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Acute Pericardial Disease

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Acute pericardial diseases include acute pericarditis and cardiac tamponade. Both conditions can be associated with acute hemodynamic instability or cardiac failure and require immediate diagnostic workup and treatment.

The pericardium surrounds the heart with two layers. The outer fibrous layer is called the parietal pericardium; the inner layer, which covers the cardiac surface, is the serous visceral pericardium. The pericardium is attached to the sternum and diaphragm by ligamentous bindings. Up to 50 mL of fluid produced by visceral pericardial cells is normally present in the pericardial space (1).

The pericardium can be affected by a wide variety of microorganisms, a nonspecific inflammatory process, and various heart and systemic diseases. Irrespective of the etiology, clinical manifestations of the pericardial diseases correspond to the pericardial inflammation, pericardial effusion with cardiac tamponade, and pericardial constriction due to calcifications (2).

Acute Pericarditis

A number of diseases, syndromes, and agents can produce a clinical syndrome termed acute pericarditis, which is the consequence of the inflammation of the pericardial layers. In a majority of cases the histopathologic changes show hyperemia, increased microvasculature, accumulation of leukocytes, deposition of fibrin, and adhesions that can be formed between layers of pericardium and adjacent structures. If an acute pericarditis is accompanied by myocarditis or pericardial effusion it can present with symptoms of acute heart

failure. The etiologic causes are listed in Table 23.1.

Clinical Picture

Acute pericarditis is characterized by progressive central chest pain, pericardial friction rub, and repolarization changes in the electrocardiogram. The chest pain occurs rapidly and is usually sharp, pleuritic, and postural, being worse while lying supine and by coughing, and frequently reduced by sitting up. It can radiate to the neck, trapezius ridge, or shoulder, and can mimic angina pectoris, making the differentiation from acute coronary syndrome more difficult. On the other hand, acute pericarditis can present with only vague precordial distress, or it may be asymptomatic. Fever and cough can also occur, and some patients report breathing problems, but true dyspnea is present only in patients with cardiac tamponade or coexisting pulmonary and cardiac diseases. Pleural effusion may also be present.

The most typical physical sign is the pericardial friction rub, which can be heard in approximately three fourths of patients. It has classically three components related to atrial systole, ventricular systole, and ventricular diastole. Since the rub usually waxes and wanes, a repeated auscultation in various positions is mandatory. Cardiac sounds can be distant or muted due to development of pericardial effusion.

Diagnosis

The basic diagnostic workup requires a history, physical examination, electrocardiography,

TABLE 23.1. Etiology of pericarditis

Infectious pericarditis
Pericarditis in systemic autoimmune diseases
Pericarditis and effusion in diseases of surrounding organs
Pericarditis in metabolic disorders
Traumatic pericarditis
Neoplastic pericardial disease
Idiopathic pericarditis

Source: Adapted from Maisch et al. (3).

laboratory tests, and chest x-ray. Electrocardiogram (ECG) is abnormal in most patients with pericarditis. Changes are present in the majority of limb and precordial leads with four characteristic phases: ST segment elevation with J point elevation and upright T waves (phase I), normalization of ECG (phase II), T-wave inversion (phase III), and normal ECG tracing (phase IV) (4). In more than 40% of patients atypical ECG changes are found. ST elevation in only a few leads can be confusing and may suggest myocardial infarction. However, in patients with pericarditis, reciprocal ST depression is usually absent.

Laboratory tests can show changes in white blood cell count, sedimentation rate, and other acute-phase reactants. Serum troponin level can be elevated, especially when pericarditis is accompanied with myocarditis (5).

Cardiomegaly on chest radiography is usually not evident, unless more than 250 mL of pericardial fluid is present.

Radionuclide studies with indium 111 or gallium 67 can be useful for confirmation of pericardial inflammation (6, 7).

