MDCT of the pericardium: A teaching primer.

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Learning objectives

- To illustrate the anatomoradiology of the pericardium on MDCT
- To demonstrate examples of pericardial recesses and their variations.
- To provide a pictorial demonstration of the wide variety of the abnormalities of the pericardium and the pitfalls in the diagnosis of pericardial diseases.
Background

Multidetector row computed tomography (MDCT) provides imaging detail of the overall cardiac morphology including the normal and diseased pericardium.

It is important for the general radiologist to be familiar with both normal and variant pericardial anatomy and with that of the pericardial recesses, which can mimic some pathological processes.

This review aims to illustrate normal pericardial anatomy, diagnostic pitfalls, commonly encountered abnormalities and some more unusual entities.
Findings and procedure details

I/Introduction:

Although often not considered the primary imaging modality for evaluation of the heart, when it comes to the pericardium computed tomography compliments and in many cases surpasses echocardiograms and magnetic resonance imaging in the evaluation of pericardial pathology.

The combination of its spatial and contrast resolution, the ability to administer enhancing agents, the lack of field of view limitations as well as our ability to infer dynamic information serve to make this modality the test of choice in many cases of pericardial disease.

The continued development of multidetector computed tomography, cardiac gated imaging and the increasing use of three-dimensional CT in the area of coronary artery evaluation make it imperative to have a clear understanding of the normal pericardium and the pathologies that affect it.

II/ Computed Tomography Technique:

The parietal pericardium is defined on CT by the low houndsfield unit epicardial fat internally and mediastinal fat and lung externally. What we are seeing on imaging is the apposed parietal serous and fibrous pericardium. In most cases where thickening, fluid or calcification are the issue oral or intravenous contrast are not required. When there are questions regarding tumor involvement, cardiac chamber effect and myocardial change intravenous contrast may be of value.

The standard CT coverage in most people is from the great vessels through the diaphragm. One should note that a high insertion on the great vessels is an anatomic variation and when there is a very large pericardial effusion it everts the central tendon and pushes the pericardium caudally, thus one may need to change coverage accordingly.

Multidetector CT may have a role in functional imaging both in perfusion and dynamic imaging of chamber size and motion but this has yet to be fully explored.

III/ Normal anatomy and function:

The normal pericardium is a double-layered fibroserosal sac embryologically forming a subdivision of the celomic cavity into which invaginates the developing heart (Fig 1).

The thickness of the normal parietal pericardium has been determined to be 1-2mm (Fig 2) by anatomic studies but it is non-uniform in thickness with most CT measurements being taken anterior to the plane of right and left atrium where it is best defined.
The visceral and parietal serous pericardium are intimately attached to the epicardium and fibrous pericardium respectively creating between these layers the pericardial space that normally has 20-25 mls of lymph fluid. The potential spaces of the pericardium are defined by sinuses and recesses.

The pericardium is anchored cranially to the adventitia of the great vessels and caudally to the central tendon of the diaphragm. Other points of fixation of the pericardium are the sternum and adjacent structures such as esophagus and spine.

The pericardium has a mechanical role holding the heart in position, preventing over-dilatation of the heart, facilitating the hemodynamic interdependence of the ventricles and providing a barrier between the heart and other thoracic structures.

IV/ Pericardial Recesses:

The visceral pericardium (epicardium) adheres to the heart and great vessels. It forms recesses and sinuses, which can be visible at cross-sectional imaging if they contain enough fluid, even in the absence of pericardial effusion (Fig 3, 4, 5).

Knowledge of the location of these recesses and sinuses will prevent mistaking them for enlarged lymph nodes or other masses.

Knowledge of the pericardial anatomy is also important because tumors and pericardial cysts may arise in these locations.

The pericardial recesses can be categorized on the basis of whether they arise from the pericardial cavity proper, the transverse sinus, or the oblique sinus.

1/ Recesses Arising from the Pericardial Cavity Proper

The pulmonic vein recesses, which are usually small, lie along the lateral borders of the heart between the superior and inferior pulmonary veins (Fig 3, 4, 5, 6, 7, 8).

The pulmonic vein recesses are in proximity to and can be mistaken for bronchopulmonary lymph nodes (Fig 7).

The postcaval recess lies posterior to and to the right of the SVC and is also usually small (Fig 3, 4, 5, 13).

