UNIT 4 DISEASES OF PERICARDIUM

Structure

4.0 OBJECTIVES

After reading this unit, you should be able to:

- understand the structure and functions of pericardium;
- describe the various diseases of the pericardium, their recognition and management;
- differentiate acute pericarditis from acute myocardial infarction;
- use echocardiography in the diagnosis of pericardial diseases;
- diagnose and manage cardiac tamponade; and
- recognize constrictive pericarditis and plan management.

4.1 INTRODUCTION

Pericardium is the sac covering the heart. Pericardium consists of two layers—the visceral pericardium (epicardium) and the parietal pericardium. The visceral pericardium directly lines the heart surface. The cavity between the two layers of the pericardium contains approximately 15 to 35 ml. of serous fluid—the pericardial fluid. The visceral pericardium is a thin membrane—a monolayer of mesothelial cells, while the parietal pericardial thickness may be up to 2.5 to 3.0 mm. Perietal pericardium is attached loosely by ligaments to sternum and vertebral bodies and firmly to central tendon of the diaphragm.

The phrenic nerves lie over the parietal pericardium and supply most of it. Vagal fibres from oesophageal plexus also supply pericardium.

The internal mammary arteries and small branches from aorta supply the pericardium.

The parietal pericardial lymphatics drain to the anterior and posterior mediastinal nodes while the visceral pericardial lymphatics drain to tracheal and bronchial mediastinal nodes.

The pericardium buttress the thinner chambers of the heart, the atria and right ventricle and maintains the normal diastolic pressures and dimensions in these chambers. The pericardium can prevent acute cavitary dilatation. The pericardial fluid reduces the friction during heart

movement. The ligamentous attachment of pericardium to the neighbouring structures prevent cardiac displacement. The normal intrapericardial pressure can vary from -5 to +5 mm. of Hg. though mostly it is negative. The pericardial pressure varies during respiration along with pleural pressure.

4.2 ACUTE PERICARDITIS

Acute pericarditis is defined as acute inflammation of the pericardium and is clinically characterized by chest pain, fever, tachycardia and pericardial friction rub. It has characteristic electrocardiographic (ECG) changes. It is more common in men.

Etiology

a) Idiopathic: Most common cause, though many of these cases could be of viral etiology.

b) Viral: The Coxsackie B and Echovirus are most common pathogens.

c) Purulent: Purulent pericarditis due to staphylococci, pneumococci or strephtococci can occur.

d) Tuberculous: Tuberculosis can infect the pericardium resulting in tuberculous pericarditis.

e) Pericarditis in acute myocardial infarction: Transmural myocardial infarction can cause pericardial inflammation in 12 – 15 per cent of cases. It is more often detected in anterior wall myocardial infarction and inferior wall with right ventricular infarction. With the widespread use of reperfusion therapy, the incidence of pericarditis in acute myocardial infarction has significantly come down.

f) Uremic: Uremic patients can develop pericarditis.

g) Neoplastic: Malignant tumours especially of lung and breast and lymphomas can involve the pericardium leading to pericarditis with effusion.

h) Collagen Disorders: Collagen disorders like lupus erythematosis, rheumatic fever and rheumatoid disease can cause pericardial inflammation.

i) Dressler's Syndrome: Occurs 2 weeks to 2 years after acute myocardial infarction. The exact cause of Dressler's syndrome is not clear, though proposed to be autoimmune in nature.

j) Post pericardiotomy Syndrome: Occurs 6 to 8 weeks after cardiac surgery. Thought to be autoimmune in mechanism.

k) Rare infecting organisms: Rarely fungal, rickettsial, spirochetal and parasitic infections can lead to pericarditis.

l) Traumatic: Traumatic perforation of the pericardium during procedures like pacemaker insertion, catheter ablation for arrhythmias or interventional procedures or pericardial perforation from penetrating chest injury or oesophageal perforation can cause pericardial inflammation.

m) Radiation: Radiation to the chest can cause pericardial inflammation.

n) Other metabolic causes: Myxoedema and gout can also cause pericarditis.

