Acute pericarditis: An overview

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Acute pericarditis is a common cardiovascular disorder which may present in primary and secondary care settings. Patients typically seek advice because of chest pains. This condition is part of the spectrum of pericardial diseases; other elements include congenital lesions, pericardial effusion, chronic pericarditis, recurrent pericarditis, cardiac tamponade and pericardial constriction. The most common pericardial disease is acute pericarditis (Spodick, 2005; Imazio et al, 2009). Acute inflammation of the pericardium may occur as an isolated clinical problem, as in viral or idiopathic pericarditis, or a manifestation of systemic disease, such as malignant neoplasms, post-myocardial infarction, renal failure, metabolic disorders, immunodeficiency or trauma. In developed countries, approximately 85% of cases of acute pericarditis are viral or idiopathic in the immunocompetent patient (Imazio et al, 2010; Sheth et al, 2010). The terms 'viral' and 'idiopathic' are used almost interchangeably, as most cases of 'idiopathic' pericarditis are actually of viral origin.

Acute pericarditis is diagnosed in approximately 0.1% of hospital admissions, accounting for up to 5% of emergency department visits for chest pain without myocardial infarction (Tingle et al, 2007). Patients may present with classic pericarditis symptoms. However, on occasion acute pericarditis may mimic an acute myocardial infarction (MI).

Most patients with viral or idiopathic pericarditis experience self-limiting symptoms that improve with pharmacological intervention—complications are rare (Imazio et al, 2010). It is possible for low-risk patients to be managed as an outpatient but hospitalisation is recommended for higher risk groups.

In contrast to other areas within the field of cardiology, there are few randomised control trials to guide the diagnosis and management of pericardial disease and therefore treatment is empirical. The only published guidelines are from the European Society of Cardiology (ESC) (Maisch et al, 2004). These guidelines present the relevant evidence in order to guide physicians to weigh up the benefits and risks of a particular diagnostic or therapeutic procedure.

ABSTRACT

Acute pericarditis is a common cardiovascular condition that may present in primary and secondary care settings. This disorder has multiple causes. However, in developed countries up to 85% of acute pericarditis is caused by viral or idiopathic origins. This article will focus mainly on this aetiology. It is important for health-care professionals involved in the assessment of chest pain and management of this group of patients to have a comprehensive understanding of the condition. This overview discusses the anatomy of the pericardium, the aetiology of acute pericarditis, clinical presentation, diagnostic procedures and treatment strategies associated with this disorder. The European Society of Cardiology Clinical Guidelines on the Diagnosis and Management of Pericardial Diseases (2004) and recent relevant research are used to underpin this article.

KEY WORDS
• Acute pericarditis • Pericardium • Chest pain • Diagnosis • Assessment

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Anatomy and physiology

The pericardium is a double-layered fibroserous sac that covers the entire myocardium and extends onto the great vessels. The sac anchors the heart in the central thorax while separating it from other mediastinal structures.

Each pericardium layer is around 1–2 mm thick (*Figure 1*): the outer layer of this sac is the tough, dense fibrous pericardium, which helps to protect the heart by acting as a mechanical and immunological barrier. It also helps limit over-filling and distention of the heart, assisting normal cardiac function. The inner layer is the serous pericardium; a thin slippery, two-layer serous membrane that forms a loose sac around the heart. Its parietal layer lines the internal surface of the fibrous pericardium and attaches to the great arteries exiting the heart. It then turns inferiorly, continuing over the external heart surface as the visceral layer, also known as the epicardium (Marieb, 2013). The visceral and parietal layers are separated by the pericardial cavity, which contains 15–50 mls of serous 'pericardial fluid'. This fluid lubricates cardiac motion and decreasing friction between the two layers during contraction and relaxation. However, the pericardium is not essential, normal cardiac function can be maintained in its absence (Khandaker et al, 2010).

