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SHORT REPORT

Simultaneous Anterior and Inferior Myocardial Infarction Due To Occlusion of the Left Anterior Descending Coronary Artery

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Acute occlusion of the left anterior descending artery (LAD) generally results in ST-segment elevations in leads V1-V6 and reciprocal ST-segment depressions in leads II, III and aVF (1). There are several reports about the clinical importance, prevalence and causes of simultaneous anterior and inferior myocardial infarction due to occlusion of the LAD (2). Here we report a patient with acute anterior and inferior ST segment elevation due to LAD occlusion.

Case Report

A 30-year-old previously healthy male was referred to our emergency department with severe crushing chest pain of 2 h onset. He had been smoking 3 packets of cigarettes a day for 15 years. He did not have any other risk factors for coronary artery disease. Initial ECG demonstrated ST-segment elevations up to 2 mm in leads II, III, aVF and V1-V6 (Figure 1a). On physical examination, blood pressure was 110/80 mmHg, and heart rate was 110/min regular. There were no abnormal findings on cardiac or other systemic examinations.

The patient was put on aspirin 300 mg (P.O.), clopidogrel 300 mg (P.O.) loading dose and also 10,000 U bolus I.V. heparin followed 1000 U/h infusion intravenously. Coronary angiography showed a critical thrombotic lesion located in the proximal LAD. The first diagonal branch (D1) had TIMI-III flow, whereas the distal part of the LAD had TIMI-I-II flow (Figure 1b). The right coronary artery (RCA) and the left circumflex artery (LCA) were normal. The LAD was wrapped around the apex, which caused both anterior and inferior ST-segment elevations. Primary direct stenting was performed (Ephesus, 3.0-12 mm, Medistar, Turkey), and TIMI-III flow was successfully achieved. His chest pain disappeared and ST-segment elevations returned to isoelectric line and Q waves evaluated in leads V1-V4, but ST-segment elevations in inferior leads persisted due to distal LAD occlusion (Figures 2a and 2b). We could not manage to pass a 0.014" guide wire through the occlusion site. Heparin infusion was followed 1000 U/h intravenously and tirofiban infusion 0.1µg/kg/min after a 30 min loading dose of 0.4 µg/kg/min was started. We maintained the activated clotting time between 300 to 350 s during the infusion of heparin and tirofiban.

All the biochemical and hematological tests including total cholesterol, HDL, LDL and triglyceride levels were normal. Cardiac specific enzymes were elevated 4-5 times above the normal limits in the first 24 h follow-up results. The peak serum troponin-I level following percutaneous coronary intervention (PCI) was 8.5 mg/l. Echocardiography showed mild apical hypokinesia and

