



Acute inferior myocardial infarction caused by left anterior descending artery occlusion.

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ABSTRACT

INTRODUCTION: Acute myocardial infarction with ST segment elevation in the inferior leads suggests an acute occlusion of the right coronary artery (RCA). Occlusion of the left anterior descending artery (LAD) usually causes ST segment elevation in the precordial leads and reciprocal ST segment depression in the inferior leads. But ST segment elevation in the inferior leads due to occlusion of the LAD is uncommon.

MATERIAL AND METHODS: It's the clinical case of 60-year-old previously healthy man presented inferior STEMI. Physical examination, urgent coronarography, some laboratory and instrumental tests were performed.

RESULTS: A 60-year-old previously healthy man presented to an outpatient clinic with complaints of intensive burning retrosternal chest pain with irradiation to the left hand that had started half an hour before. ECG revealed ST segment elevation in leads II, III, aVR, aVF, V4-V6 and reciprocal ST segment depression in the lead aVL. Coronary angiography was performed: there was left coronary artery dominance, RCA was intact, subocclusion of the proximal LAD was found; "slow flow" phenomenon was noted. A drug-eluting stent was introduced into the proximal LAD.

CONCLUSIONS: Inferior acute myocardial infarction can be caused by untypical branch of coronary artery and it's belongs to rare clinical cases.

KEY WORDS: myocardial infarction, STEMI, coronarography, electrocardiogram.

INTRODUCTION

Acute myocardial infarction with ST segment elevation in the inferior leads suggests an acute occlusion of the right coronary artery (RCA) [1]. Occlusion of the left anterior descending artery (LAD) usually causes ST segment elevation in the precordial leads and reciprocal ST segment depression in the inferior leads [2]. But ST segment elevation in the inferior leads due to occlusion of the LAD is uncommon [3]. A case of ST segment elevation in leads II, III, aVR, aVF, V4-V6 caused by subocclusion of the proximal LAD is described.

CASE PRESENTATION

A 60-year-old previously healthy man presented to an outpatient clinic with complaints of intensive burning retrosternal chest pain with irradiation to the left hand that had started half an hour before. The patient had suffered from arterial hypertension for the last three years; he had neither taken anti-hypertensive drugs nor visited a doctor for this time. So, the target values of blood pressure have not been reached. He is a smoker (about 20 pack-years). ECG revealed ST segment elevation in leads II, III, aVR, aVF, V4-V6 and reciprocal ST segment depression in the lead aVL (Figure 1).

On physical examination, his blood pressure was 130/76 mm Hg; his pulse was 88 bpm. An acute coronary syndrome was diagnosed. Morphine, aspirin, clopidogrel, rosuvastatin, metoclopramide, and enoxaparin were given. The patient was urgently transferred to the nearest PCI-capable hospital. Time from the beginning of chest pain to the moment of coronary angiography was approximately 3 hours. Coronary angiography was performed (Figure 2): there was left coronary artery dominance, RCA was intact, subocclusion of the proximal LAD was found; "slow flow" phenomenon was noted. A drug-eluting stent was introduced into the proximal LAD. Control coronary angiography showed complete restoration of the coronary blood flow.

On the third day after admission, all laboratory tests were normal, except following ones: total cholesterol – 8.5 mmol/L, LDL – 6.15 mmol/L, blood urea nitrogen – 9.1 mmol/L, creatinine – 196.1 μmol/L, bilirubin – 25.5 μmol/L, AST – 134.8 IU/L, leukocytes – $10.9 \times 10^9/L$, banded neutrophils – 7%, segmented neutrophils – 85%, lymphocytes – 5%.

ECG on the seventh day after PCI (Figure 3) showed ectopic atrial rhythm with T wave inversion in leads III and aVF. Low amplitude of the T wave was in leads II and V6. ECG on the ninth day after PCI (Figure 4) revealed sinus bradycardia and pathological Q waves in the inferior leads.