Clinical Presentation

Symptoms

Chest Pain

Chest pain is the most important symptom. It is retrosternal in location and patient usually locates the site of the pain without difficulty. The pain is severe and sharp and may radiate to the shoulder region and back. The pericardial pain increases on deep inspiration, coughing, swallowing and lying supine. Pain may be alleviated with patient sitting up and leaning forwards.

Fever

Fever is a feature of pericarditis especially of infective etiology. Viral pericarditis can present with a prodrome of upper respiratory infection, while purulent pericarditis can present with acute onset of fever with chills and rigor. Tuberculous pericarditis can have low grade prolonged fever.

Myalgia especially in viral pericarditis.

Most of the patients will have difficulty in breathing secondary to chest pain during inspiration.

Signs

- Pericardial friction rub is pathognomonic of pericarditis. It is heard as a phasic scatching sound. It may vary with phases of respiration mostly increasing on inspiration. The pericardial rub is best audible in the lower left parasternal region. Classically pericardial rub has three components corresponding to ventricular systole, early ventricular diastole and atrial systole. Most often the pericardial rub is audible as a biphasic noise — a systolic and diastolic component. In 10 per cent of cases the pericardial rub is monophasic, during systolic phase only, when it can be confused with murmur. The pericardial rub most often waxes and wanes while this does not happen with murmur.

- Low grade to high grade fever can occur depending upon the etiology.

- Tachycardia is a feature of pericarditis.

- Patients are tachypnoeic and most often have shallow breathing secondary to painful restriction of inspiration.

Investigations

1) *Blood Examination*

Erythrocyte sedementaion rate (ESR) may be elevated in tuberculous; collagen and purulent pericarditis. The Dressler's syndrome is associated with leukocytosis and elevated ESR.

2) *Electrocardiogram (ECG)*

Four stages of evolution of ECG changes may occur.

Stage 1: Acute changes. There is ST-segment elevation with concavity upwards and upright T-waves in all leads except aVR and V1. aVR may show ST-segment depression (Fig. 4.1) PR-segment depression could be seen especially in L2 and lateral chest leads (Fig. 4.2). The absence of reciprocal changes helps in distinguishing from acute myocardial infarction.

Fig. 4.1: ECG showing ST elevation in all leads except aVR which shows ST depression. Note that there are no reciprocal changes

Fig. 4.2: ECG recording of L_2 showing PR segment depression in a case of pericarditis

Stage 2: Several days later. Resolution of ST-segment and PR-segment to baseline. The T-wave flattens.

Stage 3: T-wave inversion.

Stage 4: T-wave normalizes – may take days to weeks.

3) *Chest X-ray*

In chest x-ray cardiac silhoutte will be normal unless associated with pericardial effusion in which case there will be cardiomegaly. Evidence of tuberculosis or malignancy in chest X-ray will help in etiological diagnosis.

4) *Echocardiography*

Pericarditis without effusion does not have any definite echocardiographic features. When the pericarditis lasts longer pericardial thickening may be recognized.

5) *Computed Tomography (CT)*

Magnetic resonance imaging (MRI) and transoesophageal echocardiography (TEE) have limited application.

Differential Diagnosis

Main symptom of pericarditis is chest pain and hence Acute Pericarditis must be differentiated from myocardial infarction, aortic dissection, pleuritis and pulmonary embolism. The classical features of pericardial pain will help in diagnosis: In ECG ST segment elevation does not occur in aortic dissection, pleuritis and pulmonary embolism. The ST-elevation of acute pericarditis has concavity upwards with upright T-waves and no reciprocal changes. These differentiate acute pericarditis from acute myocardial infarction (Table 4.1). Echocardiography helps to differentiate these conditions from acute pericarditis.

Table 4.1: ECG Differentiation of Acutepericarditis and Acute Myocardial Infarction

Feature	Acute Pericarditis	Acute Myocardial Infarction
ST Segment Elevation	Concavity upwards and wide spread in all leads except in aVR and V1 not restricted to a region of arterial supply	Convexity upwards and restricted to a region of arterial supply
Reciprocal ST change	Absent	Present
PR segment Depression	Can occur	Not a feature
T inversion	Shallow (Stage III)	Deep
Development of Q-wave	Never	Yes

Treatment

Symptomatic Treatment

Pain relief can be achieved with indomethacin 25 to 50 mg. thrice daily; Ibuprofen 600 mg. thrice daily for 1 or 2 weeks. In case of persistence of pain prednisolone 40 to 60 mg. orally for 5 days and then tapered over 3 weeks is useful.

