form of hypertension and hypercholesterolemia. The data obtained were tested statistically to find the meaning of the relationship with the chi-square test.

RESULT AND DISCUSSION

The research data was taken from medical records at *the Agung Jatibening Raya Clinic, Bekasi* from February 2019 to September 2019. There were 50 medical records and 32 medical records that met the inclusion criteria. Of these, 12 (37.5%) had an ECG according to STEMI, while 20 (62.5%) were included in NSTEMI. The 51-60 years age group is the largest group with ECG images according to STEMI, while for the ECG NSTEMI images are also in the same age group (Table 1).

Table 1. Distribution of Patient Age Groups based on STEMI and NSTEMI ECG Results

Age Group	STEMI	Percentage	NSTEMI	Percentage
40 – 45 year	2	16,7	5	41,67%

46 – 50 year	3	25	3	15%
51 – 60 year	6	50	11	55%
61 – 80 year	1	8,3	1	5%
Total	12	100%	20	100%

Source: Primary data for February - September 2019

Table 2. Risk factors and features of the troponin enzyme in the studied patients

	STEMI		NSTEMI	
	n	%	n	%
Risk Factors				
Hypercholesterolemia	12	25	18	25
Hypertension	11	16,7	15	20
Both	9	75	13	65
Not both	0	0	0	0
Enzyme tests				
Troponin enzymes	8	66,7	18	90

Table 2 shows that infarction is more common in patients who have two risk factors (hypercholesterolemia and hypertension). In both STEMI and NSTEMI, there is an increase in cardiac biomarkers, namely the troponin enzyme. A total of 8 STEMI patients from 12 existing patients or about 66.7%, while 18 out of 20 NSTEMI patients experienced an increase in the troponin enzyme or about 90% of the number of the patients present. This difference is not significant.

Table 3. Frequency Distribution of ECG NSTEMI Evolution with the Occurrence of STEMI

Variable	Frequency	Percentage
STEMI stagnant	7	58,3%
Evolution of NSTEMI to STEMI	5	25%
TOTAL	12	83%

Source: Secondary data for February 2019 - September 2019

In Table 3, it is found that the results of electrocardiogram examination with NSTEMI can evolve into STEMI in 5 patients of 20 STEMI patients. Complaints of chest pain can be a sign of circulatory disorders in the heart. These complaints must be followed up by doing an EKG. EKG image to confirm the presence or absence of ST elevation.

The incidence of STEMI in a hospital home is rarely an emergency case, but symptoms of chest pain should be monitored continuously. Although STEMI events often give an image of ST-segment elevation, there are times when STEMI evolves. It can also give the opposite situation, namely the EKG image without STEMI, but in the next few times, the ECG image shows STEMI, as seen in Figure 1.



Figure 1. Electrocardiogram results of a patient with chest pain and shortness of breath; age 53 years. A peaked T wave is seen in leads V₂ - V₄. Fifteen minutes later, another electrocardiogram was examined, and the results can be seen in Figure 2. The ST image elevation segment begins to appear in several leads accompanied by an inverted T wave.

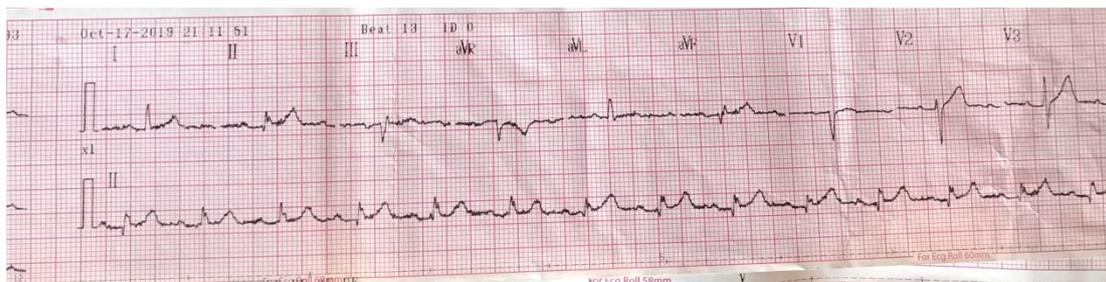


Figure 2. Results of the electrocardiogram 15 minutes after the first EKG examination.

