ONE MINUTE CONSULT

BRIEF ANSWERS TO SPECIFIC CLINICAL QUESTIONS

Q: Should we still use electrocardiography to diagnose pericardial disease?

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A: YES. ACUTE PERICARDITIS has a unique clinical presentation, physical findings, and electrocardiographic (ECG) changes. ECG is always ordered to look for ischemic changes in patients with chest pain. Acute pericarditis develops in stages, which makes it easy to differentiate from early repolarization and, more significantly, myocardial infarction. The ECG changes, along with the clinical presentation and physical findings, can make the diagnosis of pericarditis.

In atypical and complicated cases, advanced imaging studies (ie, echocardiography and cardiac magnetic resonance imaging) have been used to confirm the diagnosis and to follow the course of the disease. However, ECG remains a useful, cost-effective test.

PERICARDIAL DISEASE IS DIVERSE

The pericardium is a thin layer that covers the heart and separates it from other structures in the mediastinum.

Pericardial syndromes include acute, recurrent, constrictive, and effusive-constrictive pericarditis, as well as pericardial effusion with or without tamponade. Causes include viral or bacterial infection, postpericardiotomy syndrome (Dressler syndrome), postmyocardial infarction, primary and metastatic tumors, trauma, uremia, radiation, and autoimmune disease, but pericardial syndromes can also be idiopathic.[1]

*Dr. Klein has disclosed that he has served on a steering committee for clinical trials for Bayer.

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Acute pericarditis is the most common pericardial syndrome and occurs in all age groups. Once diagnosed, it can easily be treated with anti-inflammatory drugs. However, recurrent pericarditis, reported in 30% of patients experiencing a first attack of pericarditis, can be difficult to manage, can have a significant impact on the patient's health, and can be life-threatening.[2]

CHANGES OF ACUTE PERICARDITIS DEVELOP IN STAGES

Pericarditis can be diagnosed on the basis of ECG changes, clinical signs and symptoms, and laboratory and imaging findings.[3] ECG criteria of acute pericarditis have been published.[4,5]

The characteristic chest pain in acute pericarditis is usually sudden in onset and sharp and occurs over the anterior chest wall. The pain is exacerbated by inspiration and decreases when the patient sits up and leans forward.[4]

ECG classically shows a widespread saddle-shaped (upward concave) ST-segment elevation in the precordial and limb leads, reflecting subepicardial inflammation. PR-segment depression (with PR-segment elevation in lead aVR) can accompany or precede the ST changes and is known as the "discordant ST-PR segment sign" (FIGURES 1 AND 2). These changes are seen in 60% of patients.

Acute pericarditis classically causes widespread upward concave ST-segment elevation

The ECG changes develop in stages, making them easy to differentiate from early repolarization and, more significantly, from myocardial infarction. Four stages are apparent[1,4,6–9]:

- **Stage I** occurs in a few hours to days, with diffuse, up-sloping ST-segment elevation and upright T waves, the result of an alteration in ventricular repolarization caused by pericardial inflammation. Because of al-

Postpericardiotomy syndrome (Dressler syndrome)

