Challenges in Clinical Electrocardiography

Reframing the Approach to High-Risk Electrocardiography Patterns

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Case Presentation

A patient in their 40s presented to the emergency department with dizziness and presyncope after 1 day of large-volume hematemesis and multiple black stools. They reported profound weakness and vague, nonpleuritic chest discomfort, but no classic central pressurelike chest pain. There was no history of known coronary artery disease, diabetes, hypertension, smoking, myocardial infarction, or revascularization. On arrival, the patient appeared pale, anxious, and diaphoretic, with a blood pressure of 82/50 mm Hg, heart rate of 90 beats per minute, respiratory rate of 28 breaths per minute, and oxygen saturation of 96% on room air. An examination revealed cool extremities with weak peripheral pulses, prolonged capillary refill, mild epigastric tenderness, and melena on rectal examination, without pulmonary crackles or focal neurologic deficits. Initial laboratory testing showed a hemoglobin level of 4.8 g/dL (to convert to g/L, multiply by 10), hematocrit level of 16% (to convert to the proportion of 1.0, multiply 0.01), lactate level of 31.5 mg/dL (to convert to mmol/L, multiply by 0.111), and normal creatinine levels; the high-sensitivity troponin I concentration was less than the 99th percentile. Bedside point of care ultrasonography demonstrated preserved left ventricular systolic function without regional wall motion abnormalities and a markedly collapsed inferior vena cava that was consistent with severe hypovolemia. A 12-lead electrocardiogram (ECG) was obtained (**Figure**, A), which revealed a high-risk pattern that prompted an urgent cardiology consultation and raised the question of whether emergent coronary angiography was indicated.

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Question: What is the key abnormality on the ECG, and what is the most likely underlying pathophysiologic mechanism in this patient?

