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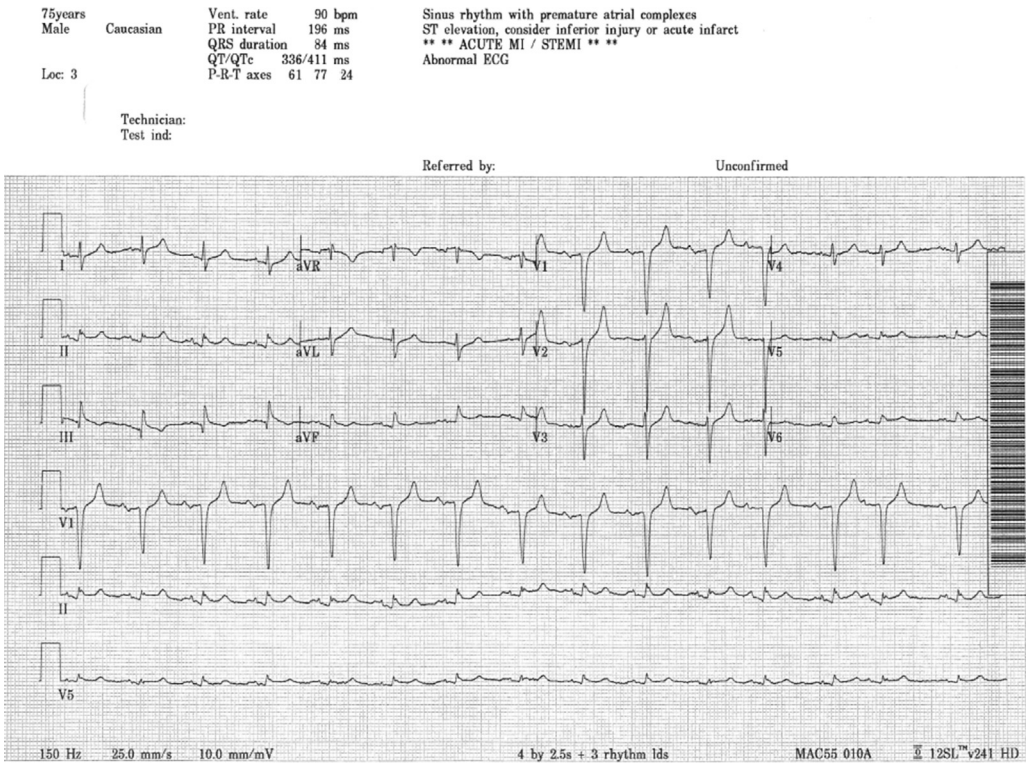

Figure 1. An ECG demonstrating ST-segment elevation in the inferior and lateral leads, along with hyperacute T waves in the anterior leads.

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A 75-year-old man with confirmed cholecystitis and a history of type 2 diabetes mellitus, hypertension, and hyperlipidemia presented to our emergency department as a transfer from an outside hospital. On arrival, he was ill appearing, febrile, tachycardic, and slightly hypotensive. He was given broad-spectrum antibiotics, and an ECG (Figure 1) was performed for surgical planning. ECG showed normal sinus rhythm with ST-segment changes. No previous ECG was available for comparison.

Should the cardiac catheterization laboratory be used?

For the diagnosis and teaching points, see page 625.
To view the entire collection of ECG of the Month, visit www.annemergmed.com

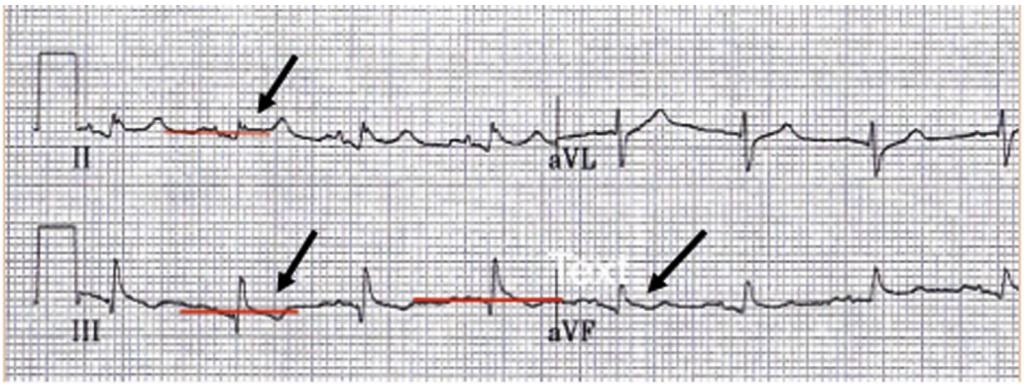

Figure 2. A close-up image of the inferior leads, highlighting the minimal but concerning ST-segment elevation, along with a nonpathologic Q wave and nonspecific T-wave inversion in lead III.