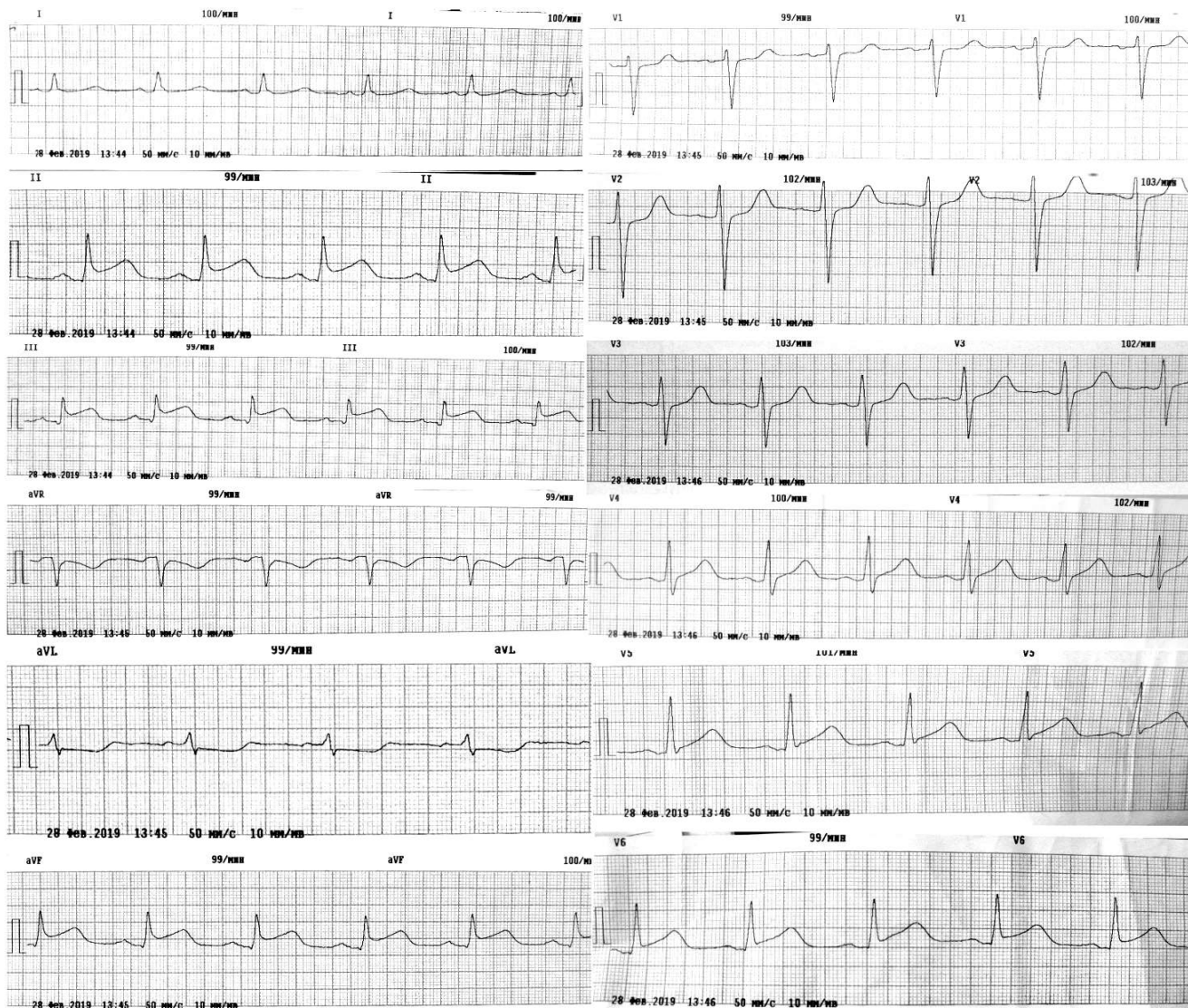


Figure 1. Single-lead ECG performed one hour after chest pain onset. ST segment elevation in leads II, III, aVF, V5-V6; ST segment depression in leads aVR, aVL.

