**Acute Pericarditis**

This is acute inflammation of the pericardium, the membranous sac which surrounds the heart.

**Anatomy**

The pericardium is composed of an outer fibrous layer (parietal pericardium) and an inner serous membrane (visceral pericardium), with approximately 50 mL of plasma ultrafiltrate in between.

It promotes cardiac efficiency by limiting dilation, maintaining ventricular compliance with preservation of the Starling curve and distributing hydrostatic forces. It aids atrial filling by creating a closed chamber, reduces external friction and acts as a barrier against infection and extension of malignancy. Finally, it anatomically fixes the heart to the sternum, diaphragm and costal cartilages.

**Pathogenesis**

In most cases the pericardium becomes acutely inflamed, with pericardial vascularisation and infiltration with polymorphonuclear leukocytes. A fibrinous reaction frequently results in exudate and adhesions within the pericardial sac, and a serous or haemorrhagic effusion may develop. In some conditions (eg, tuberculosis, sarcoidosis, fungal infections and rheumatoid arthritis), a granulomatous pericarditis develops.

**Aetiology**

<table>
<thead>
<tr>
<th>Viral Infection</th>
<th>Rheumatological</th>
<th>Primary Neoplasms</th>
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<tbody>
<tr>
<td>Coxsackievirus*</td>
<td>Sarcoïdosis</td>
<td>Sarcomas</td>
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<tr>
<td>Echovirus</td>
<td>Systemic lupus</td>
<td>Mesoïthelioma</td>
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<tr>
<td>Epstein-Barr virus</td>
<td>erythematous*</td>
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<tr>
<td>Influenza</td>
<td>Rheumatoid arthritis</td>
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<td>Human immunodeficiency virus</td>
<td>Dermatomyositis</td>
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<tr>
<td>Mumps</td>
<td>Scleroderma</td>
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<td></td>
<td>Polyarteritis nodosa</td>
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<td></td>
<td>Vasculitis</td>
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<td></td>
<td>Ankylosing spondylitis</td>
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<tr>
<th>Bacterial Infection</th>
<th>Immunological</th>
<th>Metastatic Neoplasms</th>
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<tbody>
<tr>
<td>Staphylococcus</td>
<td>Coeliac sprue</td>
<td>Breast</td>
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<tr>
<td>Haemophilus</td>
<td>Inflammatory bowel disease</td>
<td>Lung</td>
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<td>Pneumococcus</td>
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<td>Lymphoma</td>
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<td>Salmonella</td>
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<td>Tuberculosis</td>
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<td>Meningococcus</td>
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<td>Syphilis</td>
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<tr>
<th>Miscellaneous Infection</th>
<th>Drugs</th>
<th>Other</th>
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</thead>
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<tr>
<td>Histoplasmosis</td>
<td>Hydralazine*</td>
<td>Chest trauma</td>
</tr>
<tr>
<td>Blastomycosis</td>
<td>Procainamide*</td>
<td>Uraemia*</td>
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<tr>
<td>Coccidioidomyosisis</td>
<td>Isoniazid</td>
<td>Myædema</td>
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<tr>
<td>Aspergillosis</td>
<td>Methysergide</td>
<td>Aortic dissection</td>
</tr>
<tr>
<td>Echinococcus</td>
<td>Phenylzin</td>
<td>Radiation therapy</td>
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<tr>
<td>Amoebiasis</td>
<td>Anticoagulants</td>
<td>Myocardial infarction*</td>
</tr>
<tr>
<td>Rickettsia</td>
<td></td>
<td>Post-myocardial infarction syndrome (ie Dressler's syndrome, post-pericardiotomy)*</td>
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</tbody>
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*Some of the more common aetiologies

**Prevalence**

Acute pericarditis accounts for 5% of emergency department visits for chest pain in the absence of myocardial infarction\[1\]. In developed countries, around 80% of the cause of pericarditis is either postviral or idiopathic\[6\]. It is more common in men than in women and is seen more frequently in adults than in children.
Even when managed effectively, many patients with acute pericarditis present with one or more repeated episodes, which is termed recurrent pericarditis [2].