Idiopathic pericarditis is the most common form of the acute pericarditis, but in fact most cases are due to viral infection. Specific etiologic diagnosis can be confirmed by tuberculin skin testing, viral studies, rheumatoid factor, and antinuclear antibodies. Renal failure, neoplastic diseases, cardiac surgery, and secondary to chest radiotherapy should be considered as causes of secondary pericarditis. The term *autoreactive pericarditis* is characterized by elements of autoimmune response (8). Considering that idiopathic pericarditis resolves spontaneously in most patients, the tests for diagnosis of viral and autoimmune etiology (immunoglobulins, complements in pericardial fluid, virologic and

immunohistologic studies) are too complex and expensive for routine practice (2). On the other hand, suspected purulent pericarditis in the presence of bacterial chest infections should be confirmed or excluded as soon as possible (9).

Echocardiography, which should be performed in all patients, is not specific for the diagnosis of pericarditis, but it shows pericardial effusion in approximately 10% of patients. Cardiac tamponade occurs more frequently in patients with specific etiology (tuberculosis, neoplastic and purulent pericarditis) than in patients with idiopathic pericarditis (10).

In the differential diagnosis acute coronary syndrome, dissecting aortic aneurysm, and pleuritis should be considered.

In the diagnostic workup of acute pericarditis, the three-stage approach seems to be appropriate:

Stage 1 is a history, physical examination, ECG, chest x-ray, general blood analysis, and echocardiography. In patients with tamponade or prolonged pericardial effusion (more than 1 week), tuberculosis should be excluded and antinuclear antibodies should be measured. Stage 2 is pericardiocentesis and proper examination of the pericardial fluid, which should be performed in patients with suspected purulent or neoplastic pericarditis and in patients with cardiac tamponade (therapeutic indication). Stage 3 is a pericardial biopsy, which is indicated only in patients with recurrent tamponade and in patients with persistent clinical effusion and clinical symptoms without etiologic diagnosis for more than 3 weeks (2). Other authors advocate an early invasive approach with pericardioscopy and pericardial biopsy for establishing the specific etiologic diagnosis (11–13).

Treatment

Despite the fact that most patients with acute pericarditis are hospitalized, hospital admission and treatment are absolutely necessary only for patients with idiopathic or viral pericarditis, who have large effusion or cardiac tamponade, concomitant myocarditis, high fever, or subacute clinical course, and for immunocompromised patients and those with anticoagulant treatment (14).

Patients are treated with aspirin (initial dose of 500 to 1000 mg every 6 hours) or with nonsteroidal antiinflammatory agents (ibuprofen 1800 to 2400 mg/day, indomethacin 75 to 225 mg/day, or paracetamol 3 to 4 g/day) for at least 3 weeks (2, 3). Indomethacin should not be used in elderly patients and in patients with coronary artery disease. Corticosteroids should be avoided and considered only in patients in whom tuberculosis is excluded and who are resistant to treatment and have persistent symptoms for more than 1 week. Corticosteroids can be used also in patients with connective tissue diseases or autoreactive and uremic pericarditis. Intrapericardial application of corticosteroids is effective and avoids systemic side effects (8).

Recurrent pericarditis occurs in around 24% of patients, usually in the first weeks after the first episode of acute pericarditis (15). The treatment of recurrences is basically the same as for the first episode, but in patients with two or more recurrences, treatment with colchicine may be successful (16). Intrapericardial treatment with cisplatin appeared to prevent recurrences of neoplastic pericardial effusion (17).

