

## Role of CT and MRI in Diagnosis of Pericardial Diseases

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### ABSTRACT

**Background:** pericardial diseases are important causes of morbidity and mortality in cardiovascular diseases. CT and MRI are more than adjuncts to echocardiography in pericardial diseases assessment, as they provide an excellent pericardial anatomy delineation and precise pericardial lesions evaluation including; effusion, constrictive pericarditis, thickening, masses and congenital anomalies. Ideal management needs the proper imaging modality choosing ability. **Aim of the Study:** this study aimed to evaluate the role of CT and MRI versus echocardiography in the diagnosis of pericardial diseases and to show the limitations of each modality.

**Conclusion:** tissue characterization with CMR is superior to cardiac CT and echocardiography. CMR can differentiate tumor from thrombus and is often helpful to assess the perfusion of a pericardial mass with the use of gadolinium contrast. The final diagnosis depends on typical pathologic features.

**Keywords:** pericardial diseases, CT, MRI.

### INTRODUCTION

The pericardium represents a simple, two-layered, fibroelastic sac that surrounds the heart and provides lubrication and protection. Normally, it is a thin-walled structure (<3 mm) with minimal pericardial fluid (<50 ml). The normal pericardium is fairly distensible, precluding excessive constraint of the ventricles <sup>(1)</sup>. The pericardium has been described as an intracardiac pressure modulator, limiting acute distention of any cardiac chamber <sup>(2)</sup>. Pericardial diseases are important causes of morbidity and mortality in patients with cardiovascular disease. Inflammatory diseases of the pericardium constitute a spectrum ranging from acute pericarditis to chronic constrictive pericarditis. Other important entities that involve the pericardium include benign and malignant pericardial masses, pericardial cysts, diverticula, as well as congenital absence of the pericardium <sup>(3)</sup>. Although the underlying etiology of pericardial disease varies, the typical response of the pericardium is relatively nonspecific, with production of pericardial fluid. A wide range of pericardial diseases may occur, including acute or chronic inflammation, fibrosis or effusion <sup>(4)</sup>. The pericardium may be secondarily involved by a large group of systemic diseases, such as infective, autoimmune and neoplastic processes. Moreover, iatrogenic causes for example, after cardiac surgery or radiation therapy represent an important cause of pericardial related morbidity and mortality <sup>(5)</sup>. Clinical diagnosis with a detailed examination is often complemented by ECG, chest x-ray (CXR) and echocardiography, as well as

potentially hemodynamic catheterization, depending on the nature and severity of the symptoms. While these tools have a broad and well-established role in the diagnosis of pericardial disease, newer modalities, such as cardiac computed tomography (CT) and cardiovascular magnetic resonance imaging (CMR) are important methods for aiding in diagnostic evaluation <sup>(6)</sup>. Echocardiography is the method of choice for evaluating most pericardial diseases. When competently performed in patients with good acoustic windows, echocardiography accurately detects all pericardial effusions and provides clinically relevant information about their size and hemodynamic importance. The technique is less reliable than MRI and CT in detecting pericardial thickening/constriction and calcification as well as small loculated effusions, but is still extremely useful in these conditions <sup>(7)</sup>.

In the evaluation of pericardial disease, CT and MRI traditionally have been used as adjuncts to echocardiography. However, CT and MRI are particularly useful as sensitive and noninvasive methods for evaluating loculated or hemorrhagic pericardial effusion, constrictive pericarditis, and pericardial masses. Both CT and MRI provide excellent delineation of the pericardial anatomy and can aid in the precise localization and characterization of various pericardial lesions, including effusion, constrictive pericarditis, pericardial thickening, pericardial masses, and congenital anomalies. Both modalities provide a larger field of view than does echocardiography, allowing the examination of the

entire chest and detection of associated abnormalities in the mediastinum and lungs. Soft-tissue contrast on CT scans and MR images also is superior to that on echocardiograms. Given the many potential applications of these modalities in the evaluation of pericardial diseases, familiarity with the CT and MR imaging features of these diseases is important for optimal management of the patient <sup>(8)</sup>.

**The study was approved by the Ethics Board of Ain Shams University.**

### **Congenital Defects of the Pericardium**

Pericardial defects are the result of a failure of the pluro-pericardial membranes to fuse completely on one or both sides and may be associated with congenital anomalies such as patent ductus arteriosus, bronchogenic cysts, tricuspid insufficiency, atrial septal defects, left diaphragmatic hernia and pulmonary sequestration <sup>(9)</sup>.

Chronic pain may result from traction on cardiac structures in the absence of the pericardium or even compression of the left anterior descending coronary artery by the rim of a partial defect. Pericardial defects have been diagnosed noninvasively by plain chest x-ray, CT, and MRI. With the wide spread use of advanced imaging, non-operative diagnosis may redefine the prevalence of this condition <sup>(10)</sup>.

### **Pericardial Effusion**

Pericardial effusion occurs either due to increased production of pericardial fluid (infections and inflammations) or inadequate drainage of the fluid (malignancy or hypothyroidism), or from a structural abnormality that allows fluid to enter the pericardial cavity <sup>(10)</sup>.

