The assessment of pericardial disease on cardiovascular magnetic resonance imaging: A pictorial review

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Introduction

The pericardium is a two-layered fibrous membrane that envelops all four cardiac chambers and the origins of the great vessels. Many disease processes can affect the pericardium including infection, neoplasm, trauma, connective tissue disease, primary myocardial disease and congenital cardiac disease.

Echocardiography historically has been the imaging modality most commonly used for the initial assessment of suspected pericardial disease. While echocardiography has the advantage of being readily available and is particularly good in the assessment of suspected simple pericardial effusion or tamponade, it has limitations when characterising more complex pericardial pathology. As a result, cross-sectional imaging techniques are increasingly being used to evaluate for suspected pericardial disease as they provide a comprehensive assessment of the pericardium and enable the detection of associated abnormalities in the mediastinum and chest. The main advantages of cardiovascular magnetic resonance imaging (CMR) over other imaging modalities are its ability to make a functional assessment, particularly in cases of suspected pericardial constriction, and tissue characterisation sequences which allow further analysis of inflammatory conditions and pericardial tumours.

In this article, we review the anatomy of the pericardium as demonstrated on CMR. We also discuss the variety of pericardial pathologies that can be identified, including pericardial effusions and thickening with or without constriction, developmental abnormalities and tumours.

Normal pericardial anatomy and physiology

The pericardium is a relatively avascular fibrous membrane that surrounds the heart and attaches to the sternum, the diaphragm and the anterior mediastinum and envelops the origin of the great vessels and venae cavae. It consists of two layers, the visceral and parietal pericardium. The visceral pericardium is composed of a single layer of mesothelial cells that are adherent to the epicardium, while the parietal pericardium is a fibrous structure that is less than 2mm thick. The two layers of the pericardium are separated by a potential space that in normal adults contains 15-50mls of serous fluid.

The pericardium limits the spread of infection and inflammation to the heart from adjacent mediastinal structures. In addition, due to its relatively inelastic physical properties, the pericardium limits acute cardiac dilatation and enhances the mechanical interactions of the cardiac chambers. However, the pericardium can adapt to very slowly accumulating pericardial effusions and can increase in size over time. Hence, slowly accumulating pericardial effusions may become quite large without compressing the cardiac chambers to cause cardiac tamponade.

Congenital pericardial defects

Complete pericardial agenesis is rare but benign as there is no risk of cardiac herniation, which is the main complication of a focal pericardial defect. The most common partial pericardial defect is over the lateral wall of the left ventricle. These may be asymptomatic and initially present as an incidental-imaging finding. Complications of partial pericardial defect include cardiac herniation and entrapment, particularly the left atrial appendage. Surgical repair of the defect is often required in cases where cardiac herniation has occurred.

Pericardial effusion and cardiac tamponade

The accumulation of more than 50mls of fluid in the pericardial space is abnormal. Pericardial effusions can occur as a result of impaired venous or lymphatic drainage. Common causes of pericardial effusion include infection (viral, bacterial or tuberculous), neoplasm, myocardial infarction, traumatic injury, heart failure and renal insufficiency. While simple effusions may be readily identified by echocardiography, CMR can identify small or loculated effusions, especially those found in anterior locations which would otherwise be challenging to identify with echocardiography alone. In addition, breath-hold cine CMR imaging can demonstrate changes in the distribution of pericardial fluid between systole and diastole, which helps distinguish between small pericardial effusions and pericardial thickening.

Effusive pericardial effusions typically demonstrate high signal intensity on T2-weighted images. Exudative effusions often show intermediate signal intensity on T1 and T2-weighted images, which may have protein content-related differences in gravity-dependent regions that may subsequently develop into loculations. Haemorrhagic effusions exhibit high signal intensity on T1-weighted images. Acute accumulation of over 100mls can impair diastolic filling with compression of the RV free wall, causing cardiac tamponade (figure 1). The importance of early recognition of this potentially life-threatening condition cannot be overstated, with treatment aimed at emergency drainage of the pericardial fluid.

