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Pericarditis: Are There Any Changes Since 1761? A Panoramic View of Pericarditis

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Pericardial diseases have attracted the attention of physicians for centuries, who have tirelessly compared its physical findings with their observations and comments (1–3).

The parietal pericardium, composed of collagen and elastin fibers, is sac of approximately 2 mm thickness that covers the entire heart and the exit areas of the major vessels. It contains approximately 30–40 ml of serous fluid (4, 5). It keeps the heart in a fixed position in the thorax with connections to surrounding tissues and forms a mechanical barrier against the enlargement of the heart cavities and the spread of infections.

Physiologically, the right ventricular diastolic pressure affects the diastolic pressure of the left ventricle via the interventricular septum. If the right ventricular pressure increases (right ventricular infarction, pulmonary embolism, etc.), the pericardium makes this diastolic interaction more pronounced and the pulses paradoxus and Kussmaul signs (increased venous fullness of the neck in the imperium) occur. Since the pericardium is dilated during chronic dilatation of the heart cavities, it does not prevent diastolic filling of the heart (4, 5).

Acute Pericarditis

Pericardial diseases usually begin with acute pericarditis and continue as benign acute-course pericarditis or myocarditis. They may also result in pericardial effusion, tamponade, and constrictive pericarditis. Acute pericarditis is an inflammatory condition characterized by edema, thickening, pericardial fluid, and pericardial frotman (5, 6). It is responsible for 5% of non-ischemic chest pain and is associated with myocarditis in 15% of patients (7–9). Pleuritic pain, dyspnea, and cough are common symptoms of pericarditis and myocardial ischemia, muscle pain, gastro-esophageal pain, pneumonia, and pulmonary embolism should be considered in the differential diagnosis. In addition, the patient may be asymptomatic or may present with systemic infection (4, 7). Friction in the pericardium (frotman) is pathognomonic for the diagnosis of this disease. In 60% of patients, there is transient ST-elevation and/or PR-depression with an opening upward in other leads except aVR and V1 (9). Since the pericardium is electrically silent, 90% of patients with electrocardiography (ECG) findings can be detected with myocarditis and troponin height is also available to help make a diagnosis (9). Telecardiography usually does not show any features for pericarditis. Although ejection fraction is normal on the echocardiograph, pericardial fluid may be seen in 60% of patients. Computed tomography (CT) or magnetic resonance imaging (MRI) may be required to detect pericardial thickening (5, 6).

Diagnosis and Treatment

There are four important criteria for the diagnosis of acute pericarditis. If two of these are present, acute pericarditis can be diagnosed. These include: chest pain, pericardial frotman, appropriate ECG changes, and visible pericardial fluid in the echocardiograph (5). Complete blood count, serum creatinine, CRP, troponin level, and lung radiographs are the routine confirmatory tests. The prognosis of idiopathic pericarditis, which makes up 60%–90% of acute pericarditis in Western societies, is extremely accurate and to responds well to non-steroidal anti-inflammatory drugs (NSAIDs) (5, 6, 9). Ibuprofen 600–800 mg 3 times a day for 1–2 weeks, aspirin 750–1000 mg 3 times a day for 1–2 weeks, or colchicine 0.5–0.6 mg 2 times a day for 3 months treatment is sufficient for many patients. Corticosteroids 0.2–0.5 mg/kg/day may be used for 6–12 weeks in patients who do not respond to NSAIDs or colchicine, or if these drugs are contraindicated (5). There is no need for hospitalization in patients who respond to initial treatment and/or show a low level of pericardial fluid. Patients with excess effusion or an additional suspected cause of pericarditis other than idiopathic factors should be hospitalized.

In acute pericarditis, a specific cause is detected in approximately 20% of patients. After approximately 6 years of follow-up of all acute pericarditis patients, 1.8% of the constriction and 3% of tamponade is seen. Patients with idiopathic pericarditis may develop contraction at 0.5% (9).

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Recurrent Pericarditis

Recurrent pericarditis may occur 1–1.5 months or more after (up to 18 months) discontinuation of anti-inflammatory therapy (5–7). The recurrence of acute idiopathic pericarditis at 15%–30% may be seen, and drugs that were used in the first attack can be used again. Steroids can be added if there is no response to treatment. Colchicine is continued for 6–12 months. In intractable cases, azathioprine, human immunoglobulins, or anakinra may be added to the treatment (4). Pericardiotomy may be required in cases that do not improve despite all forms of conservative medical treatment.

Pericardial Effusion and Cardiac Tamponade

Interventional cardiological procedures, cardiac surgery, heart failure, pulmonary hypertension, malignancy, and acute pericarditis all cause pericardial effusions. However, there is another relevant cause (5, 10). While slow-accumulating pericardial effusion in the long term does not produce any symptoms, rapidly accumulating fluid of 150–200 cc can impair cardiac functions by acting as a barrier to diastolic expansion. If the amount of fluid accumulation is significant, right-left atrium and ventricle pressures are synchronized. Cardiac contractility increases as a result of increased sympathetic activity as a compensation mechanism, but the volume of beats does not increase due to ventricular filling restriction. Pulses paradoxus (having more than 10 mmHg drop in deep inspiration in systolic blood pressure), seen in constrictive pericarditis and pulmonary embolism, is also an important finding. However, in people with a previous history of hypertension, pulsus paradoxus may not be seen (4). The loss of the y-wave in the right atrium pressure register is important for the diagnosis of tamponade.

