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The Role of Multimodality Imaging in the Management of Pericardial Disease

David Verhaert, MD; Ruvin S. Gabriel, MBChB; Douglas Johnston, MD; Bruce W. Lytle, MD; Milind Y. Desai, MD; Allan L. Klein, MD

Pericardial pathology is commonly encountered in clinical practice and may present either as an isolated process or in association with other systemic disorders. Recognizing pericardial pathology can be relatively straightforward, particularly if the clinical manifestation is typical (eg, the patient with acute pericarditis and an audible friction rub reporting retrosternal pain, exacerbated by inspiration or in the supine position) or when an associated disease process gives a direct clue to the diagnosis (eg, the finding of a complex pericardial effusion in a patient with a known malignancy). In these situations, the diagnostic pathway can be limited to a relatively small sequence of tests to basically confirm the initial clinical suspicion.

However, pericardial disease can also result in nonspecific symptoms and equivocal physical findings. When the initial tests of choice turn out to be nondiagnostic or the course of the disease is prolonged, pericardial disease may cause considerable diagnostic dilemmas. Furthermore, established diagnostic techniques may not visualize the full extent of the pericardial disease process. In such difficult clinical situations, an integrated multimodality imaging approach may provide incremental value. Unfortunately, current guidelines do not address the role of a multimodality approach in the difficult to manage pericardial patient. This review will therefore discuss the potential role of different imaging modalities in the diagnosis and management of pericardial disorders, with a specific focus on what constitutes a rational multimodality imaging approach.

Pericardial Disease: Which Imaging Test to Choose?
The 3 imaging modalities most commonly used for evaluation of pericardial disease are echocardiography, cardiac computed tomography (CT), and cardiac MRI (CMR). The main indications and limitation for each test are summarized in Table 1. Echocardiography remains the initial imaging method of choice for the majority of patients with pericardial disease, but in many clinical scenarios, a transthoracic echocardiogram (TTE) alone is insufficient. Under those circumstances, many clinicians experience difficulties deciding which test to choose as the best next step in the diagnostic process. Each patient scenario tends to be unique, mandating a highly individualized approach from the clinician but unfortunately also explains the complete absence of consensus or general recommendations to assist clinicians in making these decisions. Sometimes it is even unclear whether patients may benefit at all from additional imaging. As a consequence, patients may get referred for an exhaustive battery of imaging tests when it is not absolutely needed. The first important role of the imaging expert is therefore to ensure that patients do not get inappropriate or unnecessary tests and avoid incomplete or nondiagnostic studies. Subsequently, he should decide when and how to combine different tests to answer important clinical questions. Table 2 offers an example of pericardial disease-specific protocols for each imaging modality; adhering to standardized protocols would potentially maximize the information derived from an imaging test and probably avoid incomplete studies.

The role and value of specific imaging modalities for the recognition and characterization of a variety of pericardial disorders will subsequently be discussed and illustrated.

Pericarditis
Clinical Scenario
A 37-year-old man with a history of recurrent pericarditis is referred with a new episode of chest pain. During the last 2 years, the patient experienced multiple flare-ups of pericarditis affecting his ability to work and perform daily activities. He has previously been treated with intermittent courses of nonsteroidal antiinflammatory drugs and short trials of prednisone therapy. Echocardiography shows only a trivial pericardial effusion. Before initiating therapy with high-dose nonsteroidal antiinflammatory medication and colchicine, a CMR is performed, confirming the presence of active pericardial inflammation. Six weeks later, it is eventually decided to gradually taper the medication dose based on the results of a new CMR scan documenting a dramatic response to therapy (Figure 1). No serious recurrences have been noted since.

Acute pericarditis implies an inflammatory reaction of the pericardium, often presenting as a sudden, pleuritic-type
Technical difficulties in case of obesity, low signal-to-noise ratio of the operator dependent worse outcome (fever initial response to standard therapy). TTE is certainly indicated when there is concern for features of constrictive acute dissection of the ascending aorta (hemopericardium) or tuberculosis. Pericarditis can also occur in association with autoimmune disease; or more uncommon infections (eg, myocardial infarction (either acute or delayed, then referred to as Dressler syndrome); uremia; hypothroidism; systemic autoimmune disease; or more uncommon infections (eg, tuberculosis). Pericarditis can also occur in association with acute dissection of the ascending aorta (hemopericardium) or as a result of neoplastic invasion of the pericardium.

