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ECHOCARDIOGRAPHY (JM GARDIN AND AH WALLER, SECTION EDITORS)

Multimodality Imaging for the Assessment of Pericardial Diseases

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Abstract

Purpose of Review The diagnosis of pericardial syndromes, while occasionally straightforward, includes a wide range of pathologies and is often a well-recognized clinical challenge. The aim of this review is to highlight the key role of the various imaging modalities for the diagnosis and management of the spectrum of pericardial diseases.

Recent Findings Cardiac imaging has become an integral part of the diagnostic process often beginning with echocardiography and supported by advanced imaging modalities including computed tomography, magnetic resonance imaging, and positive emission tomography. These modalities go beyond the simple identification of the pericardium, to identifying increased pericardial thickness, active pericardial edema and inflammation, and its effect on cardiac hemodynamics.

Summary Multimodality imaging has significantly facilitated the diagnosis and long-term management of patients with pericardial diseases. The role of these imaging modalities in overall prognosis and prevention remains to be investigated.

Keywords Acute pericarditis · Constrictive pericarditis · Tamponade · Effusive constrictive · Transient constrictive · Pericardial effusion

Introduction

Diseases of the pericardium are frequently encountered in clinical practice and include a wide range of pathologies from acute inflammation to constrictive pericarditis [1]. The diagnosis of pericardial syndromes, while occasionally straightforward, is a well-recognized clinical challenge. Moreover, diseases of the pericardium have been associated with significant morbidity and mortality, re-enforcing the need for an accurate diagnosis [2]. An integrated multimodality imaging approach has been proposed and emerging as the preferred strategy when faced with pericardial pathology. The aim of this article is to review the key role of the various imaging modalities for the diagnosis and management of pericardial diseases.

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The Normal Pericardium

The pericardium, composed of an outer fibrous sac and an inner serous sac, surrounds the heart up to the proximal portions of the great vessels. The inner serous sac has two distinct layers: a visceral layer adjacent to the epicardium and a parietal layer adjacent to fibrous pericardium. The potential space between these two inner layers normally contains less than 50 ml of serous fluid [3, 4]. The normal thickness of the pericardium described on autopsy is 0.5–1 mm; however, the pericardium appears thicker on imaging. In fact, a normal pericardium has been measured up to 4 mm in both CT and MRI largely due to cardiac motion and volume averaging [5]. Only in the presence of significant thickening of the pericardium (≥ 5 mm) does it become visible by surface echocardiography. Transesophageal echocardiogram, on the other hand, provides more reliable measurements when compared to CT imaging ($r = 0.95$). A normal pericardium was found to be 1.2 mm ± 0.8 mm thick and rarely exceeded 2.5 mm [6].

Gated computed tomography offers higher spatial resolution and more accurate delineation of the pericardium particularly when surrounded by fat. Cardiac magnetic resonance also utilizes the contrast from adjacent adipose tissue. The pericardium is easily visualized as a curvilinear line of low signal intensity between the myocardium and the high-signal-intensity pericardial fat when using black blood spin echo and gradient echo sequences. A normal thickness on CMR is typically less than 2 mm; however, the upper limit is not clearly defined [7].

Acute Pericarditis (Echo, CT, and an MRI)

Acute pericarditis is the most common disease of the pericardium and, when treated with appropriate anti-inflammatory medications, resolves within days to weeks. In the Western world, the most common form is idiopathic pericarditis. In a small percentage of cases, specific etiologies can be identified such as infectious, autoimmune, neoplastic, and iatrogenic [8••, 9, 10]. Acute pericarditis often presents with pleuritic chest pain (85–90% of cases), a pericardial friction rub (less than 33%), diffuse ST elevations and PR depressions on electrocardiogram (60% of cases), and a pericardial effusion (60%). The presence of two or more of these features is required for the diagnosis of acute pericarditis. The diagnosis can be further supported with the presence of elevated inflammatory markers, an elevated white blood cell count, or elevated troponin [11–13]. Cardiac imaging plays a key role if the diagnosis remains unclear or a definitive diagnosis is required.

It is currently suggested that all patients with acute pericarditis undergo an echocardiogram within 24 h of presentation [14] (Table 1). While the most common finding is a normal study, the presence of a pericardial effusion can aid in the diagnosis of pericarditis. Echocardiography also allows for a rapid risk stratification by assessing for the presence of a large effusion and/or tamponade physiology and constrictive physiology as well as regional anomalies of the ventricles suggestive of myocardial involvement [15, 16] (Fig. 1a). In the presence of poor prognostic features such as fever, suspicion of constriction, chest trauma, failure to respond to therapy, or an inconclusive echocardiographic study, further imaging should be considered [5].