Clinical Findings

Pericardial effusion is usually asymptomatic if it does not cause cardiac tamponade. Although there is no pulmonary congestion, it is noteworthy that dyspnea is a common complaint. Hypotension, deep heart sounds (muffled), and jugular venous fullness (Beck's triad) are important signs of tamponade. Along with the pericardial friction rub in the pericardium, a tubular respiratory sound in the left back can be heard. As cardiac output decreases, cold extremities, peripheral cyanosis, tachypnea, tachycardia, and shock may be seen. Pulses paradoxus should be investigated if tamponade is suspected (4, 5).

Laboratory Tests

In pericarditis findings on the ECG, electrical alternans and low voltage criteria can be seen (4, 5). There is no abnormality seen in the antero-posterior chest X-ray, except for a small amount of effusion. If there is a large amount of effusion, the heart is seen as a balloon filled with water (flask-like) and the lungs are oligemic. Cardiac pulsation disappears during fluoroscopy. In echocardiography, it is diagnostic of pericarditis to see less (<10 mm), moderate (10–20 mm), or severe (>20 mm) pericardial effusions during diastole. Collapse seen in the early diastole in the right ventricle wall and the late diastole in the right atrium wall are important for the diagnosis of tamponade (11). CT or Magnetic resonance imaging (MRI) may be helpful if echocardiography is insufficient, especially if there is pleural effusion or pulmonary hypertension combined with loculated effusion. CT and MRI are also more sensitive in assessing pericardial thickness.

Treatment

If tamponade has been detected, the pericardiocentesis procedure should not be delayed. Where no tamponade is detected, NSAID or cortisone and colchicine can be used together and the response to treatment can be frequently followed up, on the basis of which the need for pericardiocentesis can be decided. There is no need to perform diagnostic pericardiocentesis if there is a disease that can explain the effusion. If pericardiocentesis is needed to be delayed in the presence of tamponade, intravenous saline is started and attempts are made to increase the filling pressure (5). Echocardiographic follow-up should be performed after pericardiocentesis and the catheter should not be removed immediately. In the presence of trauma, aortic dissection, rupture after myocardial infarcts, locule effusion, and widespread fibrous band detection, closed pericardiocentesis should be avoided and open pericardiocentesis should be preferred. Percutaneous balloon pericardiotomy is the preferred choice for effusions due to malignancies and frequent recurrent effusions. Hematocrit, leukocyte count, and protein level should be studied from the sample taken, after which the culture should be taken, staining should be done, and presence of malignant cells should be investigated (5). For tuberculosis, adenosine deaminase (ADA) and polymerase chain reaction examinations should be performed (5, 12).

Constrictive Pericarditis

Constrictive pericarditis may be caused by acute pericarditis, chest radiographs, complications related to cardiac surgery, or idiopathic factors. Thickening, fibrosis, and calcification are seen in the parietal pericardium and the parietal and visceral pericardium may stick together. Pericardial thickness is normal in 20%–40% of patients (5, 11).

Due to the decrease in pericardial elasticity, filling of the heart with blood is limited and the right and left atrial mean pressures and the ventricular diastolic pressures are equalized. It is important to increase right ventricular filling, decrease left ventricular filling, and shift the interventricular septum to the right during diastole. Since ventricular relaxation is not sufficient in diastole, the ejection fraction decreases and the kidneys begin to retain water and salt due to decreased cardiac output (5). Fatigue and muscle weakness are manifested. Systemic venous congestion leads to peripheral edema and acid accumulation in the abdomen.

Clinical and Laboratory Findings

Generally, right heart failure, peripheral edema, and congestion in the liver are prominent. However, cough and dyspnea are also seen due to left heart failure. Constrictive pericarditis should be considered in patients with right heart failure (4). Presence of the Kussmaul sign and decrease in inspiratory venous pressure is seen in most patients (4) along with the rapid "y" descent seen in right atrial pressure recordings. Pericardial knock after the second heart sound is an important finding.

No specific findings are seen on the ECG and in telecardiography. Biatrial dilatation, septal bounce, dilation of hepatic veins, and pericardial thickening are important findings in echocardiographic evaluation. An increase in mitral E-wave velocity by more than 25% is an important finding. Increased E-wave in the mitral medial annulus seen using a tissue Doppler is a valuable diagnostic finding (11). Right and left heart pressures are similar in constrictive peri-

carditis, while a change of 3–5 mmHg could be noticeable. The square root mark is detected on the ventricular pressure track. CT and MRI are the most reliable methods for demonstrating pericardial thickness and calcification.

Treatment

Surgical removal of the pericardium is the most important treatment option with 2–20% mortality. After this intervention, which should be done without delay, 60% of patients also show improvement in diastolic functions. Beta blockers should not be given to these patients due to compensatory sinus tachycardia (4).

Effusive constrictive pericarditis

It is a combination of pericardial effusion and constriction. The diagnosis is made when the right atrial pressure does not decrease despite pericardial effusion evacuation. Although tuberculosis, radiation, and malignancy are common causes, it may be an idiopathic condition (13).

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