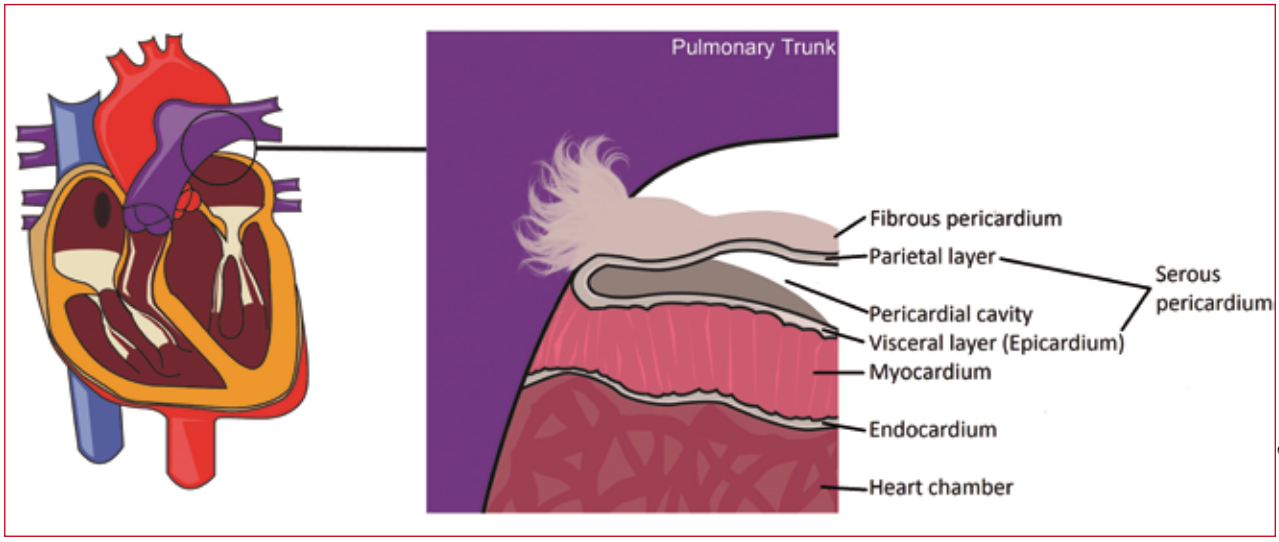

Figure 1. Structure of the pericardium

Pericarditis is a result of an inflammatory process usually affecting the inner visceral layer and the outer parietal layer of the pericardium; this may be acute or chronic. Men have a higher incidence of pericarditis than women and children, it usually occurs between the ages of 20 and 50 years (Carter and Brookes, 2005).

Aetiology

There are many causes of acute pericarditis (*Table 1*). However, up to 85% of cases have a viral or idiopathic aetiology. Pericarditis in which the cause cannot be established is considered to be 'idiopathic', although most patients have a viral pericardial syndrome. The term 'idiopathic' persists because in clinical practice it is not productive to search for specific viral aetiologies due to limitations in laboratory techniques. A definite diagnosis would require analysis of pericardial fluid or tissue, which is invasive and the influence of the findings would not change the clinical management of patients. Acute idiopathic pericarditis typically follows a brief and benign course and patients have a good prognosis. Cases are often preceded by a recent flu-like illness or gastrointestinal symptoms. The viral illness triggers the replication of the virus in the pericardium, which elicits a cellular response and leads to inflammation (Sheth et al, 2010).

In Western countries bacterial pericarditis is rare, but it is still often seen in the developing world and if left untreated is fatal. With treatment, mortality can still be as high as 40% due to complications such as bacterial sepsis and cardiac tamponade (Sheth et al, 2010).

Pericarditis can occur post MI from between 1 and 3 days as a reaction to inflammation and healing of the myocardium. An estimated 7% of post MI patients develop the condition (Futterman and Lemberg, 2006). Less commonly, pericarditis may occur 1–4 weeks post MI or cardiac surgery as part of Dressler's syndrome, a systemic inflammatory condition. Dressler's syndrome is thought to be caused by autoimmune reaction mediated by antibodies due to various myocardial antigens (Tingle et al, 2007). The patient may experience chest pain, pleurisy and electrocardiogram (ECG) changes consistent with pericarditis. The incidence of Dressler's syndrome has decreased over the years owing to more prompt reperfusion therapies.