Acute pericarditis

Acute inflammation of the pericardium can present in isolation or as part of a multi-system condition. Pathologically there is thickening of both visceral and parietal pericardial layers, with or without an associated pericardial effusion. Classically, patients with acute pericarditis present with sharp retrosternal chest pain which is worse on inspiration and in a supine position and relieved by sitting forward. A pericardial rub may be heard on auscultation. Although as many as 90% of isolated cases of pericarditis are idiopathic or viral in aetiology, other potential causes include bacterial and tuberculous infection, uraemia, connective tissue diseases, myocardial infarction, neoplasms, radiotherapy and pericardial trauma.

Acute pericarditis is identified on CMR by thickening of both visceral and parietal pericardial layers, which show low signal intensity on T1-weighted sequences, with areas of active pericardial inflammation shown by increased signal on T2 and late gadolinium enhanced imaging (figure 2).

Constrictive pericarditis

Pericardial constriction occurs when a scarred, thickened and frequently calcified pericardium impairs cardiac filling, limiting the total cardiac volume. The most frequent causes of constrictive pericarditis are mediastinal radiation, cardiac surgery and tuberculous pericarditis. Other causes include infection, trauma, neoplasm and connective tissue disease.
Patients with constrictive pericarditis frequently present with symptoms of heart failure, such as dyspnoea, orthopnea and fatigue. Constrictive pericarditis is difficult to differentiate from restrictive cardiomyopathy on clinical history, examination and echocardiography alone. In both conditions, ventricular filling is restricted, leading to an increase in diastolic filling pressure. It is vital, however, to distinguish between these two similar conditions as patients with constrictive pericarditis, unlike those with restrictive cardiomyopathy, may benefit from surgical pericardectomy.

There are a number of key imaging features depicted on CMR that help to distinguish constrictive pericarditis from restrictive cardiomyopathy (Figures 3 and 4). Pericardial thickening of greater than 4mm is abnormal and, when associated with the clinical findings of heart failure, highly suggestive of constrictive pericarditis. The central cardiovascular structures may display characteristic morphological changes in constrictive pericarditis, the ventricles appearing narrow and tubular, the right ventricular free wall appearing distorted and the aorta and both venae cavae being enlarged. CMR can also demonstrate characteristic functional manifestations of constrictive pericarditis, including abnormally rapid diastolic filling and abnormal motion of the interventricular septum during inspiration. Real-time cine images are particularly useful for demonstrating ventricular septal flattening on deep inspiration during diastole (“ventricular interdependence”), which occurs when pericardial constriction is established (Figure 3).

**Pericardial masses**

The differential diagnosis of pericardial masses is extensive and includes pericardial cysts, haematomas, benign and malignant primary neoplasm and metastatic disease. CMR can accurately define the site and extent of pericardial masses and can aid diagnosis of these masses through differences in MR signal intensity, enhancement with gadolinium, contrast and the presence or absence of blood flow on cine imaging.

Pericardial cyst and pericardial diverticula

Membrane-lined fluid-filled sacs can either be in communication with the pericardium as an out-pouching (pericardial diverticula) or have separated completely (pericardial cyst). Pericardial cysts are usually asymptomatic and approximately 80% are located in the right cardiophrenic sulcus. They exhibit low or intermediate signal intensity on T1-weighted imaging and homogenous high signal intensity on T2-weighted imaging. They do not enhance following the administration of gadolinium.

**Pericardial haematoma**

CMR is particularly good for the diagnosis of pericardial haematomas. Acute haematomas demonstrate homogenously high signal intensity on T1-weighted imaging; where as subacute haematomas (week 1-4) demonstrate heterogeneous high signal intensity on T1- and T2-weighted imaging. Chronic organising haematomas may contain low intensity foci on T1-weighted imaging with a dark peripheral rim representing haemosiderin deposition, fibrosis and calcification. Haematomas usually do not enhance with gadolinium, allowing differentiation from pericardial neoplasms, and do not contain internal flow, allowing differentiation from coronary and ventricular pseudoaneurysms.

