Pericarditis

Clinical features and management

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Background
Pericarditis is an important diagnosis to consider in a patient presenting with chest pain. It is diagnosed in 5% of patients presenting to hospital emergency departments with chest pain in the absence of a myocardial infarction.

Objective
This article describes the common features and management of pericarditis in the general practice setting.

Discussion
Characteristic clinical findings in pericarditis include pleuritic chest pain and a pericardial friction rub on auscultation of the left lower sternal border. Electrocardiography may reveal diffuse PR depressions and diffuse ST segment elevations with upward concavity. The most common aetiologies of pericarditis are idiopathic and viral, and the most common treatment for these is nonsteroidal anti-inflammatory drugs and colchicine. The complications of pericarditis include effusion, tamponade and myopericarditis. Pericardial effusion may present as a globular heart shadow on chest X-ray. The presence of effusion, constriction or tamponade can be confirmed on echocardiography. Tamponade is potentially life threatening and is diagnosed by the clinical findings of decreased blood pressure, elevated jugular venous pressure, muffled heart sounds on auscultation and pulsus paradoxus.

Keywords: pericarditis; chest pain/diagnosis

Case study

Michael, 32 years of age, presents to the hospital emergency department with chest pain of 4 hours duration.

Discussion

History and examination

Chest pain is a common presenting symptom. The initial assessment is clinical. Important elements on history are a description of the pain and its associated symptoms and presence of risk factors such as history of cardiovascular disease, renal impairment, diabetes, hypertension, dyslipidaemia, positive family history of cardiac disease, and smoking history. Cardiac and respiratory system history and focused musculoskeletal history looking specifically for trauma to area of pain are indicated. Relevant stressors and any upper gastrointestinal symptoms should be sought.

Examination includes cardiovascular examination looking for acute changes including vital signs, and particularly looking for signs of shock (clammy, tachycardia, decreased blood pressure), pulsus paradoxus, elevated jugular venous pressure (JVP), changes in heart or lung sounds and peripheral oedema. Respiratory and localised musculoskeletal and abdominal examinations are also likely to be indicated.

Common causes of chest pain are shown in *Table 1*.[1]

Case study (continued)

Michael stated that his pain was sharp and worse on inspiration. It had a sudden onset, did not radiate and was not associated with any shortness of breath. It was relieved slightly when he sat up and leaned forward.
Michael had no cardiac risk factors and no past medical conditions except for a recent 'flu-like' illness in the previous month. He had no risk factors for acquisition of HIV or tuberculosis, and no history of kidney disease.
On clinical examination, he was afebrile and, except for a soft pericardial friction rub, he had no other positive findings.

Discussion

History and examination findings

These symptoms are characteristic of pericarditis, a common condition that is diagnosed in 5% of patients presenting to hospital with chest pain in the absence of myocardial infarction.[2] Pericarditis is typically associated with sharp retrosternal pain (present in 98.3% of cases) that may radiate to the neck, shoulder or arms.[3] The pain is often worse when the patient is supine and improves when sitting upright or leaning forward. It may be aggravated by deep breathing, swallowing or coughing.

Pericardial friction rub is the most important

Table 1. Common conditions that cause chest pain[1]
Life threatening causes of chest pain
Myocardial infarction
Aortic dissection
Acute pulmonary embolism
Pericarditis with pericardial effusion/tamponade
Pneumothorax
Common aetiologies of chest pain presenting to general practice
Musculoskeletal/chest wall disease or injury (48.7% of cases)
Cardiovascular causes (16%)
Psychogenic/psychosomatic causes (11.5%)
Pulmonary causes (10.3%)
Gastrointestinal causes (8.2%)

physical sign of pericarditis (present in 35% of cases).[3] It is a high pitched, scratching sound and is heard most frequently at the left lower sternal border during expiration with the patient upright and leaning forward. Other important clinical signs to look for are fever ≥38°C and signs of tamponade (raised JVP, muffled heart sounds, decreased blood pressure). Patients with findings supportive of tamponade should be transferred to hospital urgently.

Other life threatening causes of chest pain symptoms similar to that of acute pericarditis include pulmonary embolus (PE) and acute myocardial infarction (AMI). Electrocardiogram (ECG) showing characteristic features may suggest a diagnosis. If the diagnosis is unclear, the patient should be sent to hospital for urgent assessment.

