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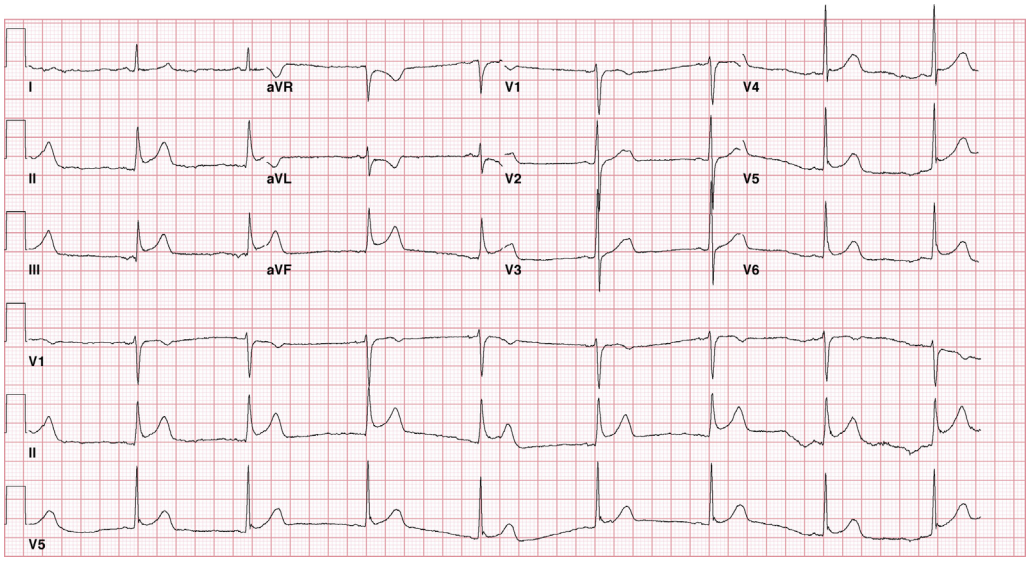

Figure 1. Initial ECG recorded on arrival to the ED.

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A 62-year-old woman with a history of prediabetes, hypertension, and hyperlipidemia presented to the emergency department (ED) with retrosternal chest pain. At approximately 9 PM, she experienced sharp chest discomfort that radiated to the anterior neck. She received naproxen, which ameliorated her pain, and went to sleep. The patient awoke suddenly approximately 3 hours later with excruciating, retrosternal chest pain and dyspnea.

The patient characterized her pain as worsening with deep inspiration and with rolling on her side. Six weeks before presentation, she had experienced an upper respiratory infection, which was treated empirically with oseltamivir, with improvement in her symptoms, although she continued to complain of cough on presentation. Vital signs included a pulse rate of 50 beats/min, blood pressure of 97/54 mm Hg, respiratory rate of 16 breaths/min, and oxygen saturation of 99% on room air. Physical examination was notable for normal vital signs, clear lungs, and normal heart sounds without murmurs, rubs, or gallops.

An ECG was performed on arrival to the ED 30 minutes later (Figure 1). A previous ECG was not available for review.

What is the best course of action at this time?

For the diagnosis and teaching points, see page 779.
To view the entire collection of ECG of the Month, visit www.annemergmed.com

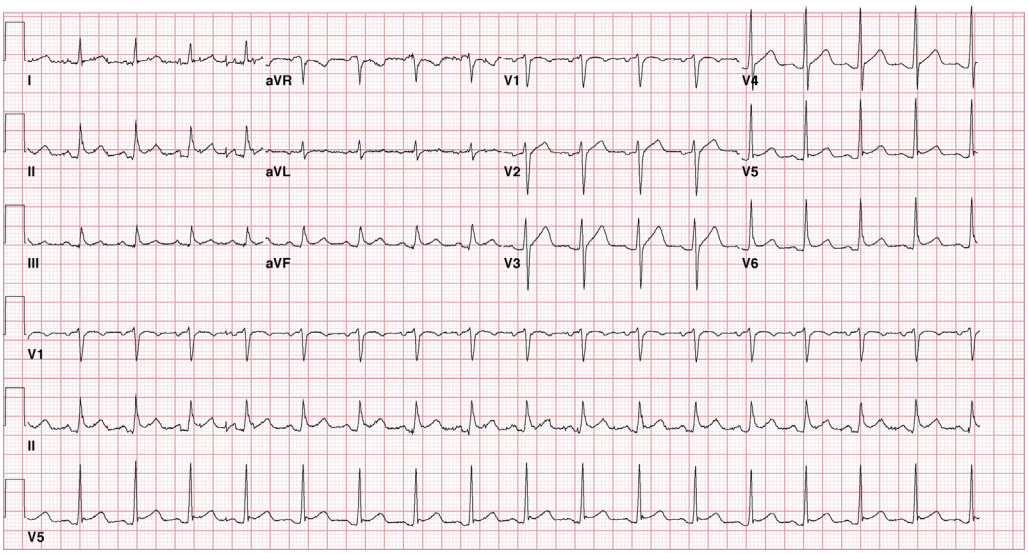

Figure 2. Repeated ECG 6 hours after presentation.