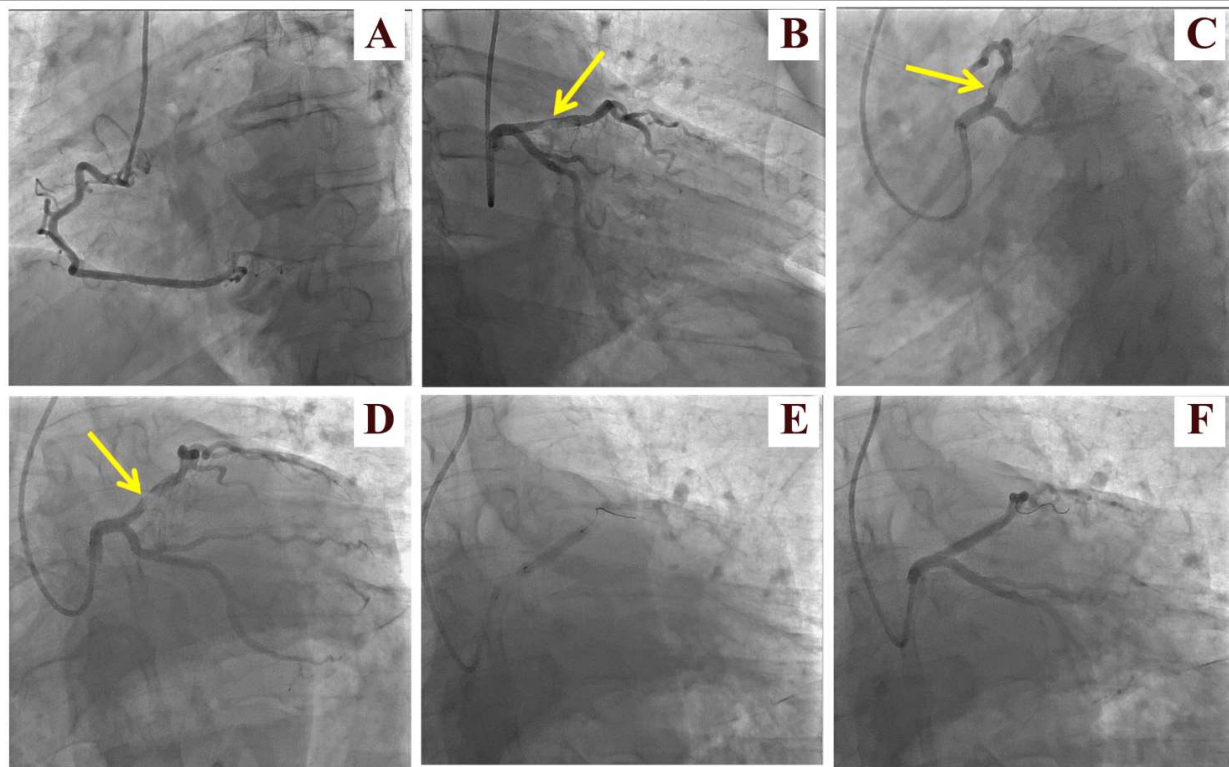


Figure 2. Coronary angiography and ad hoc PCI performed 3 hours after chest pain onset:
 A –RCA, no significant stenoses; B – D – subocclusion of proximal LAD (arrowed);
 E – PCI, balloon inflation; F – restored coronary blood flow.



Figure 3. ECG performed on the seventh day after admission. Ectopic atrial rhythm.
 Inverted T waves in leads III, aVF.