Specific Treatment

Specific treatment is directed to the etiology of pericarditis. Ideopathic and viral pericarditis do not require additional treatment.

For tuberculous pericarditis, four drugs regime is recommended for 9 months, with atleast 6 months treatment after culture conversion.

For pursulent pericarditis, appropriate antibiotic against the infecting organism should be given.

Follow Up

Most of the patients of ideopathic or viral pericarditis need follow up for atleast 1 month to make certain that the pericarditis process has completely resolved. Followup also is required to exclude the development of constrictive pericarditis especially in tuberculous and perulent pericarditis.

Complications

Recurrence ==> About 20 per cent cases of pericarditis especially idiopathic, viral or Dressler's syndrome, can recur. If pericardial effusion occurs, then it needs management (see later). Recurrent pericarditis may require treatment with steroids.

Check Your Progress 1

1) Mention four common causes of pericarditis.

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2) Describe the character of the chest pain in acute pericarditis.

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3) Describe the features of the pericardial friction rub.

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4) What are the four stages in the evolution of the ECG in acute pericarditis?

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5) How do you distinguish the ECG changes of acute pericarditis from actue ST elevation myocardial infarction?

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4.3 PERICARDIAL EFFUSION

Collection of fluid in the pericardial sac is known as Pericardial effusion. All cases of pericarditis can lead to pericardial effusion. The symptoms in a case of pericardial effusion depends upon the quantity and rate of accumulation of fluid. Rapid accumulation of large quantity of pericardial fluid can cause life threatening **cardiac tamponade**. Small to low moderate effusion, especially occurring insidiously, can be asymptomatic. Rapidly accumulating pericardial effusion of more than 200 ml. can cause haemodynamic disturbance and can produce symptoms. Slowly accumulating fluid in pericardial space may not produce symptoms even upto 1.5 to 2 litres. If the pericardium is stiff due to any pathology then smaller quantity of fluid can cause symptoms.

Etiology

All causes of pericarditis can cause pericardial effusion. (Table 4.2)

Table 4.2: Causes of Pericardial Effusion

1) Idiopathic
2) Viral infection
   - Cox sackie B virus
   - Echovirus
   - Adenovirus.
3) Tuberculous infection.
4) Other bacterial infections.
   - Staphylococcus.
   - Streptococcus
   - Penumococcus.
   - Haemophilius influenza.
5) Rickettsial infection.
   - Psittacosis.
6) Fungal infection.
   - In fungal endocarditis.

7) Radiation.
8) Malignancy
    - Primary – Mesothelioma.
    - Secondary – From lungs, breast, lymphoma and leukaemia.
9) Immunologic disorders.
    - Systemic Lupus Erythematosis.
    - Rheumatoid arthritis.
    - Ankylosing spondylitis.
    - Scleroderma.
    - Sarcoidosis.
    - Rheumatic fever (rarely).
    - Polyarteritis Nodosa.
10) Dissecting thoracic aneurysm.
11) Trauma
12) Drugs
    - Hydralazine.
    - Phenytoin.
    - Procainamide.
    - Warfarin.
13) Infiltrative disorders.
    - Amyloidosis.
14) Endomyocardial fibrosis.
15) Metabolic
    - Uraemia.
    - Myxoedema.
16) Hydropericardium.
    - Cardiac failure.
    - Hypoalbuminaemia.

Clinical Presentation

Asymptomatic: Slowly accumulating small to moderate pericardial effusion may not cause significant elevation of intra-pericardial pressure and hence are asymptomatic. Occasionally these patients complain of fatigue or exertional breathlessness.