History, physical examination, ECG, and serological evidence of systemic inflammation will usually guide the clinician to the right diagnosis. When there is a clear diagnosis of uncomplicated idiopathic pericarditis, additional imaging is usually unnecessary because of the self-limited nature of the disease. However, TTE is still widely used as a screening tool for the presence of an associated pericardial effusion, visualized as an echo-free space adjacent to the myocardium. TTE can semiquantify the size of pericardial effusions into trivial (only in systole), small (<1 cm), moderate (1 to 2 cm), and large (>2 cm) or very large (>2 cm with compression of the heart). Our threshold for ordering a TTE is usually low, particularly when pericarditis is associated with indicators of worse outcome (fever >38°, subacute course, or failure of initial response to standard therapy). TTE is certainly indicated when there is concern for features of constrictive physiology or to exclude cardiac tamponade but also to detect associated heart disease or to guide diagnostic or therapeutic pericardiocentesis.

When to Consider Added Imaging?

Figure 2 illustrates a suggested pathway for situations in which sequential imaging generally is indicated, usually after a baseline TTE has been obtained. In summary, tomographic imaging (CT or CMR) for the evaluation of pericarditis should be considered in case of:

(1) Therapeutic difficulties or complications after acute pericarditis (failure to respond to aspirin or nonsteroidal antiinflammatory drugs and evolution toward recurrent or chronic pericarditis with possible constrictive features).

(2) Traumatic etiology (penetrating injury, esophageal, or gastric perforation).

(3) Pericarditis associated with a specific disease (eg, neoplastic disorders) or with involvement of adjacent anatomic structures (aortic dissection, empyema, acute pancreatitis).

One good example of the complimentary role of both CT and CMR is their ability to clearly visualize pericardial thickening and to demonstrate inflammatory changes involving the pericardium and/or epicardial fat. As illustrated by the introductory case scenario and Figure 1, CMR may thus confirm the presence of pericarditis when the diagnosis is uncertain by TTE alone. However, it should be emphasized that TTE is frequently unremarkable in acute pericarditis, and the majority of these patients do not need additional imaging unless they present with atypical features or there is concern for a complicated course.

Similarly, CMR can be used to monitor the inflammatory process in patients with recurrent pericarditis or effusive constrictive pericarditis, thereby identifying patients with persistence of pericardial inflammation despite courses of medical therapy. Tapering or stopping antiinflammatory therapy (particularly corticosteroids) in these patients is often troublesome, as a balance needs to be made between the risk of recurrence and the risk of possible side effects related to continuation of therapy. Clinicians will often try to obtain proof of stable remission before therapy is reduced or stopped, usually by reassuring that symptoms have resolved.

Table 1. Strengths and Limitations of Various Imaging Modalities in the Evaluation of Pericardial Disease

<table>
<thead>
<tr>
<th>Echocardiography</th>
<th>Cardiac CT</th>
<th>CMR</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main indications/advantages</strong></td>
<td><strong>Main limitations/disadvantages</strong></td>
<td><strong>Main indications/advantages</strong></td>
</tr>
<tr>
<td>First-line diagnostic imaging test in the evaluation and follow-up of pericardial disease</td>
<td>Limited windows, narrow field of view</td>
<td>Time consuming, high cost</td>
</tr>
<tr>
<td>Widely available</td>
<td>Technical difficulties in case of obesity, obstructive lung disease or immediately post- cardiothoracic surgery</td>
<td>Preferably stable heart rhythms</td>
</tr>
<tr>
<td>Low cost</td>
<td>Operator dependent</td>
<td>Contraindicated in case of pacemaker or defibrillators</td>
</tr>
<tr>
<td>Safe</td>
<td>Low signal-to-noise ratio of the pericardium</td>
<td>Lung tissue less well visualized</td>
</tr>
<tr>
<td>Can be performed bedside or in hemodynamically unstable patients</td>
<td>Limited tissue characterization</td>
<td>Calcifications less well visualized</td>
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</tbody>
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Calcifications less well visualized

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or C-reactive protein has normalized. In case of doubt, we typically perform CMR and if active inflammation is still present, tapering of the antiinflammatory medication is reconsidered regardless of symptomatic improvement.

