NSTEMI Presenting with Acute Pulmonary Edema with Culprit Lession Total Occluced Left Circumfl ex: a case report

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Abstract

Current guidelines for the management of patients with acute coronary syndromes (ACSs) focus on the ECG to dichotomize patients into having ST elevation myocardial infarction (STEMI) or non-ST elevation myocardial infarction (NSTEMI)/ Unstable Angina (UA) in order to rapidly triage patients to receive reperfusion therapy. Left circumflex artery occlusion is often categorized as NSTEMI because of the absence of significant ST elevation on the 12 standard ECG leads. ST elevation is the condition 'sine qua non' for diagnosing acute total coronary occlusion causing transmural infarction. However, ST elevation when there is circumflex artery occlusion is seen on the 12 standard ECG leads in fewer than 50% of patients. We reported a 77 years old women who diagnosed with NSTEMI. Twelve lead ECG showed ST depressed in V2-V5. On angiography we found a totaly ocluded of left circumflex as culprit lession.

Keywords: NSTEMI; culprit lession; total occlusion; Left Circumflex

Introduction

Prompt restoration of blood flow in the infarct-related artery is essential to myocardial salvage and mortality reduction. As the benefits of reperfusion decline rapidly with time, prompt and accurate diagnosis of acute myocardial infarction (AMI) is very important in determining the initiation of reperfusion therapy[4]. The 12 standard ECG leads has been an initial diagnostic tool in patients with suspected AMI in the emergency department (ED) and ideally should be performed and interpreted within 10 min of arrival to the ED. However, this conventional ECG has a very low sensitivity for the detection of AMI, especially if the culprit lesion is in the left circumflex artery (LCx)[5].

ST elevation was not seen on the 12 standard ECG leads in up to 60% patients in LCx-related AMI and this could possibly lead to an unwarranted delay of therapeutic decisions. Because of lack of ECG presentation, these patients with LCx occlusion might be underdiagnosed by the physician in the emergency department.. It seems that the patients with an occluded LCx presented with less ST elevation, and their primary PCI was delayed or performed less[5,8].

Case Illustration

We reported a 77 old woman who came to ED Sardjito General Hospital complaining of short of breath (dyspnea) since 6 hours before. One day before, patient has chest pain along with diaphoresis, no dyspnea, nausea or vomitus. She went to ER in a private hospital. Standard 12-lead ECG was taken, unfortunately, we didn't have the record. The laboratory test showed normal cardiac enzyme, so she was discharged. One day after, she came to ER complaining of dyspnea but no chest pain. The 12 lead ECG showed ST depressed in lead V2-V5 (figure 1). The laboratory test showed increased cardiac enzyme (CK: 3119 IU/L, CKMB: 587 IU/L and Troponin I: 20.49 IU/L). Patient was unconciousness, blood pressure was increased (190/110 mmHg) and rales in both of lung on physical examination.

Patient was diagnoses as NSTEMI, hypertensive emergency and acute pulmonary edema. Then, patient was tranfered to catheterization laboratory. Coronary angiography demonstrated totaly occludded of LCx as culprit lession (figure 2). DES was inserted and patient was transferred to intensive cardiac care unit (ICCU).

Discussion

Dichotomize patient with acute coronary sindrome into having ST elevation or not in order to rapidly triage patients to receive reperfusion therapy. Understanding of pathophysiology of UAP/ NSTEACS and STEACS give the basic of first