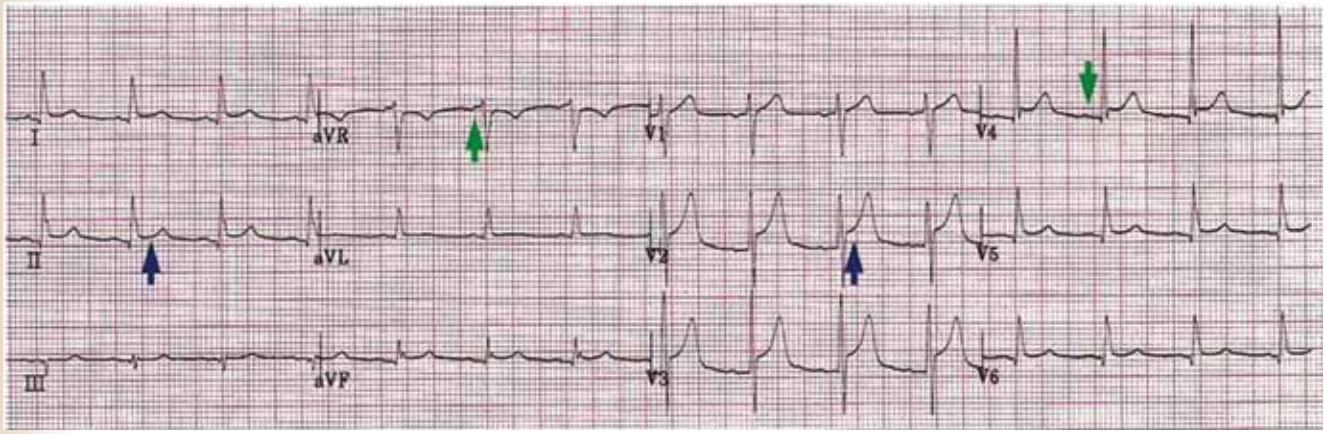

FIGURE 1. Electrocardiogram of a 63-year-old man who presented with chest pain, fever, and a friction rub 4 weeks after cardiac bypass surgery. Note the diffuse up-sloping ST-segment elevations (blue arrows), best seen in II, III, and V_2 to V_6. Subtle PR-segment deviation (positive in aVR and negative in most other leads) is also present (green arrows). Postpericardiotomy syndrome (Dressler syndrome) is acute pericarditis that occurs 4 to 6 weeks after pericardiotomy.

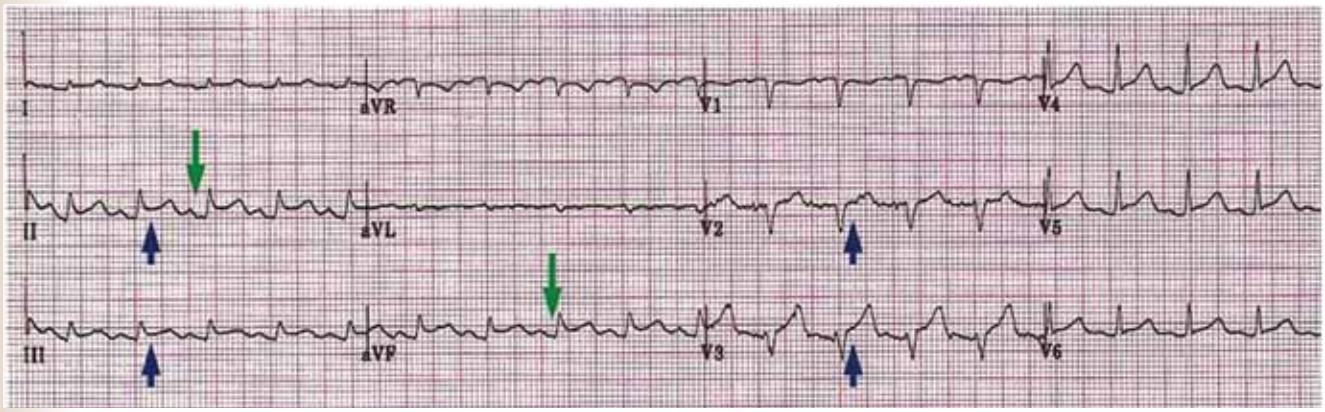

FIGURE 2. Electrocardiogram of a 19-year-old man who developed flu-like symptoms 2 weeks earlier. On the morning of this tracing he had fever, chest pain that worsened with deep breathing, and a pericardial friction rub. Note the diffuse up-sloping ST-segment elevations (blue arrows) and PR-segment depression in leads II and aVF (green arrows). Also note the low QRS amplitude, which suggests pericardial effusion.

teration in repolarization of the atrium secondary to inflammation, the PR segment is elevated in aVR and depressed in the rest of the limb and chest leads.

- **Stage II**—the ST and PR segments normalize.
- **Stage III**—widespread T-wave inversion.
- **Stage IV**—normalization of the T waves.

There is no pathologic Q-wave formation or loss of R-wave progression in acute pericarditis.

