Challenges in Clinical Electrocardiography

Diagnostic Traps—Noteworthy Electrocardiogram Patterns

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Case Presentation

A patient in their 40s was transported to the emergency department by ambulance for sudden acute chest pain that had been present for 1 hour. The patient did not have dyspnea or history of cardiac disease. The electrocardiogram (ECG) obtained in the ambulance is shown in the **Figure**, A. In the ambulance, the patient received nitroglycerin intravenously. On arrival, the patient's temperature was 36.4 °C, heart rate was 76 beats/min, and blood pressure was 90/56 mm Hg. Jugular distention was visible. No crackles were auscultated in the lungs. The initial serum troponin I and D-dimer levels were 0.03 ng/mL (normal range, ≤0.05 ng/mL; to convert to µg/L, multiply by 1.0) and 0.32 µg/mL (normal range, 0-0.55 µg/mL; to convert to nmol/L, multiply by 5.476), respectively. Peripheral oxygen saturation was 97% on room air. In the emergency department, a right ventricular and posterior wall ECG was obtained (Figure, B). Transthoracic echocardiography revealed severe global systolic dysfunction of the right ventricle (RV) with akinesia of the RV free wall and normal left ventricular systolic function.

Question: According to these ECGs, where is the culprit lesion?

Interpretation

The initial ECG (Figure, A) demonstrated accelerated atrioventricular junctional rhythm, ST-segment elevation (STE) in leads V_1 and aVR (V_1 > aVR), and diffuse ST-segment depression (STD) in leads I, aVL, and V_3 to V_6. No obvious ST-segment deviation was seen in leads II, III, and aVF. The ECG (Figure, B) obtained in the emergency department demonstrated STE in leads V_{3R} to V_{5R} and 2 ventricular premature contractions (black arrowheads).

Clinical Course

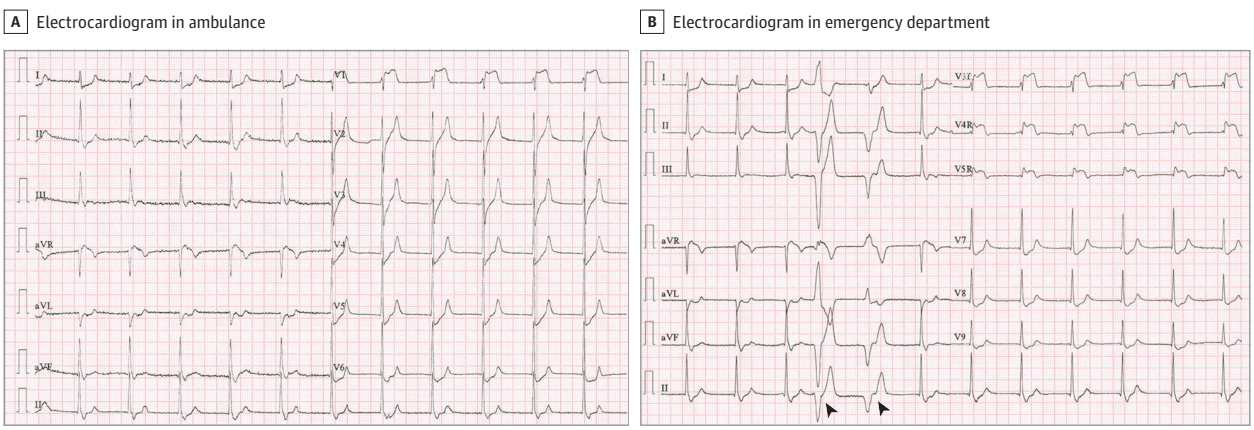
Coronary angiography showed no notable obstruction in the left main coronary artery, but total occlusion of the proximal nondominant right coronary artery (RCA) was present. A drug-eluting stent was placed in the RCA. The ECG showed that the patient had reverted to sinus rhythm after percutaneous coronary intervention. Based on the patient's symptoms (acute chest pain), signs (jugular distention), and ECG, echocardiography, and coronary angiography findings, the patient was diagnosed with isolated RV myocardial infarction (IRVMI). The patient was discharged 7 days later.