Small or loculated effusions may not always be evident on TTE; CT or CMR may then be useful, particularly when there is concern for penetrating injury (eg, a patient presenting with chest pain shortly after implantation of a transvenous pacemaker wire). CT attenuation values may occasionally give a clue to the etiology of an effusion. Serous fluid typically has a density close to water (10 Hounsfield Units [HU]), whereas higher attenuation values are more suggestive of an exudate (20 to 60 HU, as in purulent pericarditis or malignancy) or acute hemopericardium (>60 HU, as secondary to penetrat-
ing injury or acute dissection). It can occasionally be challenging to differentiate thickened pericardium from a small effusion by CT, precisely because of similar attenuation coefficients. In contrast, CMR breath-hold steady-state free precession sequence cine imaging will demonstrate a change in the distribution of pericardial fluid between systole and diastole, thereby distinguishing small effusions from pericardial thickening. CMR may also allow further characterization of a pericardial effusion. Simple transudative effusions typically have a low signal intensity on T1-weighted images, whereas hemorrhagic or exudative effusions often have a medium or high signal intensity on T1 sequences.

Cardiac Tamponade

Clinical Scenario

A 61-year-old patient receiving chemotherapy for metastatic non–small cell lung carcinoma is admitted to the emergency department because of worsening shortness of breath, excessive cough, and chest discomfort in the last few days. His blood pressure is 80/40 mm Hg, his heart rate is 110 bpm, and physical examination reveals a markedly elevated jugular venous pressure. Urgent bedside TTE demonstrates a large pericardial effusion with features of tamponade (supplemental Movie Clips 1 and 2). An echo-guided pericardiocentesis is subsequently performed before more definite treatment by surgical pericardial window formation is offered the next day.

Cardiac tamponade is a potentially fatal compression of the heart due to an accumulation of fluid in the intrapericardial space. The fluid accumulation can occur rapidly or more gradually, but once the limits of parietal pericardial stretch are exceeded, intrapericardial pressure will rise quickly and compromise diastolic filling. Patients are often anxious and may report dyspnea and chest discomfort. The classic findings in tamponade known as Beck’s triad include hypotension, jugular venous distension, and muffled heart sounds. Tachycardia is the rule, acting as a compensatory mechanism in the setting of a decreased stroke volume. Cardiac tamponade ultimately is a clinical diagnosis and must be considered in every patient with unexplained cardiogenic shock or pulseless electric activity. Because the clinical diagnosis can be challenging, echocardiography should be performed without further delay once a patient is suspected of having tamponade.

Supplemental Movie Clips 1 and 2 highlight an important feature of cardiac tamponade, which is the collapse of right-sided chambers once the intrapericardial pressure exceeds the intracavitary pressure. Because intracardiac pressures vary throughout the cardiac cycle, inversion of these chambers can be transient, typically at end-diastole for the right atrium or early diastole for the right ventricle (the latter being more specific for tamponade). Right-sided collapse will be most prominent during expiration when right heart filling is reduced. When the intrapericardial pressure continues to rise, duration and severity of collapse will further increase, eventually resulting in circulatory failure. Distension of the inferior vena cava with absent reduction in diameter during inspiration is another very sensitive, albeit nonspecific sign of tamponade. When the intrapericardial volume becomes fixed due to pericardial constraint, filling of...
one side of the heart will occur at the expense of the other. This mechanism leads to substantial reciprocal respiratory variation in mitral (>25%) and tricuspid (>50%) inflow, reflecting the accentuated ventricular interdependence. Similar reciprocal respiratory changes in flow velocity can be identified in the hepatic and pulmonary veins. Pulmonary vein diastolic forward flow will decrease with inspiration and increase with expiration. In contrast, the hepatic vein diastolic forward flow will decrease on expiration and have a large atrial reversal. An additional manifestation of increased ventricular interdependence is the leftward shift of the septum with inspiration, reversing on expiration.