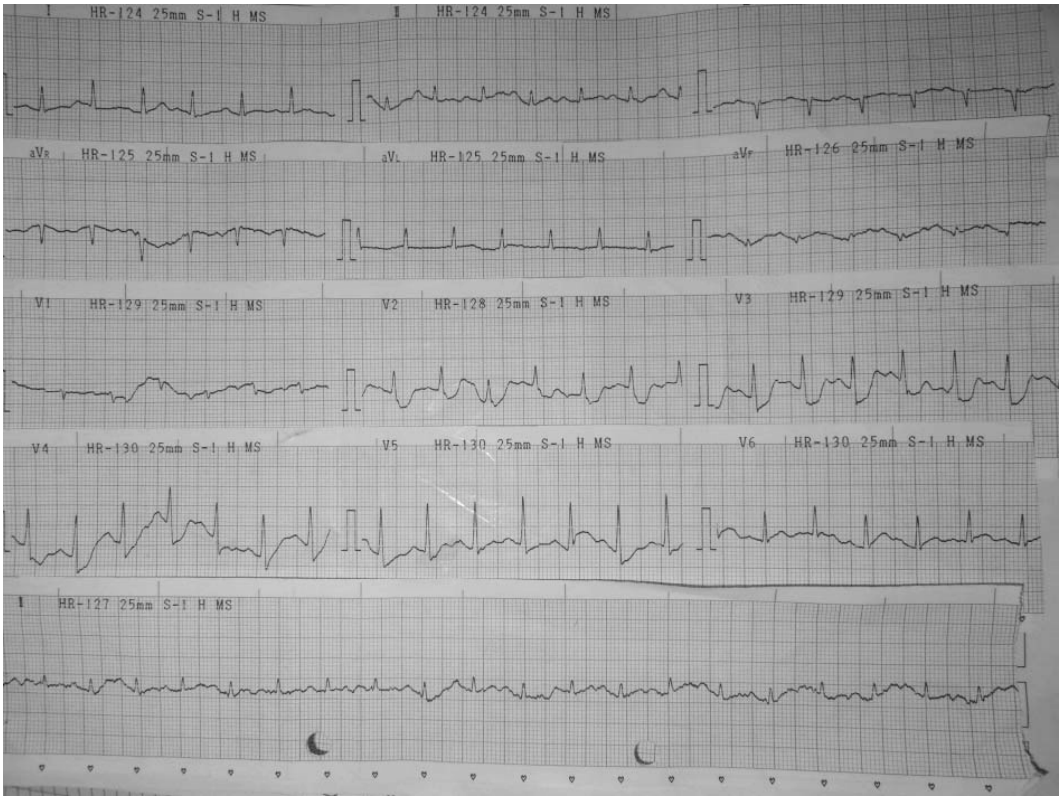

FIGURE 1. ECG recording on admission show ST depression in V2-V5

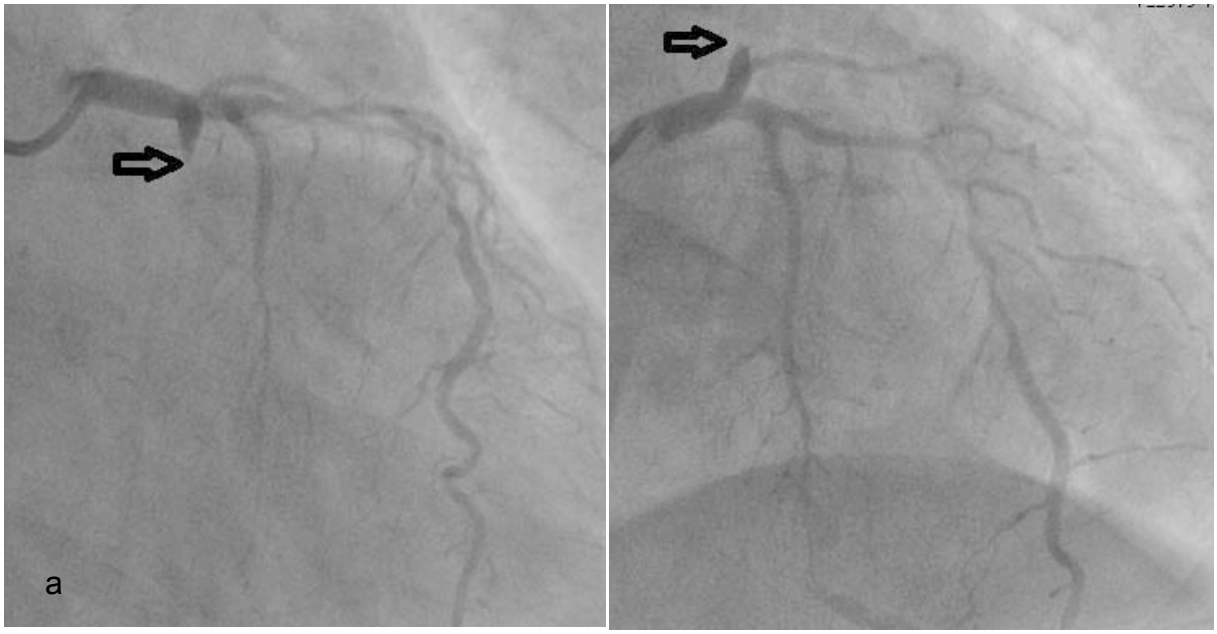

FIGURE 2. Coronary angiography show LCx total occlussion (arrow)
a.. RAO 20^0 Caudal 20^0, b. RAO 10^0 Cranial 30^0

treatment patient with ACS. Autopsy studies have shown that plaque rupture causes approximately 75% of fatal MIs, whereas superficial en-dothelial erosion accounts for the remaining 25%. After either plaque rupture or endothelial erosion, the sub-endothelial matrix (which is rich in tissue factor, a potent procoagulant) is exposed to the circulating blood; this exposure leads to platelet adhesion

