Acute Total Occlusion of the Left Circumflex Coronary Artery Presenting with Non-ST-segment Elevation Myocardial Infarction and Normal Electrocardiogram – A Case Report

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Abstract

BACKGROUND: In this case report, we report a patient with non-ST-segment elevation myocardial infarction (NSTEMI), presenting with recurrent chest pain typical of angina, a very high troponin I level despite normal electrocardiogram (ECG). On angiography, it turns out that the patient has acute total occlusion in the left circumflex artery (LCX).

CASE REPORT: A 56-year-old woman presented to the emergency department with chief complaint of recurrent chest pain typical of angina 20 h before admission. Vital signs were within normal limit. There were no murmur, additional heart sounds, and no rales or crackles. The ECG showed normal sinus rhythm, and there were no ST-T changes on serial examination. The first and second cardiac enzymes troponin I was high (>10 mg/L). Chest X-ray examination showed cardiomegaly without signs of lung edema. Patient was diagnosed with high-risk NSTEMI, hypertensive heart disease, and diabetes mellitus. Coronary showed an acute total occlusion in the LCX, which is determined as the culprit lesion for the ongoing myocardial infarction. A drug-eluting stent was deployed at the culprit lesion and the coronary flow was TIMI flow 3. There was non-significant stenosis at the mid-right coronary artery. The echocardiography showed reduced left ventricular systolic function (LVEF 50%) with hypokinetic inferior-septal and inferior-lateral segment base to apical. Post-procedural follow-up was uneventful.

CONCLUSION: One of the learning points is that ECG may fail to detect acute total occlusion and rise in troponin level, despite the absence of ST-T changes, warrant urgent invasive strategy.

Case Presentation

A 56-year-old woman presented to the emergency department with a chief complaint of recurrent chest pain since 20 h before admission. The pain radiated to back and jaw, and accompanied with heavy sweating. There was no history of dyspnea on effort, paroxysmal nocturnal dyspnea, orthopnea, or ankle edema. The patient had history of uncontrolled hypertension since 4 years before admission and diabetes. There was no history of smoking. The patient was no longer menstruating.

In the emergency department, she was moderately ill, fully alert, and had no dyspnea with recurrent chest pain. Initial examination showed normal vital signs, no rales or crackles. The ECG showed normal sinus rhythm, and there were no ST-T changes on serial examination. The first and second cardiac enzymes troponin I was high (>10 mg/L). Chest X-ray examination showed cardiomegaly without signs of lung edema. Patient was diagnosed with high-risk NSTEMI, hypertensive heart disease, and diabetes mellitus. Coronary showed an acute total occlusion in the LCX, which is determined as the culprit lesion for the ongoing myocardial infarction. A drug-eluting stent was deployed at the culprit lesion and the coronary flow was TIMI flow 3. There was non-significant stenosis at the mid-right coronary artery. The echocardiography showed reduced left ventricular systolic function (LVEF 50%) with hypokinetic inferior-septal and inferior-lateral segment base to apical. Post-procedural follow-up was uneventful.

CONCLUSION: One of the learning points is that ECG may fail to detect acute total occlusion and rise in troponin level, despite the absence of ST-T changes, warrant urgent invasive strategy.
moderate work of breathing. The blood pressure was 130/80 mmHg, heart rate 96x/min of resting heart rate, regularly and equal, respiratory rate of 20 x/minute, and with normal temperature, oxygen saturation was 99% on room air. There were no signs of heart failure on physical examination. There were no murmur, additional heart sounds, and no rales or crackles.

The electrocardiography (ECG) showed normal sinus rhythm, normal axis, and no ST-segment elevation in any lead, including RV lead and posterior, there were no ST-T changes on serial examination (Figure 1). Routine blood examination, ureum, creatinine, glucose, and electrolyte panel were within normal limits. The first and second cardiac enzymes troponin I was high (>10 mg/L). Chest X-ray examination showed cardiomegaly without signs of lung edema (Figure 2).

The patient was diagnosed with high-risk NSTEMI, hypertensive heart disease, and diabetes mellitus. Coronary showed an acute total occlusion in the LCx, which is determined as the culprit lesion for the ongoing myocardial infarction (Figure 2a). A drug-eluting stent (DES) was deployed at the culprit lesion, and the coronary flow was restored completely (TIMI Flow 3) (Figure 2b). There was non-significant stenosis at mid-right coronary artery (Figure 2c). After the procedure, the patient was transferred to the intensive cardiac care unit (ICCU).

The echocardiography showed normal all chambers with concentric left ventricle hypertrophy, reduced left ventricle systolic function (LVEF 50% biplane Simpson’s) with hypokinetic inferior-septal and inferior-lateral segment base to apical, grade I Diastolic dysfunction, normal valves, low probability of PH, normal RV contractility. Post-procedural follow-up was uneventful, the patient was hemodynamically stable, and was discharged with good condition.

Discussion

This case report showed NSTEMI patient with chest pain typical of angina and a very high troponin I level despite normal ECG. On angiography, it turns out that the patient has acute total occlusion in the LCx. Thus, ECG may fail to detect acute total occlusion and rise-and-fall in troponin level, despite the absence of ST-T changes in normal and additional leads, warrant urgent invasive strategy.

There are numerous possible causes of unequivocal ECG in LCx-related AMI. The first possible reason is due to the small infarct size. A previous study showed that a 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 decreased regional wall motion contractility [4]. However, our patient had significantly increased cardiac marker troponin I > 10 mg/dl. It was possible that the infarct size was large, as evidenced by the elevated cardiac markers; however, it is ambiguous due to normal ECG findings. Several studies showed that patients without ST-segment changes were likely due to incomplete coronary occlusion due to thrombus or vasospasm [4]. However, coronary angiography suggests the absence of vasospasm or incomplete coronary occlusion. 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 occlusion of LCx by giving collateral or dual flow and minimize infarcted area which causes minimal changes in ECG recording [4]. Coronary angiography showed right dominance in our patient, the lateral posterior segment of myocardium in our patient might be protected by the right dominant coronary
Left circumflex artery-related acute myocardial infarction has been known to be underdiagnosed with 12-lead ECG. Kim et al. conducted a study that focuses on the prognosis of occlusion in the three coronary vessels. There was no significant statistical difference among the three vessels in terms of in-hospital mortality. Multivariate analysis showed primary PCI decreased hospital mortality in patients with occluded coronary artery. In conclusion, LCx occlusion with normal ECG must be treated similarly to occlusion of other coronary arteries.

Conclusion

In this case report, we report a patient with NSTEMI, having chest pain typical of angina, a very high troponin I level despite normal ECG. One of the main reasons for not ST-segment elevation is that LCx lies in the true posterior left ventricle region, causing difficulty in detecting ischemia due to air in posterior mediastinum, a poor conductor of electricity. On angiography, it turns out that the patient has acute total occlusion in the LCx. One of the learning points is that ECG may fail to detect acute total occlusion and rise in troponin level, despite the absence of ST-T changes, warrant an urgent invasive strategy.

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