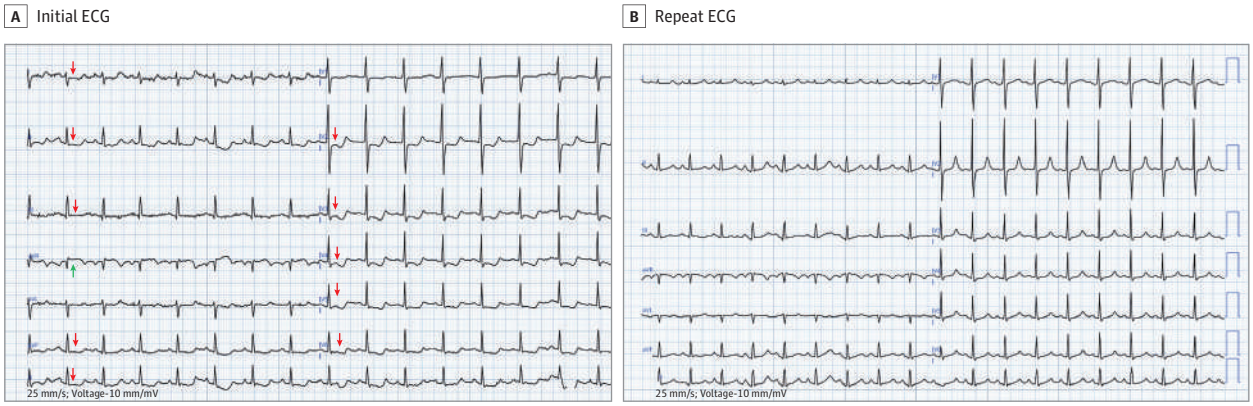
Interpretation

The initial ECG demonstrated sinus rhythm at approximately 90 beats per minute, with 1- to 2-mm ST-segment elevation in the lead aVR, an ST segment in V_1 that was higher than that in V_2, and diffuse horizontal to down-sloping ST-segment depression in leads I, II, aVL, and V_3 through V_6 (Figure, A). This pattern has been associated with left main or severe 3-vessel disease and increased mortality in non–ST-elevation acute coronary syndromes, earning a reputation as a high-risk ST-elevation myocardial infarction (STEMI)–equivalent pattern.[1-3] However, contemporary data have shown a prognostic-diagnostic paradox: the pattern predicts high mortality and extensive disease but has only modest (approximately 10%-14%) positive predictive value for acute thrombotic occlusion myocardial infarction.[3-5] Deng and colleagues[4] synthesized these data and proposed reframing STE-aVR from a vessel-specific surrogate to a high-risk marker of nonocclusive myocardial infarction and global ischemia in many hemodynamically stable patients. In this framework, STE-aVR acts as a barometer of global distress rather than a reliable locator of an acutely occluded culprit artery.[4] Within the hexaxial reference system, diffuse ST-segment depression in left-sided leads generates a net ST vector directed toward aVR; therefore, STE-aVR in this setting is the mirror of widespread subendocardial injury rather than a specific fingerprint of left main occlusion.[4,6] Clinically, STE-aVR with multilead ST-segment depression marks circumferential subendocardial ischemia from oxygen supply-demand mismatch, which may be associated with obstructive coronary disease, but also by noncoronary conditions, such as severe anemia, shock, sepsis, pulmonary embolism, or toxic-metabolic insults.[4,6]

Discussion

In the present case, point of care ultrasonography showed preserved left ventricular systolic function with no regional wall mo-

Figure. Electrocardiogram (ECG) Results



A, Initial 12-lead ECG showing sinus rhythm with 1- to 2-mm ST-segment elevation in the aVR (green arrow), ST in V_1 that was higher than V_2, and diffuse ST-segment depression (red arrows) in leads I, II, aVL, and V_3 through V_6.

B, A repeated ECG after transfusion and hemodynamic stabilization demonstrated normalization of ST segments in aVR changes.

tion abnormalities, a nondilated right ventricle, and a markedly collapsed inferior vena cava that was consistent with profound hypovolemia. Lung ultrasonography results did not reveal interstitial syndrome or consolidation, and focused venous and abdominal scans demonstrated no deep venous thrombosis or free intra-abdominal fluid.

Given the profound anemia, hypotension, absence of classic ischemic chest pain, nondynamic high-sensitivity troponin values, and normal regional wall motion, the pre-ECG likelihood of acute coronary syndrome was low. Therefore, circumferential subendocardial ischemia from hemorrhagic shock was the more probable explanation for the ECG changes. The cardiology service was consulted early, and the emergency department team prioritized resuscitation and hemorrhage control while closely monitoring for evolving myocardial infarction according to contemporary occlusion myocardial infarction pathways.[7,8] The patient received packed red blood cell transfusions, a proton pump inhibitor infusion was initiated, and vasoactive agents were used transiently to maintain perfusion pressure. Urgent upper gastrointestinal endoscopy results revealed gastroesophageal varices, which were treated with endoscopic hemostasis.

After the patient's hemoglobin levels improved to 8 g/dL and his hemodynamics improved, a repeated ECG demonstrated resolution of the diffuse ST-segment depression and normalization of the ST segments in aVR and V_1, with regression of the new right axis deviation toward the prior baseline (Figure, B). Serial high-sensitivity troponin measurements did not show a rise and fall pattern, and repeated cardiac ultrasonography continued to show preserved global systolic function without regional wall motion abnormalities. The patient's hemodynamic status remained stable; vasoactive agents were weaned, they were transferred out of the intensive care unit on hospital day 2, and they were discharged home on day 3 with gastroenterology follow-up, with no cardiac events at 6 months. From a practical emergency medicine perspective, this high-risk aVR pattern should trigger urgent risk stratification and systematic evaluation for coronary and noncoronary causes of global ischemia, but it should not automatically mandate emergent catheterization for all patients.[4,9]

Take-Home Points

- STE-aVR with diffuse ST-segment depression is a high-risk ECG pattern that is associated with adverse outcomes but is not specific for acute coronary syndrome (ACS).
- It reflects circumferential subendocardial ischemia from a global oxygen supply-demand mismatch and can arise in non-ACS conditions, such as severe anemia, shock, sepsis, pulmonary embolism, and toxic-metabolic states.
- Recognizing the prognostic-diagnostic paradox of lead aVR reframes STE-aVR from a STEMI equivalent to a high-risk, nonocclusive myocardial infarction marker that must be interpreted in a clinical context.
- In patients with a low pre-ECG likelihood of ACS, a likelihood-based strategy that prioritizes resuscitation, serial ECGs, and high-sensitivity troponins can prevent unnecessary emergent catheterization, especially when ST changes resolve and troponin and ventricular function remain stable.

ARTICLE INFORMATION

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