Symptoms and Signs

Symptoms of large pericardial effusion are:

a) Feeling of dull ache or pressure in the centre part of chest.

b) Symptoms due to compressive effect of fluid filled pericardial sac on neighbouring structures:

i) Dyspnoea – due to lung compression and collapse. Bronchial heart sounds and impaired resonance on percussion below the angle of the left scapula due to lung collapse in large pericardial effusion is known as **Ewart's sign.**

ii) Dysphagia – due to oesophageal compression.

iii) Irritating cough – due to bronchial irritation and compression.

iv) Hiccough – due to phrenic nerve stretch and irritation.

v) Hoarseness – rarely can occur due to compression of left recurrent laryngial nerve.

c) Symptoms due to systemic venous congestion like facial puffiness, right hypochondrial pain, abdominal fullness and nausea can occur.

d) Massive and rapid pericardial effusion can cause cardiac tamponade which can cause haemodynamic collapse – fall in cardice output, hypotension and shock.

Clinical Signs of PE

Jugular venous pressure may be elevated in moderate PE. 'X' descent will be more prominent than 'Y' descent. No abnormal impulses over the precordium. On percussion, the dull note may extend beyond the palpated apex beat and also on to the right side of sternum. Heart sounds may be feeble and pericardial rub may be heard.

Investigations

1) **Electrocardiogram (ECG):** Low voltage complexes is the classical ECG finding. In massive PE with tamponade, electrical alternans, (sometimes total electrical alternans) can occur.

2) **X-ray Chest:** Uniform cardiomegaly with a smooth outline is characteristic of PE (Fig. 4.3). The superior venacava is usually engorged. Pulmonary vessels will be less prominent. In large pericardial effusion, the cardio-phrenic angle will be acute.

Fig. 4.3: X-ray chest in a case of large pericardial effusion

3) **2 Dimensional Echocardiography (2DE):** Transthoracic 2DE is the investigation of choice to confirm PE and cardiac tamponade. The salient 2DE features of PE are:

a) Echo free space indicating fluid collection in the pericardial sac.

b) Decreased movement of parietal pericardium.

c) In exudative pericardial effusion like tuberculous effusion, there will be fibrous strands attached to the visceral as well as parietal pericardium.

d) If the effusion is large, the heart will be swinging in the pericardial fluid. This swinging movement is thought to be the cause of electrical alternans.

In cardiac tamponade, there will be diastolic collapse of the right ventricle (Fig. 4.4). This is because of intrapericardial pressure being higher than the right ventricular diastolic pressure in cardiac tamponade. Doppler can detect respiratory variation of mitral and tricuspid flow patterns in cardiac tamponade. The mitral flow increases on expiration while tricuspid flow increases on inspiration (Fig. 4.5).

Fig. 4.4: 2DE in a case of pericardial effusion with cardiac tamponade showing diastolic RV collapse (the arrow indicates the collapse). AOR – Aorta, LA – Left Atrium, LV – Left Ventricle, PE – Pericardial Effusion, RV – Right Ventricle

Fig. 4.5: The atrioventricular valve Doppler flow pattern in cardiac tamponade. Note the expiratory increase of mitral flow and inspiratory increase of tricuspid flow

e) Size of the PE.

   i) Small PE (approximately 100 ml.) – mostly seen in the posterior part of the heart and fluid space is less than 1 cm. in width.

   ii) Moderate PE (100 – 500 ml.) – seen all around the heart – width 1.0 cm.

   iii) Large PE (>500 ml.) – more than 1 cm. in width all around.

   iv) Tamponade – 2DE can show swinging movement of the heart, diastolic collapse of right ventricle and respiratory flow variation across the atrioventricular valves.

Conditions Mimicking Pericardial Effusion

In 2DE few conditions can mimic PE.

a) Pericardial fat—Mostly localized anteriorly. Absence of fat in the posterior part helps to differentiate from PE.

b) Pericardial cysts can mimic loculated PE—Pericardial cysts are often found in the right cardiophrenic angle which is an important distinguishing feature.

c) Pleural effusion of the left side occasionally can cause confusion in the diagnosis of PE. In the parasternal long axis view, the PE stops at the left atrioventricular junction while pleural effusion does not have such demarcation. The pleural fluid is detected posterior to and beyond the cardiac images.

d) Computerised axial tomography and Magnetic resonance imaging—There investigations are not usually required for the diagnosis of PE.

