

PERICARDIAL EFFUSION AND CARDIAC TAMPONADE

PERICARDIAL EFFUSION

Definition

Pericardial effusion refers to accumulation of pericardial fluid, blood or lymph, in excess of the 15 to 50 mL that are normally found in the pericardium. The pericardial fluid therefore can be a transudate, typically occurring in patients with congestive heart failure or an exudate which contains a high concentration of proteins and fibrin, and can occur with any type of pericarditis, severe infections, or malignancy. Classification of pericardial effusion is presented in Table 1.¹ Pericardial effusions can occur with or without cardiac tamponade.

Pathophysiology

The pressure–volume curve of the normal pericardium is a J-shaped curve. After an initial short shallow portion that allows the pericardium to stretch slightly in response to physiological events, such as changes in posture or volume status, with minimal pressure increase, then the pericardium does not allow further sudden increases of the volume without a marked increase in the intrapericardial pressure. Thus, a slowly accumulating pericardial fluid may allow pericardial distention till the accumulation of 1–2 L of pericardial fluid without the development of cardiac tamponade, whereas a sudden increase of pericardial volume of 100–200 mL, as in haemopericardium, may elevate pericardial pressure till 20–30 mmHg with acute cardiac tamponade.²

Clinical Settings

Idiopathic chronic pericardial effusion is defined as a collection of pericardial fluid that persists for more than three months and has no apparent cause. It is well tolerated for long periods in most patients, but severe tamponade can develop unexpectedly at any time.³ Large idiopathic chronic effusions (>3 months) have a 30–35% risk of progression to cardiac tamponade.³ Thus, large pericardial effusions (>20 mm) effusions should be drained if the effusion persists for more than a month or if there is right- sided collapse.

Moderate pericardial effusions without tamponade in the setting of **myocardial infarction** may indicate subacute ventricular wall rupture and carry the risk of late true wall rupture.

Neoplasia-associated effusion is seen with lung cancer, breast cancer, melanoma, lymphomas, and leukemias. Primary tumors such as mesothelioma are rare. Intrapericardial instillation of cytostatic/sclerotic agents may be needed. However, approximately 75% of

pericardial effusions in patients with malignancies are caused by other causes such as radiation or opportunistic infections.

Effusions seen with **heart failure** or in **the last trimester of pregnancy** are transudates that never progress to cardiac tamponade.

Presentation

Symptoms vary according to the speed of pericardial fluid accumulation and aetiology of the effusion. the patients may be asymptomatic or complain of dyspnea on exertion and fatigue. Nausea (diaphragm), dysphagia (oesophagus), hoarseness (recurrent laryngeal nerve), and hiccups (phrenic nerve) that have been described with large effusions are very rare nowadays.

Physical Examination

Usually, physical signs are not specific but quite heart sounds, and elevated jugular venous pressure may be seen. Pulsus paradoxus is typically a sign of tamponade.

Investigations

Most effusions are detected by transthoracic echocardiography, which together with ECG, and assessment of markers of inflammation and myocardial injury are essential (Table 3). CT and CMR may allow detection of loculated effusions, pericardial thickening and masses (Table 30. In the absence of tamponade, pericardiocentesis is indicated if purulent, tuberculous, or neoplastic pericarditis is suspected. CT is recommended to evaluate pericardial thickness, calcifications, masses, and loculated pericardial effusions, as well as concomitant pleuropulmonary diseases and chest abnormalities (ESC 2025 GL on myocarditis and pericarditis, I C).⁴

Therapy

An algorithm for management is presented in Figure 1. Admission is necessary for high-risk patients. Underlying disease may be detected in up to 60% of cases and therapy is therefore specific.² When the diagnosis is unclear or idiopathic and inflammatory markers are elevated, aspirin or a NSAID for 1-2 weeks is given (Table 5). Combination of aspirin or a NSAID for 2-4 weeks with colchicine for 3-6 months is considered for recurrent cases, while corticosteroids at low to moderate doses for 2-4 weeks may be given for specific indications such as systemic inflammatory diseases and pregnancy.² Doses are given in the Chapter on Acute Pericarditis. Intrapericardial triamcinolone has also been tried in resistant cases.²

Pericardiocentesis alone frequently results in the resolution of large **idiopathic** effusions, but recurrence is common. Pericardiectomy should be considered only in highly

symptomatic recurrences resistant to medical therapy, and in cases of chronic permanent constriction. If smaller pericardial effusions recur and the patient remains asymptomatic without hemodynamic compromise, regular follow-up is recommended. **Subcritical uremic tamponade** often responds to intensified renal dialysis, but if this approach is unsuccessful, drainage is required. When recurrent pericardial effusions are related to **chylopericardium**, the underlying etiology may be thoracic duct obstruction, requiring surgical treatment.⁵ In **neoplastic** pericardial disease, pericardial window by open surgery or thoracoscopy, balloon pericardiotomy, and sclerotic local therapy may be needed.