Cardiac Tamponade

Cardiac tamponade is the pathologic restraint of cardiac filling due to increased pericardial pressure, caused by the excess of fluid in the pericardial cavity. Typical characteristics of tamponade are equalization of left and right ventricular filling pressure, restricted diastolic filling of both ventricles, and decreased cardiac output with development of shock (1). Cardiac tamponade can occur in any disease with pericardial effusion, but it is most common in patients with pericardial effusion due to malignant diseases, renal failure, and viral pericarditis. Other medical diseases and surgical conditions that can provoke tamponade are listed in Table 23.2. Parietal pericardium can exert an important radial stress on the heart and can significantly limit the cardiac volume even in normal conditions. Pericardial restraint becomes very important under pathologic conditions. Cardiac chambers compliance is markedly decreased and diastolic ventricular interaction is augmented. The compressive effect of pericardial

TABLE 23.2. Etiology of cardiac tamponade

Medical diseases
Common
Malignant diseases
Renal failure
Viral pericarditis
Less common
Radiation therapy, anticoagulant therapy
Hypothyrosis
Rheumatoid arthritis, systemic lupus erythematosus
Tuberculosis, AIDS
Acute myocardial infarction (thrombolysis, rupture)
Purulent pericarditis
Surgical conditions
Invasive cardiac procedures with perforation
Cardiovascular surgery and postpericardiotomy syndrome
Chest trauma
Aortic dissection

Source: Adapted from Davies et al. (18).

pressure is exerted primarily on the right heart and caval vessels. The left ventricular function becomes compromised later on as a consequence of inadequate filling. The different effect on the right and left ventricle is probably related to high right ventricular compliance, the extrapericardial part of the left atrium, and the long intrapericardial segment of the caval vessels (18). In patients with classic findings of tamponade without pericardial effusion, a tension pneumopericardium must be suspected (19).

Clinical Picture

The clinical picture depends on the underlying etiology, preexistent heart and lung diseases, and the rapidity of fluid accumulation. Accumulation of the intrapericardial fluid is associated with initial small increase of the intrapericardial pressure and followed by a steep rise. Once pericardium can no longer stretch, a tamponade is typically produced with very little accumulated fluid (Fig. 23.1) (20). In acute severe hemorrhage tamponade can occur with accumulation of 100 to 300 mL. It is typically clinically presented as an obstructive shock with systemic hypotension and elevated central venous pressure (distended neck veins!) without pulmonary congestion. The diagnosis of cardiac tamponade must always be considered as a possible etiology of unexplained

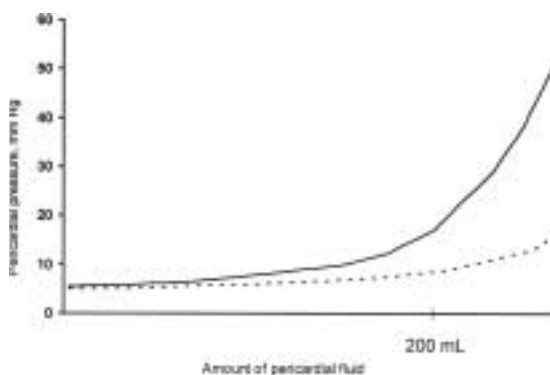


FIGURE 23.1. Schematic drawing representing the relation between amount of the accumulated pericardial fluid and pericardial pressure in acute (solid line) and subacute (dotted line) tamponade.

obstructive shock. If the pericardial fluid accumulates slowly, much larger amounts (2 L or more) can be accommodated. Dyspnea with orthopnea and signs of low output syndromes or mechanical compression of adjacent organs are the most common patient complaints (21, 22).

Acute tamponade, which is usually associated with severe hemodynamic compromise, is most frequently caused by trauma and rupture of the heart or aorta. Subacute tamponade has a less dramatic clinical presentation and is associated with idiopathic, uremic, or neoplastic pericarditis. Regional tamponade (loculated pericardial effusion or localized hematoma) occurs usually after pericardiectomy or myocardial infarction.

On physical examination, signs of elevated jugular venous pressure, tachycardia, tachypnea, and paradox arterial pulse can be found. However, pulsus paradoxus can be absent in patients with cardiac tamponade and preexistent elevation of left ventricle filling pressure, right-to-left cardiac shunt, aortic stenosis, and severe chest trauma.

Heart sounds are usually very silent or even absent; pericardial friction rub is heard in only one third of patients.