**Primary pericardial neoplasms**

Primary pericardial neoplasms of the pericardium are rare and may be benign or malignant. Benign pericardial tumours include lipoma, teratoma, fibroma and haemangioma. Lipomas characteristically have high signal intensity on T1-weighted imaging (Figure 5) while fibromas typically demonstrate low signal intensity on T1 and T2-weighted imaging, with poor enhancement with gadolinium. Deposition of fat or calcium within a pericardial mass is suggestive of teratoma.

Malignant pericardial tumours include primary pericardial mesothelioma, lymphoma and sarcoma. Pericardial mesothelioma is demonstrated on CMR as an encasing irregular pericardial mass often associated with pericardial effusion and nodules. Lymphoma and sarcoma typically appear as large irregular heterogeneous masses that display low or intermediate signal on T1-weighted imaging. Typically lymphomas heterogeneously enhance with gadolinium and are associated with pericardial effusions.

**Metastatic pericardial neoplasms**

Pericardial metastases are far more common than primary pericardial tumours. Metastatic tumours may spread to the pericardium via lymphatic or haematological spread or from direct invasion from adjacent structures within the thorax. Lung and breast cancers are the most common sources of pericardial metastases, followed by melanoma and lymphoma.

Most metastatic pericardial neoplasms demonstrate low signal intensity on T1-weighted imaging and high signal on T2-weighted imaging. Metastatic melanoma is the exception, demonstrating high signal intensity on T1-weighted imaging because of the paramagnetic effect induced by melanin. Sites of malignant pericardial disease will generally enhance with gadolinium (Figure 6). Metastatic disease is associated with haemorrhagic pericardial effusions and irregular pericardial thickening. Direct pericardial invasion by adjacent tumours can be identified by evidence of focal obliteration of the pericardium.

**Conclusion**

CMR is an increasingly useful non-invasive investigation for cardiac disease. This imaging review highlights pericardial anatomy, physiology and pathology, specifically illustrating the CMR findings of acquired and developmental pericardial disease and thus empowering the reader to confidently identify pericardial abnormalities on CMR.

**Suggested further reading**


**FIGURE 1**

a) SSFP short axis view showing circumferential pericardial effusion (arrow) and b) T1-weighted Black Blood four-chamber view showing a large pericardial effusion (*) with compression of the right ventricle leading to cardiac tamponade.
FIGURE 2
a) Late gadolinium-enhanced four-chamber view and b) late gadolinium-enhanced short axis view of a patient with acute tuberculous pericarditis showing circumferential pericardial thickening (white arrows) and pericardial effusion (Black arrows).

FIGURE 3
a) SSFP four-chamber view showing bi-atrial dilation and abnormal pericardial thickening (black arrows) in a patient with constrictive pericarditis. Still-captured images from real-time cine imaging taken in end-diastole c) during inspiration and d) expiration, illustrating flattening of the interventricular septum in inspiration (interrupted black arrow).

FIGURE 4
a) SSFP four-chamber view demonstrating gross bi-atrial dilation without pericardial thickening in restrictive cardiomyopathy. Still-captured images from real-time cine imaging taken in end diastole b) during inspiration and c) expiration illustrating preserved concavity of interventricular septum throughout respiratory cycle.

FIGURE 5
a) HASTE and b) SSFP four-chamber view showing a large pericardial lipoma (arrow) centered on the right AV groove, compressing the RA.
FIGURE 6
CMR study in a patient with known lower limb clear cell sarcoma and a pericardial effusion. a) Still-captured image from four-chamber cine imaging showing multiple pericardial (white arrows) and myocardial (black arrow) masses. b) T1-weighted Black Blood two-chamber view, c) T2-weighted Black Blood two-chamber view and d) STIR Black Blood three-chamber view demonstrate these masses to be of intermediate signal intensity on T1-weighted imaging and high signal on T2-weighted and STIR imaging. e) First pass perfusion four-chamber view shows peripheral enhancement within the pericardial mass with a central low signal. These features are typical of metastases to the pericardium.