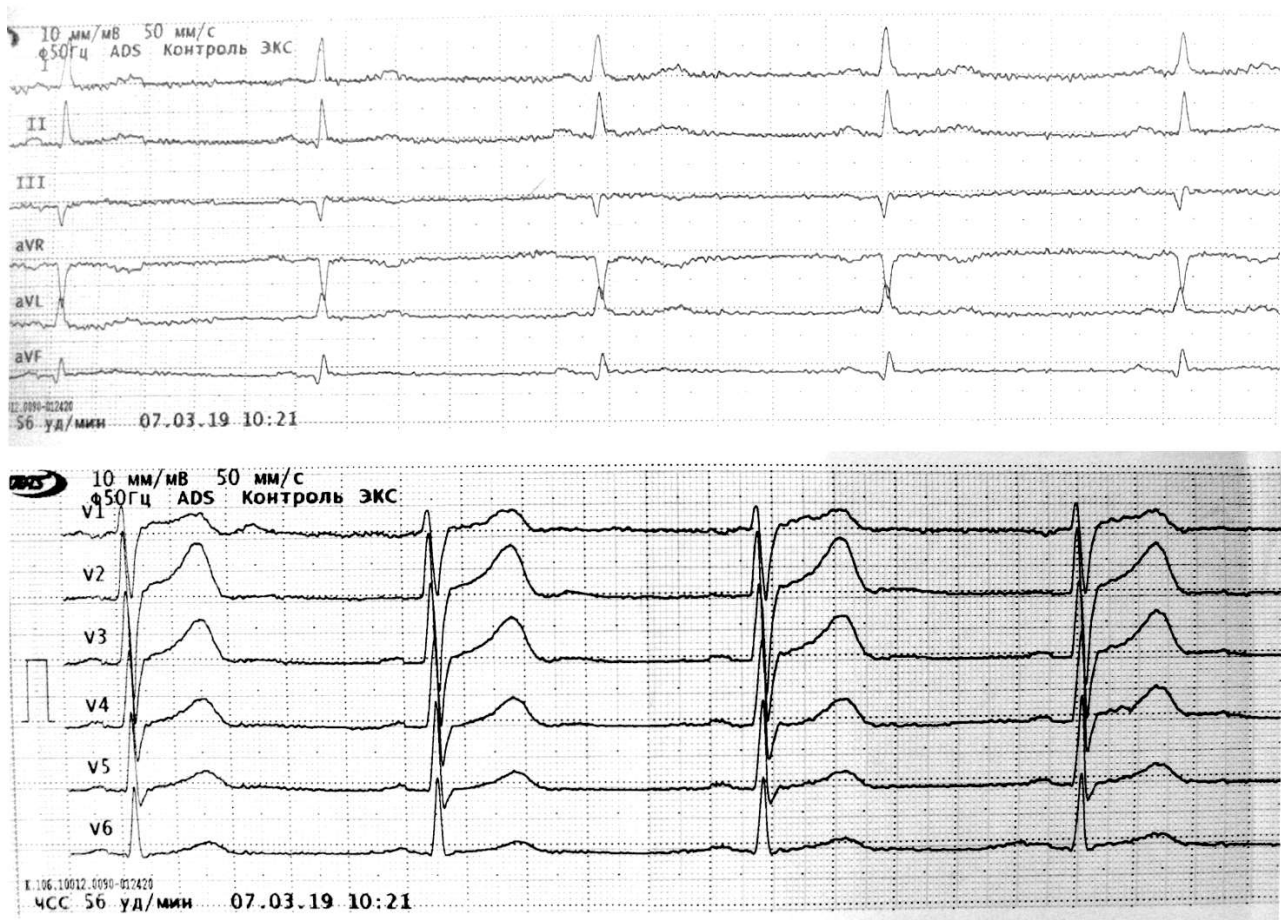


Figure 4. ECG performed on the ninth day after admission. Sinus bradycardia. Pathological Q waves and flat T waves in leads III and aVF.

DISCUSSION

Acute inferior myocardial infarction usually is caused by occlusion of the RCA, and rarely it's due to the occlusion of the left circumflex coronary artery [2]. Inferior STEMI due to occlusion of the LAD is uncommon. Distally occluded LAD that wraps around the apex may lead to ST segment elevation in the inferior leads [4]. In such cases two conditions should be present: a relatively small mass of ischemic myocardium of the anterior wall, resulting in a less amplitude of reciprocal ST segment depression in the inferior leads and concomitant transmural ischemia of the inferior wall of the left ventricle causing shift of ST segment upward in the inferior leads [1]. Cases of simultaneous anterior and inferior myocardial infarction due to occlusion of the wrapped LAD are described [5]. But in our case, the LAD is damaged in the proximal segment. It's difficult to explain such ECG changes. But it's possible, that such ECG changes

are due to relatively more significant ischemia of the infero-lateral wall of the left ventricle, as myocardium of this wall is supplied with blood in the last turn; this is the most distant part of myocardium supplied by the LAD; “slow flow” phenomenon may play a significant role in this state. So, ischemia occurs there earlier than in other parts of myocardium. Perhaps, in this case, ST segment elevation would have spread to other precordial leads, if PCI had been delayed.

CONCLUSIONS

Thus, inferior acute myocardial infarction can be caused by untypical branch of coronary artery and it's belongs to rare clinical cases.

Disclosure statement

No potential conflict of interest was reported by the author's.

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