Electrocardiographic changes are nonspecific and include reduced voltage, electrical alternans, and electrical changes, which are typical for pericarditis (23).

The chest x-ray can show an enlarged cardiac silhouette (shaped like a water bottle) and oligemic

lungs. In patients with acute tamponade, cardiac size and shape may be quite normal (24).

Definitive diagnosis of pericardial effusion and severity of cardiac tamponade can easily be accomplished by transthoracic echocardiography in almost all patients. A transesophageal approach is occasionally necessary in patients with localized tamponade or poor visibility. The size of the effusion can be graded according to the echo-free space in diastole as small (<10 mm), moderate (10 mm to <20 mm), large (≥ 20 mm), or very large (≥ 20 mm with compression of chambers). In patients with large effusions the heart is moving free in the pericardium ("swinging heart") (3).

Echocardiographic signs of tamponade are diastolic or early systolic collapse of the right atrium, diastolic collapse of the right ventricle, respiratory variation of ventricular volumes, decreased collapsibility index of the inferior vena cava, and exaggerated respiratory variation of mitral and tricuspid inflow velocity (25–27). Ninety percent of patients with typical clinical signs of tamponade had collapse of one or both right cardiac chambers, which was found also in 38% of patients without clinical tamponade. Abnormal pulmonary venous flow has a good correlation with clinical features of tamponade, with a higher sensitivity than right ventricular collapse and a much higher specificity than right atrial collapse (28). In patients after cardiac surgery, localized posterior pericardial effusion with left ventricular diastolic collapse may be responsible for cardiac tamponade (29).

Hemodynamic variables show equalization of right atrial, right ventricular diastolic, pulmonary diastolic, and pulmonary artery occlusion pressures, and inspiratory increase of right-sided pressures with a decrease of left-sided cardiac pressures. The latter is responsible for pulsus paradoxus, which is defined as inspiratory drop in systolic pressure more than 10 mm Hg.

In the differential diagnosis of obstructive shock pulmonary embolism, right ventricular infarction and tension pneumothorax should be considered. On the other hand, in patients with large pericardial effusion, congestive heart failure is the most common differential diagnostic problem.

Treatment

Medical therapy of cardiac tamponade is only a temporizing measure and includes volume expansion and inotropic support. Plasma expanders or saline infusion may be useful in hypovolemic patients, but inotropic support is usually not effective for initial hemodynamic stabilization (20). All anticoagulant medication should be discontinued, and treatment with vitamin K or protamine sulfate should be considered. Patient who absolutely need anticoagulation should receive heparin instead of warfarin. Positive pressure mechanical ventilation should be avoided before pericardiocentesis, since it further decreases the venous return.

The only effective treatment of cardiac tamponade is pericardiocentesis and evacuation of accumulated fluid. Pericardiocentesis should be done immediately in hemodynamically compromised patients. A blind subxiphoid approach may be indicated as a lifesaving procedure, but pericardiocentesis under echocardiographic guidance is preferred. Echocardiography identifies the best place and shortest approach for the puncture, which is frequently at atypical sites of the chest (30, 31). After drainage, all patients should be monitored for possible cardiac failure due to increased venous return. A pericardial catheter can be left in place for 2 to 3 days until secretion is reduced to <25 mL/day (3). It allows a reliable control of the pericardial fluid reaccumulation and reduces the need for repetitive pericardiocentesis.

Pericardiocentesis is contraindicated in patients with aortic dissection; further relative contraindications are severe uncorrected coagulopathy, thrombocytopenia, and small or loculated effusion (32).

Examination of pericardial fluid includes complete laboratory assessment, and culture and stains for bacteria, tuberculosis, and fungi, and it should be done as soon as possible to obtain an etiologic diagnosis.

If no safe access for pericardiocentesis is possible, surgical drainage may be indicated. A subxiphoid surgical approach is commonly applied, but a thoracotomy has to be done occasionally. Surgical drainage is preferable in patients with acute traumatic and purulent pericardial effusion (1).