Clinical manifestations of pericardial effusion depend on the rate of accumulation of fluid in the pericardial cavity. Rapid accumulation may cause elevated intrapericardial pressures with as little as 80 ml of fluid, while gradual accumulation of fluid can grow to 2 liters without symptoms <sup>(11)</sup>.

### **Types of pericardial effusion**

- (1) Transudative (Congestive heart failure, Myxoedema, Nephrotic syndrome).
- (2) Exudative (Tuberculosis, Spread from empyema, Malignant effusion).
- (3) Hemorrhagic (Chest trauma, Rupture of aneurysms, Malignant effusion) <sup>(10)</sup>.

Causes of pericardial effusion based upon composition of effusion:

- Serous (acute pancreatitis, chemotherapeutics, chronic disease, cirrhosis, congestive heart failure, Dressler's syndrome, hypoalbuminemia, hypothyroidism, infection, irradiation, malnutrition, nephrotic Syndrome)
- Fibrous (viral, pyogenic, tuberculous, fungal, syphilitic, protozoal, parasitic Pericarditis)
- Blood (acute myocardial infarction, anticoagulants, aortic rupture, cardiac catheterization, chemotherapeutics, coagulotherapy, heart surgery, neoplasm, perforation, trauma)
- Lymph or Chylous (benign obstruction of thoracic duct, idiopathic, neoplasm)
- Miscellaneous (cardiomyopathy, SLE) <sup>(10)</sup>.

Chylopericardium is defined as a pericardial effusion which consists of chyle. It is a rare condition in which lymphatic fluid leaks into the space around the heart. When this fluid builds up it can compress the heart and lead to poor heart function. Chylopericardium may associated with lymphangiomas, cystic hygromas, thoracic and cardiac surgery, trauma, radiation and malignancy. Pericardial effusion if untreated or if refractory to treatment can lead to accumulation of large amount of fluid around the heart, severe hemodynamic compromise and even death. If the fluid accumulates too rapidly or is too large, then cardiac tamponade may occur. Cardiac tamponade may require urgent intervention including pericardiocentesis. This complication is more common in patients with specific underlying etiologies such as malignancy, tuberculosis, or purulent effusion <sup>(11)</sup>.

### **Prognosis** <sup>(10, 12)</sup>

- (1) Idiopathic pericardial effusion is often self-plimited and most patients recover in 2 weeks to 3 months.
- (2) Tuberculous cause - The mortality rate is 8-17%. The mortality is 17-34% if the tuberculous pericardial effusion is associated with HIV.
- (3) Traumatic pericardial injury.-In penetrating injuries, pericardial effusion and tamponade may develop rapidly. Early detection and early treatment of cardiac tamponade is associated with a good prognosis. Minor perforations, isolated right ventricular wounds, and a systolic blood pressure more than 50 mm Hg are all associated with better outcomes.

- (4) Malignancy -Pericardial effusion secondary to malignancy is associated with poor outcomes and a more complicated course
- (5) Autoimmune Disease.-Pericardial involvement in scleroderma and rheumatic fever is associated with worse outcomes.
- (6) Renal Failure.-Pericardial disease secondary to renal failure is associated with significant morbidity and may result in hemorrhagic pericardial effusion.

• **Pericarditis**

It is inflammation of the pericardium. There can be an accompanying accumulation of serous or fibrinous fluid. Vascular congestion of the pericardium is also present. The underlying myocardium may or may not be inflamed as well <sup>(13)</sup>. Clinically, **acute pericarditis** presents within 6 weeks of the disease onset; **subacute pericarditis** presents within 6 weeks to 6 months of the disease onset; and **chronic pericarditis** manifests after 6 months of the disease onset. Acute pericarditis is more common than chronic pericarditis, and often occurs as a complication of viral infections, immunologic conditions, or as a result of a heart attack (myocardial infarction). Chronic pericarditis is less common, which may manifest as constrictive pericarditis <sup>(13)</sup>.

Pericarditis has many causes, including the following:

- Unknown cause "idiopathic" –The condition improves with empiric anti-inflammatory treatment (ie, aspirin, ibuprofen).
- Infection –Cardiotropic viruses usually spread to the myocardium and pericardium hematogenously and cause acute inflammation. This may cause pericardial effusion and fibrinous change of pericardium. Most patients with viral pericarditis recover completely with few developing recurrences.

**Bacterial pericarditis results from:**

Contiguous spread from infectious focus in the chest, hematogenous spread of infection, or direct inoculation as (penetrating injury or cardiothoracic surgery) <sup>(13)</sup>. Tuberculous pericarditis develop from lymphatic spread from peritracheal, peribronchial or mediastinal lymph nodes or by contiguous spread from a focus of infection in lung or pleura. The pathologic stages proceed from: Serous or serosanguineous

pericardial effusion, then organization of granulomatous caseation and thickening of pericardium occur with development of constrictive pericarditis <sup>(11)</sup>.

Pericarditis in renal failure is thought to result from inflammation of the pericardium by metabolic toxins such as urea, creatinine, methylguanidine <sup>(13)</sup>.

Radiation – Prior radiation to the chest is an important cause of pericardial disease, especially for breast cancer, lung cancer, or lymphoma.

