Challenges in Clinical Electrocardiography

Missed Posterior Myocardial Infarction Leading to Free Wall Rupture

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Case Presentation

A healthy woman in her early 60s presented with 4 hours of substernal chest pain and diaphoresis. The initial electrocardiogram (ECG) (**Figure**) was read as showing no diagnostic abnormalities, and she was referred for outpatient evaluation. Transthoracic echocardiography revealed preserved systolic function without pericardial effusion. Cardiac troponin T was elevated at 65.3 ng/L (normal range, <14 ng/L; to convert to µg/L, multiply by 1), and she was diagnosed with high-risk non–ST-segment elevation myocardial infarction (NSTEMI) with a Global Registry of Acute Coronary Events score of 110. Aspirin loading was administered, and she was admitted for planned coronary angiography within the day.

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Approximately 4 hours after presentation (1 hour postadmission), she collapsed in the cardiology ward bathroom. Cardiopulmonary resuscitation achieved return of spontaneous circulation after 5 minutes. Bedside ultrasonography revealed massive pericardial effusion. Emergency pericardial drainage and surgery identified a 15-mm rupture of the posterior left ventricular free wall, which was successfully repaired.

Following stabilization, coronary angiography demonstrated complete occlusion of the distal left circumflex artery. The patient recovered and was discharged home in stable condition. At 2-year follow-up, she had returned to normal daily activities without neurological sequelae, and her left ventricular ejection fraction remained at more than 50%.

Questions: What important abnormality was missed on the initial ECG that could have suggested posterior myocardial infarction?

Interpretation

The patient's admission ECG demonstrated sinus rhythm at a rate of approximately 70 beats per minute. The PR interval was within normal limits (approximately 160 ms), and the QRS duration was narrow (<100 ms). The QTc interval was estimated at approximately 420 ms.

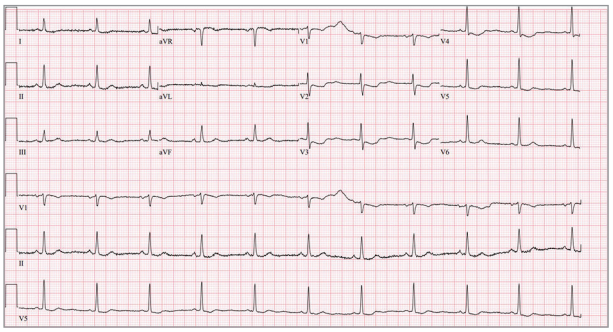
Importantly, there were subtle horizontal ST-segment depressions in leads V_2 through V_3, with a reciprocal pattern suggestive of posterior wall ischemia, and an R/S ratio greater than 1 in these leads. No clear ST-segment elevation was present in the standard leads, and T-wave morphology otherwise appeared unremarkable. There were no pathological Q waves or conduction abnormalities.

These changes can be easily overlooked, as they can be mistaken for nonspecific repolarization abnormalities. However, in the clinical context of acute chest pain, they raise strong suspicion for posterior myocardial infarction, which should prompt the use of posterior leads (V_7 through V_9) for confirmation.

Discussion

Posterior myocardial infarction (MI) represents a diagnostic blind spot in routine clinical practice, largely because the posterior wall is not directly visualized on the standard 12-lead ECG. Instead, its ischemic changes often manifest indirectly as ST-segment depression in the anterior precordial leads, most commonly V_1 through V_3. These changes are subtle, frequently misinterpreted as nonspecific or as reciprocal changes of an inferior MI, and are underrecognized by automated ECG interpretation software. This case illustrates the consequences of such a pitfall: delayed recognition of posterior MI led to a free wall rupture.

Figure. Initial Electrocardiogram at Presentation



The 12-lead electrocardiogram demonstrates sinus rhythm with nonspecific ST-segment changes. Subtle ST depressions in leads V_1 through V_3 are suggestive of posterior myocardial infarction; no overt ST elevation is present.

The initial ECG in this case demonstrated only subtle horizontal ST-segment depression in V_2 through V_3 without overt ST-segment elevation. In the context of acute chest pain, such findings should have prompted consideration of posterior ischemia and the recording of posterior leads (V_7 through V_9). Posterior leads increase diagnostic sensitivity by directly recording the posterior wall and may reveal diagnostic ST-segment elevation that reclassifies NSTEMI into a ST-segment elevation MI requiring urgent reperfusion therapy. Importantly, studies have shown that up to 20% of patients initially diagnosed with NSTEMI are later found to have posterior transmural infarction, underscoring the risk of underdiagnosis when relying solely on standard ECGs.[1,2] The reliance on automated interpretation further compounds the problem, as computer algorithms often label these changes as "nonspecific ST-T abnormalities."[3]

The implications of underrecognition are substantial. Delayed or missed diagnosis may postpone reperfusion therapy, increasing the risk of complications such as papillary muscle rupture, ventricular septal rupture, and free wall rupture. In this patient, posterior MI was misclassified as high-risk NSTEMI, leading to an inpatient monitoring strategy rather than immediate reperfusion. Within hours, she experienced free wall rupture with cardiac tamponade. While timely cardiopulmonary resuscitation and surgical intervention saved her life, the episode highlights the precarious nature of delayed diagnosis in this subset of MI. Patients with isolated posterior MI frequently do not receive timely reperfusion therapy if the diagnosis

is not suspected, largely because the classical criterion of ST-segment elevation is absent on the standard 12-lead ECG.[4]

Left ventricular free wall rupture is an uncommon but devastating complication of acute myocardial infarction, with mortality rates exceeding 80% when not promptly recognized and surgically managed.[5] Cardiac rupture most often occurs between the third and fifth day after infarction, corresponding to the period of maximal myocardial wall thinning and necrosis.[6] In this patient, however, rupture developed within 24 hours of symptom onset, an unusually early course. Such rapid progression raises the possibility of preexisting, undiagnosed chronic coronary artery disease, with the acute infarction occurring in a myocardium already weakened by prior ischemic injury. Posterior and lateral infarctions, due to their often subtle electrocardiographic manifestations, are especially prone to delayed recognition, further increasing the risk of mechanical complications such as rupture.[7] In the present case, survival was achieved only because tamponade was rapidly identified by point-of-care ultrasonography and emergent surgical repair was undertaken without delay.

The case illustrates how subtle ECG findings, if missed, can result in catastrophic outcomes. Posterior MI remains an area where misdiagnosis persists despite decades of awareness, and its consequences can be fatal. In busy emergency and hospital settings, systematic ECG interpretation, clinical correlation, and use of posterior leads remain essential to reduce the risk of misdiagnosis and prevent complications.

Take-Home Points

- Posterior MI is easily overlooked: subtle ST depression in V_1 through V_3 may represent posterior ischemia and should prompt the use of posterior leads (V_7 through V_9).
- Missed diagnosis carries catastrophic risk: delayed recognition of posterior MI can lead to fatal complications such as free wall rupture.
- Close monitoring is critical: patients with suspected high-risk MI, even without overt ST-segment elevation, should be managed in a monitored setting to allow early detection of sudden deterioration.

ARTICLE INFORMATION

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