2/ Recesses Arising from the Transverse Sinus

The superior aortic recess extends anterior to the ascending aorta and has anterior, posterior, and right lateral portions
The anterior portion of the superior aortic recess has a characteristic triangular shape as it insinuates itself between the ascending aorta and the main pulmonary artery (Fig 4, 5, 9, 10, 11).

The lateral portion similarly insinuates itself between the ascending aorta and the SVC (Fig 13).

The posterior portion lies posterior to the ascending aorta, where it is sometimes referred to as the superior pericardial recess or superior sinus (Fig 10, 11, 13).

The inferior portion of the superior aortic recess communicates with the transverse sinus. The posterior extension of the superior aortic recess can be mistaken for a lymph node.

Distinguishing features of the posterior portion of the superior aortic recess are its location directly posterior to the ascending aorta, its crescent shape, and the fact that it has fluid attenuation.

These features help distinguish fluid in the pericardial recess from precarinal lymph nodes, which tend to be round or oval and of soft-tissue attenuation. The superior aortic recess attaches directly to the aorta so that intervening fat is not identified.

This lack of a fat plane also helps distinguish fluid in the pericardial recess from precarinal lymph nodes.

Occasionally, the superior pericardial recess extends more superiorly than expected to lie in a right paratracheal location (Fig 14), where it may be mistaken for a right paratracheal lymph node or bronchogenic cyst.

The inferior aortic recess extends inferiorly from the transverse sinus posterior to the aorta and anterior to the left atrium, extending inferiorly to the level of the aortic valve.

The right and left pulmonic recesses lie inferior to the right and left pulmonary arteries, respectively. Fluid collections within the pulmonic recesses can mimic the appearance of lymphadenopathy (Fig 7).

Recesses Arising from the Oblique Sinus.

The oblique sinus (Fig 15, 16) extends superiorly behind the right pulmonary artery and medial to the bronchus intermedius, where it is called the posterior pericardial recess.

V/ Anomalies of the Pericardium:

1/ Absence:

Most commonly an absent pericardium is congenital or a result of cardiac surgery.
Congenital types are thought to be due to a premature closure of the duct of Cuvier causing vascular compromise.

Absence is classified as partial or complete with partial left sided defects being the most common.

Although in many it is an incidental finding, herniation of a portion of the chamber or coronary artery is a recognized complication (Fig 17).

CT features are an absent fibrous pericardium with lung on both sides of the right ventricle outflow tract and bulging of the main pulmonary artery to the left side. There is no preaortic recess and direct contact between heart and lung is observed (Fig 18, 19).

2/ Pericardial Effusion:

Pericardial effusion originates in the obstruction of venous or lymphatic drainage from the heart. Common causes of pericardial effusion include heart failure, renal insufficiency, infection (bacterial, viral, or tuberculous), neoplasm (carcinoma of lung or breast, or lymphoma), and injury (from trauma or myocardial infarction).

CT attenuation measurements enable the initial characterization of pericardial fluid.

A fluid collection with attenuation close to that of water is likely to be a simple effusion (Fig 20, 21). Attenuation greater than that of water suggests malignancy, hemopericardium, purulent exudate, or effusion associated with hypothyroidism (Fig 22). Pericardial effusions with low attenuation also have been reported in cases of chylopericardium.

Cardiac Tamponade: CT Findings

Large pericardial effusion with enlargement of either the SVC (diameter similar or greater than adjacent aorta) or IVC (diameter twice adjacent aorta. Periportal edema and reflux of contrast into IVC and azygous vein or enlargement of hepatic and renal veins

3/ Pneumopericardium:

Iatrogenic and traumatic are among the more common etiologies of pneumopericardium (Fig 24).

Cardiac surgery, pericardiocentesis and esophageal sclerotherapy represent the majority of iatrogenic causes.
Direct connections have been identified such as; alveolar-pericardial, pleuro-pericardial, peritoneo-pericardial, as well as enteric fistulae particularly esophageal.

In discriminating air collections CT surpasses echocardiography and MRI studies. Pneumopericardium just like effusions can result in a tamponade effect.

4/ Cysts:

Congenital pericardial cysts are formed when a portion of the pericardium is pinched off during early development. Pericardial cysts usually have thin smooth walls without internal septa. At CT, they have the same attenuation as water and do not enhance after contrast material administration (Fig 25). Pericardial cysts may occur anywhere in the mediastinum, although they usually are found in the right cardiophrenic angle. A pericardial cyst in an unusual location may be indistinguishable from a bronchogenic cyst or thymic cyst.

