

Is It ST-Segment-Elevation Myocardial Infarction?

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A 76-year-old man presented with acute-onset chest pain and dyspnea. He had a history of coronary artery disease, ischemic cardiomyopathy (left ventricular ejection fraction, 25%), recent non-ST-segment-elevation myocardial infarction (NSTEMI), and stenting of the left anterior descending coronary artery (LAD) one month before the current presentation. Soon thereafter, he underwent stenting of the left main coronary artery (LMCA), obtuse marginal branch (OMB), and left circumflex coronary artery. His chest pain subsided soon after arrival at the emergency department. His cardiac troponin I level was 22 ng/mL (normal level, <0.03 ng/mL), and his brain natriuretic peptide level was 841 pg/mL (normal level, <100 pg/mL). Figure 1 shows his electrocardiogram (ECG).

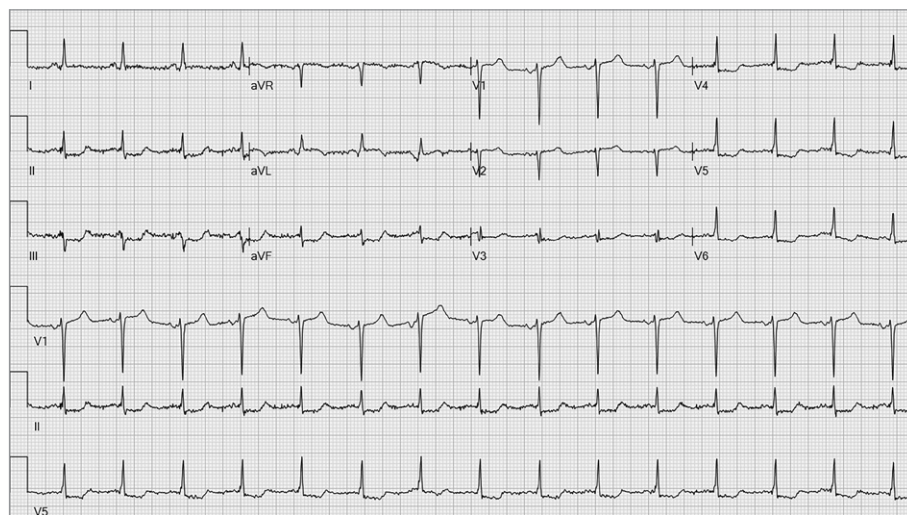


Fig. 1

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What is the diagnosis?

- A) NSTEMI due to LMCA stent thrombosis
- B) ST-segment-elevation myocardial infarction (STEMI) due to proximal LAD occlusion
- C) STEMI due to first OMB or ramus intermedius occlusion
- D) Takotsubo cardiomyopathy
- E) Ischemic cardiomyopathy without specific ECG changes

See next page for the answer.

FOCUS on ECGs: Answer #28

C) STEMI due to first OMB or ramus intermedius occlusion

The ECG shows sinus rhythm, mild ST-segment elevation (STE) with T-wave inversion in lead aVL, ST-segment depression with positive T waves in leads II, III, aVF, and V₃ through V₆, and an isoelectric ST segment in leads I and V₁ through V₂. The Fourth Universal Definition of Myocardial Infarction¹ says, “More profound ST-segment shifts or T-wave inversions involving multiple leads/territories are associated with a greater degree of myocardial ischaemia, and a worse prognosis. For example, ST-segment depression ≥ 1 mm in 6 leads, which may be associated with STE in lead aVR or lead V₁ and hemodynamic compromise, is suggestive evidence of multivessel disease or left main disease” (eliminating answer A). This is considered a high-risk ECG pattern for which invasive intervention within 2 hours is recommended, in accordance with the 2020 European Society of Cardiology guidelines for managing acute coronary syndromes without persistent STE.² Our patient’s ECG shows ST-segment depression in 7 leads but no significant STE in lead aVR or V₁. In contrast, STE with negative T waves is seen in lead aVL.

Patients with a nonischemic early repolarization pattern can have STE in lead aVL. However, the T waves are usually positive, and there is often a J-point notch. The pattern here suggests a reperfused or more advanced stage of STEMI,³ and the patient’s clinical presentation (symptom resolution after hospital arrival) supports this diagnosis.

New STE in 2 contiguous leads is necessary for the diagnosis of STEMI.¹ However, this is an epidemiologic (not pathophysiologic) criterion, to minimize false-positive diagnosis. Acutely occluded arterial branches may cause STE in only one lead with diffuse ST-segment depression.⁴ The STE in leads aVL and V₂ through V₅ is caused by LAD occlusion proximal to the first diagonal branch (eliminating answer B),⁴ and STE in leads aVL and V₂ without STE in leads V₃ through V₅ suggests an occluded first diagonal branch. The ST-segment depression in the anterolateral and inferior leads is incompatible with the typical presentation of takotsubo cardiomyopathy (answer D). The STE in lead aVL, incompatible with ischemic cardiomyopathy (answer E), is accompanied by ST-segment depression in lead V₂, suggesting obstruction of the first OMB. Our patient had an isoelectric ST segment in lead V₂, so the differential diagnosis is an occluded OMB or ramus intermedius.⁴ A coronary angiogram on the second day of admission showed subacute thrombotic occlusion of the first OMB (Fig. 2, arrow), and stenting restored patent flow.

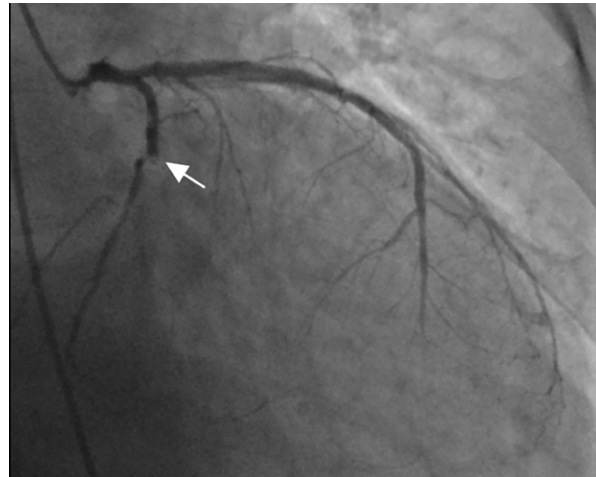


Fig. 2

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