Patients presenting with PEff without inflammatory marker elevation have an increased risk of a neoplastic aetiology (likelihood ratio 2.9).[615] In chronic effusions with no definite aetiology, and without inflammatory markers, empirical anti-inflammatory treatment is futile.[1,621] In worsening large effusions without evidence of systemic inflammation, the exclusion of cancer with advanced imaging (mainly CT) is warranted.[196] C-reactive protein should be measured before pericardiocentesis, since it can increase after the procedure.[622] Notably, the classical Light criteria used for the classification of pleural effusions should not be applied to PEff due to the high rate of misclassification of fluids as exudates, since pericardial fluid is rich in mesothelial cells, proteins, albumin, and lactate dehydrogenase (LDH).[623,624]

11.7.4. Therapy

The treatment of PEff is shown in *Figure 19*. Therapy of PEff should be targeted at the aetiology, as much as possible. In the absence of pericardial inflammation (namely in the absence of C-reactive protein elevation and evidence of pericardial inflammation by an imaging technique) anti-inflammatory treatment is not recommended. According to recent data, the usefulness of pericardial drainage in asymptomatic or oligosymptomatic, large, chronic, idiopathic effusions without evidence of pericarditis has been revised.[49,621] Conservative treatment improves outcomes, the risk of CTP being only 2.2%/year without reported deaths.[625] In addition, survival after recurrence or complications is significantly better in patients treated conservatively without interventions. Nevertheless, if during follow-up the PEff becomes symptomatic and haemodynamic impairment is observed by echocardiography, drainage is warranted.

11.7.5. Prognosis and follow-up

The prognosis of PEff is essentially related to its aetiology.[626] Moderate to large effusions are more common for specific aetiologies.[105,615] In general, PEff should be considered as a marker of the severity of the underlying disease.[584] Small, idiopathic, asymptomatic PEff have an overall good prognosis with a very low risk of complications, although not all studies concur.[613] Those individuals should be reassured about the benign nature of the condition, and should not restrict their physical activity if C-reactive protein is normal.[11] The follow-up of PEff is mainly based on the evaluation of symptoms, using focused echocardiography to assess size changes, and haemodynamic issues, as well as C-reactive protein. Asymptomatic patients with mild idiopathic effusions do not generally require specific monitoring. In asymptomatic patients with at least moderate effusions, a reasonable follow-up is every 6 months, ideally in specialized centres. The latter patients should be instructed to seek medical advice in case of symptom appearance, such as dyspnoea or fatigue, and/or chest pain suggesting pericarditis.

11.8. Cardiac tamponade

Cardiac tamponade is a pericardial syndrome occurring when PEff impairs diastolic filling of the heart until cardiac output is reduced. The size of the effusion as well as its distribution may vary. Since the pericardium is relatively stiff, if pericardial fluid collects quickly, such as in haemopericardium, the limit of pericardial stretching is reached quickly with volumes of 200–300 mL. In contrast, slowly accumulating PEff may reach 1 to 2 L before the development of CTP (Supplementary data online, *Figure S4*). This pathophysiology explains that CTP is a last-drop phenomenon. Thus, a small increase of pericardial volume may precipitate the syndrome, and the aspiration of small amounts of pericardial fluid with pericardiocentesis may greatly improve the clinical condition.[118]

Pericardial effusions are usually circumferential, but after trauma or cardiac surgery, they may be loculated and even be responsible for localized compression and the development of CTP.

11.8.1. Presentation

Beck identified a triad of signs, consisting of hypotension, increased JVP, and quiet heart sounds as presenting symptoms.[118,619]

This triad was classically identified in 'surgical tamponade' with acute CTP due to intrapericardial haemorrhage because of trauma, or myocardial/aortic rupture. Beck's triad may be lacking in patients with 'medical tamponade' with slowly accumulating pericardial fluid. Acute CTP is usually associated with tachycardia and low blood pressure (<90 mmHg) that is only slightly reduced in subacute, chronic tamponade.[118,619] On physical examination, classical signs include neck vein distention with elevated JVP, pulsus paradoxus, and diminished heart sounds. Pulsus paradoxus is defined as an inspiratory reduction of the systolic blood pressure by at least 10 mmHg.[118,619] Pulsus paradoxus is due to exaggerated ventricular interdependence occurring in CTP, when the overall volume of ventricles becomes unable to expand, and any change in the volume on one side of the heart causes opposite changes on the other side. On ECG, the patient usually shows tachycardia, low QRS voltages, and electrical alternans due to the damping effect of pericardial fluid and swinging heart.[118,619]

11.8.2. Aetiology and diagnosis

Cardiac tamponade shares the same causes of PEff. In clinical practice, the most common aetiologies include cancer, TB, purulent infections, trauma, iatrogenic complications of cardiovascular interventions (e.g. ablation of arrhythmias, device implantation, PCIS), acute aortic disease, systemic inflammatory diseases, and renal failure.[118] In the setting of AP with a viral or idiopathic aetiology, CTP is rare (1%–2% of cases) and is more common with pericarditis associated with a non-idiopathic aetiology (20%).[347] The diagnosis of CTP is a clinical diagnosis based on the combination of a suggestive history, symptoms, signs, and imaging confirmation by echocardiography (*Table 17*).[118]

Table 17 Echocardiographic signs of cardiac tamponade

Echocardiographic feature	Sensitivity	Specificity
Large pericardial effusion with swinging heart	n.a.	n.a.
Diastolic collapse of the RA	50%–100%	33%–100%
Duration of diastolic collapse of the RA as a ratio of the cardiac cycle length >0.34	>90%	100%
Diastolic collapse of the RV	48%–100%	72%–100%
Respiratory changes of the mitral E velocity >25%–30%, tricuspid E velocity >40%–60%	n.a.	n.a.
Inferior vena cava plethora (dilatation >20 mm and <50% reduction of diameter with respiratory phases), as well as hepatic vein dilatation	97%	40%

n.a., not available; RA, right atrium, RV, right ventricle.