- Trauma to the chest may be **blunt**, as with a steering wheel injury, or **sharp**, as with a bullet or knife wound. Invasive cardiac procedures, and rarely, cardiopulmonary resuscitation (CPR).
- Myocardial infarction can cause pericarditis.
- Drugs and toxins.
- Metabolic disorders – The major cause of metabolic-related pericarditis is kidney failure.
- Rheumatic diseases – SIE, rheumatoid arthritis, systemic sclerosis, and mixed connective tissue disease are the most common rheumatic causes of pericarditis.
- Gastrointestinal diseases – Pericarditis may occur in patients with inflammatory bowel disease (ulcerative colitis or Crohn's disease) <sup>(14)</sup>.

The prognosis depends on the complications of pericarditis, the underlying etiology, and the associated co-morbidities.

Constriction can occur after almost any pericardial process. Historically, the most common etiology was tuberculosis, but in the modern age, this cause now accounts for <2% of cases <sup>(15)</sup>.

**Table 1: constrictive pericarditis incidence rates <sup>(15)</sup>.**

Idiopathic/viral pericarditis	0.76 cases per 1000 person
Connective tissue disease/pericardial injury	4.40 cases per 1000 person
Malignant pericarditis	6.33 cases per 1000 person
Tuberculous pericarditis	31.65 cases per 1000 person
Purulent pericarditis	52.74 cases per 1000 person

Constrictive pericarditis is most commonly caused by conditions or events that cause inflammation to develop around the heart, including:

- (1) Coronary artery bypass graft surgery
- (2) Radiation therapy to the chest, postsurgical
- (3) Tuberculosis, Post Viral Pericarditis, Incomplete drainage of purulent pericarditis, Fungal and Parasitic Infections
- (4) Chronic Renal Failure, Connective Tissue Disorders
- (5) Neoplastic pericardial infiltration, Mesothelioma
- (6) Following pericarditis associated with ST elevation myocardial infarction (Dressler's syndrome)
- (7) In Association with pulmonary asbestosis
- (8) Bacterial, viral, or surgical infections that cause abnormal buildup in the covering of the heart<sup>(13)</sup>.

Constrictive pericarditis' complications include; heart failure, pulmonary edema and scarring of the heart muscle. Constrictive pericarditis may be life threatening if left untreated<sup>(13)</sup>.

#### **Benign Pericardial Tumors**

Benign pericardial tumors include teratoma, fibroma, fibrous histiocytoma, lipoma and hemangioma<sup>(16)</sup>.

#### **A- Pericardial teratoma**

It is a benign germ cell neoplasm that typically affects infants and children, who present with respiratory distress and cyanosis secondary to pericardial tamponade and compression of right-sided vascular structures (aortic root, superior vena cava, right atrium, and pulmonary artery). Pericardial teratomas are usually right-sided multi cystic masses, which typically connect to one of the great vessels via a pedicle.

Most teratomas of the heart lie within the pericardial sac; rarely, they can be intramyocardial. The tumors can be very large and are almost always associated with a pericardial effusion<sup>(17)</sup>.

#### **B- Pericardial fibromas**

They are cellular, fibroblast-rich tumors with little collagen in infants, whereas tumors in adults are composed of collagen. In approximately 50% of the cases, spots of calcification and less commonly ossification may be found. Examinations of resected cardiac fibromas reveal firm or rubbery masses<sup>(18)</sup>.

#### **Malignant Pericardial Tumors**

#### **A- Primary**

##### **1- Pericardial mesothelioma:**

It is a malignant primary neoplasm that arises from the mesothelial cells of the pericardium. The term is used to describe those tumors localized to the pericardium and does not apply to primary pleural tumors that secondarily invade the pericardium. Although pericardial mesotheliomas represent less than 1% of all malignant mesotheliomas, they account for 50% of all primary pericardial tumors<sup>(19)</sup>.

##### **Primary cardiac sarcomas**

Sarcomas are rare malignant mesenchymal neoplasms; however, they constitute the majority of primary cardiac neoplasms and the second most common primary cardiac tumor. By definition primary cardiac sarcomas are confined to the heart. Although all types of sarcomas affect the heart, the most common cell types are angiosarcoma (37% of cases), undifferentiated sarcoma (24%), malignant fibrous histiocytoma (MFH) (11%–24%), leiomyosarcoma (8%–9%), and osteosarcoma (3%–9%)<sup>(20)</sup>.

##### **Primary cardiac lymphomas (PCL)**

They are typically of the non-Hodgkin type. By definition, these tumors involve only the heart or pericardium at the time of diagnosis, with no evidence of extracardiac lymphoma. They usually involve the right chambers and pericardium<sup>(21)</sup>. It composes only 1.3 % of all cardiac tumours. This disease is more common in the elderly age.

#### **B- Secondary**

Secondary or metastatic heart tumors occur comparatively more frequently, with an at least 100 times higher incidence than primary tumors of the heart. Cardiac metastases predominantly occur in patients in the sixth and seventh decade of life. The most common tumors with pericardial metastatic potential are carcinomas of the lung, the breast, malignant lymphoma, leukemia, and malignant melanoma. Metastases are present at autopsy in 10–12% of patients with known neoplasia<sup>(22)</sup>.

#### **Miscellaneous**

##### **Pericardial cysts**

They are an uncommon benign congenital anomaly in the middle mediastinum. They represent 6% of mediastinal masses, and 33% of mediastinal cysts<sup>(23)</sup>.