Clinical presentation and diagnosis

The classic symptoms of acute pericarditis are chest pain and possible indicators suggestive of a recent viral infection

Table 1. Causes of acute pericarditis

- Idiopathic
- Infectious pericarditis
  - Viral (coxsackie B, influenza, echovirus, Epstein-Barr)
  - Tuberculosis
  - Bacterial infections
- Post-myocardial infarction pericarditis
- Dressler's syndrome
- Post-cardiac surgery
- Neoplasms (primary or secondary)
- Connective tissue disease (systemic lupus erythematous)
- Drug induced
- Hypothyroidism
- Radiation
- Uraemia

Table 2. Diagnostic criteria (two or more should be present)

- Typical chest pain
- Suggestive ECG changes (typically ST-segment elevation, PR depression)
- Pericardial rub
- Pericardial effusion

(Adapted from Sheth et al, 2010)

Table 3. Presenting pain in acute pericarditis compared to myocardial ischaemia/infarction

	Pericarditis	Myocardial ischaemia
Location	Retrosternal area and left precordium	Same as pericarditis
Radiation	Back, trapezius ridge(s)	Across chest, shoulders, arms, neck, throat, jaw and back. Not trapezius ridge
Character	Sharp, pleuritic,	Heavy, crushing, pressure
Inspiration	Pain increases	No effect
Body movement	Pain worse on laying flat; eases when sitting up and leaning forward	Usually no effect
Duration	Persistent	Intermittent
Onset	More often sudden	Can be crescendo with ischaemia or sudden with infarction
Glycerine-trinitrate	No relief	Usually pain relieved (not with infarction)

Table 4. ECG changes in acute pericarditis*

Stage	ECG change
Stage 1	Diffuse concave ST-segment elevation during the first few days of pericardial inflammation, lasting up to 2 weeks. The appearance of the ST-segment elevation of pericarditis differs from the usual convex appearance of acute myocardial ischaemia
Stage 2	Return of ST-segments to baseline and flattening of the T wave lasts from days to several weeks
Stage 3	Inversion of the T waves begin at the end of the second or third week
Stage 4	Gradual resolution of T wave that may last up to 3 months

*Some cases do not include all four stages

(Adapted from Conover, 2003)

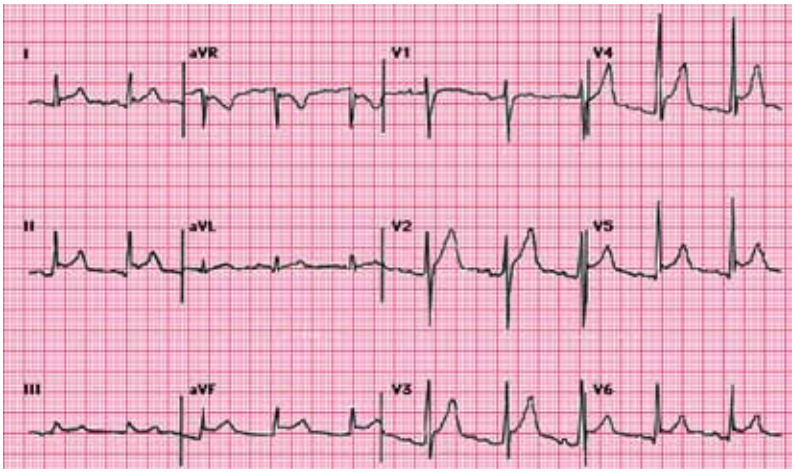

Figure 2. A 12-lead ECG from a patient with acute pericarditis

such as fever, fatigue, respiratory or gastrointestinal symptoms. In clinical practice the diagnosis of acute pericarditis is based on four main criteria (*Table 2*). It has been suggested that two or more features should be present for the diagnosis of acute pericarditis (Imazio and Trinchero, 2007; Sheth et al, 2010). Tests and investigations that assist diagnosis include an electrocardiogram, chest X-ray, echocardiography and laboratory testing.

Chest pain

Patients with acute pericarditis usually present with sudden onset chest pain, which is sharp and stabbing in nature and is typically located in the retrosternal or left precordial area. The pain is usually aggravated by inspiration and coughing. This is because the pericardium is attached to the sternum, great vessels and the diaphragm; during inspiration, the diaphragm pulls on the inflamed pericardium resulting in increased pain. Lying flat also increases pain while it can be eased by sitting up and leaning forward. Frequently, pericardial pain radiates to one or both of the trapezius muscle ridges because the phrenic nerves that innervate these muscles pass through the pericardium. Spodick (2008:398) states that 'trapezius ridge pain is an almost 100% giveaway that the pericardium is diseased'.

Pericarditis chest pain can also mimic pain associated with myocardial infarction, as the pain can radiate to the neck, arms or the left shoulder making it difficult to distinguish from ischaemia (Khandaker et al, 2010; Sheth et al, 2010). Classical differences are shown in *Table 3*.

