Left Circumflexus Coronary Artery Total Occlusion with Clinical Presentation as NSTEMI and Acute Pulmonary Oedema

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ABSTRAK

Pedoman manajemen pada pasien dengan sindroma koroner akut tergantung pada pembagian diagnosis menjadi infark miokard dengan elevasi segmen ST (IMA-EST) atau infark miokard akut tanpa elevasi segmen ST (IMA-NEST)/angina pektoris tidak stabil (APTS). Pasien dengan IMA-EST seawal mungkin dilakukan terapi reperfusi koroner untuk melisiskan trombus yang oklusif. Elevasi segmen ST merupakan kondisi 'sine qua non' untuk mendiagnosis oklusi total akut pada segmen arteri koroner yang menyebabkan infark miokard transmural. Oklusi total pada arteri circumflexus kiri (LCx) sering dikategorikan sebagai IMA-NEST karena tidak adanya elevasi segmen ST yang bermakna pada sadapan standar elektrokardiogram. Elevasi segmen ST ditemukan kurang dari 50% pada pasien dengan oklusi total LCx, sehingga terapi reperfusi terlambat diberikan. Kami melaporkan seorang wanita berusia 77 tahun yang didiagnosis IMA-NEST dengan gambaran elektrokardiogram 12 sadapan berupa depresi segmen ST di sadapan V2-V5. Pada angiografi koroner ditemukan lesi culprit berupa oklusi total pada LCx.

Kata kunci: *infark miokard dengan elevasi segmen ST (IMA-NEST), lesi culprit, oklusi total, circumflexus kiri.*

ABSTRACT

Current guidelines for the management of patients with acute coronary syndromes (ACSs) focus on the electrocardiogram to divide patients into ST-elevation acute myocardial infarction (STEMI) or non-ST-elevation acute myocardial infarction (NSTEMI)/unstable angina (UA). Patients with STEMI in the earliest time will receive reperfusion therapy to destruct occlusive thrombus. An ST segment elevation is the 'sine qua non' for diagnosing acute total coronary occlusion causing transmural myocardial infarction. Left circumflex coronary artery (LCx) occlusion is often categorized as NSTEMI because of the absence of significant ST-elevation on the 12 lead standard electrocardiogram. An ST segment elevation is presented in fewer than 50% of patients with LCx total occlusion, such that the reperfusion therapy is delayed. We reported a 77 years old woman whom being diagnosed with NSTEMI because a 12 lead electrocardiogram showed ST segment depression in lead V2-V5. On coronary angiography, we found a total occlusion in the LCx artery as the culprit lession.

Keywords: *ST-elevation acute myocardial infarction (STEMI), culprit lession, total occlusion, left circumflex.*

INTRODUCTION

A prompt restoration of blood flow in the infarct-related artery is essential to rescue the myocardium and reduce mortality following ST-elevation acute myocardial infarction (STEMI). Since the benefits of reperfusion therapy decline over time, a prompt and accurate diagnosis of STEMI is very important in determining the initiation of reperfusion therapy.[1] The 12 lead standard electrocardiography (ECG) has been an initial diagnostic tool in patients with suspected AMI presenting in the emergency department (ED) and ideally should be performed and interpreted within 10 minute of arrival to the ED. Based on the ECG recording, patients will be divided into STEMI or non-ST-elevation acute myocardial infarction (NSTEMI)/unstable angina (UA). However, this conventional ECG has a very low sensitivity for detecting STEMI if the culprit lesion is in the left circumflex coronary artery (LCx).[2]

An ST segment elevation is not seen on the 12 lead standard ECG in up to 60% patients in LCx-related AMI. Therefore, the patients will not be cathegorized as having STEMI but NSTEMI instead and can possibly lead to an unwarranted delay of therapeutic decisions especially reperfusion therapy. Because of the lack in ECG presentation, the patients with LCx total occlusion might be underdiagnosed by the physician in the ED. As consequences, the patients with a totally occluded LCx present with less ST-elevation in ECG and the primary PCI as an earliest reperfusion therapy they should be entitled is delayed or performed less as compared with other coronary segment occlusions.[2,3]

CASE ILLUSTRATION

We report a 77-year old woman who came to the Emergency Department (ED) of Dr. Sardjito General Hospital, Yogyakarta complaining of shortness of breath since 6 hours before admission. In the previous day, the patient had a chest pain along with diaphoresis. The pain was non radiating. She complained no dyspnea, nausea nor vomitus. She went to ED in a private hospital. A 12-lead standard ECG was taken; however, we did not have the record. The laboratory test was done and showed normal cardiac enzyme, so she was discharged by the attending physician in the private hospital.

