ACUTE PERICARDITIS AND CARDIAC TAMPONADE

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Abstract

Acute pericarditis is caused by an inflammatory process in the pericardial tissue, and as a response to the inflammation, a pericardial effusion occurs. Acute accumulation of even smaller amounts of pericardial fluid can lead to cardiac tamponade. Pericardiocentesis is a procedure used to urgently evacuate pericardial effusion and treat patients with cardiac tamponade.

Keywords: acute pericarditis, pericardial effusion, cardiac tamponade, pericardiocentesis

Introduction

Diseases of the pericardium can be categorized based on the clinical course into acute (lasting less than 6 weeks), subacute, and chronic (lasting more than 6 months). According to clinical presentation, pericardial diseases are classified into syndromes: acute pericarditis, pericardial effusion and cardiac tamponade, recurrent pericarditis, and constrictive pericarditis (transient and chronic). It is well-known from clinical practice that patients with acute pericarditis, in whom pericardial effusion does not develop, generally have a favorable prognosis. However, progressive accumulation of even a small amount of fluid in the pericardial space can lead to cardiac tamponade within a few hours of the onset of initial symptoms of the disease[1, 2]. Clinically, this disease most commonly presents with dyspnea, hypotension, tachycardia, distended neck veins, muffled heart sounds, and paradoxical pulse[3, 4]. This urgent condition requires prompt evacuation of the pericardial effusion. If intervention is delayed, it can lead to cardiac arrest in diastole as a result of cardiac tamponade and a fatal clinical outcome.

Acute pericarditis

The visceral pericardium is a serous membrane separated by a small amount (15-50 mL) of fluid (ultrafiltrate of plasma) from the fibrous portion - the parietal pericardium. In healthy individuals, the pericardium prevents sudden dilation of the cardiac chambers during exertion and in cases of hypervolemia. As a result of the normally negative intrapericardial pressure during the ejection phase of the cardiac cycle, the pericardium assists with atrial filling. Additionally, the pericardium maintains the anatomical position of the heart, reduces friction between the heart and surrounding structures, prevents kinking of major blood vessels, and hinders the spread of infection from the lungs and pleural space to the heart. Acute pericarditis is the most common pathological process affecting the hearts sac. It has been established that various types of viruses, such as Coxsackie virus, adenovirus, and echovirus, can induce acute inflammation of the cardiac sac[5, 6]. In clinical autopsies, acute pericarditis can be found as an incidental finding in about 1% of patients. The prevalence of pericarditis in the general population, regardless of the disease's etiology, is estimated to be around 0.2% to 0.5%[4, 7].

Acute pericarditis is most commonly a result of infection by cardiotropic viruses, less frequently by bacteria and other microorganisms, but it can also be caused by local or systemic autoimmune reactions, neoplastic infiltration, and metabolic disorders. Acute inflammation of the cardiac sac rarely occurs following radiotherapy for chest neoplasms or after fungal and parasitic infections.

The inflammatory process in pericardial tissue leads to severe pain due to the dense network of nerve endings from the phrenic nerve (*n. phrenicus*), which also causes the pain to radiate to the trapezius muscle ridge. Changes in the electrocardiogram, as well as potential disturbances in rhythm and conduction, result from the inflammatory reaction in the myocardium, as the pericardium is electrically neutral. Pericardial effusion occurs in acute pericarditis as a response to inflammation. A normal pericardium is permeable to water and electrolytes, and pericardial fluid is in dynamic equilibrium with the blood. Inflammation in the pericardial tissue disrupts this balance. Local production of inflammatory mediators stimulates fluid production from the visceral pericardium and the release of large molecules that attract additional fluid and hinder its resorption. The diagnosis of acute pericarditis is made if at least two of the following symptoms or signs are present: chest pain that intensifies upon inhalation, changes with body movements, and radiates to the trapezius ridge; pericardial friction; typical ECG changes (diffuse concave ST elevation and PR depression,

Image 1. Typical electrocardiographic changes most commonly involve diffuse concave ST elevation with PR segment depression - left image. Radiographic finding of pericardial effusion - right image.