Table 1. Differential diagnosis of ST-segment elevation.

Diagnosis	ECG Findings
CORONARY ARTERY OCCLUSION	
STE-ACS	Concave with hyperacute T waves rapidly evolving to convex ST-segment elevation in leads corresponding to a vascular territory, often with reciprocal ST-segment depression
Prinzmetal's angina	Identical to STE-ACS but classically transient with evidence of spasm on coronary angiography[24]
NON-OCCLUSIVE MYOCARDIAL INJURY	
Acute pericarditis and myocarditis	Diffuse concave ST-segment elevation in a patient with sharp, positional (nonanginal) chest pain, often associated with widespread PR-segment depression and PR-segment elevation in aVR (knuckle sign)
Stress cardiomyopathy (including Takotsubo's syndrome)	Indistinguishable from STE-ACS but often associated with diffuse, deep T-wave inversions and QT prolongation[25]
Pulmonary embolism	ST-segment elevation may be present in the precordial leads, often accompanied by S1Q3T3 pattern and other signs of right-sided heart overload[26]
PATTERNS NOT CHARACTERIZED BY MYOCARDIAL INJURY	
Early repolarization	J point elevation with concave ST segment and a notched or slurred terminal R wave
Left bundle branch block and ventricular pacing	Concave ST-segment elevation discordant from the predominant QRS vector, generally less than 5 mm (Antzelevitch and Yan[27])
Brugada's syndrome	Coved or saddle-back ST-segment elevation usually observed in the right precordial leads[28]
LVH	Concave discordant ST-segment elevation in the anterior leads, associated with ECG findings of LVH

STE-ACS, ST-segment elevation acute coronary syndrome; *LVH*, left ventricular hypertrophy.

ECG OF THE MONTH

(continued from p. 777)

DIAGNOSIS:

Interpretation

Triage ECG revealed sinus bradycardia at 50 beats/min. The J point was elevated 1 mm above the isoelectric line in the inferior leads, associated with slurred terminal R waves and convex elevation of the ST segment itself. A 1- to 2-mm J-point elevation was also present in leads V4, V5, and V6 and was preceded by notched terminal R waves with similar ST-segment elevation. ST-segment depression was present in aVR and aVL. These changes were consistent with early repolarization. However, the ST-segment depression in aVR and aVL could have represented reciprocal changes from infarction, and thus ST-segment elevation acute coronary syndrome remained on the differential.

CLINICAL COURSE

The initial tracing was interpreted by ECG as inferolateral ST-segment elevation acute coronary syndrome, which was also suspected by the emergency physician and the cardiology service. The patient was taken urgently to the cardiac catheterization laboratory, where angiography revealed mild coronary luminal irregularities, no evidence of vasospasm, and a normal ventriculogram result. Serum troponin I level on presentation was less than 0.02 μg/L.

The patient continued to experience chest discomfort after catheterization, and a second ECG (Figure 2) was obtained on arrival to the ward 6 hours after the initial tracing.

Consider how the repeated ECG changes the differential diagnosis. What is the best course of action now?

The repeated ECG was interpreted as sinus tachycardia at 103 beats/min; PR-segment (defined as the end of the P wave to the beginning of the QRS complex) depression in leads II, III, aVF, V3, and V4; and PR-segment elevation in aVR. Concave J point ST-segment elevation was present in leads II, V5, and V6, which is associated with notching of the terminal component of the preceding R wave. This tracing was consistent with acute pericarditis superimposed on early repolarization.

Troponin I testing performed at the same time as the repeated ECG revealed a level less than 0.02 μg/L. Urine toxicology screen result was negative. Transthoracic echocardiogram revealed a left ventricular ejection fraction of 75%, no wall motion abnormalities, concentric left ventricular hypertrophy, and no evidence of pericardial effusion.

In accordance with the additional history obtained, the 2 ECG results, and the cardiac catheterization findings, the patient received a diagnosis of acute pericarditis. Ibuprofen was administered, which resulted in significant improvement in her chest discomfort. She was discharged home with ibuprofen 600 mg 3 times daily and close cardiology follow-up.

DISCUSSION

This patient presented with retrosternal chest pain and ST-segment elevation in more than 2 contiguous leads but had no evidence of obstructive coronary disease, highlighting the importance of ST-segment and QRST morphology in the diagnosis of ST-segment elevation acute coronary syndrome. The subsequent clinical evolution to pericarditis reinforces the importance of serial ECG recordings and having a diagnostic schema for ST-segment elevation.