Patients may be dyspnoeic, which is not related to exertion but to the reluctance of the patient to take a deep inspiration because of increased levels of pain. Shortness of breath is also more apparent in the rare complications of pericarditis and can be a sign of pericardial effusion and cardiac tamponade.

Pericardial rub

On physical examination, a pericardial rub is common, caused by the rub between the visceral and parietal surfaces. Sheth et al (2010) describe the pericardial rub as having three distinct components that correspond to the phases of greatest cardiac movement: atrial contraction, ventricular contraction and ventricular relaxation, respectively. The pericardial rub has a high-frequency sound, generally strongest at the left lower to middle sternal edge, with a peculiar grating, scratching quality. Interestingly, Imazio et al (2004) studied a cohort of patients with acute pericarditis and confirmed a pericardial rub in only 35% of patients, demonstrating this diagnostic criteria has a poor sensitivity.

Electrocardiogram

An ECG is the most useful diagnostic tool in acute pericarditis. ECG changes can occur quickly in a few hours or as long as several days after the onset of chest pain (Ratib et al, 2011). The ECG changes occur in four

stages; however, these changes may not be seen in all cases (*Table 4*). Patients usually present in normal sinus rhythm or sinus tachycardia. Classically, the ECG reveals widespread upward concave or 'saddle-shaped' ST-segment elevation in most leads, except V1 and AVR. PR depression is evident, reflecting the abnormal repolarisation that develops secondary to pericardial inflammation (*Figure 2*). PR segment elevation in AVR is also a hallmark of acute pericarditis (Riera et al, 2011). In contrast, during an MI there is a convex appearance of the ST-segment that occurs during the acute injury stage, where there is specific arterial territory ST elevation. For example, an inferior MI would show ST elevation only in II, III and AVF often with reciprocal changes.

In some cases the diagnosis of acute pericarditis may be challenging as symptoms may mimic an acute MI, due to the presence of chest pain and ST elevation. Prompt revasculisation is necessary for ST elevation MI to preserve myocardium, so performing urgent coronary angiography may be necessary when the diagnosis is uncertain. It has been reported that up to 20% of patients with pericarditis are taken to the cardiac catheter laboratory as an initial diagnosis could not be differentiated from acute myocardial infarction (Salisbury et al, 2009). This highlights the importance of a thorough history-taking and clinical examination in order to aid a correct diagnosis.

Chest X-ray

In many cases, the chest X-ray (CXR) is normal in uncomplicated acute pericarditis. However, a number of changes are possible:

- A pericardial effusion may develop and if large (usually greater than 250 mls) will result in enlargement of the cardiac shadow, which gives a globular shape (Goyle, 2002)
- Pulmonary congestion could be present in associated myocarditis
- Pleural effusions may be seen.

Echocardiography

When pericarditis is suspected, the ESC Guidelines (Maisch et al, 2004) recommend echocardiography to aid in management and diagnosis. Transthoracic echocardiography can determine the presence and size of a pericardial effusion and contractile dysfunction from myocarditis, as well as haemodynamic changes associated with tamponade and constrictive pericarditis.

In cases where it is unclear if the patient is suffering from acute pericarditis or MI, echocardiography can provide further information on territorial myocardial hypokinesia or akinesia evident in MI, which would assist diagnosis.

Laboratory tests

Non-specific blood tests can provide evidence of active inflammation. This includes white cell count (WCC), erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), which are usually elevated in acute pericarditis.

Markers of myocardial damage, such as Troponin I and Creatinine Kinase MB fraction (CK-MB), can also be elevated in acute pericarditis, which suggests some degree of myocardial involvement known as myopericarditis. The inflammation of the two structures coexists, sharing the same viral aetiology but not of equivalent intensity. Studies have shown that between 14 and 32% of patients with viral or idiopathic acute pericarditis have a raised Troponin I, suggesting some myocardial involvement (Imazio et al, 2003; Imazio, 2008; Machado, 2010). This may be superficial involving the epicardium causing a minimal Troponin rise or a higher level of Troponin generally observed in myopericarditis, where there may be evidence of ventricular wall abnormalities on the echocardiogram.