4.4 MASSIVE PERICARDIAL EFFUSION – CARDIAC TAMPONADE

Cardiac tamponade is that situation where increase in pericardial fluid raises the intrapericardial pressure which interferes with diastolic filling. This leads to decreased ventricular filling resulting in low cardiac output.

Pathophysiology

Progressive increase in pericardial fluid results in progressive increase in intrapericardial pressure, till a critical volume is reached beyond which small increases result in significant increase in intrapericardial pressure. The compliance of the pericardium and rate of accumulation of fluid decides the critical volume. The increase in intrapericardial pressure results in decreased ventricular distension and decreased ventricular filling. This results in decrease in stroke volume. In order to maintain cardiac output, there will be sinus tachycardia. In severe cardiac tamponade, this compensatory mechanism will be inadequate resulting in low output, hypotension, hypoperfusion, and shock state. The reduction in coronary circulation can further reduce the myocardial performance.

Clinical Features

The clinical presentation will be that of a low output state with anxiety, restlessness, dyspnoea, sweating, cold extremities and drowsiness.

Signs

a) Elevated Jugular venous pressure (JVP)-With the increase in intrapericardial pressure the JVP also gets elevated with prominent ‘X’ descent and reduced or absent ‘Y’ descent.

b) Tachypnoea.

c) Sinus tachycardia to compensate the low stroke volume.

Pulsus Paradoxus is a Feature of Cardiac Tamponade: Pulsus paradoxus is diagnosed if decline in systolic pressure during normal inspiration exceeds 10 mm. of Hg. (Pulsus paradoxus is an exaggerated normal response). In extreme cases, the pulse may not be felt during inspiration. Though pulsus paradoxus can be detected by palpation of pulse, blood pressure recording during inspiration and expiration and demonstrating the decline of more than 10 mm. of Hg. in systolic pressure during inspiration confirms this physical finding.

Mechanism: During inspiration there is increase in systemic venous return to the right side of the heart resulting in greater right ventricular filling. Because of the elevated intra-pericardial pressure in cardiac tamponade, in order to accommodate the extra volume of blood during inspiration in the right ventricle, the interventricular septum is pushed to the left ventricle resulting in reduced left ventricular filling. The decreased pulmonary venus return during inspiration adds on to the reduced left ventricular volume which results in reduced stroke volume and reduced systolic pressure.

Pulsus paradoxus can occur in other conditions like effusive constrictive pericarditis and obstructive pulmonary disease.

Investigations

Electrocardiogram (ECG)

Low voltage complexes with electrical alternanes, especially total electrical alternanes, is a feature of cardiac tamponade.

2 Dimensional Echocardiogram (2DE)

The 2 DE features of cardiac tamponade are:

1) Massive pericardial effusion.

2) Swinging heart motion in the pericardial fluid.

3) Right ventricular (RV) early diastolic collapse—This is seen in the anterior free wall of RV as a depression during early diastolic (Fig. 4.4) and is best seen in parasternal long and short axis views. This sign is highly sensitive in detecting cardiac tamponade. Increase in RV volume and or pressure as in pulmonary arterial hypertension or RV infarction can delay or negate the occurrence of RV diastolic collapse.

4) Right atrial diastolic collapse is also a highly sensitive sign of cardiac tamponade (Fig. 4.6). This is best detected in apical 4 chamber view.

Fig. 4.6: Note the prominent right atrial collapse (arrow) in a case of cardiac tamponade. LA – Left Atrium, LV – Left Ventricle, PE – Pericardial Effusion, RA – Right Atrium, RV – Right Ventricle.

5) Respiratory variation of atrioventricular valve flow patterns – expiratory increase in flow across mitral valve and inspiratory increase in flow across tricuspid valve (Fig. 5) are features of cardiac tamponade. Mitral E decrease of 25 per cent during inspiration is highly suggestive of cardiac tamponade.

6) Inferior vena cava plethora – Lack of normal inspiratory collapse of 50 per cent in the proximal inferior vena cava is a feature of cardiac tamponade.

Transoesophageal Echocardiography is not indicated in Cardiac tamponade.

Haemodynamic Studies: With the availability of accurate diagnosis by 2DE, there is no indication for cardiac catheterization to confirm the diagnosis of cardiac tamponade.