Pericarditis preceded by viral respiratory or gastrointestinal symptoms is suggestive of a viral aetiology. These are self limiting and respond well to analgesia and anti-inflammatory treatment.

Features suggestive of nonviral pericarditis include fever ≥38°C (bacterial pericarditis), kidney disease (uraemic pericarditis), unsafe sexual practices and intravenous drug use (HIV pericarditis), history of malignancy, autoimmune disease and risk factors for previous tuberculosis acquisition. Suspicion of a nonviral nonidiopathic aetiology of the pericarditis should prompt referral for review by a cardiologist.

Complications

Complications of acute pericarditis include pericardial effusion (present in 60% of cases[2]), tamponade (5% of cases[2]), myopericarditis and recurrent pericarditis. Pericardial effusion is the abnormal accumulation of fluid in the pericardial cavity. Cardiac tamponade occurs when this fluid accumulates under pressure and obstructs diastolic filling of the heart. Myopericarditis is another possibly serious complication, with extension of the inflammation to the myocardium which can be associated with creatinine kinase and troponin elevations and regional wall motion abnormalities on the echocardiogram.

Case study (continued)

Michael's ECG and chest X-ray are unremarkable. He had mild leukocytosis and moderately elevated C-reactive protein (CRP).

Discussion

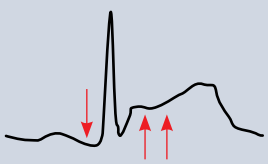
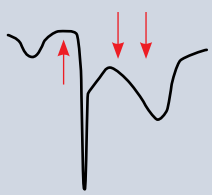
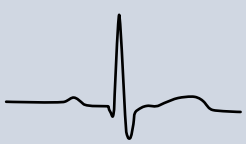
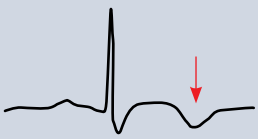
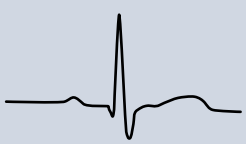
Electrocardiogram

Findings in pericarditis can include diffuse PR depression and diffuse ST elevation with upward concavity (AMI typically produces ST elevation with upward convexity). The ST elevation usually involves more than one coronary vascular territory and there is usually an absence of reciprocal ST changes between leads III and aVL (*Table 2, Figure 1*).[4]

Chest X-ray

Chest radiography is used to rule out pericardial effusion and abnormalities in the mediastinum or lung fields that may be responsible for a pericardial effusion (eg. lung carcinoma). It may also help to exclude other causes of chest pain (eg. pneumothorax).

Table 2. Stages of development of ECG changes in pericarditis

Stage	ECG change	
I	(a) Diffuse ST segment elevation (double arrows) and PR segment depression (single arrow) (b) Reciprocal PR segment elevation (single arrows) and ST segment depression (double arrows) in aVR and occasionally V1	(a)  (b) 
II	Normalisation of the ST segment and PR segments	
III	Widespread T wave inversions (single arrow)	
IV	Normalisation of the T waves	

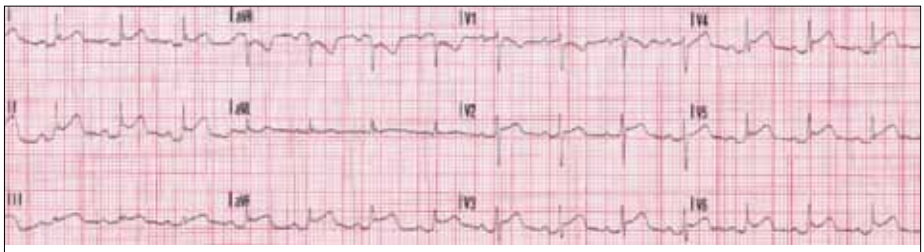


Figure 1. Diffuse ST elevations and PR depression in all leads except aVR and V1, which show reciprocal ST depression and PR elevation

A large cardiac silhouette with predominant increase in transverse diameter (globular or 'water bottle' shape) may indicate pericardial effusion. This is measured by dividing the maximal transverse width of the cardiac silhouette by the maximal internal thoracic diameter. A cardiothoracic ratio of 0.5 is the upper limit of normal. Unlike the enlarged cardiac silhouette seen in heart failure, in pericardial effusion the lung field usually appears clear (*Figure 2*). Comparisons with previous chest X-rays are useful. It is important to remember that a chest X-ray may appear normal in the presence of a small pericardial effusion (<250 mL).