5/ Pericardial Thickening
Pericarditis/Calcification/Constriction

In general the pericardium reacts to a wide variety of insults in a limited fashion that includes fluid exudation, fibrin production and cellular proliferation.

Pericarditis may be primary or secondary with primary idiopathic being the most common. Of the many secondary causes infection (especially tuberculosis), renal failure, radiation, myocardial infarction and collagen vascular disorders are the most clinically relevant.

In many cases pericarditis is self limited or limited with medication alone and has little consequence. However in some the natural history results in calcification and/or constriction.

Two patterns of calcification have been described linear or amorphous. It involves the visceral and parietal layers and most commonly is found near the right and left atrio-ventricular grooves.

Calcification may be seen independent of constriction and vice versa.

Normal pericardial thickness is less than 2 mm (Fig 2). Pericardial thickness of 4 mm or more indicates abnormal thickening and, when it is accompanied by clinical findings of heart failure, is highly suggestive of constrictive pericarditis (Fig 26).
A normal pericardium by CT, in a patient with the correct clinical picture excludes constriction and makes a restrictive cardiac disease more likely.

With constrictive pericarditis, CT will reveal consequences of constriction including elongated right ventricle, bowed septum, enlarged right atrium, dilated vena cavae and possibly liver congestion (Fig 27).

Pericardial thickening may be limited to the right side of the heart or to an even smaller area, such as the right atrioventricular groove.

Neither pericardial thickening nor calcification is diagnostic of constrictive pericarditis unless the patient also has symptoms of physiologic constriction or restriction.

6/ Pericarditis without Constriction:

Pericardial thickening may occur in the absence of constrictive pericarditis. Pericardial thickening may result from inflammation caused by a variety of conditions, including acute pericarditis, uremia, rheumatic heart disease, rheumatoid arthritis, sarcoidosis, and mediastinal irradiation. At contrast-enhanced CT, enhancement of the thickened pericardium indicates inflammation (Fig 26). A limitation of CT imaging of the pericardium is the occasional difficulty in differentiating a small effusion from pericardial thickening. Echocardiography may depict the effusion more clearly in such cases.

5/ Pericardial Neoplasms:

Benign and malignant pericardial solid masses are equally common. Teratoma and malignant mesothelioma are the leading primary solid masses.

Secondary malignancies are far more common than primary with seventy percent due to spread from lung, breast and lymphoproliferative disorders.

Although primary tumors more commonly affect the myocardium than the pericardium the reverse is true of secondary tumors. In those with pericardial metastases 25% have reduced cardiac function and for the majority tamponade is the commonest cause of death.

CT features of masses that may elucidate their etiology include; morphology, location, extent, cyst or solid character, their effect on cardiac chambers as well as their enhancement characteristics and the amount of extracardiac disease (Fig
28, 29, 30). It is in the setting of malignancy with its ability to evaluate the whole thorax that CT has much to offer.
Fig. 1: Cross section of the heart wall and pericardium showing the fibrous pericardium, the parietal layer and the visceral layer.

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Fig. 2: Axial CT scan of the heart: Normal pericardium with a thickness not exceeding 2 mm.

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Fig. 3: Line diagram demonstrating the posterior pericardial reflections, which form the transverse and oblique sinuses and their associated recesses.

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**Fig. 4:** Pericardial Sinuses and Recesses: at level of main pulmonary artery

**Fig. 5:** Pericardial Sinuses and Recesses: at level of right pulmonary artery


**Fig. 6:** Normal cross-sectional anatomy of pericardial sinus and recess. a. Cross-sectional drawing of pericardial sinus and recess. b. Due to fluid collection in rSAR, right lateral margin of rSAR migrate into right lateral margin of SVC, wrapping one half of SVC (arrow). rSAR = right lateral portion of superior aortic recess, aSAR = anterior portion of superior aortic recess, pSAR = posterior portion of superior aortic recess, LPR = left pulmonary recess.
Fig. 7: Reconstructed Coronal CT image of the mediastinum demonstrating the superior right pulmonic vein recess (arrow head. It could be misdiagnosed as lymph node.
Fig. 8: Axial contrast-enhanced CT scan shows a small amount of fluid in the transverse sinus (T) posterior to the ascending aorta (AA). The transverse sinus extends laterally, where it communicates with the left pulmonic recess (arrow) inferior to the left pulmonary artery (PA)

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**Fig. 9**: Axial contrast-enhanced multidetector CT scan shows left pulmonic recess (arrow) of transverse sinus (asterisk). Pericardial fluid is also seen anterior to aorta and pulmonary artery forming characteristic cleft as it indents between great vessels (arrowheads).