CT is of particular utility in these circumstances. A non-calcified thickened smooth pericardium with an associated pericardial effusion and iodinated contrast enhancement of the visceral and parietal surfaces is suggestive of acute inflammation of the pericardium. The presence of irregular pericardial contours signifies a more subacute or chronic inflammatory process [17]. Similarly, with CMR imaging, the presence of a thickened pericardium and a pericardial effusion on the T1-weighted black blood sequence is suggestive of acute pericarditis. Moreover, the signal intensity on spin-echo images is inversely related to chronicity of the pericardial inflammation [2, 7]. An increased signal on T2 weighted short tau inversion recovery sequences (STIR) suggests ongoing edema which confirms the acuity [18]. The presence of late gadolinium enhancement (LGE), representing abnormal vascular permeability and fibroblast proliferation, indicates active inflammation of the pericardium with a reported sensitivity of 94–100% [19–21]. It has also been found to correlate with neovascularization on histology (Fig. 1b, c). Lastly, dynamic CMR tagging of the myocardium can further help in assessing pericardial adhesions often seen with inflammation [22]. CMR, like echocardiography can aid in excluding other diagnoses such as acute myocardial infarction, myocarditis, or myopericarditis.

The treatment of acute pericarditis includes the use of non-steroidal anti-inflammatory drugs (NSAIDs) or corticosteroids. Colchicine reduces the rate of recurrence after both the initial event and any recurrence and is now a mainstay of therapy. Immunosuppressive agents, anakinra, and pericardiectomy are often reserved for frequently recurring disease [23, 24]. Imaging has emerged as a tool to direct therapy particularly when tapering or stopping anti-inflammatories. The decision to taper anti-inflammatories is commonly based on biochemical and clinical resolution of inflammation; however, in case of doubt, CMR can inform the clinician if the pericardium is still actively inflamed and can help with the speed of tapering and the number of anti-inflammatory drugs [1] (Fig. 1d–f).

Chronic and Recurrent Pericarditis

After a first episode of acute pericarditis, the probability of developing a first recurrence within 18 months is estimated to range between 15 and 30%. After an initial recurrence of pericarditis, additional recurrences occur in 25% to 50% of patients [11, 25]. Echocardiography remains the initial imaging modality in recurrent or chronic pericarditis (Table 1). The findings are also similar to those in acute pericarditis; however, special attention to constrictive physiology is often warranted. These would include respiro-phasic motion of the interventricular septum or the presence of a septal bounce [26]. Both CT and MRI can confirm the presence of an inflamed pericardium with contrast enhancement when the diagnosis is in question. CMR also has a good negative predictive value when assessing pericardial thickness and inflammation and has clinical utility when following pericardial inflammation longitudinally to guide therapy [26, 27].

Pericardial Effusion

The pericardial sac contains between 10 and 50 ml of plasma ultra-filtrate that acts as a lubricant between the pericardial layers. Various conditions including acute pericarditis and hypothyroidism result in an accumulation of this fluid.

Table 1 Various modalities for imaging the pericardium and current recommendations

Imaging modality	Advantages	Disadvantages	Most notable utility	Recommendations								
				Society	Acute pericarditis	Recurrent pericarditis	Pericardial effusion	Tamponade	Effusive constrictive	Constrictive pericarditis	Pericardial masses	Congenital
Echocardiography	• Safe • Rapid acquisition • Low cost • Portable/available in hemodynamically unstable patients	• Operator dependent • Limited windows • Patient-specific technical difficulties such as obesity, mechanical ventilation, COPD • Limited tissue characterization	• Detection of effusion • Guiding pericardiocentesis • Assessing diastolic function • Assessing systolic function • Respiratory changes	*ASE* *ESC*	1st line *Recommended* Diagnosis and follow-up	1st line *Recommended* Diagnosis and follow-up	1st line *Recommended* Diagnosis and follow-up	1st line *Recommended* Diagnosis and pericardiocentesis	1st line *Recommended* Diagnosis and follow-up	1st line *Recommended* Diagnosis and follow-up	1st line *Recommended* Diagnosis	1st line *Recommended* Diagnosis
Cardiac CT	• Spatial resolution • Superior anatomic imaging • Calcium detection • Adjacent structures • Fluid characterization	• Ionizing radiation • Iodinated contrast often needed • Functional images require more time/radiation • Technical difficulties with rapid/irregular heart rate • Hemodynamically stable patients only • Need breath hold	• Assessing pericardial thickness • Assessing pericardial calcium • Effusion detection	*ASE* *ESC*	2nd line *Reasonable* for complicated course*	2nd line *Reasonable* for diagnosis	2nd line *Reasonable* for diagnosis	Not recommended *Reasonable* if suspected aortic dissection or trauma	2nd line *Reasonable* for diagnosis	2nd line 1st line for preoperative planning *Reasonable* for diagnosis and operative planning	2nd line *Recommended* for diagnosis	2nd line *Reasonable* for diagnosis CT Not recommended for follow-up
Cardiac MRI	• Safe • Temporal and spatial resolution • Excellent anatomic imaging • Evaluation of adjacent structure • Tissue/fluid characterization	• Time consuming and costly • Contrast agent (CI in renal failure) • Technical difficulties with rapid/irregular heart rate • CI with some pacemaker/defibrillators (epicardial leads, fractured leads, abandoned leads) • Calcification not well visualized • Hemodynamically stable patients only • Need breath hold	• Assessing pericardial thickness • Assessing pericardial inflammation • Effusion detection • Pericardial masses • Tissue characterization • Systolic function • Respiratory changes	*ASE* *ESC*	2nd Line *Reasonable* for complicated course* Recommended if suspicion of myocarditis	2nd Line *Reasonable* for diagnosis	2nd Line *Reasonable* for diagnosis	Not Recommended *Reasonable* if suspected aortic dissection or trauma Reasonable for follow-up	2nd Line *Reasonable* for diagnosis and follow-up	2nd Line *Reasonable* for diagnosis and reasonable for follow-up	2nd Line *Recommended* diagnosis	2nd Line *Reasonable* for diagnosis