Other investigations

A full blood count may reveal leukocytosis. C-reactive protein is usually elevated, although it is not specific. A troponin rise is detectable in more than 30% of cases as inflammation of the epicardium can lead to troponin release. However, unlike acute coronary syndrome, elevated troponin in pericarditis is not a negative prognostic marker.[5]

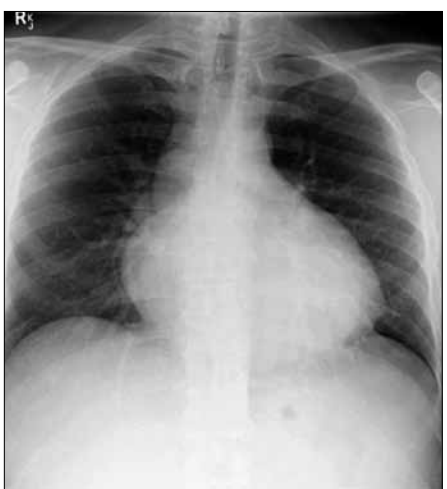


Figure 2. A large heart silhouette on chest X-ray due to pericardial effusion. Other common causes of a large heart silhouette include dilated cardiomyopathy

Case study (continued)

On the basis of his clinical findings, recent viral infection and low risk for ischaemic heart disease, Michael was diagnosed with viral pericarditis. His symptoms improved with 400 mg oral ibuprofen four times daily.

Management

Nonsteroidal anti-inflammatory drugs (NSAIDs) are the mainstay of treatment. Ibuprofen is preferred for its favourable effect on the coronary flow and large dose range. Depending on severity and response, 300–800 mg every 6–8 hours may be initially required and can be continued for days or weeks, ideally until the effusion has disappeared.[6] Gastrointestinal protection may be considered.

Colchicine (0.5 mg twice daily) added to an NSAID or as monotherapy also appears to be effective for the initial attack and the prevention of recurrences. It is well tolerated with fewer side effects than NSAIDs.[6]

The role of systemic corticosteroids in pericarditis is controversial. High dose corticosteroids (ie. prednisolone 1 mg/kg/day) with a 2–4 week taper is considered in pericarditis secondary to connective tissue disease, uraemia or autoreactivity.[6]

Case study (continued)

Two weeks later, Michael presented to the hospital with chest discomfort and dizziness on exertion. Clinical examination revealed muffled heart sounds, decreased blood pressure and a pericardial rub on cardiac auscultation. Jugular venous pressure was elevated and pulsus paradoxes of 25 mmHg was present.

Discussion

Michael's clinical findings are consistent with tamponade from a pericardial effusion. Clinical manifestations of pericardial effusion are dependent on the rate of accumulation of fluid in the pericardial sac. Rapid accumulation of small amounts of pericardial fluid may cause symptomatic elevation of intrapericardial pressures, while large but slowly progressing effusions can be asymptomatic. The classic triad of pericardial tamponade includes:

- dilated neck veins

Table 3. Clinical signs in the diagnosis of pericardial effusion

Cardiovascular	• Pulsus paradoxus: an exaggeration of physiologic respiratory variation in systemic BP, defined as a decrease in systolic BP >10 mmHg with inspiration, due to reduced cardiac output during inspiration • Kussmaul sign is a rise in JVP on inspiration. Kussmaul sign is seen in conditions in which right ventricular filling is limited by pericardial fluid, and in noncompliant pericardium or myocardium • Tachycardia • Hepatojugular reflux: this can be observed by applying pressure to the periumbilical region. A rise in the JVP of >3 cm H_2O for more than 30 seconds suggests elevated central venous pressure (transient elevation in JVP can be normal) • Pericardial rub
Respiratory	• Tachypnoea • Decreased breath sounds
Abdominal	• Hepatosplenomegaly
Other	• Weakened peripheral pulses • Oedema • Cyanosis

