Acute Pericarditis: Appendicitis of the Heart?

Acute pericarditis is a common cardiovascular condition that is largely self-limited and effectively treated with nonsteroidal anti-inflammatory therapy. Despite simple and effective treatment, the diagnosis is often challenging because clinical symptoms can be misleading. Classically, acute pericarditis presents with sharp pleuritic chest pain that often is positional and varies with respiration. However, pericardial pain can also manifest as dull radiating chest discomfort that may mimic symptoms of myocardial ischemia.

Physical examination at bedside can provide insight, most notably by the presence of a pericardial friction rub. However, only a minority of patients will have an audible rub at presentation.[1,2] Auscultatory findings, if present, tend to be transient and variable. A study of a cohort of patients with acute pericarditis confirmed poor sensitivity of a pericardial friction rub, which was found in only 35% of the cohort.[1] Despite the obvious limitations of physical findings, a pericardial friction rub is still one of the key diagnostic criteria.[3]

The characteristic electrocardiographic pattern is considered part of the diagnostic armamentarium, but its specificity is less clear.[3] Because of global superficial myocardial inflammatory injury, serial electrocardiographic changes occur, usually commencing with PR depression (PR elevation in lead aVR), followed by diffuse ST-segment elevation. ST-segment elevation is noted in 65% to 70% of all cases of acute pericarditis, but it is often difficult to distinguish from ST-segment elevation myocardial infarction (STEMI).[2,4] Although there can be differentiating electrocardiographic traits, providing a complete assessment may be difficult because many features in pericarditis are temporal.[5,6] Reciprocal ST-segment changes may favor STEMI but are not always present. If an acute myocardial infarction (AMI) is considered as an alternative diagnosis, time for treatment may be limited, and additional strategies will be needed to determine the cause of the patient's condition.

Cardiac biomarkers are frequently considered part of the diagnostic tool kit but are sometimes elevated in patients with acute pericarditis because of the inflammatory process involving the epicardium with subsequent myocardial necrosis. In fact, the incidence of elevated cardiac troponin I levels in patients with viral or idiopathic acute pericarditis has been reported to be 32.2%; of these patients, 23.7% had a troponin I level at admission that was beyond the AMI threshold.[4] Furthermore, the temporal relationship of troponin elevation may be remarkably similar to that seen in AMI.[4,7] The prognostic implication of elevated troponin levels is largely benign in acute pericarditis.[4] An elevated cardiac biomarker in pericardial disease is not unusual and further complicates the diagnosis, raising suspicion for alternative etiologies of troponin elevation.

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Continued efforts in cardiac imaging have advanced assessments of pericardial disease. Chest computed tomography and cardiac magnetic resonance imaging offer superior image quality of the pericardium, although access is often limited and delayed. Echocardiography provides a relatively simple, noninvasive assessment of the pericardium at the time of patient presentation. Although the presence of a pericardial effusion is common, the absence of an effusion does not exclude diagnosis. Pericardial effusions are present in approximately 60% of cases of acute pericarditis, with 80% being mild, 10% being moderate, and 10% being severe.[1] Thus, bedside echocardiography may be of particular benefit, especially if there are diagnostic dilemmas. If ischemia is suspected, regional wall motion abnormalities suggest an ischemic process. However, a substantial number of patients will have no identifying features, which may prompt further diagnostic modalities such as coronary angiography when the diagnosis is in doubt.

In this issue of *Mayo Clinic Proceedings*, Salisbury et al[8] provide great insight into both the frequency and the predictors of urgent coronary angiography in patients with acute pericarditis. This single-center retrospective analysis involved adult patients with viral or idiopathic pericarditis who had electrocardiographic changes that were potentially compatible with pericardial inflammation. Of a total of 238 patients, 16.8% underwent diagnostic coronary angiography; higher frequencies were noted in those with ST-segment elevation on their presenting electrocardiogram. Using univariate logistic regression, positive predictors of coronary angiography included typical angina, ST-segment elevation on the index electrocardiogram, previous percutaneous coronary intervention, elevated troponin T level on admission, diaphoresis, and male sex. Negative predictors were pleuritic or positional chest pain.

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The study by Salisbury et al is the most thorough study published that describes the frequency of coronary angiography in patients with acute pericarditis. Certainly, it adds to the growing body of evidence supporting alternative conditions that result in ST-segment elevation.[9] Recently, a 14% "false-positive" rate of cardiac catheterization was found among patients with suspected STEMI.[10] In patients with negative cardiac biomarkers, common causes include early repolarization, nondiagnostic electrocardiography, pericarditis, and previous myocardial infarction. In patients with positive cardiac biomarkers, stress cardiomyopathy, myocarditis, and STEMI due to emboli/spasm may present as alternative conditions.[10]

Performing urgent coronary angiography may be necessary when the diagnosis of acute pericarditis is uncertain. Symptoms may be misleading, and the electrocardiogram may be suggestive of an acute coronary syndrome (ACS). In fact, in approximately one-third of patients with myopericarditis, symptoms may suggest an ACS.[11] In the current era of prompt reperfusion therapy, performing urgent angiography may be necessary to exclude AMI. The major complication rate of diagnostic cardiac catheterization for death, myocardial infarction, or stroke is well below 1%.[12] Thus, the risk-benefit profile of patients with suspected ACS may favor diagnostic cardiac catheterization.

The study by Salisbury et al reports a 4.8% rate of prior thrombolytic therapy in patients with acute pericarditis who were transferred to Mayo Clinic. Others have reported thrombolytic therapy rates as high as 19% for patients with myopericarditis that was mistaken for AMI.[13] Given the heightened risk of pericardial effusion and tamponade after fibrinolytic therapy in patients with pericarditis, thrombolytic therapy should be avoided.[14] In fact, patients in whom the diagnosis of pericarditis is uncertain should be promptly transferred for cardiac catheterization.

The important observations by Salisbury et al remind us of the diagnostic challenges we still face with acute pericarditis.[8] As such, AMI remains a plausible alternative diagnosis. Thus, diagnostic coronary angiography still plays an important role in discrimination and risk stratification. However, other diagnostic modalities should be considered during the acute phase of clinical presentation. Although electrocardiography may duplicate ST-segment ischemic changes, the progressive sequence of ST-T changes through 4 stages is well established.[15] As well, PR-segment deviations are usually seen on the initial electrocardiogram and are present in approximately 80% of patients with acute pericarditis.[5,16] Echocardiography may be useful for excluding STEMI, which often presents with wall motion abnormalities. In fact, Salisbury et al report an exceedingly low rate of regional wall motion abnormalities (4.5%) in approximately half of their patients with pericarditis who underwent echocardiography before receiving treatment.[8] This rate is consistent with previously reported rates of 7% of patients with acute pericarditis having echocardiographic diffuse or localized abnormal ventricular wall motion.[2,4] Cardiac biomarkers may be of limited benefit and should not be relied on once STEMI is considered because of restricted time assessments.

Differentiating pericarditis from STEMI can be challenging. This situation is similar to removing a normal appendix so that a real case of acute appendicitis will not be overlooked. Occasionally, it is necessary to perform cardiac catheterization in a patient with acute pericarditis to rule out AMI.

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