Pericardial cysts occur at the rate of 1 person per 100,000. Seventy five percent of them have no associated symptoms, and are usually found incidentally during routine chest x-ray or echocardiography. 70% of them are located at the right cardiophrenic angle, 22% in the left, and the rest are in the posterior or anterior superior mediastinum<sup>(13)</sup>.

### **Pericardial fat pad**

It is a small lump of fatty tissue on the outside of the heart. Cardiologists generally consider it as of little or no significance.

### **Calcification**

Calcification of the pericardium is a relatively rare occurrence, yet can be a signpost of pericardial constriction. It is significant since it indicates a treatable cause for cardiac dysfunction. Historically, tuberculous pericarditis used to be the most common cause of pericardial calcification. Most recent cases of calcific pericarditis have been idiopathic and have been thought to represent sequelae of previous viral pericarditis. Uremic pericarditis, connective tissue diseases with their concurrent polyserositis, radiation treatment, and cardiac surgery occasionally lead to calcific constrictive pericarditis.

Pericardial calcification should be distinguished from:

- Coronary artery calcification, a marker of atherosclerosis.
- Myocardial calcification which can occur following myocardial infarction.
- Valvular calcification of the mitral valve or aortic valve.
- Aortic calcification as can be seen in syphilitic aortitis<sup>(24)</sup>.

### **Pneumopericardium:**

It is a medical condition where air enters the pericardial cavity and a well-recognized clinical and radiologic entity. It can be congenital, or introduced by a wound<sup>(25)</sup>.

## **CT and MRI**

### **Normal Pericardium:**

With cardiac computed tomography (CCT), the normal pericardium is best imaged in systole and appears as a line of average thickness of 1.3 to 2.5 mm (almost always  $\leq 4$  mm), CCT delineates the pericardium as a bright, linear structure that is

easily detectable in both contrast- and noncontrast enhanced examinations because of its visibility against the low attenuation of the surrounding fat. Visualization of the pericardium varies with location and is sometimes difficult against the lateral, posterior, and inferior left ventricular wall because of a paucity of pericardial fat<sup>(26)</sup>.

### **Pericardial defects**

Although the pericardium is usually sufficiently thick to be identified on CCT and CMR, visualization at the most common site of pericardial defects, the lateral, posterior, and inferior left ventricular wall, can be poor because of a paucity of fat. Several indirect morphologic signs have been accepted as diagnostic of pericardial defects. Interposition of lung tissue between the aorta and pulmonary artery or between the diaphragm and the base of the heart is the most specific sign and occurs in all patients with complete left pericardial defects or partial defects overlying these anatomic structures<sup>(26)</sup>.

### **Pericardial Effusion**

CT and MR imaging often allow for detection of the amount and distribution of fluid accumulation. In general, a pericardial thickness greater than 4 mm is regarded as abnormal. However, because fluid can accumulate in the pericardial recesses, a relationship between the measured width of the pericardial space and the total volume of fluid is not clear cut. For instance, a pericardial space anterior to the RV that is greater than 5 mm corresponds to a moderate effusion of 100–500 mL of fluid<sup>(27)</sup>.

Characterization of pericardial fluid can, to some extent, be achieved by measuring attenuation values on CT images and signal intensity on MR images. If the CT attenuation value is greater than that of water, then an effusion is more likely to be due to hemopericardium, malignancy, purulent exudates, or hypothyroid-related effusion<sup>(22)</sup>. Low-attenuation pericardial effusions have been reported in cases of chylopericardium<sup>(16)</sup>.

Transudates typically manifest with low signal intensity on T1-weighted MR images and with high signal intensity on T2-weighted images. Exudates, having high protein and cell content, increase the rate of T1 relaxation (higher signal intensity) and shorten the rate of T2 relaxation (lower signal intensity). Hemopericardium can be suspected in patients who have previously undergone aortic or cardiac surgery or who have a history of trauma or neoplastic disease and is characterized by high

signal intensity on T1-weighted images and inhomogeneous low signal intensity on cine SSFP images. Bright-blood dynamic cine MR imaging often allows a better appreciation the intrapericardial contents, such as the visualization of fibrinous strands or of the presence of coagulated blood <sup>(27)</sup>.

**Pericarditis**

On CCT, pericardial thickening is suggestive of acute pericarditis. As the duration of pericardial inflammation increases, the once smooth pericardium may develop irregular contours <sup>(26)</sup>. In the case of a pericardial effusion, attenuation measurements enable its initial characterization. Simple serous effusions (transudates) usually have the same attenuation as water but vary with cell and protein concentration (0 to 25 HU). Attenuation >25 HU suggests a nonserous fluid composition (exudate) such as those seen in malignancy, hemopericardium, purulent exudates, or effusion-associated hypothyroidism. Fat hazing and lymphadenitis were critical CT signs in diagnosing an acute component to the pericarditis <sup>(28)</sup>.

On CMR, The signal intensity of the thickened pericardium on electrocardiogram-gated CMR images is variable in acute pericarditis, and there is no pathognomonic intensity pattern. Signal intensity on spin echo images is inversely related to the chronicity of the pericardial inflammation. Enhancement of the thickened pericardium after the administration of contrast material may aid in its visualization and usually suggests active inflammation characteristic of acute pericarditis. However, pericardial contrast enhancement is nonspecific and must be interpreted within the clinical context. On CMR, transudative effusions show a low signal intensity on T1-weighted spin echo images and a high intensity on gradient echo images such as balanced steady-state free precession (bSSFP).