Treatment

Percutaneous Pericardiocentesis

Cardiac tamponade is an acute emergency and percutaneous pericardiocentesis must be done as immediately as possible. Ideally this should be done under 2DE guidance with haemodynamic monitoring. The percutaneous pericardiocentesis can be done from the subxiphoid region with needle directed towards the 2nd left costochondral junction. Fluid should be removed slowly. Removal of even small quantity of fluid can relieve the tamponade. Complications are damage to coronary arteries and laceration to heart and lungs.

See also under cardiac emergencies.

Surgical Pericardial Drainage

Surgical drainage of the pericardial fluid is considered in recurrent pericardial effusion. Surgical drainage gives an opportunity for pericardial biopsy which can confirm the etiology of PE. Pericardial window can be created for drainage of PE to left pleural cavity. This is done for recurrent large PE.

Specific Treatment

Depending upon the etiology of PE, specific treatment must be given.

Check Your Progress 2

1) What are the echocardiographic features of pericardial effusion and cardiac tamponade?

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2) What is pulsus paradoxus and what is its mechanism?

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4.5 CONSTRICTIVE PERICARDITIS

Constrictive pericarditis is the sequelae of chronic fibrosis and thickening of the pericardium as a result of chronic inflammation.

Etiology

The etiology of constrictive pericarditis is similar to acute pericarditis with effusion. (Table 4.3).

Table 4.3: Causes of Constrictive Pericarditis

1) Tuberculous pericarditis.
2) Pyogenic pericarditis.
3) Viral pericarditis.
4) Fungal pericarditis.
5) Trauma.
6) Radiation.
7) Immunologic disorder.
    - Systemic Lupus erythmatosis.
    - Rheumatoid disease.
8) Idiopathic.

Pathophysiology

The thickened and rigid pericardium causes constriction of the heart and restricts ventricular dialatation and diastolic filling of the ventricles. The thickened rigid pericardium can cause dissociation of intracardiac and intrathoracic pressures and elevation of diastolic intracardiac pressures. This results in systemic and pulmonary venous congestion. Since the early phase of ventricular relaxation is normal, the early filling of the ventricles take place normally. Further relaxation of ventricles are restricted by the thickened pericardium resulting in acute elevation of diastolic ventricular pressure which gives a square root (“) appearance to the diastolic ventricular

pressure tracing. Uniform constriction of all the four cardiac chambers results in equalization of diastolic pressures in all the four chambers. This is different from restrictive cardiomyopathy where the difference in diastolic pressure between ventricles will be more than 5 mm. of Hg.

The myocardium is usually normal in structure and function. This results in good systolic function.

The ventricular filling takes place during early filling phase only as further filling is prevented by the thickened and rigid pericardium.

Clinical Features

Constrictive pericarditis (CP) has an insidious onset with features of early cardiac failure like fatigue, reduced exercise tolerance, exertional beathlessness and oedema of the legs. Abdominal distension secondary to chronic hepatomegaly and acites can occur.

Signs

1) Jugular venous pressure (JVP) is elevated in all patients of CP which reflects the elevated diastolic pressure of the right atrium. In CP, the JVP can engorge during inspiration, known as **Kussmaul's sign;** there can be prominent 'Y' descent representing early rapid filling of the right ventricle, **Friedrich's sign.**

2) One third of CP patients have irregularly irregular pulse due to atrial fibrillation. In patients with effusive CP there can be pulsus paradoxus.

3) Praecordial examination may reveal cardiomegaly by percussion. The first heart sound may be muffled. A pericardial knock, which is due to early cessation of ventricular filling due to pericardial restriction is audible. This is a high pitched sound audible in diastolic phase earlier than the timing of S3. Murmurs are not common even though atrioventricular valve regurgitations due to alteration of ventricular geometry have been described.

4) Hepatomegaly and ascites are almost always present. Splenomegaly is detected in chronic cases.

Investigations

Electrocardiogram

Low voltage complexes can occur. Left atrial enlargement may be seen. Atrial fibrillation occurs in about one fourth of patients.