Figure 2. Suggested diagnostic approach to investigate acute pericarditis and its complications from an imaging perspective. ESR indicates erythrocyte sedimentation rate; NSAIDs, nonsteroidal antiinflammatory drugs; TTE, transthoracic echocardiogram; and TEE, transesophageal echocardiogram.

Figure 3. Classic anatomic findings of pericardial constriction by CMR and CT. CMR findings of constriction demonstrated in black blood T2-weighed spin-echo (A) and steady-state free precession sequence cine sequence (B) in the horizontal long-axis view. Pericardial thickening (6 mm) is seen adjacent to the right atrium and ventricle (arrow), with characteristic tubular deformity of the right and left ventricles (RV/LV) and dilation of the right atrium. Other contributory findings by CMR (not illustrated here) are tethering of the underlying myocardium to the pericardium (demonstrated on tagged gradient echo cine sequences), a diastolic septal bounce, and subjective abrupt cessation of diastolic filling on steady-state free precession sequence cine images. C, Cardiac CT demonstrating the presence of circumferential pericardial calcification on a multiplanar reconstructed short-axis image (arrows). D, Three-dimensional volume-rendered imaging showing the extent and anatomic distribution of pericardial calcium.
When to Consider Added Imaging?

Once the presence of cardiac tamponade has been established by echocardiography, there is basically no need for confirmatory testing by CT or CMR as any delay in appropriate treatment could be harmful to the patient. Occasionally TTE is inconclusive, in our experience particularly during the early postoperative period when the acoustic windows can be very poor. However, in these situations, transesophageal echocardiography (TEE) will almost always allow exclusion of a postoperative loculated pericardial effusion or intrapericardial clot. Occasionally, a significant effusion is initially detected first on postoperative CT or CMR. In this case, the multimodality imaging specialist must be able to recognize signs of imminent tamponade, which are essentially similar to those outlined above.

Constrictive Pericarditis

Clinical Scenario

A 44-year-old man presents with increasing shortness of breath since the last 8 months. More recently he also noticed worsening leg edema and abdominal girth. His medical history is insignificant except for mild asthma, and he has no risk factors for coronary artery disease. An echocardiogram shows normal left ventricular systolic function (ejection fraction, 60%) but restrictive diastolic filling. Mild biatrial enlargement and a plethoric inferior vena cava are also seen. An abnormal septal bounce is noted (supplemental Movie Clip 3), but in the absence of significant respiratory variation of mitral or tricuspid inflow, further evidence is deemed necessary to confidently make the diagnosis of constrictive pericarditis. A cardiac CT is then ordered, excluding obstructive coronary artery disease and additionally demonstrating extensive circumferential pericardial calcification (Figure 3C and 3D). The patient is subsequently referred for surgical pericardiectomy.

Chronic inflammation can occasionally result in fibrous thickening and calcification of the pericardium and lead to a condition where ventricular filling is severely impaired due to loss of pericardial compliance. The etiology of constrictive pericarditis is most commonly idiopathic; however, it can be associated with prior cardiac surgery, mediastinal radiation, or scarring related to tuberculous pericarditis. Although constriction occasionally develops acutely (within days) or subacutely (3 to 12 months)—mainly after cardiac surgery—a long delay between the initial insult and the onset of constriction is the more common scenario.

The diagnosis of constriction remains challenging and although clinical clues (related to the presence of unexplained systemic venous congestion) are important in considering the disease, further confirmation by other tests is always necessary. No single diagnostic approach can or should be used to diagnose all cases of constrictive pericarditis, and clinicians therefore must tailor their diagnostic strategy according to each patient. The algorithm shown in Figure 4 suggests an investigative strategy in case of clinical suspicion for constrictive pericarditis and will subsequently be discussed in more detail.