CARDIAC TAMPONADE

Definition

Cardiac tamponade is a life-threatening, slow or rapid compression of the heart due to the pericardial accumulation of fluid, pus, blood, clots, or gas, as a result of effusion, trauma, or rupture of the heart.⁶ Occurrences of tamponade can be acute, subacute, regional, or characterized by low pressure.

Pathophysiology

The true filling pressure is the myocardial transmural pressure, which is intracardiac minus pericardial pressure. The increased intrapericardial pressure equals or exceeds the pressure in the right heart chambers, leading to reduced chamber diastolic compliance and impaired filling, collapse of the right atrium and ventricle during diastole and diminished cardiac output. Expansion of the RV is limited to the interventricular septum, resulting in bulging of the RV into the LV, reduced LV compliance and decreased filling of the LV during inspiration (ventricular interdependence). Maximal pericardial pressure in tamponade occurs during end-diastole, when RA volume is minimal. When fluid collection is slow, the pericardium can stretch to accommodate a large volume with minimum compromise of cardiac function, partly due to systemic neurohumoral responses that compensate for reduced cardiac filling. Thus, intrapericardial hemorrhage from wounds or cardiac rupture occurs in the context of a relatively stiff pericardium and quickly overwhelms the pericardial capacity to stretch before most compensatory mechanisms can be activated, whereas in the case of a slow increase in pericardial volume as a result of inflammation, 2 liters or more may accumulate before critical, life-threatening tamponade occurs.⁶

Clinical Settings

Causes of cardiac tamponade are presented in Table 2.

Pericarditis. Tamponade is reported in about 15% of patients with idiopathic pericarditis but in as many as 60% of those with neoplastic, tuberculous, or purulent pericarditis.⁷

Myocardial Infarction. When pericardial effusion in a parasternal short-axis view exceeds 10 mm in the setting of myocardial infarction, the risk of free wall rupture is high and pericardial aspiration for measurement of hematocrit in the effusion is useful.⁸ Pericardial effusion indicates increased mortality in this setting and early pericardiocentesis may be life saving.

Iatrogenic. In the current era, invasive cardiac procedures that require anticoagulation and left atrial access using transseptal puncture have become one of the most common causes of acute tamponade.⁹ Its incidence is not exactly known, but it is estimated 1-6% (Table 7). In iatrogenic cardiac perforation the development and perforation of tamponade depends on the state of anticoagulation, LV and PA pressures, and chamber perforated. RA perforation in the absence of anticoagulation may be well tolerated. LA perforation is more serious because trans-septal access involves anticoagulation, and LA pressure is higher than the RA pressure. RV (RV wall ≥ 4 mm) and especially LV perforation (LV wall ≥ 10 mm) may be tolerated unless if severe pulmonary hypertension or aortic stenosis preexist, respectively.⁹

Postoperative tamponade is more frequent after valve surgery than CABG and may be seen early or late (5 days-2 weeks) after the operation. Reported incidence of postoperative pericardial effusions is 1.5% and of tamponade 0.7%.¹⁰ Later after cardiac surgery the pericardium may be adherent to the myocardium and thus prevent the development of tamponade; however, posterior localized effusion that may not be accessible to pericardiocentesis, may be seen.

Tuberculosis and other *infections, neoplasms, collagen vascular disease, radiation, uremia, aortic dissection*, and *pneumopericardium* are other rare causes of tamponade.¹

Presentation

Presentation depends on the type and severity of tamponade. Patients are usually weak and faint with tachypnea and possibly chest pain. The initial symptom may be also one of the complications of tamponade such as renal failure or right upper quadrant pain due to hepatic congestion. Cardiac tamponade should also be considered in patients in cardiogenic shock, especially if they have increased jugular venous pressure or pulseless electrical activity.

Physical examination

Systemic arterial hypotension, tachycardia, quiet heart sounds, elevated jugular venous pressure, and pulsus paradoxus are the hallmarks of cardiac tamponade. However, most physical signs are not specific.