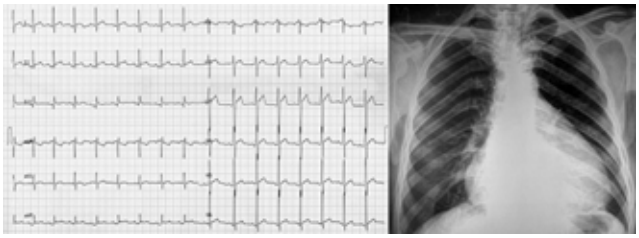


Image 1); echocardiographically diagnosed pericardial effusion, and elevated C-reactive protein (CRP).

The treatment of acute pericarditis caused by viral infections is aimed at alleviating disease symptoms, preventing complications, and addressing the underlying cause of the illness. Initially, analgesic and anti-inflammatory therapy is administered as long as the patient has chest pain, followed by a gradual tapering off of the medication. High doses of nonsteroidal anti-inflammatory drugs (such as ibuprofen 600-800 mg, three times a day; indomethacin 25-50 mg, three times a day) or acetylsalicylic acid 750-1.000 mg, three times a day, are the mainstay of therapy for acute pericarditis. Simultaneously, colchicine is administered for the following three months at a dose of 0.5 mg twice a day, or half that dose for patients weighing less than 70 kg (0.25 mg, twice a day). In the case of recurrent forms, colchicine therapy is extended to six months or longer, along with proton pump inhibitor protection[6, 8, 9]. Corticosteroids are not recommended in the acute phase of the disease as they increase the risk of recurrence. These drugs are commonly used in patients for whom first-line therapies do not lead to an adequate therapeutic response. In some patients, the use of biological therapy is possible (such as IL-1 receptor antagonists like anakinra). However, when corticosteroids (0.2-0.5 mg/kg/day of methylprednisolone) and anakinra (2 mg/kg/day up to 100 mg/day) are employed, careful monitoring for potential complications, including the development of bacterial superinfections, is necessary[4, 6, 10]. Biological therapy is still a subject of ongoing research, but there are already clear clinical confirmations of its effectiveness and benefits, particularly in patients with recurrent pericarditis. When disease recurrences are frequent, multiple, debilitating, and last for more than two years, surgical treatment - pericardiectomy - is often considered.

Cardiac tamponade

Cardiac tamponade refers to the accumulation of a certain amount of pericardial effusion that leads to obstruction of blood flow into the heart. The most common etiological factors causing this condition are neoplastic and viral pericarditis. Tamponade can also result from intrapericardial bleeding (trauma, neoplasms, aortic dissection, post-infarction ruptures, and heart perforations during interventional procedures). In clinical practice, it is recognized that the acute accumulation of a small amount of pericardial fluid, as little as 200 mL, can lead to cardiac tamponade. With chronic fluid accumulation, the pericardium can tolerate up to 2.000 mL of effusion, depending on myocardial thickness, parietal pericardium, and intracardiac pressures. The classic Beck's triad, hypotension, increased venous pressure, and soft heart sounds, are usually found only in severe, acute cardiac tamponade[2, 11]. When the disease develops slower, clinical manifestations resemble heart failure (dyspnea, orthopnea, enlarged liver, swollen jugular veins). It is important to note that such patients never have congestive changes auscultated in the lungs[4, 12]. Echocardiographically, during the development of cardiac tamponade, the collapse of the right atrium is detected first, followed by the right ventricle, and less commonly the compression of the left atrium and left ventricle. In cases of large effusions, the phenomenon of "heart swinging" or "floating heart" can be observed (Image 2).

