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Focus on ECGs: Case #27

Electrocardiographic Changes in a Man With Acute Pancreatitis: A Myocardial Infarction?

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A 69-year-old man with a history of heavy alcohol use presented with abdominal pain and was diagnosed with alcohol-induced acute pancreatitis. On admission, he had not reported chest pain, palpitations, or dyspnea. Initial laboratory tests showed a lactic acid level of 8.3 mmol/L (normal, 0.5–2.2 mmol/L), normal renal function, and normal cardiac enzyme levels. An electrocardiogram (ECG) on admission showed sinus tachycardia. His symptoms and lactic acidosis resolved after aggressive fluid resuscitation and bowel rest. Two days later, sudden-onset dyspnea, tachycardia, and hypoxia necessitated the administration of 2 L of supplemental oxygen. A chest radiograph showed acute pulmonary edema. The patient's serum cardiac troponin level peaked at 0.35 ng/mL (normal, <0.03 ng/mL), and his brain natriuretic peptide level was 235 pg/mL (normal, <100 pg/mL). His ECG showed changes (Fig. 1).

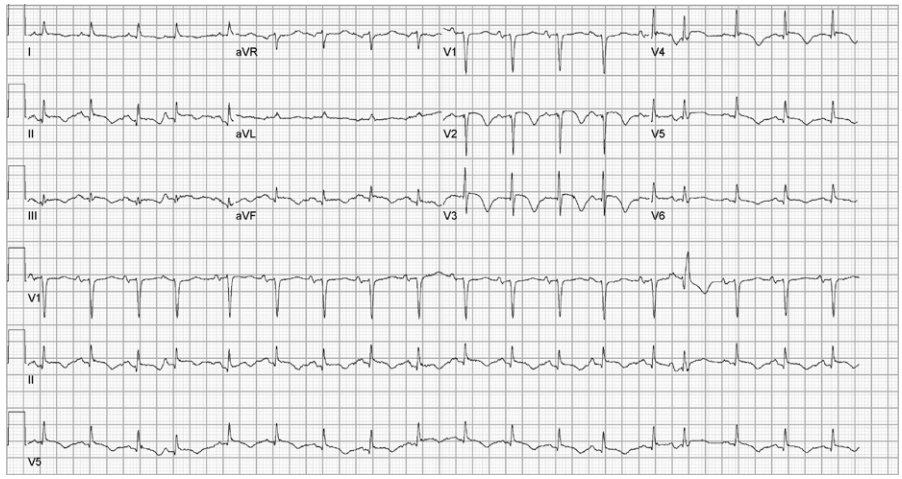

Fig. 1

What is the most likely diagnosis?

A) ST-segment-elevation myocardial infarction (STEMI)

B) Stress-induced (takotsubo) cardiomyopathy

C) Acute pulmonary embolism

D) Critical stenosis of the left anterior descending coronary artery (Wellens syndrome)

See next page for the answer.

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B) Stress-induced (takotsubo) cardiomyopathy

Figure 1 shows sinus tachycardia with premature atrial contractions, new ST-segment elevations (STEs) in the inferior leads and in leads V_2 through V_6, and T-wave inversions throughout. Figure 2 shows the admission ECG.

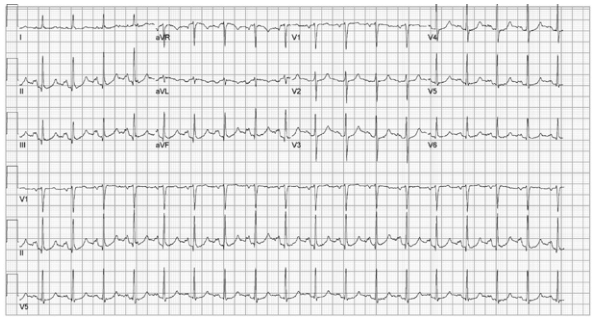

Fig. 2

According to the Mayo Clinic criteria, the most common ECG changes associated with takotsubo cardiomyopathy (TTC) are STE and T-wave inversion. These changes are seen frequently in the precordial leads.[1] Clinically, distinguishing TTC from acute coronary syndrome is challenging because of overlapping symptoms, elevated cardiac enzyme levels, and similar ECG changes. Certain ECG patterns indicate TTC rather than acute coronary syndrome, including the absence of ST-segment depression in the reciprocal leads; diffuse STEs extending beyond the territory of a single coronary artery; and STE in lead aVR with STE in the inferior, anterior, or anteroseptal leads.[2]

Patients with suspected TTC may have known coronary artery disease (CAD) or associated risk factors; therefore, STEMI must be ruled out when STE is seen on an ECG.[3] If angiograms reveal no fixed obstructive CAD, acetylcholine testing may be considered, because spasm-induced coronary obstruction has been proposed as the mechanism underlying TTC, with atherosclerotic CAD providing a protective "stenting" effect.[4] Pulmonary embolism may trigger sinus tachycardia and T-wave inversions, but without STE or pulmonary edema. Wellens syndrome can cause isolated deep T-wave inversions in leads V_1 through V_3, but it cannot explain inferior-lead STE and T-wave changes.

The pathophysiology of TTC involves transient stunning and reversible left ventricular (LV) systolic dysfunction after physiologic or emotional stress. Fewer than 20 cases of TTC have been reported as complications of acute pancreatitis. Of note, pancreatitis itself can cause STE, typically in the inferior leads.[5] In our patient's case, an echocardiogram and left ventriculogram revealed severe apical hypokinesis and an LV ejection fraction of 40%; however, a coronary angiogram did not reveal obstructive CAD, so the diagnosis was TTC. Two months later, he had normal LV ejection function, consistent with expected recovery timelines.

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