ECG OF THE MONTH

(continued from p. 624)

DIAGNOSIS:

Interpretation

Acute Cholecystitis. The ECG in Figure 1 demonstrates a sinus rhythm with minimal but concerning ST-segment elevation in leads II, III, and aVF (Figure 2), along with V5 and V6, yet without reciprocal changes in I and aVL. There was a nonpathologic Q wave and nonspecific T-wave inversion in lead III (Figure 2) and hyperacute-appearing T waves in the anterior leads. Findings were concerning for an acute ST-segment elevation myocardial infarction, particularly so without a previous ECG for comparison.

CLINICAL COURSE

Given that the patient was septic with known cholecystitis, a point-of-care troponin test was ordered; the level was undetectable. Emergency cardiology consultation was obtained and a bedside echocardiogram was performed, which showed no wall-motion abnormalities. Further review of the patient's medical records indicated that he had undergone a nuclear stress test 2 weeks earlier, whose results were negative for evidence of ischemia or infarction. Given the reassuring cardiac evaluation, he proceeded to the surgical ICU for further resuscitation and percutaneous cholecystostomy. No further ECGs were completed during the patient's hospitalization.

DISCUSSION

This patient presented with confirmed cholecystitis, and a 12-lead ECG, obtained for surgical planning, was completed and demonstrated the aforementioned ST-segment elevation in the inferior and lateral leads. These findings prompted inquiry in regard to appropriate next steps in management and highlighted the challenges surrounding interpretation of ST-segment elevation.

The challenge surrounding interpretation of ST-segment elevation stems from the controversy about PR-segment versus TP-segment relation to the ST segment. Historical consensus suggests the TP segment serves as the isoelectric baseline and subsequent comparison of the ST segment of an ECG.[1] Conversely, the American Heart Association recommends measuring the ST-segment level relative to the PQ-segment junction during exercise stress testing, and others have taken this as standard measure.[2] The American Heart Association, American College of Cardiology Foundation, and the Heart Rhythm Society have noted that the ST- and T-wave amplitudes are referenced against

either the TP or PR segments of the ECG and make no formal recommendation.[3] In clinical practice, the proximity of PR and ST segments leads to technically easier comparison than the TP segment; however, the presence of PR-segment depression in the provided ECG lends to the interpretation of more worrisome ST-segment elevation, whereas if the TP segment were used, the ST-segment elevation would be even more minimal.

Although this ECG highlights the challenges of evaluating ST-segment elevation, it also highlights a unique cause of ST-segment changes of which the emergency physician should be aware. Acute cholecystitis can present with signs and symptoms similar to those of coronary ischemia. ST-segment elevation, although most concerning for cardiac ischemia, can be mimicked by pancreatitis, gastric distention, and acute cholecystitis in the setting of normal coronary angiography result.[4]

In a retrospective study of acute cholecystitis mimicking or accompanying cardiovascular disease among 5,552 Japanese patients hospitalized in a cardiology department between 2010 and 2014, 16 patients ultimately received a diagnosis of acute cholecystitis. Five of those patients initially had suspected cardiovascular disease, which later turned out to be incorrect. Two of these 5 patients had ECG findings concerning for coronary ischemia: inverted or flat T waves in V2 to V6 and ST-segment depression in lead II-aVF, with normal coronary arteries demonstrated by coronary angiography. The remaining 11 patients were found to have both acute cholecystitis and cardiovascular disease, without specifics in regard to corollary ECG findings. The investigators concluded that acute cholecystitis, albeit rarely, may cause ECG changes concerning for coronary ischemia, leading to misdiagnosis as a cardiovascular disorder, or may coexist with a cardiovascular disease.[5]

The exact cause of ECG changes in cholecystitis is unknown, but the most recently proposed mechanism comes from the study of gallbladder distention in anesthetized pigs. Investigators found that gallbladder distention causes a reflex plasma renin activity involving vagal afferent pathways, with an efferent limb in renal nerves involving β-adrenergic receptors. The response to gallbladder distention produces reflex vasoconstriction, along with reflex mesenteric and iliac vasoconstriction. Minor deficiencies in coronary circulation may become prominent in ECG changes when accentuated by reflex vasoconstriction.[6]

Although acute cholecystitis has been known to cause dynamic ECG changes that mimic ischemia, there appear to be 7 cases noting ST-segment elevation in the setting of acute cholecystitis.[3-8] Of these 7 cases, 4 patients underwent coronary angiography, all instances of which demonstrated normal coronary arteries. Three of these 4 patients' ST-segment elevations resolved after cholecystectomy, and 1 patient's ECG changes resolved with antibiotic administration.

ST-segment elevation in the right clinical context is justification alone for the emergency physician to initiate reperfusion therapy; however, thrombolysis and rescue angioplasty are not without risks of significant morbidity and mortality.[4,7,8] Awareness of these alternative differentials is crucial to ensuring appropriate diagnostic investigations. In the case of our patient, appropriate intervention included prompt percutaneous cholecystostomy and treatment of septic shock in the surgical ICU with vasopressors and broad-spectrum antibiotics.

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