X-ray Chest

Cardiac enlargement occurs in almost half of the cases. Pericardial calcification is seen in chronic cases (Fig. 4.7). Pericardium over the inferior surface and free wall of left ventricle, over the right ventricle and atrioventricular groove are common sites for calcification. Calcification is best seen in lateral film.

Fig. 4.7: X-ray chest showing pericardial calcification (arrow).

2-Dimensional Echocardiography

Thickened pericardium can be detected. In about a third of patients there will be associated some degree of pericardial fluid. Sudden anterior motion of interventricular septum following atrial systole is a feature. On Doppler evaluation, respiratory variation of atrioventricular valve flow will be detected – there will be significant decrease in mitral flow and increase in tricuspid flow during inspiration. This difference can be 33 per cent and above. Opposite changes are seen during expiration. Pulmonary venous flow in CP will show decreased systolic to diastolic flow ratio. There will be significant increase in pulmonary venous flow during expiration. Similarly the venous flow pattern in inferior venacava can show decreased systolic to diastolic flow ratio and marked increase in flow during inspiration. In restrictive cardiomyopathy, the prominent respiratory variation of the venous flow does not happen.

Haemodynamic Study

Simultaneous right and left heart studies are useful. Due to exaggerated waves in atria, W-shaped atrial pressure tracing will be seen. There will be equalization of end diastolic pressure in all the four chambers of the heart (difference will be less than 5 mm. of Hg.). The ventricular diastolic pressure wave will show the square root sign (“). The end diastolic pressure in both the ventricles will be elevated and almost equal with the difference being less than 5 mm. of Hg. The right ventricular diastolic pressure will be usually more than one third the right ventricular systolic pressure.

Treatment

Medical Treatment

1) Judicious use of diuretics to alleviate systemic congestion. This may be enough in some cases of mild CP.

2) Specific treatment of etiology. Any treatable etiology has to be treated, e.g., tuberculosis.

Surgical Treatment

Pericardiectomy will relieve constriction in more than 80 per cent of patients and will provide good symptomatic relief. The procedure carries 10 per cent — 20 per cent mortality. The risk is higher in patients with greater functional disability.

Check Your Progress 3

What are the changes in the neck veins and abdominal examination in constrictive pericarditis?

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4.6 LET US SUM UP

Pericardium is the covering sac of the heart. The pericardium prevents acute cardiac chamber dilatation. Acute pericarditis, pericardial effusion and constrictive pericarditis are the major diseases of the pericardium. The acute pericarditis can be diagnosed by the pericardial friction rub and classical ECG changes. It is important to differentiate acute pericarditis from acute myocardial infarction. For the dianosis of PE, transthoracic 2DED is the investigation of choice. Large PE can cause cardiac tamponade which if left untreated leads to fatal outcome. Percutaneous pericardiocentesis is life saving in cardiac tamponade. Constrictive pericarditis is the sequelae of chronic fibrosis of pericardium. Patients present with features of severe systemic venous congestion. Pericardiectomy can relieve the constriction in more than 80 per cent of patients of constrictive pericarditis. In every case of pericardial disease if etiology is treatable, it should be treated.

FURTHER READINGS

Shabetai, R., "Diseases of the Pericardium" in *Hurst's The Heart*, p. 2169, 9th edn., 1998.

Spodick, D.H., "Pericardial Diseases" in *Heart Disease*, Braunwald, E., Zipes, D.P., Libby, P. P., (eds.), 1823, 6th edn., 2001.

4.9 ANSWERS TO CHECK YOUR PROGRESS

Check Your Progress 1

1) a) **Idiopathic:** Most common cause, though many of these cases could be of viral etiology.

   b) **Viral:** The Coxsackie B and Echovirus are most common pathogens.

   c) **Purulent:** Purulent pericarditis due to staphylococci, pneumococci or strephtococci can occur.

   d) **Tuberculous:** Tuberculosis can infect the pericardium resulting in tuberculous pericarditis.

   e) **Pericarditis in acute myocardial infarction:** Transmural myocardial infarction can cause pericardial inflammation in 12 –15 per cent of cases. It is more often detected in

anterior wall myocardial infarction and inferior wall with right ventricular infarction. With the widespread use of reperfusion therapy, the incidence of pericarditis in acute myocardial infarction has significantly come down.

f) **Uremic:** Uremic patients can develop pericarditis.

g) **Neoplastic:** Malignant tumours especially of lung and breast and lymphomas can involve the pericardium leading to pericarditis with effusion.