Presentation

Symptoms
Chest pain is the cardinal symptom. The pain may be:

- Dull, sharp, burning or pressing.
- Either barely perceptible or up to a severe level.
- Felt in the substernal or precordial region.
- Radiating to the neck, trapezius ridge (especially the left) or shoulders.
- Aggravated by inspiration, swallowing, coughing and lying flat.
- Relieved by sitting up and leaning forward.

Other symptoms can include a nonproductive cough, chills and weakness.

Signs
A pericardial friction rub is present on auscultation in 60-85% of cases. Clinically, the presence of a pericardial friction rub is pathognomonic - often a rub can be heard even when a pericardial effusion is present. It is often intermittent and evanescent, so frequent examination should take place if it is absent but clinical suspicion is high.

- It has been described as a scratchy superficial sound, heard most strongly in the midline and lower left parasternal edge.
- It varies in strength with respiratory movements and is usually louder in inspiration.
- More than 50% of friction rubs have presystolic, systolic and diastolic components.
- 24% of patients with rubs are biphasic. This happens in tachycardia, when an early diastolic rub merges with the atrial component.
- Rarely, a monophasic rub can occur, particularly in patients with atrial fibrillation. It is sometimes mistaken for a murmur.

Other signs can include tachypnoea, tachycardia and fever.

- Dyspnoea and orthopnoea are particularly noticeable when cardiac tamponade develops (haemodynamic changes resulting from restriction to the movement of heart muscle, secondary to pericardial effusion).
- Patients with tamponade may exhibit Beck’s triad (hypotension, elevated systemic venous pressure (often with jugular venous distention) and muffled heart sounds).
- Slowly developing tamponade may also result in pulsus paradoxus (defined as a 10 mm Hg decrease in arterial systolic pressure with inspiration).

Abdominal pain occurs occasionally in children.

Differential diagnosis

Other causes of chest pain - in particular:

- Myocardial infarction or ischaemia.
- Pleuritic pain.
- Pulmonary infarction.
- Peptic ulcer disease or oesophagitis.

Investigations

ECG
Serial ECGs are sometimes helpful in detecting these changes, in cases of diagnostic difficulty or in monitoring the development of cardiac tamponade.

Four stages have been recognised, although all four are only present in 50% of patients:

- **Stage I**: diffuse concave-upward ST-segment elevation with concordance of T waves; ST-segment depression in aVR or V1; PR-segment depression; low voltage; absence of reciprocal ST-segment changes.
- **Stage II**: ST segments return to baseline; T-wave flattening.
- **Stage III**: T-wave inversion.
- **Stage IV**: gradual resolution of T-wave inversion.

When tamponade complicates acute pericarditis, electrical alternans may involve the P, QRS, or T complexes. Most often, only the QRS is involved; the QRS amplitude increases and decreases on alternate beats. In some cases of tamponade, electrical alternans is associated with variation in cardiac position (swinging heart). Electrical alternans is not seen in most cases of cardiac tamponade.
CXR
An enlarged 'flask-shaped' cardiac silhouette may be the first sign of a large pericardial effusion. This may not be evident in patients with small effusions (less than a few hundred millilitres) and may present with a normal silhouette.

Blood tests
- FBC with differential (may show leukocytosis).
- ESR and CRP levels (usually raised).
- U&E levels (uraemia).
- Cardiac enzymes (associated myocarditis or myocardial infarction).

In one study, an elevated troponin I level was found in 32% of patients with viral or idiopathic pericarditis. The troponin I level was related to the extent of myocardial inflammation but was not a negative prognostic marker.

NB: resolving acute pericarditis (one week or less) usually does not need any further investigations. However, if symptoms persist, the following may be indicated in order to look for an underlying cause:
- Blood cultures.
- Tuberculin testing with sputum for acid-fast bacilli.
- Antistreptolysin titre.
- Rheumatoid factor, antinuclear antibody, and anti-DNA values.
- Thyroid function (if significant pericardial effusion is present).
- Antibodies for HIV, Coxsackievirus, influenza virus and echovirus.
- Examination of pericardial fluid or pericardial biopsy specimens for fungi and malignant cells.
- Pericardial biopsy for culture and microscopic examination in cases of recurrent or persistent effusion.

Echocardiography
This is appropriate if pericardial effusion or tamponade is suspected. M-mode demonstrates persistence of the echo-free space between the parietal pericardium and the epicardium during the cardiac cycle. Fluid adjacent to the right atrium is an early indicator of an effusion. Other causes of echo-free space that must be considered include pericardial masses, pleural effusion and epicardial fat.