On the day of admission, she came to ED complaining of shortness of breath during rest, but without chest pain. The 12 lead standard ECG showed ST segment depression in lead V2-V5 (**Figure 1**). The laboratory test showed

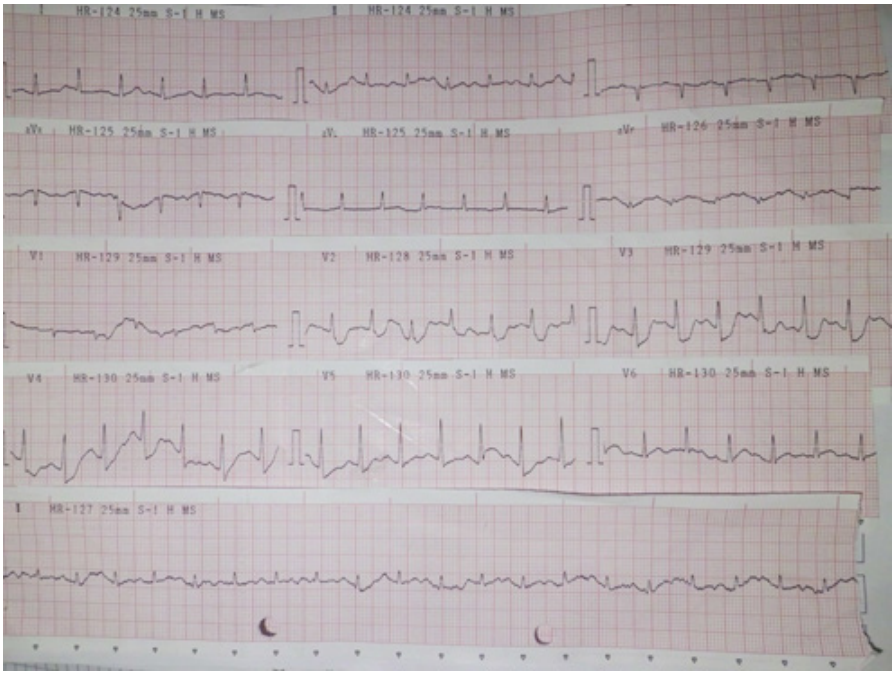

Figure 1. Electrocardiogram on admission showed an ST segment depression in leads V2-V5

increased cardiac enzyme (creatine kinase : 3119 IU/L, creatine kinase MB isoenzymes: 587 IU/L and troponin I: 20,49 IU/L). The patient general condition was weak and slightly somnolence. The blood pressure was increased (190/110 mmHg), pulse rate was 130 times per minute, respiratory rate was 36 times per minute and basal rales was present in both of lung fields.

Patient was diagnosed as NSTEMI, hypertensive emergency and acute pulmonary edema. Due to haemodynamic instability caused by myocardial infarction, patient was tranfered to catheterization laboratory. Coronary angiography was performed and demonstrated a total occlusion in LCx. The totally occluded LCx was the culprit lesion responsible for the current myocardial infarction (**Figure 2**). A drug eluting stent (DES) was implanted into the culprit lesion and the coronary flow was restored completely (TIMI Flow 3) (**Figure 3**). After the procedure the patient was transferred to intensive cardiac care unit (ICCU).

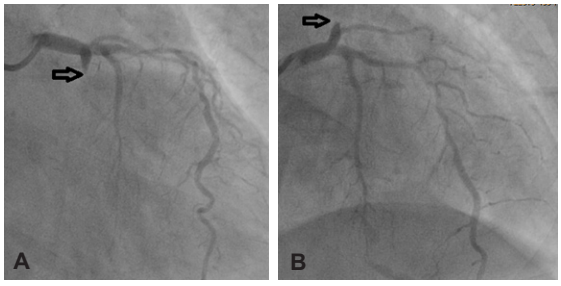

Figure 2. Coronary angiogram showed an LCx total occlussion (arrow). a. RAO 20^0 Caudal 20^0 view and b. RAO 10^0 Cranial 30^0 view

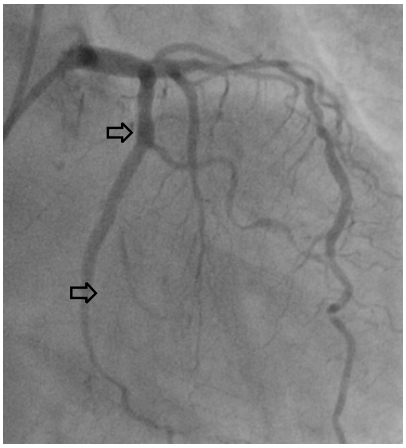

Figure 3. Coronary angiogram post DES insertion showed a flow in the LCx (arrow)