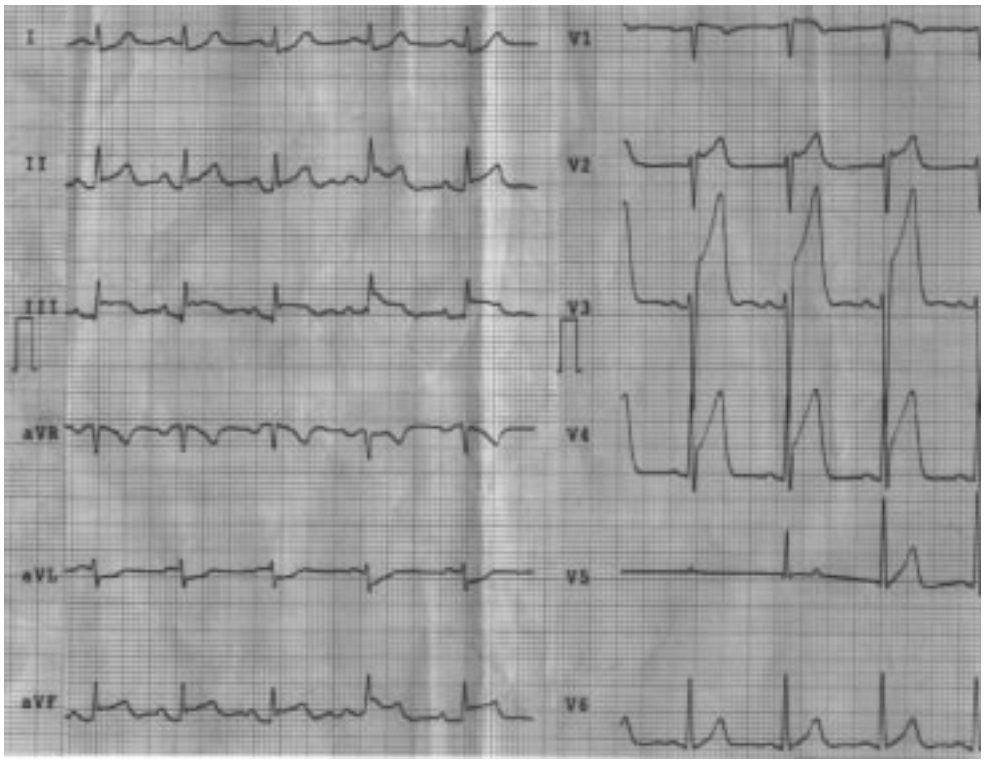


Figure 1a. ECG shows ST-segment elevation in anterior and inferior leads

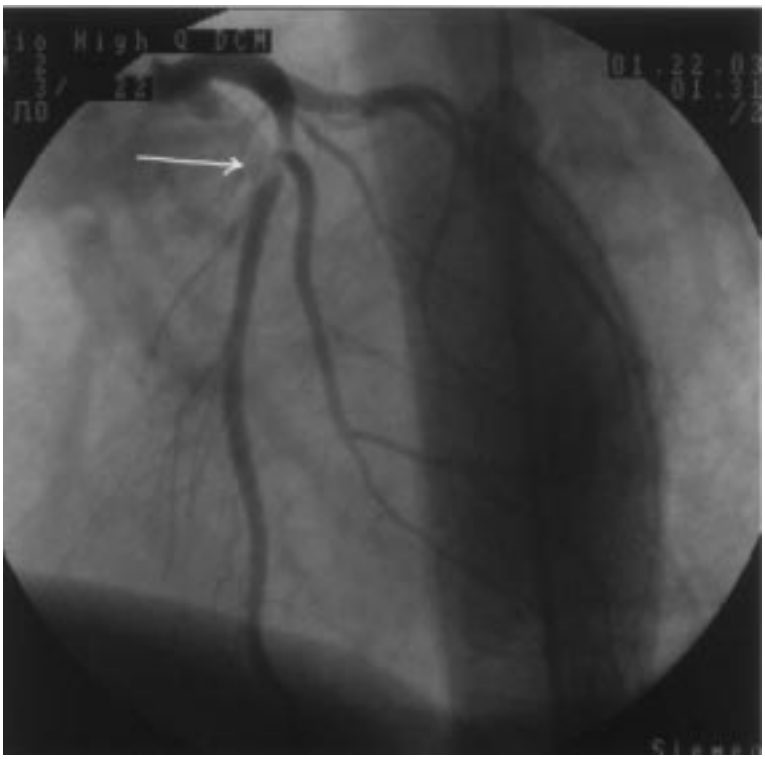


Figure 1b. The arrow shows thrombotic occlusion at proxymal portion of LAD

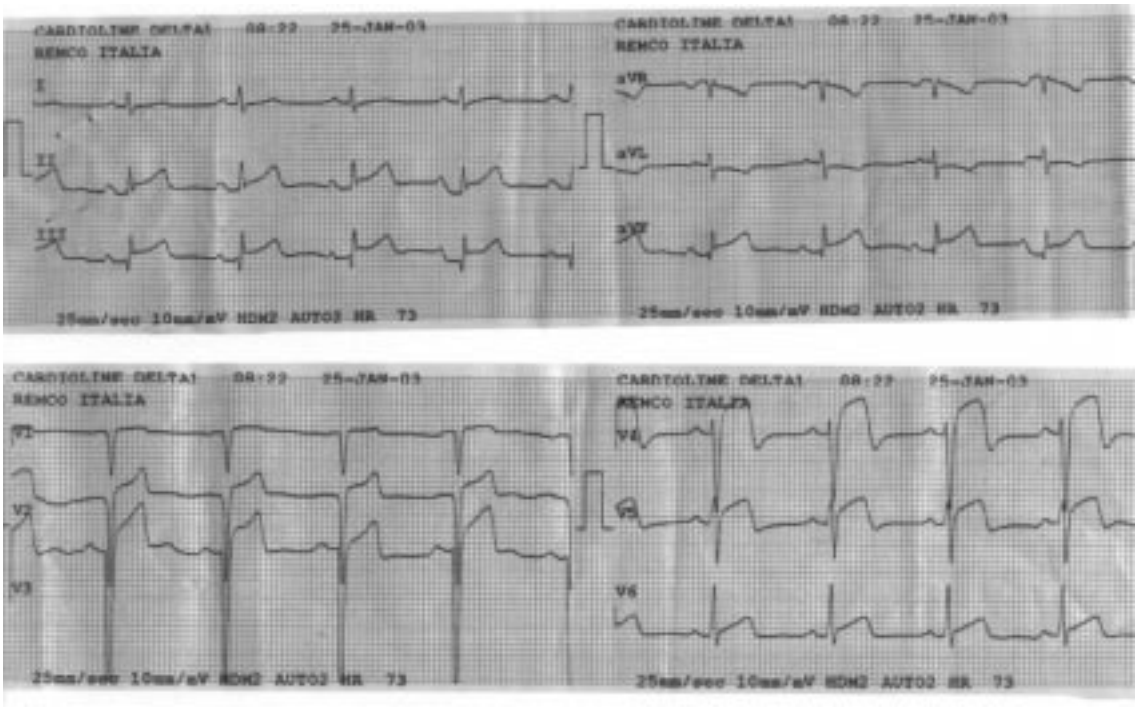


Figure 2a. Persistance of ST – segment elevation in inferior leads after a successful stenting for proximal lesion.

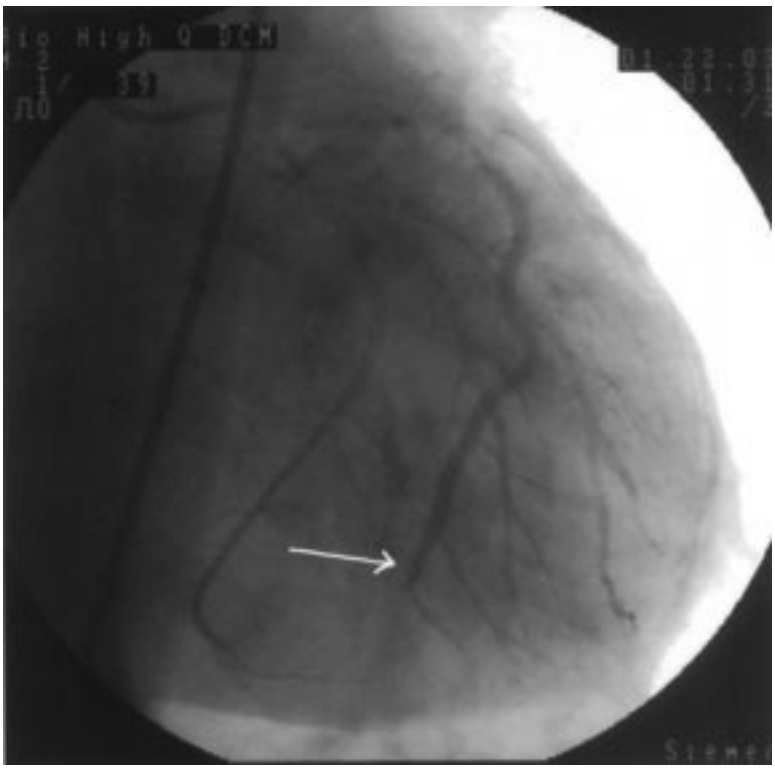


Figure 2b. The arrow shows a second thrombotic occlusion at distal portion of LAD

inferior akinesia and the ejection fraction was 50%. Our patient has been asymptomatic and orally taking aspirin 300 mg/day, clopidogrel 75 mg/day, beta blocker 50 mg/day and simvastatin 10 mg/day for the last 2 months.

The characteristics and distribution of ST segment elevation and accompanying reciprocal ST segment depressions in acute MI allow us to determine the corresponding occluded artery (1,2). In anterior wall acute myocardial infarction, elevation of the ST segment may be seen in the chest leads, and ST-segment depression is sometimes observed simultaneously in the inferior leads. Inferior lead ST-segment depression associated with anterior wall AMI is generally considered to be a reciprocal change of ST-segment elevation in the chest leads. Previous studies have shown that inferior lead ST-segment depression in patients with anterior wall AMI is related to the severity of infarction (3). Recent attention has therefore focused on the relation between inferior lead ST-segment changes in anterior wall AMI and LAD artery morphology. Studies to date suggest that the inferior lead ST-segment changes seen during anterior wall AMI are linked to the size of the anterior wall AMI and the severity of inferoapical wall AMI, determined by the length of the LAD (4). As a general rule, occlusion of LAD causes ST segment elevation in anterior leads. Inferior ST depression is not related to remote ischemia, but represents an electrocardiographic phenomenon reciprocal to ST elevation in aVL; this ST elevation in aVL is related to anterolateral extension of the infarct secondary to D1 branch ischemia. If ST segment elevation occurs more prominently in V2-V3 and if it is accompanied by ST segment depression in leads aVL and D1 this suggests with 99% sensitivity that the infarction related artery is LAD and the occlusion site is proximal to D1(2).