2) Chest pain is the most important symptom. It is retrosternal in location and patient usually locates the site of the pain without difficulty. The pain is severe and sharp and may radiate to the shoulder region and back. The pericardial pain increases on deep inspiration, coughing, swallowing and lying supine. Pain may be alleviated with patient sitting up and leaning forwards.

3) It is heard as a phasic scatching sound. It may vary with phases of respiration mostly increasing on inspiration. The pericardial rub is best audible in the lower left parasternal region. Classically pericardial rub has three components corresponding to ventricular systole, early ventricular diastole and atrial systole. Most often the pericardial rub is audible as a biphasic noise – a systolic and diastolic component. In 10 per cent of cases the pericardial rub is monophasic, during systolic phase only, when it can be confused with murmur. The pericardial rub most often waxes and wanes while this does not happen with murmur.

4) *Stage 1* – Acute changes. There is ST segment elevation with concavity upwards and upright T-waves in all leads except aVR and V1. aVR may show ST-segment depression. PR segment depression could be seen especially in L2 and lateral chest leads. The absence of reciprocal changes helps in distinguishing from acute myocardial infarction

Stage 2 – Several days later. Resolution of ST-segment and PR segment to baseline. The T wave flattens.

Stage 3 – T-wave inversion.

Stage 4 – T-wave normalizes – may take days to weeks.

5)

Table: ECG Differentiation of Acutepericarditis and Acute Myocardial Infarction

Feature	Acute Pericarditis	Acute Myocardial Infarction
ST Segment Elevation	Concavity upwards and wide spread in all leads except in aVR and V1 not restricted to a region of arterial supply	Convexity upwards and restricted to a region of arterial supply
Reciprocal ST change	Absent	Present
PR segment Depression	Can occur	Not a feature
T inversion	Shallow (Stage III)	Deep
Development of Q-wave	Never	Yes

Check Your Progress 2

1) 2 Dimensional Echocardiography (2DE): Transthoracic 2DE is the investigation of choice to confirm PE and cardiac tamponade. The salient 2DE features of PE are:
   a) Echo free space indicating fluid collection in the pericardial sac.
   b) Decreased movement of parietal pericardium.
   c) In exudative pericardial effusion like tuberculous effusion, there will be fibrous strands attached to the visceral as well as parietal pericardium.
   d) If the effusion is large, the heart will be swinging in the pericardial fluid. This swinging movement is thought to be the cause of electrical alternans.

   In cardiac tamponade, there will be diastolic collapse of the right ventricle. This is because of intrapericardial pressure being higher than the right ventricular diastolic pressure in cardiac tamponade. Doppler can detect respiratory variation of mitral and tricuspid flow patterns in cardiac tamponade. The mitral flow increases on expiration while tricuspid flow increases on inspiration.

2) Pulsus paradoxus is diagnosed if decline in systolic pressure during normal inspiration exceeds 10 mm. of Hg. (Pulsus paradoxus is an exaggerated normal response). In extreme cases, the pulse may not be felt during inspiration. Though pulsus paradoxus can be detected by palpation of pulse, blood pressure recording during inspiration and expiration and demonstrating the decline of more than 10 mm. of Hg. in systolic pressure during inspiration confirms this physical finding.

   Mechanism: During inspiration there is increase in systemic venous return to the right side of the heart resulting in greater right ventricular filling. Because of the elevated intra-pericardial pressure in cardiac tamponade, in order to accommodate the extra volume of blood during inspiration in the right ventricle, the interventricular septum is pushed to the left ventricle resulting in reduced left ventricular filling. The decreased pulmonary venus return during inspiration adds on to the reduced left ventricular volume which results in reduced stroke volume and reduced systolic pressure.

   Pulsus paradoxus can occur in other conditions like effusive constrictive pericarditis and obstructive pulmonary disease.

Check Your Progress 3

The jugular venous pressure is raised .With inspiration there is a further rise in the JVP.

The liver is enlarged and there is often ascites.