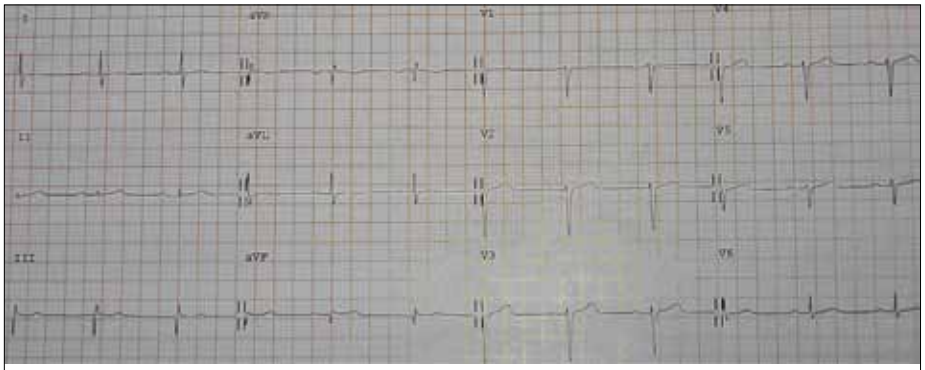


Figure 3. ECG showing poor R-wave progression and electrical alternans, best demonstrated in V1–V4

- a fall in blood pressure, and
- muffled heart sounds.

Other clinical findings of pericardial effusion are shown in *Table 3*. Fever ≥38°C, large pericardial effusion or cardiac tamponade, or failure to improve with aspirin or NSAIDs are risk factors associated with complications.[3]

Mechanism and measurement of pulsus paradoxus

During inspiration, the negative intrathoracic pressure increases, resulting in increased venous return to the heart. The right ventricle distends, causing the interventricular septum to bulge into the left ventricle. This results in a decrease in the left ventricular end diastolic volume, thus decreasing stroke volume and arterial pressure during systole. As intrapericardial pressures rise this effect becomes pronounced. The converse is true for expiration.

To measure pulsus paradoxus the sphygmomanometer cuff is inflated above systolic pressure. Korotkoff sounds are sought over the brachial artery while the cuff is deflated slowly. Initial Korotkoff sounds are heard only during expiration. The cuff is then deflated to the pressure at which Korotkoff sounds become audible during both inspiration and expiration. When the differences between these two levels exceeds 10 mmHg during quiet respiration, pulsus paradoxus is present.

Case study (continued)

An ECG and chest X-ray were performed. Initial blood tests were similar to those of his first presentation. Michael was admitted and a transthoracic echocardiogram was performed which showed up to 23 mm pericardial effusion with early tamponade (*Figure 4*).

Discussion

Electrical alternans is an alternation in the amplitude of QRS complexes. It is thought to be due to the swinging movement of the heart within the pericardial cavity (*Figure 3*). The combination of low voltage QRS complexes (≤5 mm) in limb leads and tachycardia should always raise concern about tamponade. Other causes of this combination include chronic obstructive pulmonary disease and pleural effusion.

Referral for echocardiography is useful for confirming the presence and size of an effusion. In circumstances where clinical evaluation is not reliable, echocardiography can be useful in detecting cardiac tamponade.

Case study (continued)

Pericardiocentesis was performed and 900 mL of blood stained fluid was successfully drained. Michael's symptoms were relieved, and he recovered over the next day. He was discharged on oral colchicine and ibuprofen with a follow up echocardiogram in 10 days.

Management

Pericardiocentesis is indicated for effusions that are moderate to large and are symptomatic, where medical management has been unsuccessful, or where pericardial fluid is needed for diagnostic purposes. Prompt referral to hospital for pericardiocentesis in hemodynamically significant pericardial tamponade may be lifesaving.

Colchichine at 0.6 mg twice daily for 3–6 months as an adjunct to conventional treatment has been shown to be effective for preventing recurrent pericarditis. There is little evidence of its efficacy as monotherapy and it is ineffective when used in patients with chronic pericardial effusions with a normal CRP.[7]

Case study (continued)

Michael's 10 day follow up transthoracic echocardiogram showed an asymptomatic re-accumulation of 1.7 cm of fluid and 250 mL of blood stained fluid was drained and collected for investigation. When recovered, Michael was discharged with an appointment for specialist outpatient follow up in 2 weeks. At this appointment further tests were ordered including tumour markers (alpha fetoprotein, carcinoembryonic antigen [CEA], carcinoma antigen [CA] 15.3), blood and pericardial fluid cultures for tuberculosis