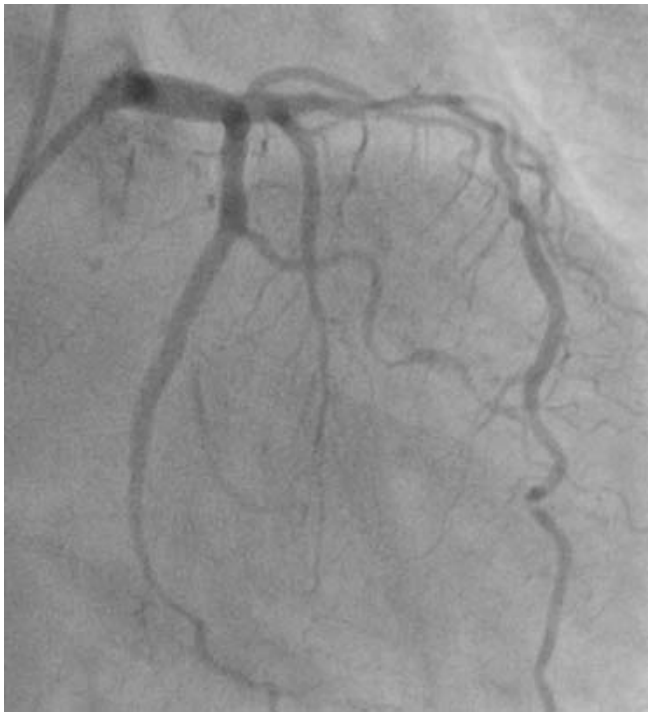

FIGURE 3. Coronary angiography post PCI show flow in LCx (arrow)

followed by platelet activation and aggregation and the subsequent formation of a thrombus. Two types of thrombi can form: a platelet-rich clot (referred to as a white clot) that forms in areas of high shear stress and only partially occludes the artery, or a fibrin-rich clot (referred to as a red clot) that is the result of an activated coagulation cascade and decreased flow in the artery. Red clots are frequently superimposed on white clots, and this characteristic causes total occlusion[1].

The differences in the underlying pathophysiology of UA/ NSTEMI and STEMI call for different therapeutic goals and approaches. In UA/ NSTEMI, the goal of antithrombotic therapy is to prevent further thrombosis and to allow endogenous fibrinolysis to dissolve the thrombus and reduce the degree of coronary stenosis. Revascularization is frequently used to increase blood flow and prevent reocclusion or recurrent ischemia. In contrast, in STEMI, the infarct-related artery is usually totally occluded, and immediate pharmacological or catheter-based reperfusion is the initial approach, with the goal of obtaining normal coronary blood flow[1,6,9]

Total oclussion of coronary artery was assosiated with STEMI, but total occlussion can be found in UAP or NSTEMI. Apps *et al.* in March 2013 published a study which show of 308 patient underwent immediate angiography and primary PCI, total aclussion was found in 75% patient ACS presenting with ST elevation, 73% in patient with ST depressed or T inverted and 63% total oclussion found in patient ACS without any ST-T changes[1].

In this case, coronary angiography showed total oclussion of LCx. Our finding accordingly with study above. But, these case may be a STEMI inferobasal (posterior) which didn not diagnose because absence of ECG posterior lead as additional lead to diagnose this case. Approximatelly 48% total oclussion LCx related AMI has ST elevation on ECG recording, and 30% has not significant ST-T changes[2]. According to algorithm management of ACS, this is potentially patients to be treated inappropriately as having a NSTEACS without having primary percutaneous coronary intervention or receiving early administration of fibrinolytic therapy[8]

Data from randomized clinical trials of STEMI have repeatedly shown that LCx is the least frequent culprit artery. But, failure to detect LCx related AMI have consequences because LCx supply significant area of left ventricel[7]. LCx supplies the inferobasal area of the myocardium.