Although ST-segment elevation is present in a number of pathologic processes (Table), it has long been recognized as a sign of complete coronary artery obstruction resulting in transmural infarction[1] and, in the correct clinical context, is an indication for urgent coronary angiography.[2] ST-segment elevation concerning for ST-segment elevation acute coronary syndrome is defined as new elevation at the J point in at least 2 contiguous leads.[3] To meet criteria, ST-segment elevation must be greater than or equal to 1 mm (0.1 mV) in all leads except V2 and V3, in which some degree of ST-segment elevation (up to 2.5 mm in men and 1.5 mm in women) is normal.[4] Patients with early repolarization pattern, stress cardiomyopathy, acute pericarditis, left ventricular hypertrophy, left bundle branch block, and Brugada's syndrome are excluded from this definition.[3] This is especially pertinent because ECG machine algorithms are very accurate for acute coronary syndrome under normal circumstances but can be confounded by other causes of ST-segment elevation. For example, at our institution, we use ECG machines that have

manufacturer-reported diagnostic accuracy in acute coronary syndrome of 95.8% (sensitivity 79.3%, specificity 98.6%). In contrast, in left ventricular hypertrophy with ST-segment elevation, the sensitivity for ST-segment elevation acute coronary syndrome is reported as 53.7% and the specificity is 60.9%.[5]

Pericarditis and myopericarditis are generally considered to be uncommon conditions that may result from a number of underlying processes, including infection (most commonly viral), autoimmune diseases (eg, systemic lupus erythematosus), neoplasms (most often metastatic tumors), trauma, postsurgical inflammation, metabolic derangements (eg, uremia), and adverse drug reactions. The presentation is characterized by positional or sharp, often pleuritic, chest pain; suggestive ECG changes; pericardial friction rub; and new or worsening pericardial effusion. At least 2 of these features should be present to confirm the diagnosis.[6] As opposed to pericarditis, myopericarditis is characterized by myocardial injury as evidenced by elevated serum cardiac troponin levels and may be associated with severe cardiac dysfunction, resulting in new-onset heart failure.[7] The treatment of choice for pericarditis is nonsteroidal anti-inflammatory drugs with or without colchicine, although steroids may be used in patients with contraindications to these medications.[6] Although myopericarditis is typically treated in the same manner, to our knowledge no randomized trials have assessed this approach, and animal models suggest that nonsteroidal anti-inflammatory drugs may actually be harmful to such patients.[7]

High-quality data in regard to ECG changes in pericarditis and myopericarditis are limited. These conditions are classically associated with diffuse, concave ST-segment elevations in leads that do not correspond to a single vascular territory.[8-10] In published reports, PR-segment depression is present in the majority of patients with pericarditis[11] and is useful in differentiating pericarditis from ST-segment elevation acute coronary syndrome when present in both the precordial and limb leads.[12] PR-segment depression has also been observed in asymptomatic patients with pericardial effusions, suggesting that this finding may be present in subclinical pericarditis.[13] Similarly, PR-segment elevation in aVR (known as the "knuckle sign"), which was present in this case (Figure 2), has been reported to be highly specific for pericarditis but lacks sensitivity.[12]

In contrast, ST-segment elevation acute coronary syndrome is usually characterized by convex (tombstone) or straight ST-segment elevation in leads corresponding to the infarcted vascular territory.[14] There are several exceptions to this rule: the ST-segment may be concave very early in the evolution of ST-segment elevation acute coronary syndrome and can be differentiated from early repolarization by the presence of hyperacute T waves ("coronary T waves") and ST-segment elevation above the J point.[15] Additionally, several small retrospective studies suggest that anterior ST-segment elevation acute coronary syndrome may occasionally present with concave ST-segment elevation.[16,17]

The terms "J point elevation" and "early repolarization" are applied in clinical practice with a not insignificant amount of heterogeneity. The early repolarization pattern has been defined in a consensus article as the combination of an end-QRS notch or slur on the downslope of a prominent R wave with J point elevation greater than 1 mm in 2 or more contiguous leads, excluding V1, V2, and V3, in the setting of a QRS duration less than 120 msec.[18] Early repolarization is not associated with PR-segment changes per se. The early repolarization pattern was until recently thought to be a nonpathologic normal variant. However, during the past decade, it has been found to be associated with arrhythmic sudden cardiac death and is thought to be related to Brugada's syndrome.[19-22]

The ECG should always be interpreted in the context of the history, physical examination, and appropriate diagnostic testing; ST-segment elevation acute coronary syndrome is a highly morbid condition, and advanced diagnostic and therapeutic modalities such as biomarker measurement, echocardiography, and coronary angiography may all be necessary to exclude it from the differential diagnosis, especially in high-risk patients.

In this case, the J point elevations in the inferior and lateral leads on the initial tracing (Figure 1) were a result of early repolarization. The ECG findings of pericarditis did not become manifest until a repeat tracing was obtained (Figure 2), and, indeed, electrical changes in pericarditis are highly variable and in some cases may be entirely absent.[23] Our patient's history, including the character of her pain, antecedent upper respiratory infection, and improvement with nonsteroidal anti-inflammatory drugs, cemented the clinical diagnosis, which was confirmed by negative angiogram results and the evolution of her ECGs.

PEARLS

The early repolarization pattern is a common cause of J point and ST-segment elevation that often complicates the diagnosis of ST-segment elevation acute coronary syndrome by both ECG machines and clinicians.

PR-segment depression is useful to differentiate pericarditis and myopericarditis from other causes of chest pain with ST-segment elevation.

PR-segment elevation (knuckle sign) in aVR is another clue to the diagnosis of pericarditis.

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