Several studies have investigated the cause and clinical significance of simultaneous anterior and inferior ST-segment elevation in the setting of anterior AMI (1-4). Sapin et al. found that simultaneous anterior and inferior ST-segment elevation was seen in 7 out of 42 consecutive patients with anterior AMI, concluding that ST-segment changes in the inferior leads seen during anterior wall AMI occur as a result of competition between reciprocal changes caused by infarction of the high lateral wall due to lesions in the proximal LAD, which depress the ST segment, and inferoapical wall AMI due to a wrapped LAD, which elevates the ST segment (2).

Although the simultaneous anterior and inferior ST-segment elevation in the setting of AMI may give an impression of widespread ischemia due to a critical mass of myocardial injury, this ECG pattern frequently results from a relatively distal LAD lesion and therefore suggests a better long-term prognosis (5).

Sasaki et al. evaluated 159 patients with occluded LAD and found that 25 patients had simultaneous ST elevation in anterior and inferior leads (6). They have defined "wrapped LAD" as an LAD from a post-reperfusion coronary angiogram that perfuses at least one fourth of the inferior wall of the left ventricle in the right anterior oblique projection (Figure 1). There are 4 conditions in cases of LAD occlusion according to the existence of a wrapped LAD: 1) If the patient has a wrapped LAD and the location of the occlusion is proximal to D1, ST segments are elevated in anterior leads and remain within isoelectric lines in inferior leads; 2) If the patient does not have a wrapped LAD and the location of the occlusion is distal to D1, ST is elevated in anterior leads and remains within isoelectric lines in inferior leads; 3) If the patient does not have a wrapped LAD and the location of the occlusion is proximal to D1, ST is elevated in anterior leads and reciprocal ST depressions occur in inferior leads (4); and if the patient has a wrapped LAD and the location of the occlusion is distal to D1, the ST segment is elevated in anterior and inferior leads simultaneously (Figures 3a-e) (6).

Our patient had a wrapped LAD and a culprit lesion just below D1. In a patient with a wrapped LAD, revascularization is thought to resolve ST elevations in both anterior and inferior leads. In our patient, this did not happen due to a second lesion in a distal LAD. Recanalization could not be achieved and normalization of the ST elevation in inferior leads was not possible. In