Discussion

Unlike the research on left ventricular myocardial infarction (MI), studies focusing on IRVMI as a separate entity began relatively late (in 1974). It occurs in fewer than 3% of all patients with MI, and its diagnosis may be challenging.[1] The most common culprit lesions causing IRVMI include occlusion of nondominant RCA or the occlusion of RV branch or RV marginal branch of RCA because these vessels supply the RV free wall. Clinical presentation of IRVMI may be very distinct. The classic triad consists of hypotension, clear lung fields, and raised jugular venous pressure.[2] The ECG plays an important role in establishing the diagnosis. There are 2 main ECG patterns of IRVMI.

The first main pattern is STE in right-sided leads. This ECG pattern constitutes STE in leads aVR and V_1 (V_1 > aVR), either accompanied or not by modest STE in lead III, and extensive STD in other leads. If right-sided leads are placed, STE in leads V_{3R} to V_{5R} can be observed. Anatomically, the RV forms the right anterior and inferior region of the heart. When IRVMI occurs, the transverse ST vec-

Figure. Electrocardiograms Obtained in the Ambulance and in the Emergency Department



A, The initial electrocardiogram demonstrated accelerated atrioventricular junctional rhythm, ST-segment elevation (STE) in leads V_1 and aVR (V_1 > aVR), and diffuse ST-segment depression in leads I, aVL, and V_3 to V_6. No obvious ST-segment deviation was seen in leads II, III, and aVF. B, The electrocardiogram obtained in the emergency department demonstrated STE in leads V_{3R} to V_{5R} and 2 ventricular premature contractions (black arrowheads).

tor points rightward and anteriorly, resulting in STE in right-sided leads. The frontal ST vector may point horizontally to the right; therefore, there will be STE in lead III but no other inferior leads. In addition, because lead V_1 faces the anterior region of the RV as well as the right upper paraseptal region,[3] STE in lead V_1 is greater than that in lead aVR.

This ECG pattern can be confused with the ECG changes of multivessel ischemia or left main coronary artery obstruction. An STD of 1 mm or greater present in 8 or more surface leads, coupled with STE in aVR and/or V_1 (aVR > V_1), suggests multivessel ischemia or left main coronary artery obstruction.[4] In the ECG of the current patient, the STE in aVR was smaller than that in V_1, and STD occurred in only 6 leads. Therefore, these findings do not resemble the ECG changes of multivessel ischemia or left main coronary artery obstruction.

The second main pattern is STE in anterior leads. This ECG pattern includes decreasing STE from V_1/V_2 to V_4/V_5 with no apparent Q waves and no reciprocal STD in inferior leads.[5,6] When IRVMI leads to RV enlargement, there will be clockwise transposition of the heart, resulting in STE in anterior leads. Because V_1 is located directly over the RV, STE in V_1 is greater than that in V_2 and V_3. In addition, when the frontal ST vector points horizontally to the front, there will be no ST-segment deviation in inferior leads. This ECG pattern can be easily misdiagnosed as acute anteroseptal or anterior MI. Differentiation of IRVMI from these MIs can be achieved by observing whether the amplitudes of STE in anterior leads are increasing or decreasing from V_1 to V_5 and/or whether STDs are present in inferior leads.

Differentiating between RV and left ventricle infarctions is essential because an adequate preload is needed in the former, whereas vasodilators should be avoided.[7] In patients who are hypotensive with RV failure, the first-line treatment is augmentation of RV preload by administering boluses of intravenous fluids. Nitrates and diuretics may worsen the hemodynamic status of these patients because they cause venodilation, which reduces the RV preload. In the case presented, the emergency personnel wrongly administered nitroglycerin, which led to a drop in blood pressure. This should be avoided as much as possible.

Take-home Points

- An IRVMI occurs in fewer than 3% of all patients with MI, and its diagnosis may be challenging.
- The classic triad of IRVMI consists of hypotension, clear lung fields, and raised jugular venous pressure.
- There are 2 main ECG patterns of IRVMI: (1) STEs in right-sided leads and (2) STEs in anterior leads.
- Differentiating between IRVMI and left ventricular infarctions is important for formulating an appropriate management plan.
- Nitrates and diuretics may worsen the hemodynamic status of patients with IRVMI.

ARTICLE INFORMATION

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