After drainage of the pericardial fluid, the treatment depends on the etiology. In patients with inflammatory diseases, aspirin, antiinflammatory drugs, and rarely steroids are used. In patients with uremic pericarditis, hemodialysis must be intensified. In purulent pericarditis rinsing of pericardial cavity together with systemic antibiotic treatment is mandatory. Instillation and irrigation with streptokinase or urokinase can liquefy the pericardial effusion and allow better drainage (33). Intrapericardial installation of chemotherapeutic drugs or tetracycline may be useful in patients with malignant pericardial effusion (34). Cardiac perforation or rupture and purulent pericarditis require immediate surgical management.

Clinical Case

A 37-year-old man who is an alcoholic and has insulin-dependent diabetes was admitted to the pulmonary department because of fever and abdominal and lower back pain. During hospitalization (12 days) he was hemodynamically stable, without respiratory or renal failure. Electrocardiogram was normal and no infiltrations were found on the chest x-ray. Abdominal ultrasound was normal. Laboratory findings show mild anemia (Hb 110 g/L), leukocytosis ($19.7 \times 10^9/L$), and slightly pathologic liver tests. C-reactive protein (CRP) was elevated (156 mg/L). He was treated with ciprofloxacin 400 mg b.i.d., and after 1 week became afebrile.

Neuropathy was suspected and the patient was transferred to the neurology department. Neuropathy was excluded, but after 3 days the fever returned and he became hypotensive (95/70 mm Hg). An ECG was reported as normal, but cardiac enlargement without pulmonary infiltration or congestion was found on the chest x-ray. The laboratory tests revealed anemia (Hb 95 g/L), leukocytosis ($18.6 \times 10^9/L$), progressive liver failure, and elevated CRP (335 mg/L).

The patient was transferred to the gastroenterology department. The next day, clinical signs of shock with distended jugular veins developed. On abdominal ultrasound a liver congestion with small amount of ascites was found, and cardiac tamponade as a reason for shock was suspected.