The high amplitude of the T wave, as shown in Figure 1, is an early indicator of coronary occlusion in the myocardium without necrosis. Under normal circumstances, repolarization runs from the epicardium (outer layer) to the endocardium (inner layer) [8;9;10;11]. The repolarization that extends from the epicardium causes an increase in the repolarization vector in the same direction, increasing the T wave. Besides, an image with a high T wave is an early sign of STEMI without accompanying abnormalities in other leads; usually, there is no increase in serum troponin or CK-MB [12;13;14;15]. The evolution that occurs as in Figure 2 is associated with the existence of a diastolic theory according to Lilly LS in a book entitled "Pathophysiology of Heart Disease" which states that repolarization that is not entirely completed in the ischemic area of the myocardium will produce more negative electricity when compared to normal myocardial tissue. When a normal myocardium has finished repolarizing during the diastolic phase, there will be a change in electricity, so that the negative electric current from the ischemic area will go to the normal area. This current causes a negative electrical potential to surround the area in the heart during the diastolic phase so that the depressed TQ segment will appear [16;17;18;19]. When the myocardial cells have finished depolarizing during the systolic phase, no electrical potential is detected, so this will provide a more positive electrical picture above the baseline. This theory finally concludes that the ST-segment elevation is a manifestation of an exchange of downward shifts in electrical voltage.

The theory of the systolic current theory explains that the occurrence of ST-elevation is an early repolarization event [20;21;22;23]. Damaged myocardium has a more rapid repolarization pattern that results in repeated electric currents in ischemic areas. The implication of ST elevation is a marker from the ECG, which is very sensitive for the manifestation of AMI (Acute Myocardial Infarction). ST-segment elevation seen in several leads can be used to predict the size of the ischemic area [24;25;26]. The number of patients who come complaining of chest pain and with STEMI ECG results usually have IVCD (Intraventricular Conduction Defect), PVC (Premature Ventricular

Complex), AV Junction and RBBB (Right Bundle Branch Block) pattern. The appearance of defects in normal pacemakers is usually a complication of infarction [27;28;29]. Heart block can develop in people with acute myocardial infarction. It is mainly found in inferior infarction because the right coronary artery supplies the myocardium in that area and the junction network. When heart block is a complication of anterior infarction, mortality is usually high. This block is due to damage to both bundle branches and usually coincides with extensive myocardial damage. A more severe complication of inferior infarction is the occurrence of complete heart block [30;31;32]. Conditions that cause heart block occur in about 5% of people with acute myocardial infarction. It is especially true in inferior infarction because the right coronary artery supplies the myocardium there and the junction network. Grade 1 AV block is of little significance, except as an indication of further blockage. Grade 2 AV block, usually of the Wenkebach type, is potentially dangerous because of the risk of progression to complete AV block or ventricular asystole [33;34;35]. The occurrence of HDAVB (High-Degree AV Block) or complete heart block increases the mortality rate, but on the other hand, it can also be cured quickly by administering thrombolytics or by repositioning through PCI [36;37;38]. Such rhythm and conduction disturbances usually occur in 95% of patients with acute myocardial infarction. Sinus tachycardia is common and is an indicator of the severity of the disease. Sinus bradycardia is expected at the time of acute infarction, is sometimes part of the vasovagal syndrome, primarily associated with inferior myocardial infarction and may also be provoked by morphine or digitalis. Reperfusion therapy that can be given to STEMI patients is reperfusion of areas experiencing ischemia. The existing collateral vessels are also very influential for reperfusion in left ventricular function [39;40;41]. These vessels provide sufficient perfusion for the myocardium to slow cell death and may be of greater importance in patients who undergo reperfusion more than 1-2 hours after coronary occlusion. Although reperfusion has been completed, and no myocardial cells can not recover, there can still be dysfunction in myocardial contractile fibres which is called myocardium stunning [42;43]. Time for reperfusion in myocardial ischemic states is significant. Myocardial oxygen consumption should be minimized by resting the patient physically and emotionally through mild sedatives and adjusting the atmosphere. The use of adrenergic agonists should be avoided. All forms of tachyarrhythmias require proper management because this condition can increase the need for oxygen consumption to the heart muscle. Heart failure should also be treated in a way that minimizes the increase in adrenergic tone and hypoxemia. If the ischemia persists, then the state of severe anaemia (severe anaemia) should be corrected by administering packed red blood cells, accompanied by administration of diuretics if there is left ventricular failure. Other complications that may accompany it are the presence of infection accompanied by tachycardia, fever, which ultimately leads to an increase in the need for oxygen consumption.

CONCLUSION

From the results of research and analysis of data from medical records, it was found that NSTEMI can evolve into STEMI. STEMI conditions require urgent action to be given management, at least by administering nitroglycerin to reduce pain intensity. The presence of pathological Q waves is also an indicator of the occurrence of ischemia in the myocardium, without necrosis. The inverted T wave is also an indicator for diagnosis with prolonged myocardial infarction, epicardial ischemia, and transmural infarction. STEMI must also be distinguished from the occurrence of early repolarization,

electrolyte disorders, such as hyperkalemia. Within 12 hours of starting STEMI, the elevated ST segment could return to baseline. ST-elevation segments were mapped every 2 hours at the time of AMI (Acute Myocardial Infarction). Return of the ST segment to baseline indicates that the cells in the myocardium are dead, which reduces the current of injury manifested by ST elevation.

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