DISCUSSION

The cathegorization of patients with ACS into an ST-segment elevation or non ST-segment elevation is an important step in order to rapidly triage patients candidate for primary reperfusion therapy. Understanding the pathophysiology of STEMI and NSTEMI/UAP gives the basic knowledge of first treatment strategy in patient with ACS.[4] Autopsy studies have shown that plaque rupture causes nearly 75% of fatal myocardial infarction, whereas superficial endothelial erosion was responsible for the remaining 25%.[4] After either plaque rupture or endothelial erosion, the sub endothelial matrix is exposed to the circulating blood and leads to platelet activation and the subsequent formation of a thrombus. Two types of thrombi can be formed, i.e. a platelet-rich clot, or a white clot, that is formed in areas of high shear stress and only partially occludes the artery and a fibrin-rich clot, or a red clot that is the result of an activated coagulation cascade. Red clots are frequently superimposed on white clots and responsible for total occlusion.[5]

The differences in the pathophysiology of STEMI and UA/NSTEMI consequently cause different therapeutic goals and approaches. In UA/NSTEMI, the goal of antithrombotic therapy is to prevent further thrombosis and to allow endogenous fibrinolysis dissolving the thrombus. Revascularization in UA/NSTEMI is often required to increase blood flow and prevent reocclusion or recurrent ischemia. In contrast, in STEMI, the infarct-related artery is usually totally occluded. Immediate pharmacological or catheter-based reperfusion is the initial approach to restore normal coronary blood flow.[4-6]

Total oclussion of coronary artery is associated with STEMI; however, total occlusion can also be found in NSTEMI. Apps et al.[6] reported a total oclussion was found in 75% ACS patients presenting with ST elevation, 73% in patient with ST depression or T invertion and 63% in ACS patients without any ST-T changes.

In our case, coronary angiogram showed total occlusion ing the LCx. Approximately, 48% of patients with total occlusion in LCx present with ST-segment elevation on ECG recording and 30% have no significant ST-T changes.[7]

According to management guideline for ACS, the latter will be treated inappropriately as NSTEACS without having primary percutaneous coronary intervention or receiving fibrinolytic therapy at earliest time.[3]

Data from several reports showed that LCx is the least frequent culprit artery.[2,7] Failure to detect LCx related AMI may have great consequences because LCx supplies significant area in the left ventricle.[8] An LCx supplies the inferobasal area of the myocardium. In the ECG, the posterior leads, reflecting the basal part of the left ventricle wall which lies on the diaphragm, can be easily detected by leads V7–V9 in the back. Because the anterior ECG leads are relatively in the opposite direction of the inferobasal leads, an anterior ST depression is often the mirror image of an inferobasal ST elevation. None of the 12 standard ECG leads reflect the inferobasal wall, therefore an isolated inferobasal STEMI often masquerades as a NSTEMI.[9]

In our case, the patient was diagnosed as NSTEMI based on ECG recording which showed ST segment depression in leads V2-V5. It was possible that the ECG in the previous hospital showed normal ECG, such that the previous physician didn't diagnose as ACS and managed the patient based on ACS guideline.

The lack of ECG presentation in patients with LCx-related AMI is multi-factorial. One possible reason of the absence of ST segment changes is due to smaller infarct size. A previous study showed total mass of myocardium lost in LCx-related AMI is smaller than in other anatomic distributions, notably anterior MI. Infarct size could be estimated by the amount of serum cardiac marker released and ejection fraction.[2] However, our case had increased cardiac marker and acute pulmonary edema which reflected low ejection fraction. The LCx usually supplies the lateral and posterior walls of the left ventricle, which are not well detected by the 12 standard ECG leads. There were some studies suggested that patients without ST segment changes were likely due to incomplete coronary occlusion from thrombus or vasospasm.[2] Again, our case confirmed the total LCx occlusion in coronary angiography. The coronary artery dominance may also obscure the ECG finding in LCx-related AMI. Right coronary dominance may act as a protective factor in acute occlussion of LCx by giving colateral or dual flow and minimize infarcted area which cause minimal changes in ECG recording.[2]

The additional chest lead ECG recording is not routine in ACS. The ESC guideline recommends to record additional ECG leads, i.e. V3R, V4R and V7–V9, when routine 12 lead ECG are inconclusive.[10] It is recommended to record the V7-V9 leads to diagnose inferobasal or posterior STEMI.[10] However, in our case, no posteror ECG was recorded at time of admission.

The management of patient with NSTEMI based on the risk stratification. Invasive management is recommended when the patient has high risk profiles. In our case, invasive management was performed due to acute pulmonary edema and haemodinamic instability. In coronary arteriography, we found the total occlusion in LCx which was a culprit lesion and subsequently restored the coronary flow by implanting DES in the culprit vessel.

CONCLUSION

We reported a 77-year old woman with NSTEMI based on clinical presentation, the 12 lead standard ECG recording and cardiac enzyme, but in the coronary angiography we found a total occlussion of LCx as the culprit lesion. The 12 lead standard ECG does not adequate to diagnose LCx-related AMI, therefore the physician must be aware of this condition and rule out the LCx when there is no ECG changes in patient suspected with ACS.

ACKNOWLEDGMENTS

The authors would like to thank Anggoro Budi Hartopo, MD, PhD for the assistance to conduct this case report.

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