An important reason to use echocardiography early in the diagnostic process is to rule out other more common causes of right-sided heart failure, including left or right ventricular systolic dysfunction, severe pulmonary hypertension, or unrecognized left-sided valvular disease. Encasement of the heart by a stiff pericardium leads to an abrupt termination of ventricular diastolic filling due to pericardial constraint,

Figure 4. Suggested diagnostic approach for the evaluation of patients with constrictive pericarditis. LV indicates left ventricular; RV, right ventricular; TEE, transesophageal echocardiogram; and TTE, transthoracic echocardiogram.
isolates the heart from respiratory changes in intrathoracic pressure, and causes exaggerated ventricular interdependence. These pathophysiologic abnormalities are represented in typical 2D and Doppler findings (Figure 5), and their recognition is pivotal not only to make a correct diagnosis but also to differentiate constrictive pericarditis from restrictive cardiomyopathy as both conditions share many clinical features. A precise measurement of pericardial thickness may be difficult (particularly by TTE), but an early diastolic “septal bounce,” a respiratory shift in the position of the interventricular septum, inferior vena cava plethora, and the presence of myocardial tethering are all classic 2D features associated with pericardial constriction (none of them particularly sensitive or specific, however). Doppler interrogation of transmitral flow velocity shows early rapid restrictive filling, but the difference with restrictive cardiomyopathy is often made by demonstrating reciprocal respiratory changes between transmitral and transthruscipid flow (or between pulmonary vein and hepatic vein flow) using simultaneous respirometry in constriction. Color M-mode imaging (measuring early diastolic flow propagation velocity, Vp) and tissue Doppler (measuring the early diastolic mitral annular velocity E [Figure 5D]) allow further differentiation from restrictive cardiomyopathy by documenting preserved myocardial relaxation in constrictive pericarditis (Vp >45 cm/s or E’ >8 cm/s). These methods are especially useful when no substantial respiratory variation of Doppler recordings is observed; for instance, in patients with atrial fibrillation having irregular R-R intervals or in patients with markedly elevated filling pressures when respiration has only little effect on the transmitral driving pressure, even with head-up tilt.
When to Consider Added Imaging?

Remaining Diagnostic Uncertainty
When the picture is complete, comprehensive echocardiography may provide conclusive evidence of constrictive pericarditis. Unfortunately, echocardiographic findings are not uncommonly equivocal in patients with a possible diagnosis of pericardial constriction. In these situations additional testing is needed to make the diagnosis with more confidence.

CT Versus MRI
Both CT and CMR provide excellent anatomic delineation of the pericardium from adjacent tissue and enable a precise measurement of pericardial thickness. A thickened pericardium (>4 mm) in the proper clinical setting certainly adds further support to the diagnosis and is a valuable piece of information if the echocardiography was inconclusive (although absence of pericardial thickening does not necessarily rule out constrictive pericarditis).22 Other important features are abnormalities in the contour of the pericardium; conical deformity (“tubing”) of the ventricles; right atrial (sometimes bialtrial) enlargement; and inferior vena cava plethora. Compared with CMR, CT is far superior in detecting calcification (Figure 3C), but calcification can occur without constriction and vice versa. CMR, on the other hand, better differentiates small effusions from pericardial thickening; has a better temporal resolution (enabling detection of rapid hemodynamic processes such as a septal bounce or respirophasic variation in septal excision (Figure 5A and supplemental Movie Clip 4); and may reveal ongoing pericardial inflammation. CMR myocardial tagging sequences can demonstrate pericardial-myocardial adherence, whereas abrupt cessation of diastolic filling can be visually appreciated on steady-state free precession sequence cine images as another (subjective) sign of a stiff pericardium. Phase encoding velocimetry provides information similar to Doppler echocardiography, although flow curves are averaged over several cardiac cycles precluding assessment of respirophasic variation. For these reasons, CMR is sometimes preferred over CT for patients with a high clinical suspicion of constriction and a nondiagnostic echo.