Conversely, exudative effusions have a high protein and cell content and therefore relatively higher T1-weighted signal intensity on spin echo images <sup>(16)</sup>.

Cine images with bSSFP are able to demonstrate cardiac motion within the pericardial space and delineate the bright appearance of pericardial fluid. This sequence allows the differentiation of structures with a high T2/T1 ratio, such as fluid, from ones with low ratios (myocardium), and enables a better delineation of the pericardium and the surrounding structures without the need for

contrast administration . Both CCT and CMR imaging are useful adjuncts to TTE in the characterization of a pericardial effusion and tamponade. Both CCT and CMR provide more detailed quantification and localization of the effusion than TTE, and they are especially useful to guide pericardiocentesis, since loculated or regions of calcified pericardium can be identified.

When hemopericardium complicates aortic dissection, CCT and CMR can readily identify the lesions, thereby preventing potentially catastrophic pericardiocentesis. Real-time cine CCT and CMR imaging can also provide information similar to TTE with respect to interventricular septal (IVS) motion and chamber collapse. Modalities such as CMR, which require relatively prolonged imaging times, may not be appropriate for clinically unstable patients with suspected tamponade <sup>(3)</sup>.

**Summary of the imaging findings in tamponade is shown in table 2.**

**Table 2: imaging findings in pericardial tamponade <sup>(3)</sup>**

Modality	Imaging Findings in Pericardial Tamponade
TTE	Pericardial effusion RV collapse in diastole; right or left atrial collapse in systole Respiratory variation in mitral (>25%) and tricuspid (>40%) inflow Ventricular interdependence Plethora of IVC Prominence of diastolic reversals in hepatic veins by pulsed Doppler
CCT/CMR	Presence of pericardial effusion, even if loculated or localized
Cine CCT or CMR	Ventricular interdependence Chamber collapse

A CCT scan is able to detect subtle amounts of calcification, a finding highly suggestive of constrictive pericarditis. Irregular calcification may be found anywhere over the surface of the heart, but is primarily found in regions where pericardial fat is abundant, i.e., the atrioventricular groove. Other findings of constrictive pericarditis on CCT include a pericardial thickness of >4 mm (diffuse or localized), narrowing and tubular deformation of the RV, normal or small ventricular size, and straightening of the IVS. Additional secondary findings include signs of impaired diastolic filling of the RV: dilatation of the IVC, hepatic veins, and

RA, as well as hepatosplenomegaly, ascites, and pleural effusions. If there is clinical evidence of impaired filling, pericardial thickening is virtually diagnostic of constrictive pericarditis. The absence of thickening, however, does not rule out the presence of constriction <sup>(29)</sup>.

In patients being considered for pericardiectomy, detailed descriptions of the location and severity of thickening and calcification will aid the surgeon with respect to both risk stratification and planning of the procedure <sup>(3)</sup>.

Spin echo sequences are useful to detect thickened pericardium, whereas focal pericardial thickening and pericardial effusions are better visualized using cine gradient echo images sensitive to pericardial fluid. Abnormal diastolic septal motion on CMR yields a sensitivity of 81% and a specificity of 100% for the diagnosis of constrictive pericarditis <sup>(30)</sup>.

Constrictive pericarditis is summarized in Table 3

**Table 3: imaging findings in constrictive pericarditis** <sup>(3)</sup>

Modality	Imaging Findings
Chest radiography	Pericardial calcification
	Pulmonary vascular congestion
TTE	>35% respiratory variation of peak early diastolic MV inflow velocity
	Augmented hepatic vein diastolic flow reversals after the onset of expiration
	Inter-ventricular septum bounce
	Pericardial thickening ( $\geq 3$ mm)
	2D TTE
	Trans-esophageal echocardiography
CCT/CMR	Pericardial calcification, CCT
	Pericardial thickening $\geq 4$ mm
	Abnormal diastolic septal motion
	Narrow]wing, tubular deformation of right ventricle
	Systemic venous hypertension
	Pericardial adhesions

**Benign Pericardial Tumors**

**A- Pericardial teratoma**

On CT, they appear as multicystic, heterogeneous tumors typically with an associated pericardial effusion. Lipid or calcific densities can also be present on CT which often provide useful clues to the diagnosis. Cardiac CT may demonstrate their intrapericardial location, extent, and relation to vascular structures to which they are adherent, assisting planned surgical interventions. On MR

images, teratomas are heterogeneous masses with signal intensity characteristics that correspond to those of their components <sup>(31)</sup>.

**B- Pericardial fibroma**

Pericardial fibromas are may grow to a large size. CT detects it as a massive, heterogeneously enhancing soft tissue mass outside the cardiac chambers, directly abutting or compressing the adjacent structures. Fibromas are characteristically hypointense on T2-weighted MR images and isointense on T1-weighted MR images. On MR images obtained after the administration of gadolinium, fibromas demonstrate little heterogeneous enhancement <sup>(31)</sup>.