Historically, there have been conflicting views on the prognosis of myopericarditis in comparison to acute pericarditis. However, a recent study (Imazio et al, 2013) of 486 patients with acute pericarditis with or without a myocardial inflammatory syndrome showed that unlike acute coronary syndromes, raised Troponin I does not indicate a negative prognostic marker in this group of patients. However, myopericarditis may put the patients at higher risk of short-term complications (Imazio, 2007).

Other specific haematological, biochemical and serological investigations may be requested to assist diagnosis dependent on suspected aetiology (Ratib et al, 2011):

- Urea and electrolytes (U&Es)
- Antistreptolysin O titres, anti-Dnase B titre, throat swabs (acute rheumatic fever)
- Blood culture
- Acute and convalescent viral titres, monospot or Paul-Brunnell test (Epstein-Barr virus), cold agglutinins (mycoplasma), fungal precipitins
- Sputum, urine and faecal samples for microbiology
- Heaf test (tuberculosis)
- Autoantibodies (lupus, rheumatoid arthritis, systemic sclerosis)
- Thyroid function tests.

Management and treatment

The treatment of acute pericarditis is directed at the underlying cause. For patients with idiopathic or viral pericarditis, therapy is focused on symptom relief with analgesia, bed rest and reduction of physical activity. Management is largely empirical owing to the relative lack of guidance. The only existing guidelines from the ESC state that 'hospitalisation is warranted for most patients to determine aetiology, to observe for cardiac tamponade, and to start anti-inflammatories and symptomatic treatment' (Maisch et al, 2004: 4). However, there is evidence to support that low-risk patients, who are haemodynamically stable without high-risk clinical features (*Table 5*) can be discharged home safely with outpatient medical management, which includes adequate

pain relief and rest. (Imazio et al, 2007; Frexia, 2010). A clinical risk stratification tool has been produced to facilitate the above and can be completed on the patient's presentation (*Figure 3*).

Medical management consists of three major agents; non-steroidal anti-inflammatory drugs (NSAIDs), colchicine and corticosteroids (*Table 6*).

Non-steroidal anti-inflammatory drugs

NSAIDs are the main therapy for viral pericarditis in order to relieve pain and resolve inflammation. Ibuprofen or aspirin have been the most commonly used and provide prompt relief of pain in most patients (Khandaker et al, 2010). In patients who do not respond to NSAID therapy in 1 week, an aetiology other than idiopathic or viral should be sought (Imazio et al, 2004). The ESC guidelines (2004) favour ibuprofen as the anti-inflammatory due to its rare side effects, favourable effect on the coronary flow and the large doses of 300–800 mgs every 6–8 hours, which can be initiated and continued for days or weeks. Indomethacin has been commonly prescribed 25–50 mg three times a day.

Aspirin is the recommended treatment of pericarditis post MI because of the requirement of antiplatelet therapy. The anti-inflammatory actions of a NSAID other than aspirin may interfere with myocardial healing and scar formation. Indomethacin should be avoided by patients with coronary artery disease as it decreases coronary artery flow (Schifferdecker and Spodick, 2003).

When using NSAIDs, it is important to consider the medication's side effects, such as platelet inhibition, renal effects and gastrointestinal upset/bleeding. Gastrointestinal protection should be prescribed in all patients.

Colchicine

Previously, colchicine has been effective in the treatment of recurrent pericarditis by reducing the inflammatory response. However, recently Imazio et al (2013) conducted a randomised trial for patients presenting for the first time with acute pericarditis. When given colchicine in addition to conventional anti-inflammatory treatment, the study showed a significant reduction in the rate of persistent or recurrent pericarditis in this group of patients. This combination treatment is now commonly used in current practice—usually 0.5 mg twice daily for 3 months.

Steroids

In general, corticosteroids are reserved for patients with an auto-immune/rheumatological aetiology and patients who have had no response or a contraindication to NSAIDs and colchicine (ESC, 2004; Khandaker, 2010).