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**Fig. 10:** Chest CT shows moderate amounts of fluid collection in rSAR (long arrow) and connection between rSAR and pSAR (short arrow). Small amounts of pleural effusion.

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**Fig. 11:** Axial(a) and sagittal(b) CT: posterior aortic pericardial recess (yellow arrow).

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Fig. 12: Chest CT shows moderate amounts of fluid collection in rSAR (long arrow) and connection between rSAR and pSAR (short arrow). Small amounts of pleural effusion. PA = posterior-anterior, rSAR = right lateral portion of superior aortic recess, pSAR = posterior portion of superior aortic recess

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**Fig. 13:** multidetector CT scan delineates uid in postcaval recess (long arrow) and posterior (arrowheads) to ascending aorta. Pericardial uid also extends anterior (short arrows) to aorta (A) and pulmonary artery (PA) as well as in left pulmonic recess (asterisk) of transverse sinus. S = superior vena cava.

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Fig. 14: Axial contrast-enhanced multidetector CT scan at level of left brachiocephalic vein (LBV) shows cephalad extension of superior aortic recess adjacent to trachea. This "high-riding" variant (arrow) can be misinterpreted as adenopathy when slice thickness precludes seeing anatomic contiguity.

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Fig. 15: The transverse sinus (T) and oblique sinus (*) are separated by pericardial reflections. The pulmonic vein recesses (arrows) lie between the superior and inferior pulmonary veins.

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**Fig. 16:** Axial chest CT scan showing the oblique sinus containing small amount of fluid and lying posterior to left atrium.

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**Fig. 17:** Illustration of partial and total absence of left pericardium
Fig. 18: Axial chest CT showing bulging of the main pulmonary artery to the left side. There is no preaortic recess and direct contact between heart and lung is observed.

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**Fig. 19:** Chest radiograph (A) and chest cardiac computed tomography scan (B) demonstrating superior and lateral displacement of the apex without identifiable pericardium over the apex of the heart consistent with congenital absence of the pericardium

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**Fig. 20:** Axial chest CT scan showing mild pericardial effusion

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**Fig. 21:** Chest film showing the fat pad sign and Axial chest CT demonstrating marked pericardial effusion.

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**Fig. 22:** Axial enhanced chest CT scan showing high density of the pericardial hematique collection.

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Fig. 23: Axial chest and abdomen CT scans: Cardiac tamponade with Large pericardial effusion, enlargement of IVC (diameter twice adjacent aorta) and Reflux of contrast into IVC.

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Fig. 24: Axial chest CT scan showing post traumatic pneumopericardium

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**Fig. 25:** Chest film and axial chest CT scan demonstrating large cystic lesion in the right cardiophrenic angle representing a pericardial cyst.

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**Fig. 26:** Axial chest CT scan showing pericardial thickening with multifocal calcifications.

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**Fig. 27:** Cardiac 3D reconstruction showing pericardial calcifications with marked compression of cardiac ventricular cavities due to constrictive pericarditis

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**Fig. 28:** Axial enhanced chest CT image showing anterior mediastinal lymphoma with pericardial invasion.

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**Fig. 29**: Axial chest CT scan demonstrating local extension of mediastinal immature germ cell tumour into pericardium with subsequent pericardial effusion.

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**Fig. 30:** Axial chest scans after IV contrast injection showing hypervascular pericardial tumoral mass around right heart cavities representing metastasis from melanoma. Note also associated left lung metastatic nodules.

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Conclusion

MDCT has augmented our ability to identify and characterise the pericardium, allowing us to differentiate between the normal anatomy and pathology with a precise relationship to other anatomical structures. Computed tomography has much to offer in the evaluation of pericardial disease. Effusions and thickening of the pericardium are the most common findings and an appreciation of the normal anatomy helps differentiate these entities from other mediastinal pathologies.

For calcified pericarditis and pneumopericardium there is no better test than CT.

ECG-gated cardiac MDCT should be considered as part of the diagnostic armamentarium in the context of suspected pericardial disease.
References


