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Acute Pericardits and its Diagnosis

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Abstract: The normal pericardium is a double-layered sac; the visceral pericardium is a serous membrane that is separated by a small quantity (15–50 mL) of fluid, an ultrafiltrate of plasma, from the fibrous parietal pericardium. The normal pericardium, by exerting a restraining force, prevents sudden dilation of the cardiac chambers, especially of the right atrium and ventricle, during exercise and with hypervolemia. Acute pericarditis, by far the most common pathologic process involving the pericardium, may be classified both clinically and etiologically.

Keywords: pain, pericardit, electrocardiograph, echocardiography, pericardial friction rub, cardiac tamponade.

Introduction

Pain, a pericardial friction rub, electrocardiographic changes, and pericardial effusion with cardiac tamponade and paradoxical pulse are cardinal manifestations of many forms of acute pericarditis. Chest pain is an important but not invariable symptom in various forms of acute pericarditis; it is usually present in the acute infectious types and in many of the forms presumed to be related to hypersensitivity or autoimmunity. Pain is often absent in slowly developing tuberculous, postirradiation, neoplastic, or uremic pericarditis. The pain of acute pericarditis is often severe, retrosternal and left precordial, and referred to the neck, arms, or the left shoulder. Often the pain is pleuritic, consequent to accompanying pleural inflammation, i.e., sharp and aggravated by inspiration, coughing, and changes in body position, but sometimes it is a steady, constricting pain that radiates into either arm or both arms and resembles that of myocardial ischemia; therefore, confusion with acute myocardial infarction (AMI) is common. Characteristically, however, pericardial pain may be relieved by sitting up and leaning forward and is intensified by lying supine. The differentiation of AMI from acute pericarditis becomes perplexing when, with acute pericarditis, serum biomarkers of myocardial damage such as creatine kinase and troponin rise, presumably because of concomitant involvement of the epicardium in the inflammatory process (an epi-myocarditis) with resulting myocyte necrosis. However, these elevations, if they occur, are quite modest, given the extensive electrocardiographic ST-segment elevation in pericarditis. This dissociation is useful in the differentiation between these conditions.

The pericardial friction rub, audible in about 85% of patients, may have up to three components per cardiac cycle, is high-pitched, and is described as rasping, scratching, or grating; it can be elicited sometimes only when the diaphragm of the stethoscope is applied firmly to the chest wall at the left lower sternal border. It is heard most frequently at end-expiration with the patient upright and leaning forward.The rub is often inconstant, and the loud to-and-fro leathery sound may disappear within a few hours, possibly to reappear on the following day. A pericardial rub is heard throughout the respiratory cycle, whereas a pleural rub disappears when respiration is suspended.

The electrocardiogram (ECG) in acute pericarditis without massive effusion usually displays changes secondary to acute subepicardial inflammation. It typically evolves through four stages. In stage 1, there is widespread elevation of the ST segments, often with upward concavity, involving two or three standard limb leads and V2 to V6, with reciprocal depressions only in aVR and sometimes V1, as well as PR-segment depression. Usually there are no significant changes in QRS

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complexes. In stage 2, after several days, the ST segments return to normal, and only then,or even later,do the T waves become inverted (stage 3). Ultimately, weeks or months after the onset of acute pericarditis, the ECG returns to normal in stage 4. In contrast, in AMI, ST elevations are convex, and reciprocal depression is usually more prominent; QRS changes occur, particularly the development of Q waves, as well as notching and loss of R-wave amplitude; and T-wave inversions are usually seen within hours before the ST segments have become isoelectric. Sequential ECGs are useful in distinguishing acute pericarditis from AMI. In the latter, elevated ST segments return to normal within hours. Early repolarization is a normal variant and may also be associated with widespread ST-segment elevation, most prominent in left precordial leads. However, in this condition, the T waves are usually tall and the ST/T ratio is importantly, this ratio is higher in acute pericarditis. Depression of the PR segment (below the TP segment) is also common and reflects atrial involvement.

Diagnosis: echocardiography is the most effective imaging technique available since it is sensitive, specific, simple, noninvasive, may be performed at the bedside, and can identify accompanying cardiac tamponade. The presence of pericardial fluid is recorded by two-dimensional transthoracic echocardiography as a relatively echo-free space between the posterior pericardium and left ventricular epicardium in patients with small effusions, and as a space between the anterior right ventricle and the parietal pericardium just beneath the anterior chest wall in those with larger effusions. In the latter the heart may swing freely within the pericardial sac. When severe, the extent of this motion alternates and may be associated with electrical alternans. Echocardiography allows localization and estimation of the quantity of pericardial fluid. The diagnosis of pericardial fluid or thickening may be confirmed by computed tomography (CT) or magnetic resonance imaging (MRI). These techniques may be superior to echocardiography in detecting loculated pericardial effusions, pericardial thickening, and the presence of pericardial masses.

Cardiac tamponade. The accumulation of fluid in the pericardial space in a quantity sufficient to cause serious obstruction to the inflow of blood to the ventricles results in cardiac tamponade.This complication may be fatal if it is not recognized and treated promptly. The three most common causes of tamponade are neoplastic disease, idiopathic pericarditis, it should be considered in any patient with hypotension 223 and elevation of jugular venous pressure. Otherwise unexplained enlargement of the cardiac silhouette (especially in subacute or chronic tamponade), reduction in amplitude of the QRS complexes, and electrical alternans of the P, QRS, or T waves each should raise the suspicion of cardiac tamponade.

Paradoxical Pulse: This important clue to the presence of cardiac tamponade consists of a greater than normal (10 mmHg) inspiratory decline in systolic arterial pressure. When severe, it may be detected by palpating weakness or disappearance of the arterial pulse during inspiration, but usually sphygmomanometric measurement of systolic pressure during slow respiration is required. Since both ventricles share a tight incompressible covering, i.e., the pericardial sac, the inspiratory enlargement of the right ventricle in cardiac tamponade compresses and reduces left ventricular volume; leftward bulging of the interventricular septum further reduces the left ventricular cavity as the right ventricle enlarges during inspiration. Thus in cardiac tamponade the normal inspiratory augmentation of right ventricular volume causes an exaggerated reciprocal reduction in left ventricular volume. Also, respiratory distress increases the fluctuations in intrathoracic pressure, which exaggerates the mechanism just described. Right ventricular infarction may resemble cardiac tamponade with hypotension, elevated jugular venous pressure, an absent y descent in the jugular venous pulse, and, occasionally, pulsus paradoxus. Paradoxical pulse occurs not only in cardiac tamponade, but also in approximately one-third of patients with constrictive pericarditis. This physical finding is not pathognomonic of pericardial disease because it may be observed in some cases of hypovolemic shock, acute and chronic obstructive airways disease, and pulmonary embolus. Low-pressure tamponade refers to mild tamponade in which the intrapericardial pressure is increased from its slightly subatmospheric levels to +5 to +10 mmHg; in some instances, hypovolemia coexists. As a consequence, the central venous pressure is normal or only slightly elevated, whereas arterial pressure is unaffected and there is no paradoxical pulse. The patients are asymptomatic or complain

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of mild weakness and dyspnea.The diagnosis is aided by echocardiography, and both hemodynamic and clinical manifestations improve after pericardiocentesis.

In conclusion, acute pericarditis is painful inflammation of the pericardium, the fluid-filled pouch surrounding your heart. The pain usually gets worse when you're lying down or when you breathe in. Depending on the cause, it's almost always treatable, and most people with this condition will recover with few or no complications.

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