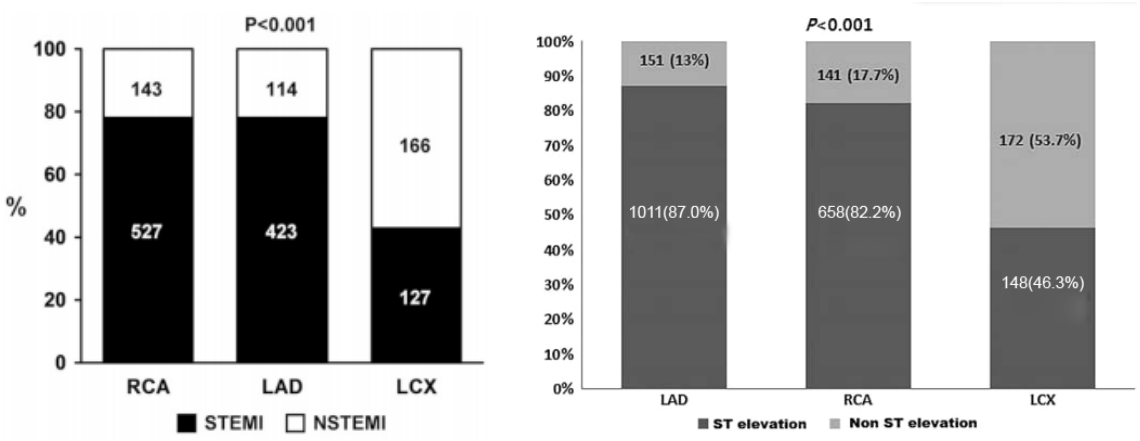

FIGURE 4. Presentation STEMI and NSTEMI due tu culprit lession
a. From *et al*., 2011 b. Kim *et al*., 2011

The term posterior, to reflect the basal part of the left ventricle wall that lies on the diaphragm and it can easily assessed by leads V7–V9. Because the anterior leads are relatively opposite in direction to the inferobasal leads, anterior ST depression is often the mirror image of inferobasal ST elevation. None of the 12 standard ECG leads face the inferobasal wall so an isolated inferobasal STEMI often masquerades as a non-STEMI[10]

In this case, patient was diagnose as NSTEMI based on ECG recoding which showed ST depression in V2-V5. It is possible the ECG records in previous hospital show normal ECG, so the physician didn't diagnose as ACS and patient didn't receive ACS's management.

The lack of ECG presentation in patients with LCx-related AMI is multi-factorial. One possible reason is that absence of ST segment changes was explained by 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 assessed by the amount of serum cardiac marker (creatine kinase-MB, troponin) and ejection fraction[5], but this case have increased cardiac marker and presence of acute pulmonary edema which reflected low ejection fraction. Second, LCx usually supplies the lateral and posterior walls of the left ventricle, which are areas not well detected by the 12 standard ECG leads. Third, there were some trials suggesting that patients without ST segment deviation were likely because of incomplete coronary occlusion due to thrombus or vasospasm[5], but this study confirmed complete LCx occlusion during coronary angiography. Forth, the coronary artery dominance, right coronary dominance may act as protective factor in acute occlussion LCx by giving colateral or dual flow, minimize area of infarc which lead no changes in ECG recording[5]. This finding suggested that this case may have large infarc size due to a STEMI inferobasal (posterior).

Recording the additional chest lead does not include in first routine chart to diagnose ACS. The ECS guideline recommended to record additional ECG leads (V3R, V4R, V7–V9) when routine leads are inconclusive. Wong, 2011, recommend to record V7-V9 to diagnose STEMI inferobasal (posterior). Unfortunatelly, in this case, no posteror ECG was recorded. The management of patient with NSTEMI based on risk stratification. Invasif management was recomennden when patient have high risk (table 1 and 2). In this case, invasif management was performed due to acute pulmonary edema and haemodinamic instability.

Conclusion

We reported a 77 years old women with diagnose as NSTEMI due to the 12 standard ECG leads record, but in the coronary angiography we found total occlussion LCx as culprit lession. The 12 standard ECG leads does not enough to diagnose LCx related AMI, so the physician must notice this condition. The clinician must role LCx when there is no ECG changes in patient suspected ACS.

Dichotomy patient to having ST elevation or not does not reflected having total or non total occlussion culprit lession because total occlussion can also be found in patient with UAP and NSTEMI.

TABLE 1. Criteria for high risk with indication for invasive management[3]

Primary
• Relevant rise or fall in troponin[a] • Dynamic ST- or T-wave changes (symptomatic or silent)
Secondary
• Diabetes mellitus • Renal insufficiency (eGFR <60 mL/min/1.73 m²) • Reduced LV function (ejection fraction <40%) • Early post infarction angina • Recent PCI • Prior CABG • Intermediate to high GRACE risk score (*Table 5*)

TABLE 2. Recommendations for invasive evaluation and revascularization[3]

Recommendations	Class[a]	Level[b]	Ref[c]
An invasive strategy (within 72 h after first presentation) is indicated in patients with: • at least one high-risk criterion (*Table 9*); • recurrent symptoms.	I	A	148
Urgent coronary angiography (<2 h) is recommended in patients at very high ischaemic risk (refractory angina, with associated heart failure, life-threatening ventricular arrhythmias, or haemodynamic instability).	I	C	148, 209
An **early** invasive strategy (<24 h) is recommended in patients with a GRACE score >140 or with at least one primary high-risk criterion.	I	A	212, 215

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