Systemic arterial hypotension is the rule but aortic pressure may increase in the early stages of acute tamponade in hypertensive patients due to sympathetic response to pericardial irritation.⁶ Patients may also be normotensive with low pressure tamponade in patients with hypovolemia or systemic disease.

Tachycardia may not be present in patients with uremia or hypothyroidism. Bradycardia due to vasovagal reaction may also be initially seen, especially in iatrogenic tamponade.

Heart sounds are quiet, but patients with pre-existing cardiomegaly and anterior or apical pericardial adhesions may have active pulsations.

JVP is usually elevated with preservation of the x descent but absence of the y descent.

However, in acute hemopericardium there is insufficient time for blood volume to increase and JVP pulsations may be exaggerated without distention.

Pulsus paradoxus (>10 mmHg fall in systolic pressure during inspiration) may also be seen with pulmonary embolism, chronic obstructive pulmonary disease, constriction, and rarely in pregnancy. Conditions that can impede the detection of pulsus paradoxus in tamponade are: pericardial adhesions, marked left or right ventricular hypertrophy, severe AR, and ASD.

Pericardial rub can be heard in inflammatory effusions.

Kussmaul sign (JVP elevation during inspiration) can be seen but unusually in the absence of pericardial constriction.

Investigations

ECG may show low QRS voltage (<0.5 V in limb leads) and signs of pericarditis. Electrical alternans, defined as the alteration of QRS complex amplitude or axis between beats, is specific for cardiac tamponade, especially when combined with P wave alteration.⁶

Chest X-ray shows cardiomegaly with effusions > 250 ml.

Echocardiography reveals diastolic collapse of the RA and early diastolic collapse of the RV when the intrapericardial pressure exceeds intracavitary pressure (Table 2 and Figure 2). RA collapse may also be seen in patients with hypovolemia, but persistence of RA collapse for more than one-third of the cardiac cycle is highly sensitive and specific for tamponade. During inspiration, the interventricular septum bulges into the LV due to increased systemic venous return to the RV and limited expansion of the RV free wall. With expiration, the transmitral pressure gradient increases and systemic venous return decreases with reversal of diastolic flow in the hepatic veins.

Left atrial collapse is very specific but not sensitive for tamponade.⁵

The inferior vena cava is dilated, with less than a 50% diameter reduction during inspiration. When cardiac tamponade is due to aortic dissection or cardiac free wall rupture, a coagulated mass may be seen within the pericardium.

Cardiac CT or transesophageal echocardiography can diagnose loculated pericardial effusions or hematoma, especially after cardiac surgery, not detectable by transthoracic echocardiography.

Cardiac catheterization, if performed, reveals inspiratory increase of the right and concomitant decrease of the left pressure, progressively increasing RA pressure and intracardiac diastolic pressure equilibration (15-30 mm Hg).

Therapy

The management of cardiac tamponade is presented in Figure 3. The presence of hemodynamic compromise requires urgent pericardiocentesis or surgical removal of pericardial fluid (Table 3). A scoring system for risk stratification of patients has been proposed,¹¹ but has been considered as based on expert consensus and requires additional validation in order to be recommended in clinical practice.¹

For **pericardiocentesis**, the needle is usually inserted between the xiphoid process and the left costal margin at a 15-degree angle to bypass the costal margin, and then its hub is depressed so that the point is aimed toward the left shoulder and is advanced slowly, until fluid is aspirated. Ideally this should be done in the catheterization laboratory or under echocardiographic guidance and with concomitant administration of IV saline. Attaching an electrode to the needle may provide misleading results. For prolonged drainage, an angiographic pigtail catheter may be left in the pericardium until the amount of fluid drained is <50 ml /day. Pericardiocentesis is relatively contraindicated in tamponade due to aortic dissection which represents a surgical emergency. Inadvertent puncture of a cardiac vessel, right ventricle or liver are rare complications.

Surgical drainage is preferable in patients with intrapericardial bleeding or with clotted pericardium.

In hypotensive patients, rapid **volume expansion** with saline or dextran should be provided, especially if the systolic blood pressure is <100 mm Hg.¹² In patients without hypotension and hypovolemia, fluid infusion may precipitate tamponade.⁶

Inotropic support is controversial since inotropic stimulation of the heart is often already maximal in tamponade.

Mechanical ventilation with positive airway pressure should be avoided because it further decreases cardiac output. In patients with cardiac arrest and a large amount of pericardial

fluid, external cardiac compression has little or no value, because there is little room for additional filling and because even if systolic pressure rises, diastolic pressure falls and, in doing so, reduces coronary perfusion pressure.⁶

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