Complications

Generally, acute idiopathic or viral pericarditis is benign and self-limiting. Recurrence is the most common complication, affecting up to 30% of individuals after a first attack (Soler-Soler et al, 2004; Markel et al, 2013). Recurring attacks of pericarditis are usually associated

Table 5. High-risk pericarditis patients

- Large circumferential pericardial effusion (total echocardiographic depth ≥ 2 cm)
- History of anticoagulant treatment
- History of, or concurrent, malignancy, particularly lung, breast, lymphoma and leukaemia
- Internal or external chest trauma (including pericarditis after surgery and recent catheter or device implantation)
- Fever exceeding 38°C
- Sub-acute onset (days to weeks)
- Immunosuppression, including HIV infection
- Evidence of myopericarditis
- Atypical electrocardiographic evolution
- Pulsus paradoxus
- Significantly increased acute phase reactants and very high cardiac Troponin

(Reproduced from Spodick (2008) with permission)

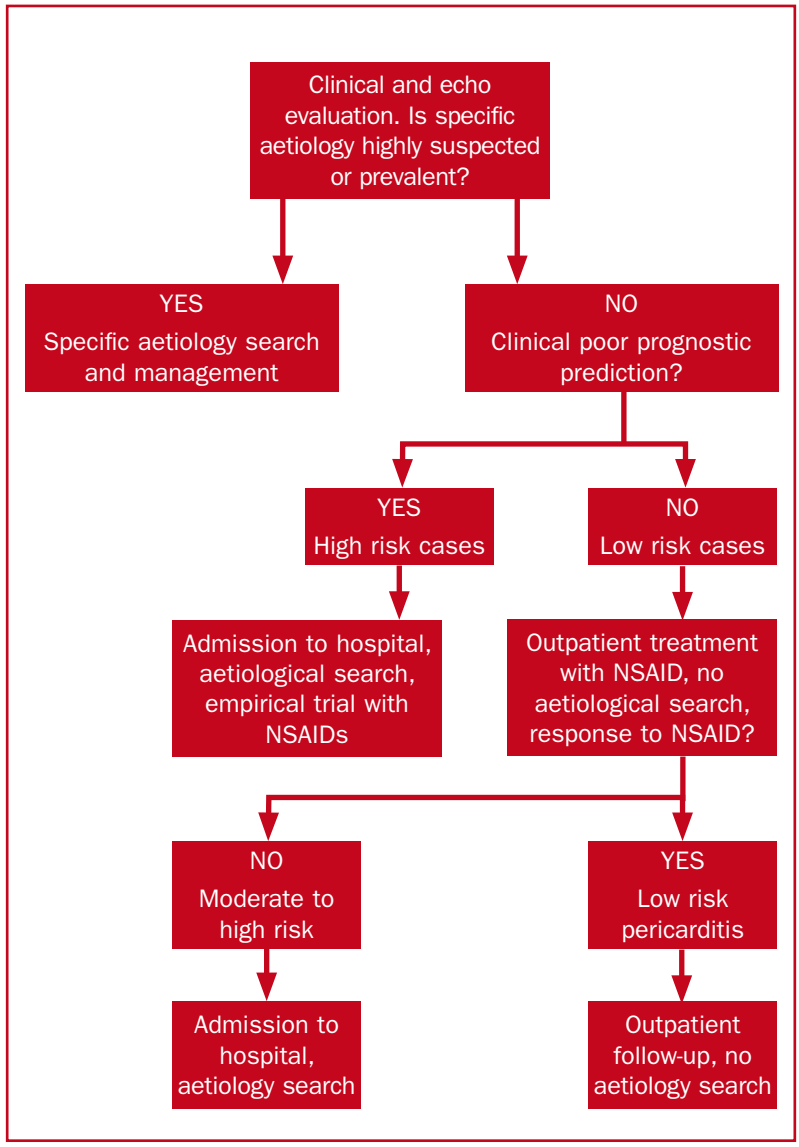

Figure 3. Acute pericarditis triage (Imazio and Trincero, 2007)

with a progressive reduction in the intensity of the clinical manifestations. The more serious complications of acute pericarditis are cardiac tamponade and constrictive pericarditis; these complications are rare but seen more commonly in specific aetiology pericarditis or in patients with clinical high-risk features (*Table 5*) (Spodick, 2008). For patients within this group hospitalisation is necessary to monitor clinical evolution and a full aetiological search is also warranted.

Chronic pericarditis

Unlike acute pericarditis, which begins suddenly, chronic pericarditis builds up gradually and lasts longer than 3 months. There are two main types of chronic pericarditis:

- Chronic effusive-constrictive pericarditis, in which fluid slowly accumulates in the space between the two layers of the pericardium causing inflammation and constriction.
- Chronic constrictive pericarditis, a rare disease that develops scar-like, fibrous tissue throughout the pericardium, which impedes normal diastolic filling due to the loss of elasticity. Constrictive pericarditis can occur following any pericardial disease; in developed countries cardiac surgery, idiopathic or viral, and radiation therapy are the most common aetiologies (Imazio et al 2010; Khandaker et al, 2010).

