Circulation

ECG CHALLENGE

Acute Inferior Wall Myocardial Infarction: What Is the Culprit Artery?

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ECG CHALLENGE

A 68-year-old man presented to the emergency department with sudden-onset chest pain, which had been persistent for 8 hours. He had been experiencing exertional shortness of breath in the preceding year. His medical history was notable for hypertension, atrial fibrillation, and cerebral infarction. A 12-lead ECG on admission is shown in Figure 1. The high-sensitivity cardiac troponin-I was 0.2618 ng/mL (normal range, 0.0000–0.0342 ng/mL). He was immediately transferred to the catheterization laboratory for coronary angiography and percutaneous coronary intervention. According to the electrocardiographic information, what are the abnormal findings, and what is the culprit artery?

Please turn the page to read the diagnosis.

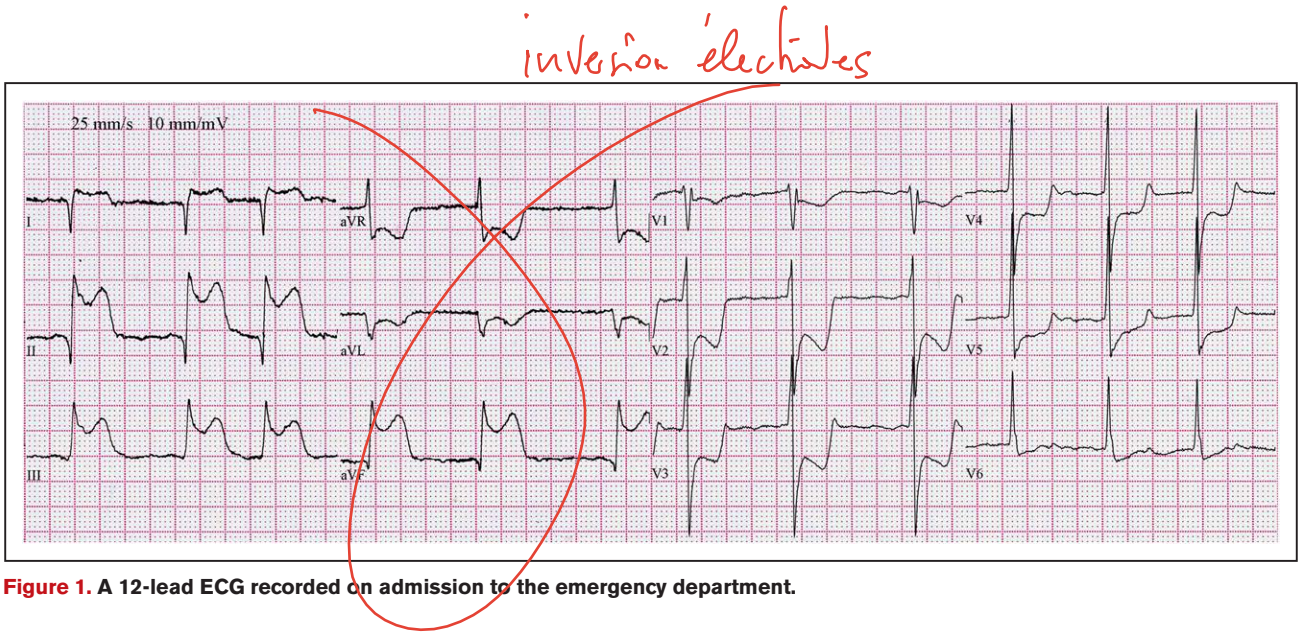

Figure 1. A 12-lead ECG recorded on admission to the emergency department.

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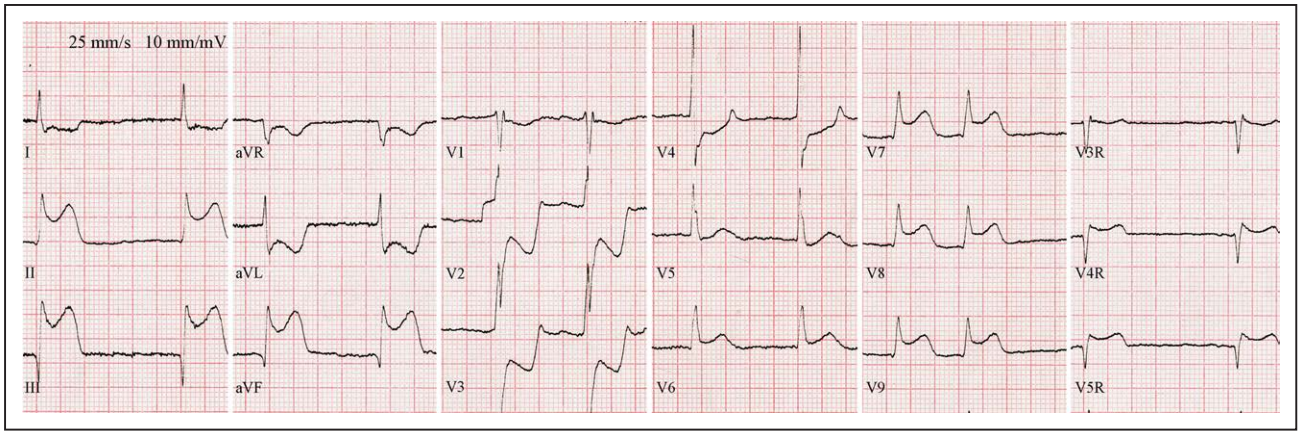