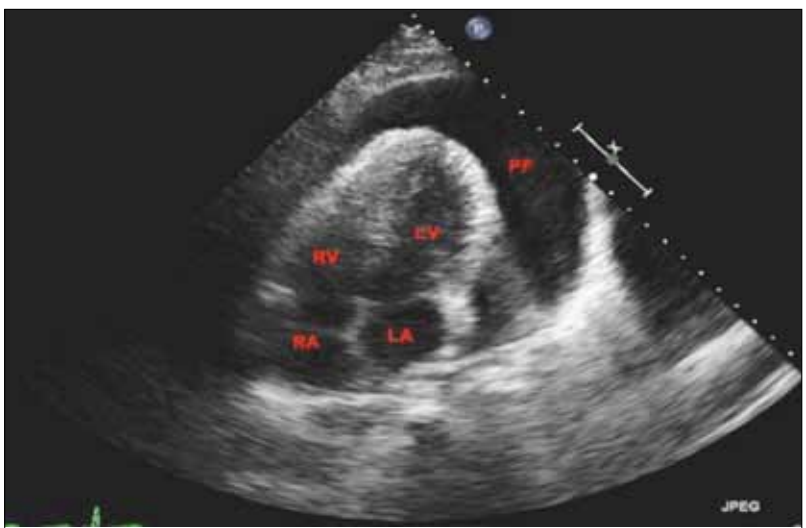


Figure 4. Michael's initial echocardiogram showing pericardial effusion of up to 23 mm fluid posteriorly with early tamponade
RV = right ventricle; RA = right atrium; LV = left ventricle; LA = left atrium; PF = pericardial fluid

and pericardial fluid cytology. These were negative and computerised tomography (CT) scan of the abdomen/chest did not show malignancy or lymphadenopathy and HIV serology was negative. Michael was also investigated for autoimmune disease. However, other than a mild unspecific elevation in antinuclear antibodies (ANA), autoimmune markers (ENA, pANCA, cANCA, dsDNA) were negative.

Discussion

Recurrence of hemorrhagic effusion is concerning, and when it occurs it is important to exclude serious causes of pericardial effusion such as malignancy, tuberculosis and HIV. Michael's diagnosis on discharge was recurrent viral pericarditis. As malignancy is a common cause of recurrent hemorrhagic pericarditis, pericardial tissue biopsy was considered to rule this out as he had re-presented with pericardial effusion.[8] Other causes of pericardial effusion are described in *Table 4*.

Key points

- Pericarditis is a common disease with characteristic clinical findings.
- Clinical diagnosis is made by the presence of characteristic chest pain symptoms, the presence of a pericardial friction rub and characteristic ECG findings.
- In Australia, it is most commonly of viral or idiopathic origin, both of which generally have a brief and benign course.
- NSAIDs and colchicine remain the mainstay of treatment for pericarditis.
- Cardiac tamponade is a serious complication of pericarditis and can be diagnosed clinically by the presence of decreased blood pressure, elevated JVP, muffled heart sounds on auscultation, and pulsus paradoxus.

Table 4. Causes of pericarditis/pericardial effusion

Cause	Prevalence	Test
Idiopathic	Most common	Often relates to lack of extensive diagnostic evaluation. Many patients with idiopathic pericarditis have had a recent viral infection
Infectious Viral/HIV	Most common cause of infectious pericarditis. Common organisms include coxsackie virus A and B and echovirus. Pericarditis may be preceded by a prodrome of upper respiratory symptoms	Serologic test for HIV in high risk patients Serology for other viral organisms not routinely performed, as it is low yield and the result is unlikely to influence management
Bacterial	May occur following pneumonia Suggested by fever ≥38°C	High mortality. Prompt blood and pericardial fluid culture may help find the offending organism
Tuberculosis	The aetiologic spectrum of pericarditis is different in developing countries, which have a higher prevalence of tuberculosis	Chest radiography, tuberculin test, histology, cultures
Autoimmune	Rheumatoid arthritis, systemic lupus erythematosus	Rheumatoid factor, complement levels, antinuclear antibodies
Neoplastic	Malignancies with the highest prevalence of pericardial effusion include lung (37%), breast (22%), and leukaemia/lymphoma (17%)	Chest radiography, pericardial fluid cytology, tumour markers Hemorrhagic effusion
Postoperative	Common after cardiac surgery	History, evidence of polyserositis, high ESR
Following MI	Often associated with a large anterior myocardial infarction	History Echocardiography
Aortic dissection	Rare	Trans-oesophageal echocardiography, CT aortogram, magnetic resonance imaging
Uraemia	Patients with chronic kidney disease, before or after dialysis	Urea and creatinine

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