CT or MRI scanning
This is helpful in cases of diagnostic doubt. A pericardial thickening >5 mm may be seen. When pericardial thickening or fluid cannot be demonstrated, the diagnosis of restrictive cardiomyopathy should be considered.

Surgical procedures
Pericardiocentesis under echocardiographic monitoring is occasionally used in cases of diagnostic difficulty, especially in cases of impending tamponade. This would be recommended if there were echocardiographic evidence of right atrial or ventricular diastolic collapse, irrespective of whether clinical signs of tamponade were present.

If tamponade recurs, a pericardial biopsy should be performed with histological and bacteriological examination. Endomyocardial biopsy and cardiac catheterisation may also be indicated in cases of diagnostic difficulty.

Management
Stable patients can often be managed in the outpatient setting without the need for hospital admission.

Patients with any of the following are associated with a higher risk and should usually be admitted to hospital:
- Fever (>38°C) and leukocytosis.
- Evidence suggesting cardiac tamponade.
- A large pericardial effusion (ie an echo-free space of more than 20 mm).
- Immunosuppressed state.
- Patients taking warfarin.
- Acute trauma.
- Failure to respond within seven days to non-steroidal anti-inflammatory drugs (NSAIDs).
- Elevated cardiac troponin, which suggests myopericarditis.

Outpatient/community management
Treatment should be given for the underlying cause.
- All patients should be advised to rest and avoid any demanding physical activity to help minimise symptoms.
- NSAIDs are usually given as first-line - eg, naproxen 250 mg 6- to 8-hourly.
- Treatment with colchicine has been shown to result in significantly fewer recurrences and longer symptom-free periods.
- In addition, when attacks occur, they are weaker and shorter in nature.
- Colchicine, alone or in combination with an NSAID, can be considered for patients with recurrent or continued symptoms beyond 14 days.
- NSAIDs are usually given for 7-14 days; then the dose should be tapered until resolution of symptoms and also improvement of serum inflammatory markers such as ESR and CRP.
• Gastric protection with proton pump inhibitors (PPIs) should be considered, especially in those patients taking high doses of NSAIDs.
• Cessation of possible causative drugs - eg, phenytoin.
• Anticoagulants should be avoided unless the pericarditis is secondary to acute myocardial infarction, as they can cause intrapericardial bleeding and possibly fatal tamponade.
• Bacterial or mycotic infections should be treated with appropriate antimicrobials.
• Uraemic pericarditis may respond to increased frequency of dialysis, to systemic or local steroid therapy and to aspiration therapy. Intrapericardial triamcinolone may be useful.
• Corticosteroids should not be used for initial treatment of pericarditis unless they are indicated for the underlying disease, the patient's condition has no response to NSAIDs or colchicine or both agents are contra-indicated.

Management of complications
• Immediate pericardiocentesis may be required when cardiac tamponade develops rapidly with falling blood pressure and shock developing. This may be done without echocardiographic guidance in an emergency; however, open thoracotomy is safer.
• Symptomatic constrictive chronic pericarditis usually requires pericardial resection.
• Recurrent or persistent symptomatic effusive pericarditis may be treated with balloon pericardiostomy, a surgical pericardial window, or sclerosis with tetracycline.
• Recurrent pericarditis - idiopathic pericarditis or pericarditis due to cardiac surgery, myocardial infarction, or trauma, may recur. Colchicine, 1 mg a day, may be helpful, in addition to NSAIDs. The role of steroids is controversial. Uncommonly, pericardial resection is required.

Prognosis
This will depend on the aetiology. Most patients with viral and idiopathic pericarditis experience self-limited symptoms that improve spontaneously within days to weeks [4]. A rapid response to aspirin or other NSAIDs usually leads to a favourable prognosis in acute pericarditis and an unlikely progression to complications.

One or more recurrences arise in 20-50% of patients after an initial episode of acute pericarditis [7].

Pericarditis associated with neoplasm, purulent effusion or tuberculosis has a complicated course and worse outcome. The mortality rate approaches 85% for untreated tuberculous pericarditis.

Further reading & references
3. 2015 ESC Guidelines for the diagnosis and management of pericardial diseases; European Society of Cardiology (August 2015

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