Image 2. Echocardiographic finding in impending cardiac tamponade. The "swinging heart" or "floating heart" sign, where the heart moves like a pendulum within a large pericardial effusion. **RV** - Right Ventricle; **RA** - Right Atrium; **LV** - left ventricle; **LA** - left atrium.

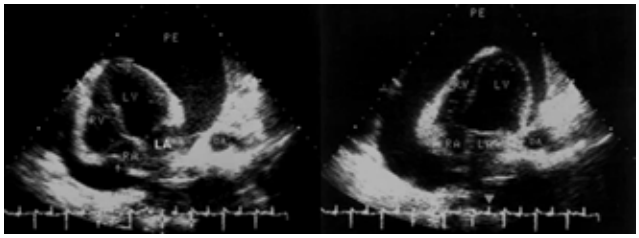


Image 3. Pericardiocentesis kit (left image). Intercoastal puncture is performed along the upper border of the lower rib to avoid injury to intercostal arteries (right image).

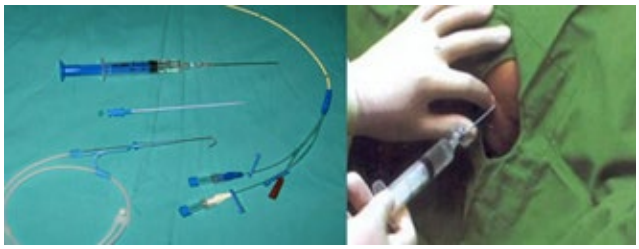
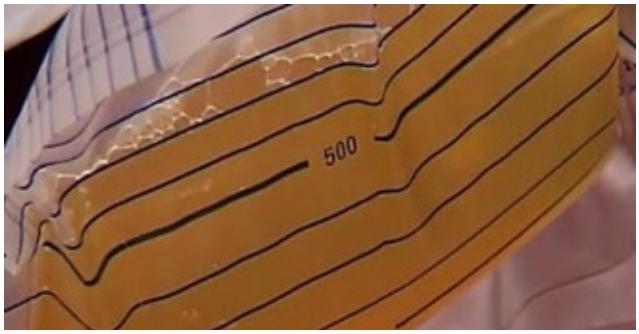


Image 4. Macroscopic presentation of pericardial effusion during pericardiocentesis. Large serous effusion immediately after evacuation from the pericardial space into the drainage bag.



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Cardiac tamponade is an absolute indication for urgent pericardial drainage through pericardiocentesis (Image 3) or surgical intervention - pericardiotomy. Medication therapy serves as a temporary measure until the pericardial effusion is evacuated. Hypovolemic patients receive continuous fluid infusions. Patients who are hypoxic and at risk of respiratory arrest must be intubated and ventilated while preparing for pericardiocentesis[13, 14].

For every patient following pericardiocentesis, it is essential to perform a comprehensive biochemical, bacteriological, and cytological analysis of the pericardial fluid to determine the cause of the pericardial effusion (Image 4).

Conclusion

The prognosis for patients with acute pericarditis is generally good. Acute pericarditis can occur in patients of any age, but it is more common in younger individuals and males. The symptoms of the disease are usually mild to moderate, but in some patients, initial discomfort is pronounced, and pericardial friction is often present. The illness typically lasts from a few days to four weeks, but one or more recurrences may occur in approximately a quarter of patients. Although a significant pericardial effusion is detected in some patients, cardiac tamponade rarely occurs.

Constrictive pericarditis is a rare but possible complication, most commonly associated with disease recurrences. Urgent clinical assessment by an experienced invasive pericardiologist is considered crucial in managing such severe cases. Echocardiography remains the gold standard for rapidly detecting patients with acute cardiac tamponade, alongside standard clinical, electrocardiographic, and radiographic evaluation. Pericardiocentesis, performed via subxiphoid or apical access, is the procedure of choice for the urgent management of patients with cardiac tamponade. Delaying this procedure in the acute phase of the disease can lead to cardiac arrest and a fatal clinical outcome.

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