Table 6. Medical therapy for acute pericarditis

- Non-steroidal anti-inflammatory drugs (NSAIDs)
  - Aspirin
  - Ibuprofen
  - Indomethacin
- Colchicine
- Prednisolone
- Gastrointestinal protection

Key Points

- There are a number of causes of acute pericarditis, idiopathic and viral aetiology are the most common accounting for up to 85% of cases
- The clinical diagnosis of acute pericarditis is confirmed when at least two of the four clinical criteria are present: typical chest pain, pericardial friction rub, widespread ST-elevation, and pericardial effusion
- Trapezius ridge pain is very specific to pericardium disease
- Differentiating pericarditis from a myocardial infarction with ST segment elevation (STEMI) can be challenging. Pericardial pain can manifest as dull radiating chest pain that may mimic symptoms of an acute myocardial infarction (AMI). Early recognition of STEMI is essential for prompt revasculisation
- Myopericarditis is not uncommon in patients with acute viral or idiopathic pericarditis. Myocardial damage is more common in younger male patients
- Medical management consists of three major agents: NSAIDs, colchicine and corticosteroids
- Colchicine, when used in addition to conventional anti-inflammatory therapy in patients with a first episode of acute pericarditis, reduces the rate of persistent or recurrent pericarditis
- Pericarditis is usually self-limiting and resolves with no long-term effects

Implications for nursing practice

Cardiac nurses involved in the assessment and ongoing management of acute chest pain will often encounter patients with suspected or diagnosed acute pericarditis. Symptoms and signs that would alert nurses to the possibility that the patient is suffering from this condition are: precordial chest pain radiating to the trapezius, intensified on deep inspiration and lying supine; a pericardial friction rub; temperature elevation; and concave widespread ST elevation on the patient's ECG (*Figure 2*).

The nursing management of pericarditis focuses on the promotion of bed rest and providing appropriate analgesia while monitoring its effect. Assisting patients into the most comfortable position is helpful; as previously mentioned, the upright leaning forward position is likely to relieve chest pain and dyspnoea if present. Nursing staff should be aware of the rare potential complications of acute pericarditis, cardiac tamponade being the most serious. The patient's cardiovascular status needs to be monitored accordingly to assess for signs and symptoms of this complication, which includes: hypotension, tachycardia, tachypnoea, raised jugular venous pressure and restlessness. This complication is more likely to happen in higher-risk patients (*Table 5*).

Psychological support for patients with acute pericarditis is paramount. Patients will need reassurance that the chest pain is not due to an MI, and a full explanation of their condition and its management is required. Furthermore, patients should be counselled on the high recurrence rate (30%) of acute pericarditis.

This article has provided an overview of the clinical manifestations, assessment, diagnosis and treatments associated with the condition, focusing mainly on viral and idiopathic pericarditis. Such information will enable cardiac nurses to develop a fuller understanding of the condition which will enhance practice and help educate patients about the disease process and its management.

Conclusion

The aetiology of pericarditis is varied with viral or idiopathic being most common in clinical practice. There is limited evidence-based data available to direct the management of acute pericarditis with the ESC guidelines

(2004) being the only published standards. Diagnostic evaluation for all patients with suspected pericarditis includes a thorough history, being mindful that differentiating pericarditis from a MI can often be challenging; followed by a physical assessment including auscultation, ECG, CXR, transthoracic echocardiogram, inflammatory markers and Troponin I.

Idiopathic pericarditis is often self-limiting and can be treated effectively with bed rest, NSAIDs and colchicine. The evidence suggests that clinical risk stratification of acute pericarditis can be performed in order to identify low-risk patients who may be treated as an outpatient safely. Patients with high risk features more associated with specific aetiologies or complications should be hospitalised.