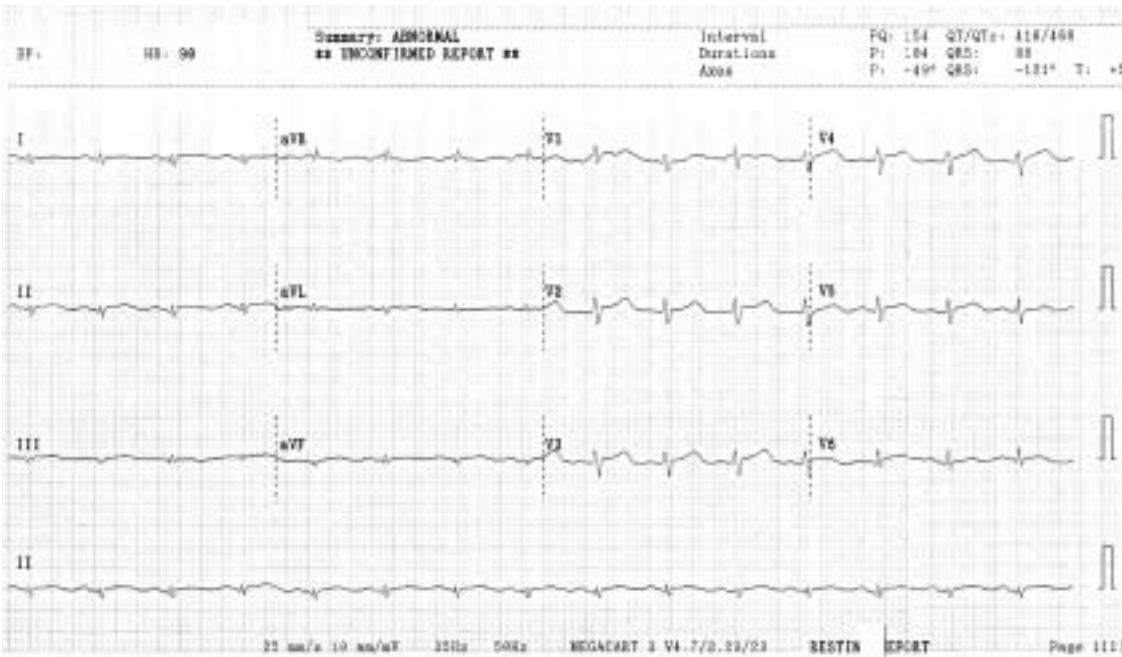


FIGURE 23.2. Electrocardiogram shows normal sinus rhythm and only discrete ST segment elevation in standard and precordial leads.

The patient was transferred to the intensive care unit. On admission he was hypotensive (90/70 mm Hg), with distended neck veins (CVP + 27.5 cm of water); his skin was pale, cool, and clammy; breathing sounds were normal; heart sounds were distant with no friction rub; and abdominal palpation was slightly tender, but no muscular defense was present. Saturation with pulse oximetry was 92% while breathing 60% oxygen. Laboratory tests showed anemia (Hb 92 g/L, Ht 0.29), leukocytosis ($17.9 \times 10^9/L$), mild renal failure (urea 20.8 mmol/L, creatinine 125 $\mu\text{mol/L}$), liver failure (bilirubin 75 $\mu\text{mol/L}$, alanine aminotransferase [ALT] 3.4 $\mu\text{cat/L}$, aspartate aminotransferase [AST] 1.28 $\mu\text{cat/L}$, gamma GT 4.21 $\mu\text{cat/L}$, NH_3 103 $\mu\text{mol/L}$, prothrombin time 2.6 international normalized ratio [INR]), elevated serum lactate (4.39 mmol/L), CRP (361 mg/L), and PCT (6.6 $\mu\text{g/L}$). The ECG showed sinus rhythm 90/min and ST segment elevation in standard and precordial leads (Fig. 23.2). On chest x-ray the heart was enlarged and the lungs were clear (Fig. 23.3). Transthoracic and transesophageal echocardiograms showed localized pericardial effusion with a cauliflower-like appearance of the fluid and

compression of the right heart chambers (Fig. 23.4).

The patient received 2L of crystalloids for hemodynamic stabilization and four units of fresh froze plasma for correction of coagulopathy. Afterward, pericardiocentesis was performed with an anterior approach, and 920 mL of purulent



FIGURE 23.3. Chest x-ray showing enlarged cardiac silhouette with clear lungs.



FIGURE 23.4. Large cauliflower-like pericardial effusion in front of right ventricle on transesophageal echocardiography.

effusion with pH of 6.42 was evacuated. Immediately after drainage the cardiac silhouette was smaller on chest x-ray and marked hemodynamic improvement was observed (heart rate 107/min, arterial blood pressure 135/75 mm Hg, CVP +11 cm of water, SatO₂ 96%). Direct examination of pericardial fluid showed granulocytes and grampositive cocci. *Enterococcus faecalis* and *Acinetobacter baumannii* were isolated in the culture. Drainage was left in place and the pericardium was irrigated every 6 hours with netilmicin and vancomycin. Systemic antibiotic treatment was started as well.

In the next 2 days the patient improved, but septic shock developed again on day 3 after pericardiocentesis. Diffuse purulent peritonitis was diagnosed with abdominal fluid examination and gastric ulcer by endoscopy. He was treated with repetitive surgical intervention, and perforation tecta contained perforation of the gastric ulcer with peritonitis was confirmed. The gastric ulcer perforation was also the cause of the purulent pericarditis, which was the first manifestation of the disease. He needed prolonged intensive care because of septic multiple organ failure.

After 4 weeks he was discharged to the ward and after 8 weeks from the hospital. No recurrent pericardial effusion or signs of constriction occurred 1 year after discharge.

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