**Malignant Pericardial Tumors**

**A. Primary**

**1-Pericardial mesothelioma**

On both CT and MR images, pericardial mesothelioma appears as a heterogeneously enhancing mass that involves both the parietal and visceral layers of the pericardium, with possible invasion of the adjacent vascular and anatomic structures <sup>(32)</sup>.

**2-Primary cardiac sarcomas**

CT is helpful in the evaluation of cardiac sarcomas as it demonstrates the broad-based tumor attachment; myocardial, pericardial, and mediastinal invasion; as well as extension into the great vessels and pulmonary metastases, when present. Angiosarcomas have been described as highly vascular right atrial tumors with pericardial extension and low-attenuation areas <sup>(20)</sup>.

CT reveals the vascular and aggressive nature of angiosarcomas. These lesions are usually lobulated, vegetated masses that heterogeneously enhance at CT with areas of necrosis and may show avid enhancement with a “sun ray” appearance <sup>(33)</sup>.

On T1-weighted MR images, the signal intensity of angiosarcoma is heterogeneous and variable, depending on the degree of hemorrhage and necrosis in the tumor; however, angiosarcoma is predominantly isointense relative to myocardium. On T2-weighted and steady-state free precession images, these masses appear heterogeneous and hyperintense relative to myocardium <sup>(34)</sup>.

**3-Primary cardiac lymphomas**

On CT images, the soft-tissue component of pericardial lymphoma may appear iso- to hypoattenuating with heterogeneous enhancement.

At MR imaging, it appears hypointense with T1-weighted sequences and iso- to hyperintense with T2-weighted sequences with variable heterogeneous enhancement. On occasion, a discrete mass is not seen, and the sole imaging finding may be diffuse pericardial thickening or effusion, which is commonly hemorrhagic. Hemorrhagic pericardial effusion appears as hyperattenuation on CT images.

On T1-weighted MR images, hemorrhagic fluid demonstrates high signal intensity; it has intermediate signal intensity on T2-weighted images. Strands of fibrin and coagulated blood, with or without loculations, can be seen on steady-state free precession images <sup>(35)</sup>.

#### **B-Secondary**

On CT metastatic involvement is suggested by an irregularly thickened pericardium or pericardial mass associated with an effusion <sup>(36)</sup>. On MRI tumor invasion of the pericardium causes focal obliteration of the pericardial line. The signal intensity in most neoplasms is low in T1 and high on T2 <sup>(35)</sup>.

An exception is metastatic melanoma, which may have high signal intensity on T1 weighted image because of the paramagnetic metals bound by melanin. Most metastasis show significant contrast enhancement <sup>(36)</sup>.

#### **Miscellaneous**

##### **Pericardial cysts**

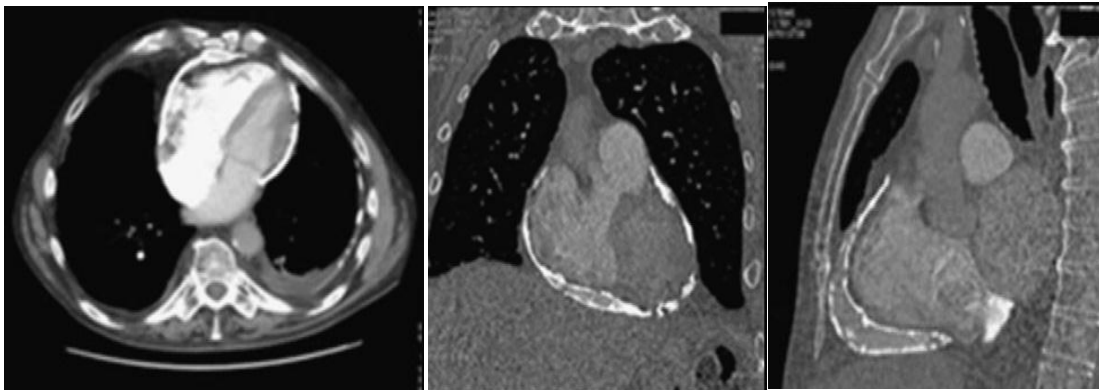
On CT scan, pericardial cysts appear as thin-walled, sharply defined, oval homogeneous masses of low attenuation. They fail to enhance with intravenous contrast. On MR scan pericardial cyst appear as cystic mass having the signal characteristics of neither a vascular lesion nor a lipoma, in direct contact with pericardium <sup>(37)</sup>.

##### **Pericardial fat pad**

It appears as a mass of fat density with low Hounsfield (negative) numbers on CT.

##### **Calcification**

CT is the preferred imaging modality (**Fig. 2**)



**Figure 2: computed tomography confirmed the highly calcified pericarditis and only a small antero-apical region free of calcification <sup>(38)</sup>.**



## CASE 1

A 55-year-old woman presented to the emergency department with complaints of breathing difficulty that had persisted for 2 days. The patient is a home maker with no history of exposure to asbestos.

On examination, the patient was in sinus rhythm with a blood pressure of 110/80 mmHg. Ultrasound identified bilateral mild to moderate pleural effusion.

The patient was then shifted to the ICU because of severe breathing difficulty. Echocardiogram showed mild pericardial effusion. The patient was evaluated with CT.