Figure 2. An 18-lead ECG with correct limb leads placement recorded after admission.
There is ST-segment elevation in leads II, III, aVF, V_7 through V_9, V_{4R}, and V_{5R}, suggesting acute inferoposterior wall myocardial infarction with right ventricular infarction.

RESPONSE TO ECG CHALLENGE

The 12-lead ECG on admission shows atrial fibrillation. There is ST-segment elevation in leads II, III, aVF, and I, with ST-segment depression in leads aVL and V_1 through V_6, suggesting acute inferoposterior wall myocardial infarction. The degree of ST-segment elevation is greater in lead II than in lead III, suggesting left circumflex coronary artery occlusion.[1] Paradoxically, the ST-segment elevation in lead I (QS complexes in leads I and aVL) suggests a lateral wall infarction; however, the precordial leads look more like a posterior wall than a lateral wall infarction. The limb and precordial discordance should raise the suspicion of the presence of electrode reversal. When limb leads recorded after admission were normal polarity, the lead III elevations are greater than lead II, as shown in Figure 2. The 18-lead ECG confirms posterior wall (V_7–V_9) with right ventricular (V_{4R} and V_{5R}) infarction as well. The electrocardiographic manifestations are consistent with a proximal right coronary artery (RCA) occlusion.[1,2] Such changes also confirm right arm–left arm lead reversal on admission ECG. Subsequent coronary angiography (Figure 3) was performed, revealing a 60% left anterior descending (proximal to middle portion) obstruction (Figure 3A) and a 100% proximal RCA occlusion (Figure 3B), which was successfully stented (Figure 3C). The left circumflex coronary artery was patent. Postoperatively, the patient received secondary prevention therapies.

Projection of the frontal plane vector on any lead axis produces the electrocardiographic pattern displayed in the respective limb lead. The waveforms of limb leads in normal sinus rhythm are shown in Figure 4A. In a patient with a right arm–left arm lead reversal, the electrocardiographic changes are as follows (Figure 4B): (1) The P-QRS-T waves are inverted in lead I, namely lead I should be flipped; and (2) the pattern of lead aVR resembles a normal aVL, and lead II resembles a normal lead III. These electrocardiographic features in normal sinus rhythm are typical and easy to diagnose. When it is complicated by arrhythmia and depolarization or repolarization abnormalities, electrocardiographic manifestations of right arm–left arm lead reversal are atypical, and it is easy to cause a misdiagnosis. In this case of atrial fibrilla-

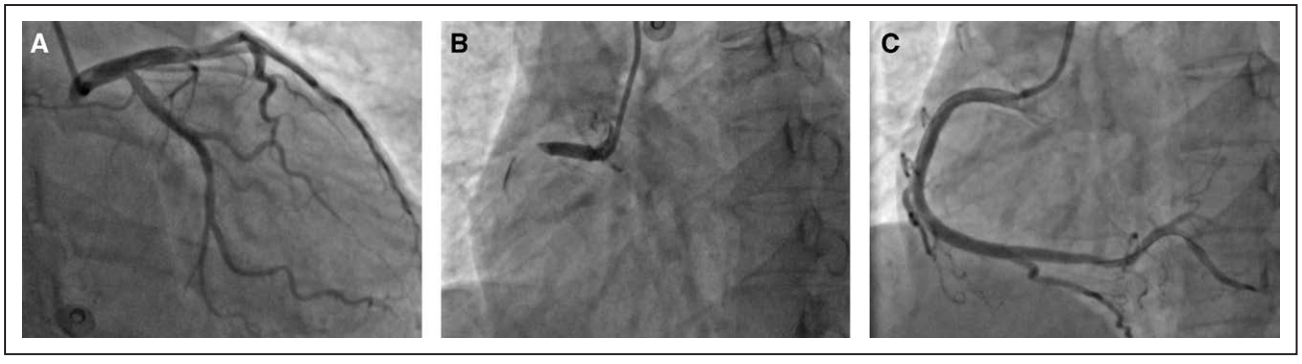

Figure 3. Coronary angiography.
Coronary angiography showed a 60% left anterior descending (proximal to middle portion) obstruction (**A**). Coronary angiography showed a 100% proximal right coronary artery occlusion (**B**). Coronary angiography after stent implantation revealed that the right coronary artery was patent (**C**).