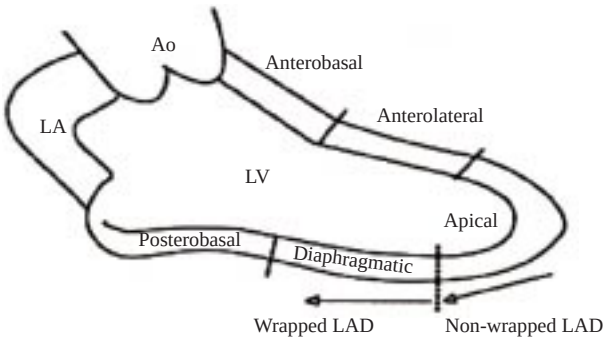
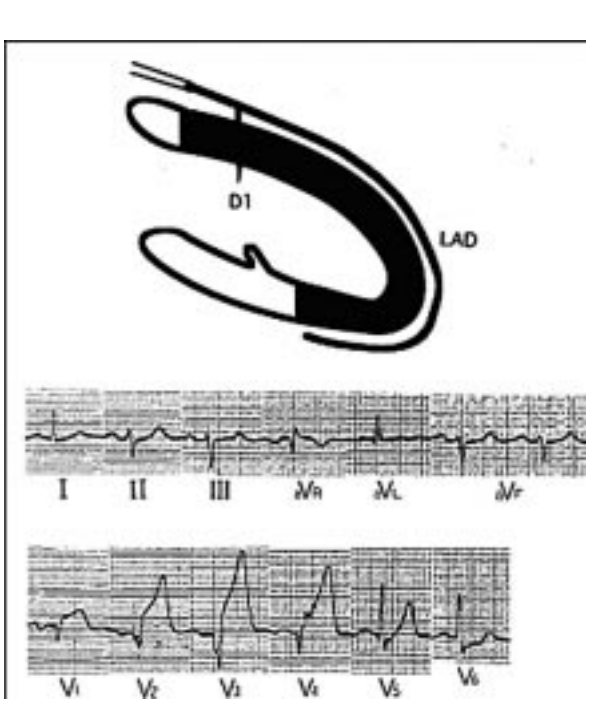
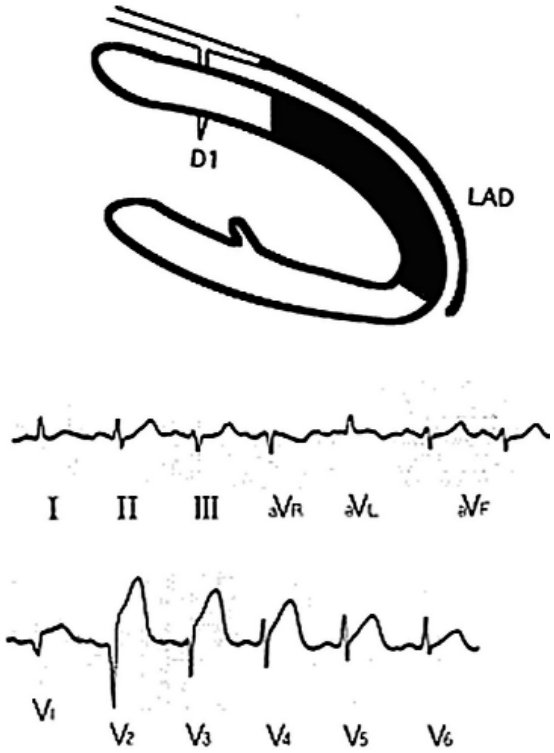
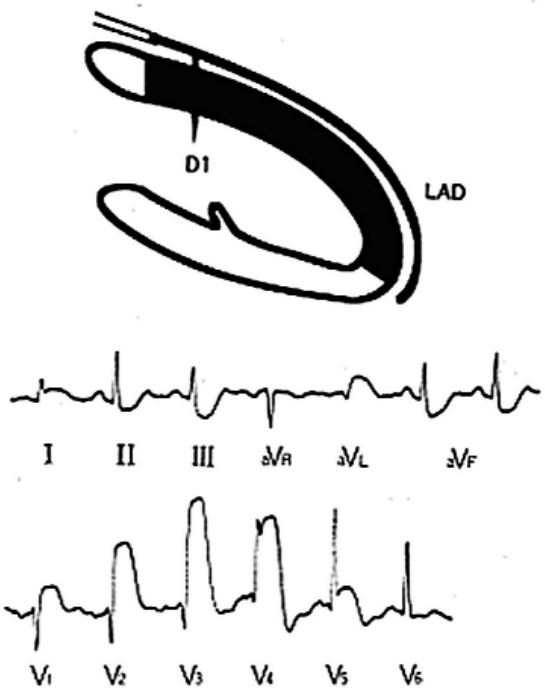


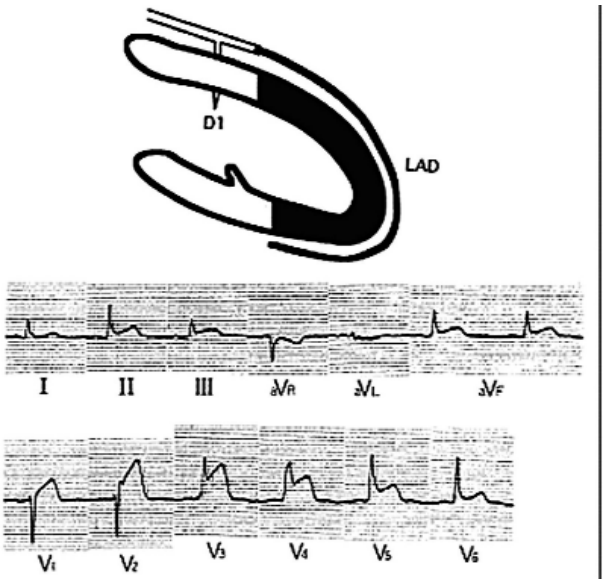
Figure 3a. Schematic demonstration of "wrapped LAD"





Figures 3 b,c. Schematic demonstration of ECG changes in patients with wrapped LAD occluded at proximal to D1 and non-wrapped LAD occluded at distal to D1.





Figures 3d,e. Schematic demonstration of ECG changes in patients with non-wrapped LAD occluded at proximal to D1 and wrapped LAD occluded at distal to D1.

conclusion; proximally located thrombotic occlusion of the LAD excluding D1 may cause simultaneous anterior and inferior ST segment elevation in AMI, which should be taken into account in revascularization procedures.

Authors Comment

Figures 1-3 are reprinted from American Journal of Cardiology, Vol 87: 1340-1345, Sasaki et al "Relation of ST-segment changes in inferior leads during anterior wall acute myocardial infarction to length and occlusion site of the left anterior descending coronary artery" Copyright 2001 Excerpta Medica (by permission).

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