**Diagnosis: picture suggestive of primary malignant pericardial mesothelioma**



**Figure 3: a and b** -Contrast-enhanced axial reformatted and coronal CT images of the thorax show lobulated thickening of the pericardium (arrow head) and fluid collections within the pericardial sleeves (arrow). **c and d**- Contrast-enhanced axial and coronal reformatted CT images of the thorax show enlarged prevascular (arrow head) and pericardial lymph nodes (arrow) <sup>(39)</sup>.

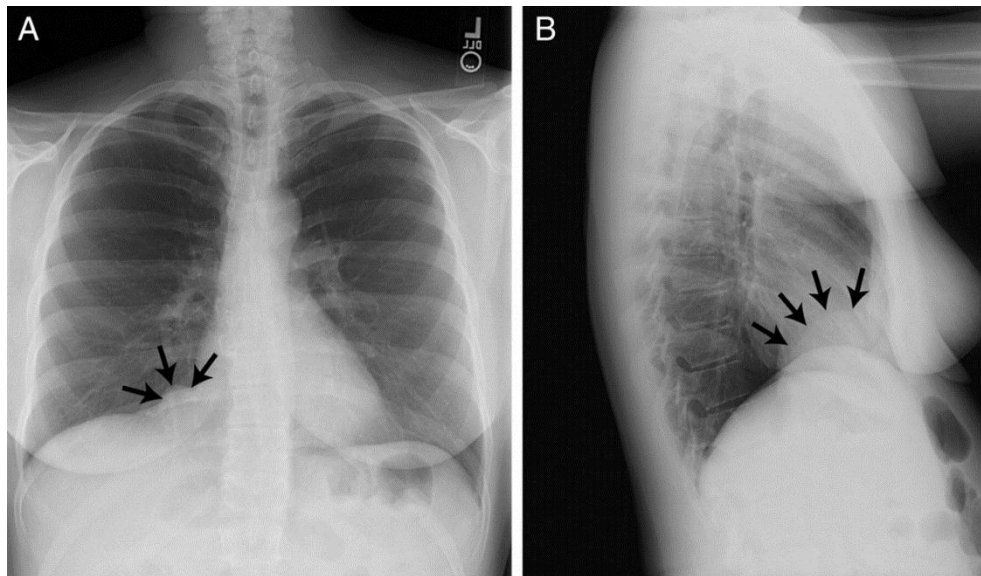
## CASE 2

A 48-year-old woman sought care for chest discomfort, generalized fatigue, and dyspnoea with minimal exertion. The symptoms were of 10 months' duration. A chest radiograph revealed a right cardiophrenic mass, with one of the differential considerations being a pericardial cyst (**Figure 4**).

The apical four-chamber view of the transthoracic echocardiogram showed an echolucent space next to the right atrium at the right cardiophrenic angle, consistent with a pericardial cyst. The right and left ventricular chamber size and function were normal. CT of the chest confirmed the diagnosis of a pericardial cyst (**Figure 4**).

The post operative histopathologic results were consistent with a pericardial cyst.

**Diagnosis:** picture of pericardial cyst.



**Figure 4:** chest radiograph. Posteroanterior (A) and lateral (B) views show a right cardiophrenic mass (arrows) at a typical location for a pericardial cyst <sup>(40)</sup>.

### CONCLUSION

Tissue characterization with CMR is superior to cardiac CT and echocardiography. CMR can differentiate tumor from thrombus and is often helpful to assess the perfusion of a pericardial mass with the use of gadolinium contrast. The final diagnosis depends on typical pathologic features.

### REFERENCES

1. Troughton RW, Asher CR and Klein A L (2004): Pericarditis. *Lancet*, 363: 717–727.
2. Czum JM, Silas AM and Althoen MC (2014): Evaluation of the pericardium with CT and MR. *ISRN Cardiology*, 214:11.
3. Yared K, Baggish AL, Picard MH *et al.* (2010): Multimodality imaging of pericardial diseases. *J. Am. Coll. Cardiol. Img.*,3: 650-660.
4. Dudzinski DM, Mak GS and Hung JW(2012): Pericardial diseases. *Curr Probl Cardiol.*, 37: 75–118.
5. Maisch B, Seferovic PM and Ristic AD(2004): Guidelines on the diagnosis and management of pericardial diseases executive summary: the Task Force on the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology. *Eur. Heart J.*, 25: 587-610.
6. Foster E, Manninig WJ and Downey BC (2011): Echocardiographic evaluation of the pericardium. [www.uptodate.com](http://www.uptodate.com).
7. Yared K, Baggish AL, Picard MH *et al.* (2010): Multimodality Imaging of Pericardial Diseases. *J. Am. Coll. Cardiol. Img.*,3: 650-660.
8. Cuccuini M, Lisi F, Consoli A *et al.* (2013): Congenital defects of pericardium. *IJA E.*, 118: 136-150.
9. Drury NE, De Silva RJ, Hall RM *et al.*(2007): Large Congenital Defects of the Pericardium. *Ann Thorac Surg.*,83: 1552-1553.
10. Saddala P, Kumar V and Patel C(2013): Pericardial effusion. <http://wikidoc.org>.