Multimodality Imaging Versus Catheterization
As illustrated in the case scenario above, cardiac catheterization can sometimes be avoided by the rational use of multimodality imaging. Nevertheless, despite the availability of CT and CMR, constrictive pericarditis continues to be a diagnostic challenge, particularly in patients presenting after prior radiation therapy or open heart surgery who may have combined pericardial, myocardial, and valvular disease. As recently shown by Talreja et al,23 in this difficult subgroup of patients, catheterization may offer valuable diagnostic information by showing dynamic respiratory changes of the ventricular pressure curves reflecting enhanced ventricular interaction. Ultimately, cardiologists need to make a synthesis of the available (and occasionally discrepant) pieces of information to make a clinical judgment whether or not a patient is likely to benefit from pericardiectomy.

Preoperative Planning in Patients With a Definite Diagnosis of Constrictive Pericarditis
Even if a diagnosis of constrictive pericarditis has unequivocally been established by routine testing, cardiac CT can assist in preoperative planning in the following situations:

1. Patients with history of prior cardiothoracic surgery: Sternal reentry and dissection of mediastinal structures can be a major challenge due to the formation of dense adhesions. The proximity of bypass grafts, the ascending aorta, innominate vein, and right ventricle to the back of the sternum pose an additional risk factor for catastrophic hemorrhaging during re sternotomy. These high-risk findings are readily detected by preoperative CT and allow the surgeon to adopt preventive
surgical strategies and thus potentially improve outcomes in these patients.\textsuperscript{24}

(2) Radiation-induced constrictive pericarditis: CT will also determine the extent of associated lung injury due to radiation.

(3) Preoperative evaluation of the localization and extent of pericardial calcification (Figure 5C and 5D): The presence of circumferential or severe posterolateral calcification may push the surgical approach toward a bilateral thoracotomy. Particularly in patients with circumferential constriction, the left ventricular pericardium should be removed first to prevent provocation of sudden pulmonary edema.

(4) The occasional patient with constrictive pericarditis and a low-to-intermediate probability of underlying coronary artery disease, to obviate the need for preoperative coronary angiography.

**Effusive Constrictive Pericarditis**

**Clinical Scenario**
A patient presents with symptoms of right-sided heart failure shortly after an episode of acute pericarditis that was complicated by a large pericardial effusion requiring pericardiocentesis. A postprocedure echocardiogram shows a normal right ventricular systolic function, but now there is a septal bounce and increased respiratory variation of the transmural (30\%) and tricuspid Doppler flow velocity (50\%), suggesting presence of constrictive physiology. Subsequently, CMR is performed, which confirms inflammatory thickening of the pericardial layers (Figure 6A). Based on these findings, antiinflammatory therapy is initiated with gradual improvement of the patient’s symptoms.

Effusive constrictive pericarditis is a relatively uncommon pericardial syndrome, characterized by impaired diastolic filling due to concomitant tamponade caused by a tense effusion and constriction by the visceral pericardium.\textsuperscript{25,26} Constrictive hemodynamics typically persist in these patients after the pericardial fluid has been removed or resolved. Based on the results of a consecutive series reported by Sagrista-Sauleda,\textsuperscript{27} effusive constrictive pericarditis may have a prevalence close to 8\% in patients presenting with cardiac tamponade. Most commonly associated with idiopathic pericarditis, effusive constrictive pericarditis can also be caused by malignancies or radiation.

Effusive constrictive pericarditis is difficult to diagnose by echocardiography at the time of presentation with tamponade. The diagnosis is therefore traditionally made by cardiac catheterization (either after but ideally at the time of pericardiocentesis). Failure of the right atrial pressure to fall \( \geq 50\% \) after the intrapericardial pressure has been lowered to near 0 mm Hg (in other words, an increase in right atrial transmural pressure after pericardiocentesis) and the finding of a diastolic ventricular dip and plateau morphology have been considered diagnostic hemodynamic criteria.\textsuperscript{26}

**When to Consider Added Imaging?**
Catheterization at the time of pericardiocentesis is not always feasible. The diagnosis of effusive constrictive pericarditis is therefore increasingly being made by noninvasive imaging techniques. Echocardiography performed after pericardiocentesis will not only confirm effective removal of pericardial fluid but will also enable screening for the presence of residual constrictive physiology. However, the presence of
abnormal pericardial thickening or inflammation on the other hand is best demonstrated by CT or CMR (Figure 6). Similar to transient constrictive pericarditis, patients with idiopathic effusive constrictive pericarditis may experience gradual improvement of their symptomatic process (either spontaneously or after treatment with antiinflammatory agents). By documenting resolution of the inflammatory process, CMR may potentially provide important prognostic information with respect to the need for future pericardiectomy. However, this remains to be tested in subsequent studies.