Further research and updated guidelines are needed to improve diagnosis and the management of acute pericarditis. BJCN

References

Carter T, Brooks CA (2005) Pericarditis: Inflammation or infarction? *J Cardiovasc Nurs* **20**(4): 239–44

Conover MB (2003) *Understanding Electrocardiography* (8th edn.) Mosby, London

Freixa X (2010) Evaluation, management, and treatment of acute pericarditis and myocarditis in the emergency department. *Emergencias* **22**(4): 301–6

Futterman LG, Lemberg L, (2006) Pericarditis. *Am J Crit Care* **15**(6): 626–30

Goyle KK, Walling AD (2002) Diagnosing Pericarditis. *Am Fam Physician* **66**(9): 1695–702

Imazio M, Demichelis B, Cecchi E, Belli R, Ghisio A, Bobbio M, Trinchero R (2003) Cardiac Troponin I in Acute Pericarditis. *J Am Coll Cardiol* **42**(12): 2144–8

Imazio M, Demichelis B, Parrini et al (2004) Day-hospital treatment of acute pericarditis: A management program for outpatient therapy. *J Am Coll Cardiol* **43**(6): 1042–6

Imazio M, Trinchero R (2007) Triage and Management of Acute Pericarditis. *Int J Cardiol* **118**(3): 286–94

Imazio M, Demichelis B, Chinaglia A, Ierna S, Demarie D, Ghisto A, Pomari F, Beli R, Trinchero R, (2008) Myopericarditis versus viral or idiopathic Acute Pericarditis. *Heart* **94**(4): 498–501

Imazio M, Brucato A, Trinchero R, Alder Y (2009) Diagnosis and management of pericardial diseases. *Cardiology* **6**: 743–51

Imazio M, Spodick DH, Brucato A, Trinchero R, Markel G, Adler Y (2010) Diagnostic issues in the clinical management of pericarditis. *Int J Clin Pract* **64**(10): 1384–92

Imazio M, Brucato A, Antonio MD et al (2013) A randomised trial of colchicine for acute pericarditis. *N Engl J Med* **369**(16): 1522–8

Khandaker MH, Espinosa RE, Nishimura RA, Sinak LJ, Hayes SN, Melduna RM, Oh JK (2010) Pericardial Disease: Diagnosis and Management. Mayo Clin Proc **85**(6): 572–93

Machado S, Roubille F, Gahide G et al (2010) Can troponin elevation predict worse prognosis in patients with acute perricarditis? *Ann Cardiol Angeiol* (Paris) **59**(1): 1–7

Maisch B, Seferović PM, Ristić AD et al (2004) Guidelines on the Diagnosis and Management of Pericardial Diseases. The European Society of Cardiology. *Eur Heart J* **25**(7): 587–610

Marieb EN, Hoehn K (2013) *Human Anatomy and Physiology* (9th edn.). Pearson, London

Markel G, Imazio M, Brucato A, Alder Y (2013) Prevention of Recurrent Pericarditis with Colchicine in 2012. *Clin Cardiol* **36**(3): 125–8

Ratib K, Bhatia G, Uren N, Nolan J (2011) *Emergency cardiology: An evidence guide to acute cardiac problems* (2nd end.). Hodder Arnold, London

Riera ARP, Ferreira C, FilhoCF, Dubner S, Barros RB, Femenia F, Baranchuk A (2011) Clinical value of lead aVR. *Ann Noninvasive Electrocardiol* **16**(3): 295–302

Salisbury AC, Olalla-Gomez C, Rihal CS, Bellwezosa G, Oh JK (2009) Frequency and predictors of urgent coronary angiography in patients with acute pericarditis. *Mayo Clin Proc* **84**(1): 11–5

Sheth S, Wang DD, Kasapis C (2010) Current and emerging strategies for the treatment of acute pericarditis: A systematic review. *J Inflamm Res* **3**: 135–42

Schifferdecker B, Spodick DH (2003) Nonsteroidal anti-inflammatory drugs in the Treatment of Pericarditis. *Cardiol Rev* **11**(4): 211–7

Soler-soler J, Sagrista-Sauleda J, Permanyer-Miralda G (2004) Relapsing Pericarditis. *Heart* **90**(11): 1364–8

Spodick DH (2005) Pericardial disease. In: Rossendorf C (ed.) *Essential Cardiology: Principles and Practice.* Humana Press, New Jersey: 653–61

Spodick DH (2008) Risk prediction in pericarditis: Who to keep in hospital. *Heart* **94**(4): 398–9

Tingle LE, Molina D, Calvert CW (2007) Acute pericarditis. *Am Fam Physician* **76**(10): 1509–14