11. **Mayosi BM, Burgess LJ and Doubell AF(2005):** Tuberculous pericarditis. *Circulation*, 23: 3608-3616.
12. **Ilan Y, Oren R and Ben-Chetrit E(1991):** Acute pericarditis: etiology, treatment and prognosis. A study of 115 patients. *Jpn Heart J.*, 3: 315-321.
13. **Patel H, Maddaleni M, Patel C et al.(2013):** Pericarditis. <http://wikidoc.org>.
14. **Imazio M, LeWinter MM and Downey BC (2015):** Pericarditis. [www.uptodate.com](http://www.uptodate.com).
15. **Imazio M, Brucato A, Maestroni S et al.(2011):** Risk of Constrictive Pericarditis After Acute Pericarditis. *Circulation*, 124: 1270-1275.
16. **Wang ZJ, Reddy GP, Gotway MB et al.(2003):** CT and MR Imaging of Pericardial Disease. *RadioGraphics*, 23: S167-S180.
17. **Cohen RA, Loarte P, Navarro V and Mirrer B(2012):** Mature Cardiac Teratoma in an Adult. *Cardiology Research*, 3: 97-99.
18. **Stolzmann P, Marincek B and Alkadhi H(2009):** MDCT: Evaluation of Congenital and Acquired Diseases of the Pericardium. *Integrated Cardiothoracic imaging with MDCT. Published by M. Remy-Jardin. Springer.*
19. **Remy-Jardin M and Remy J(2009):** Integrated Cardiothoracic Imaging with MDCT. *Springer-Verlag Berlin Heidelberg, 1<sup>st</sup> edition.*
20. **Grebenc ML, Rosado de Christenson ML et al.(2000):** Primary Cardiac and Pericardial Neoplasms: Radiologic-Pathologic Correlation. *RadioGraphics*, 20: 1073-1103.
21. **Baztarrica GP(2010):** Primary Cardiac Lymphoma. *Circulation*, 121: 2249-2250.
22. **O'Leary SM, Roobottom CA, Manghat NE et al.(2010):** Imaging the pericardium: Appearances on ECG-gated 64-detector row cardiac computed tomography. *The British Journal of Radiology*, 83: 194-205.
23. **Lau CL and Davis RD(2004):** The Mediastinum. *Sabiston's Textbook of Surgery 17<sup>th</sup> edition. Philadelphia, Elsevier.*
24. **Gibson CM (2011):** Pericardial calcification. <http://wikidoc.org>.
25. **Gibson CM, Zorkun C and Almonacid A (2009):** Pneumopericardium. <http://wikidoc.org>.
26. **Yared K, Baggish AL, Picard MH et al.(2010):** Multimodality Imaging of Pericardial Diseases. *J. Am. Coll. Cardiol. Img.*,3: 650-660.
27. **Ordovas KG and Higgins CB(2006):** Pericardial diseases. *MRI and CT of the Cardiovascular System. 1st edition. Edited by McAllister L & Barrett K. Philadelphia, PA: Lippincott.*
28. **Dwyer RT and Khalil T(2003):** Computed Tomographic Diagnosis of Unsuspected Pericarditis, Case report. *J HK Coll Radiol.*,6: 36-39.
29. **Talreja DR, Edwards WD and Danielson GK(2003):** Constrictive pericarditis in 26 patients with histologically normal pericardial thickness. *Circulation*, 108: 1852-1857
30. **Giorgi B, Mollet NR and Dymarkowski S(2003):** Clinically suspected constrictive pericarditis: MR imaging assessment of ventricular septal motion and configuration in patients and healthy subjects. *Radiology*,228: 417-424.
31. **Restrepo CS, Vargas D, Ocazonez D et al.(2013):** Primary Pericardial Tumors. *Radiographics*, 213: 33.
32. **Suman S, Schofield P and Large S(2004):** Primary pericardial mesothelioma presenting as pericardial constriction: a case report. *Heart*, 204: 90.
33. **Burke A(2008):** Primary malignant cardiac tumors. *Semin Diagn Pathol.*,25: 39-46.
34. **Holtan SG, Allen RD and Henkel DM(2007):** Angiosarcoma of the pericardium presenting as hemorrhagic pleuropericarditis, cardiac tamponade, and thromboembolic phenomena. *Int J Cardiol.*, 207: 115.
35. **Rajiah P(2011):** Cardiac MRI: part 2, pericardial diseases. *AJR Am J Roentgenol.*,197: 621-634.
36. **Eisenberg RL(2010):** Clinical Imaging: An Atlas of Differential Diagnosis. Lippincott Williams & Wilkins.
37. **Kaul P, Javangula K and Farook SA(2008):** Massive benign pericardial cyst presenting with simultaneous superior vena cava and middle lobe syndromes. *Journal of Cardiothoracic Surgery*, 3: 32.
38. **Flecher E, Heautot JF, Abouliatim I et al. (2009):** Calcified chronic constrictive pericarditis: an egg in the chest. *European Journal of Cardio-thoracic Surgery*, 35: 174.
39. **Ramachandran R, Radhan P, Santosham R et al.(2014):** A Rare Case of Primary Malignant Pericardial Mesothelioma. *J Clin Imaging Sci.*,4: 47.
40. **Najib MQ, Chaliki HP, Raizada A et al.(2011):** Symptomatic pericardial cyst: a case series. *European Journal of Echocardiography*, 12: 43.