**Pericardial Cysts**

Pericardial cysts are encapsulated structures most commonly located in the right anterior cardiophrenic angle, although they can be found throughout the mediastinum. Generally congenital in origin, pericardial cysts are also occasionally seen after previous cardiac surgery. These cystic structures are usually asymptomatic unless they cause compression of adjacent structures.

*When to Consider Added Imaging?*

Pericardial cysts are commonly detected first by plain chest radiography or echocardiography, but additional imaging by CT or CMR is often helpful to obtain a better idea of their exact size, location, and relation with the surrounding anatomy, particularly when a decision needs to be made whether to intervene on these lesions.

Pericardial cysts typically appear as thin-walled, unilocular nonenhancing structures, with near water attenuation values by CT (Figure 7). On CMR, they usually demonstrate a low signal intensity on T1-weighted images (unless the content of the cyst is proteinaceous) and a high signal intensity on T2-weighted STIR images (Figure 7). Pericardial cysts are generally benign, although it is important to recognize possible associated soft-tissue components that can be related to a malignant process.

**Pericardial Tumors**

Neoplastic involvement of the pericardium in patients with an established malignancy can occur by hematogenous or lymphatic spread but also by direct invasion from the lung or mediastinum. Although commonly detected during autopsy, pericardial metastatic disease frequently goes unnoticed until late in the disease process when patients may present with symptoms related to pericarditis or even tamponade. The most common causes of metastatic pericardial disease are breast and lung cancers, followed by renal cell carcinomas, lymphomas, and melanomas.

Primary pericardial tumors are much more uncommon, with mesothelioma (usually presenting as a pericardial effusion accompanied by pericardial nodules or plaques) and sarcomas (appearing as heterogeneous masses often associated with a serosanguineous effusion) being the most frequent. Benign tumors include fibromas, lipomas, hemangiomas, and benign teratomas.

*When to Consider Added Imaging?*

Although echocardiography may be appropriate to screen for malignant involvement of the pericardium or to monitor serial changes in the size of a malignant effusion, CT or CMR is generally necessary to accurately delineate the tumor implantation and to better evaluate the extent of tumor spread (Figure 8). Disruption of the pericardial lining, presence of an associated hemorrhagic effusion, and invasion of the tumor into the epicardial fat tissue, myocardium, or into a cardiac chamber (rather than causing displacement of these structures) are characteristics of a lesion with an aggressive nature. Associated lymphadenopathy is another important finding suggesting malignancy.
Tissue characterization with cardiac CMR is superior to cardiac CT and echocardiography. CMR can differentiate tumor from thrombus; characterize fatty tumors such as lipomas or liposarcomas (high signal intensity both on T1- and T2-weighted images with a characteristic signal reduction by fat suppressed inversion recovery sequences); and is often helpful to assess the perfusion of a pericardial mass with the use of gadolinium contrast. Sites of malignant disease usually demonstrate high signal intensity on T2-weighted STIR imaging and late gadolinium enhancement images (Figure 8).

Conclusion

Clinicians increasingly rely on cardiac imaging in the diagnostic workup of patients with pericardial disease. Continuous advances in cardiac CT and CMR technology allow for an excellent visualization and characterization of pericardial pathology, making these tomographic techniques complimentary to echocardiography. An integrated multimodality imaging strategy is sometimes needed to answer specific clinical questions, but the rational use of such an approach also requires good knowledge of the strengths and limitations of each technique. Given the paucity of evidence-based guidelines, more clinical studies are needed to better define the role of cardiac imaging in the management of patients with pericardial disease.

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Disclosures

None.

References


Key Words: pericardial disease ■ echocardiography ■ CT ■ CMR ■ multimodality imaging