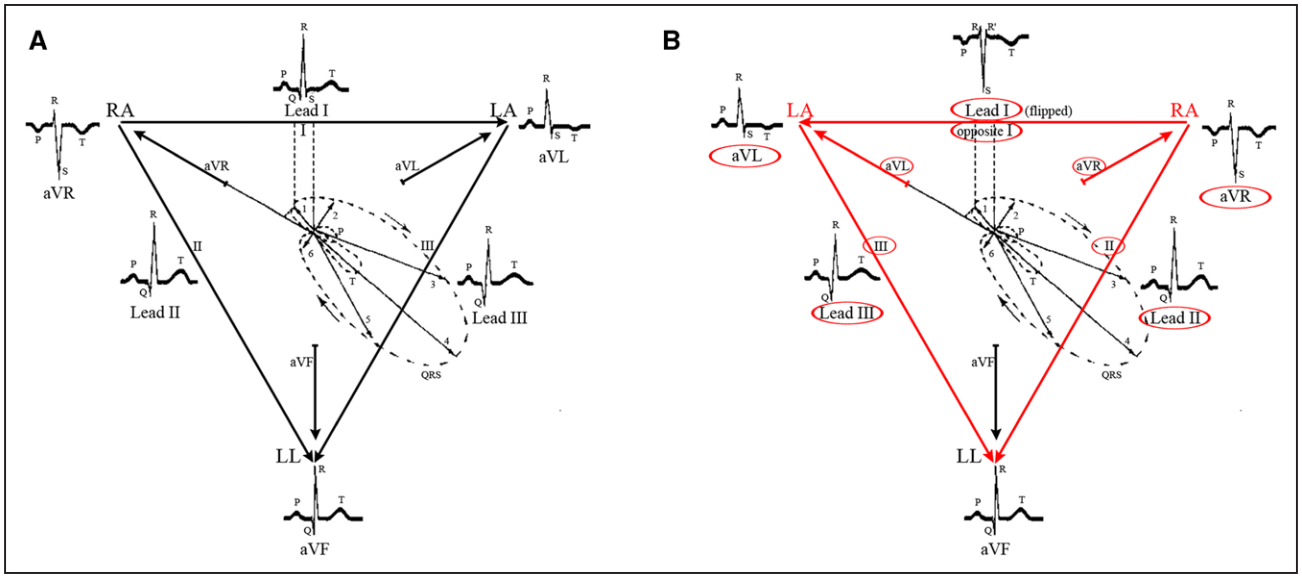

Figure 4. Schematic representation of the limb lead axis direction and electrocardiographic pattern formation.
A, Normal electrode placement, the frontal limb lead axis direction and electrocardiographic pattern. **B**, A right arm–left arm lead reversal manifested as follows: (1) Lead I is flipped; and (2) the pattern of lead aVR resembles a normal aVL, and lead II resembles a normal lead III. LA indicates left arm; LL, left leg; and RA, right arm.

tion, the absence of P waves and greater ST-segment elevation in lead II than in lead III result in incorrect interpretation of the culprit artery and lead to limb and precordial discordance. When the RCA is occluded, ST-segment elevation is greater in lead III than in lead II. When the left circumflex coronary artery is occluded, the ST-segment may be elevated to a greater extent in lead II than in lead III.[1] ST-segment elevation in the right ventricular leads helps us to identify RCA occlusion. At this time, we should suspect the presence of electrode misplacement, which can help elucidate the conflicting electrocardiographic findings. Important lessons learned from this case include the fact that the paradoxical changes in electrocardiographic manifestations of acute myocardial infarction should raise the suspicion of a spurious pattern attributable to electrode misplacement.

In patients with acute inferior wall myocardial infarction, the most important element is to recognize culprit artery and infarct size, which is essential in the treatment. As for acute inferior combined with right ventricular infarction attributable to occlusion of the proximal RCA, vasodilators, including nitroglycerine, should be avoided. To counter hypotension and cardiogenic shock, intravenous fluid boluses should be administered.[3]

ARTICLE INFORMATION

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Sources of Funding

None.

Disclosures

None.

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