The ECG changes of pericarditis vary widely from one patient to another, depending on the extent and severity of pericardial inflammation and the timing of the patient's presentation. Changes vary in duration. In some cases, ST elevation returns to baseline within a few days without T-wave inversions; in other cases, T-wave inversions can persist for weeks to months. Sometimes the abnormalities resolve by the time symptoms develop.

ASSOCIATED CONDITIONS

Myocardial involvement

In acute myocarditis, findings on ECG can be normal unless the pericardium is involved. Changes that can be seen in myocarditis and that indicate a deeper involvement of inflam-

mation include ST-segment abnormalities, arrhythmias (eg, premature ventricular or atrial contractions), pathologic Q waves, intraventricular conduction delay, and right or left bundle branch block.[1,10–12]

Elevated troponin and new focal or global left ventricular dysfunction on cardiac imaging indicates myocarditis, especially in a patient with a normal coronary angiogram.[10–13]

Pericardial effusion: Tachycardia and low QRS voltage

Pericardial effusion is often a complication of pericarditis, but it can also develop from other conditions, such as myxedema, uremia, malignancy, connective tissue disease, aortic dissection, and postpericardiotomy syndrome, and it can also be iatrogenic.

The most common ECG sign of pericardial effusion is tachycardia and low voltage of the QRS complexes. Low voltage is defined as a total amplitude of the QRS complexes in each of the six limb leads less than or equal to 5 mm, and less than or equal to 10 mm in V_1 through V_6. However, low voltage is not always present in the chest leads.

Mechanisms proposed to explain low QRS voltage associated with pericardial effusion include internal short-circuiting of the electrical currents by accumulated fluids within the pericardial sac, greater distance of the heart from body surface electrodes, reduced cardiac size caused by effusion, and change in the generation and propagation of electrical current in the myocardium.[14,15]

Cardiac tamponade: Tachycardia, electrical alternans, low QRS voltage

Sinus tachycardia and electrical alternans are specific but not sensitive signs of pericardial tamponade (**FIGURE 3**).[16,17] Electrical alternans is characterized by beat-to-beat alterations in the axis of QRS complexes in the limb and precordial leads as a result of the mechanical swinging of the heart in a large pericardial effusion.[17] There is evidence to suggest that low QRS voltage is more the result of the tamponade than the effusion.[18]

Treating tamponade with pericardiocentesis, surgical creation of a fistula ("window") between the pericardial space and the pleural cavity, or anti-inflammatory drugs can resolve low QRS voltage within 1 week.

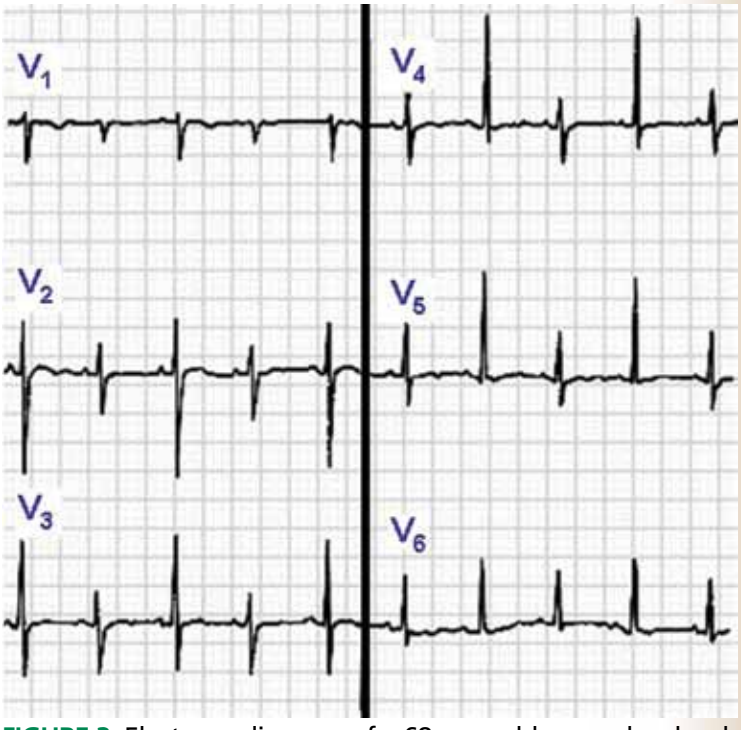

FIGURE 3. Electrocardiogram of a 68-year-old man who developed shortness of breath 1 day after cardiac bypass surgery. Cardiomegaly was found on plain chest radiography. This tracing shows sinus tachycardia with electrical alternans: beat-to-beat alterations in the axis of the QRS complexes, suggesting a large pericardial effusion with tamponade.

DIFFERENTIAL DIAGNOSIS OF ACUTE PERICARDITIS

Acute myocardial infarction

ECG changes in acute pericarditis differ from those in acute myocardial infarction in many ways.

ST-segment elevation in pericarditis rarely exceeds 5 mm, in contrast to acute myocardial infarction, in which ST elevation at the J point has to be more than 2 mm and in two anatomically contiguous leads.[19]

In pericarditis, the changes occur more slowly and in stages, reflecting the evolving inflammation of different areas of the pericardium.

The ST segment is elevated diffusely in the precordial and limb leads in pericarditis, indicating involvement of more than one coronary vascular territory, differentiating it from characteris-

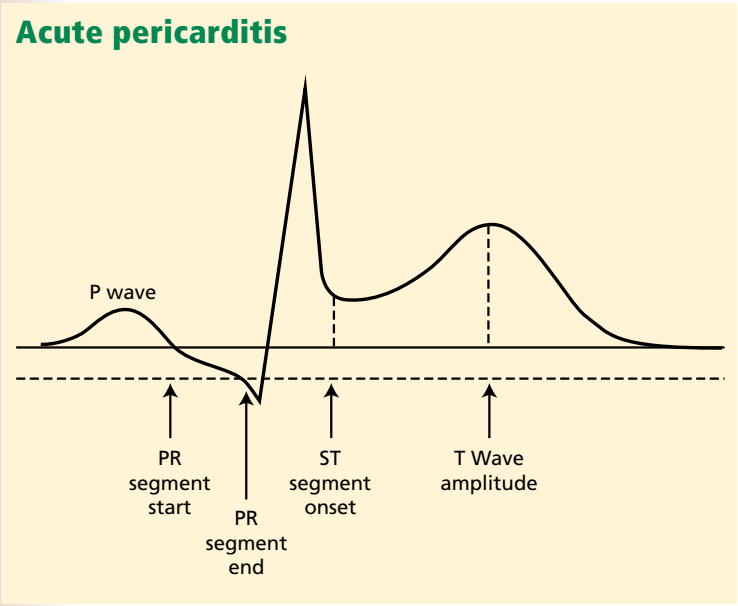

FIGURE 4. A distinctive feature of acute pericarditis is the ratio of ST elevation to T-wave amplitude in leads I, V_4, V_5, and V_6. If the ratio exceeds 0.24, acute pericarditis is present. The end of the PR segment is used as the baseline for the ST-segment onset and T-wave maximal amplitude.

tic regional changes in myocardial infarction.[19,20]

If concomitant atrial injury is present with acute pericarditis, then PR elevation in aVR with PR depression in other leads may be seen.

Finally, pathologic Q waves or high-grade heart block reflects acute myocardial infarction.

Early repolarization: Elevation of the J point

Early repolarization is sometimes seen in healthy young people, especially in black men.

Early repolarization is characterized by elevation of the J point (ie, the junction between the end of the QRS complex and the beginning of the ST segment). Elevation of the J point causes elevation of the ST segment in the mid to lateral precordial leads (V_3–V_6) with an upright T wave.[21]

Acute pericarditis tends to cause ST-segment elevation in both the limb and precordial leads, whereas ST elevation in early repolarization mainly involves the lateral chest leads.

The PR segment is more prominent in acute pericarditis, especially in lead aVR.

Another finding that strongly favors acute pericarditis is the ratio of the height of the ST-segment junction to the height of the apex of the T wave of more than 0.25 in leads I, V_4, V_5, and V_6 (**FIGURE 4**).[5,8,22]

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