*High fever (greater than 38 °C), subacute course, failure to respond to therapy, immunosuppression

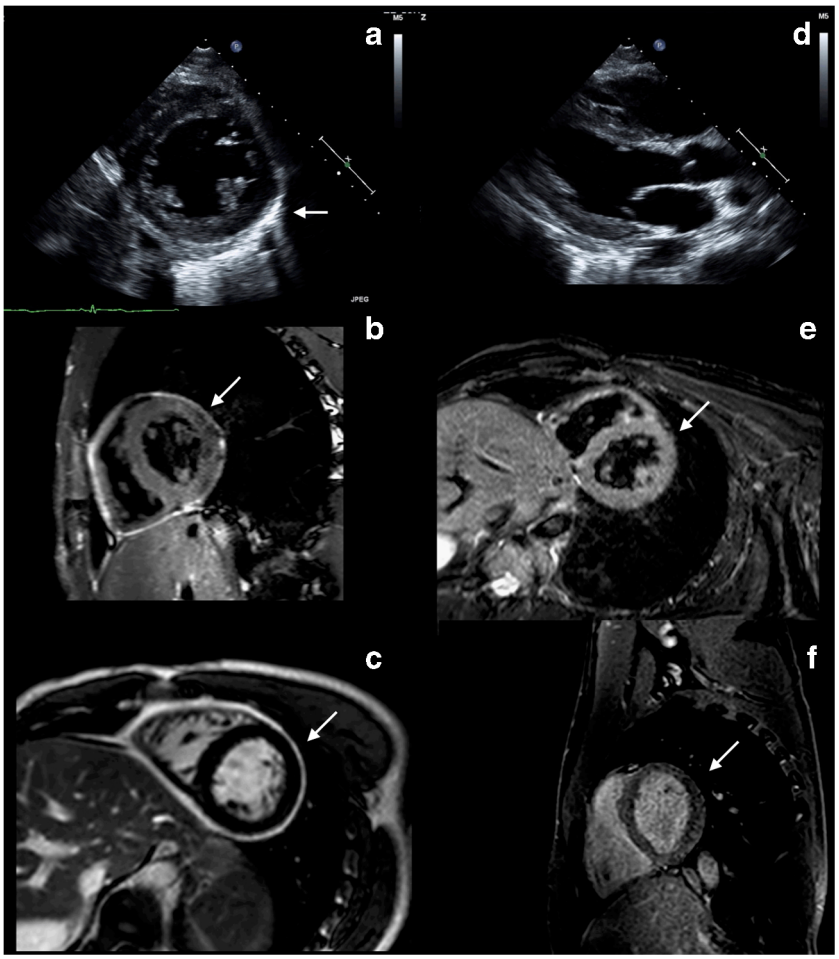

Fig. 1 28-year-old gentleman with clinical history suggestive of idiopathic/viral pericarditis. **a** Parasternal short-axis view of the heart on transthoracic echocardiogram without effusion and a hyperechoic posterior pericardium (*arrow*). **b** Short-axis view of the left ventricle on T2 sequences with increased signal intensity of the pericardium (*arrow)* demonstrating edema of the pericardium. **c** Short-axis view on late enhancement sequences with increased signal intensity from pericardium (*arrow*) signifying ongoing inflammation of the pericardium. Post anti-inflammatory treatment with NSAIDs and colchicine. **d** Parasternal long-axis view of the heart on transthoracic echocardiogram with no effusion or enhancement of the posterior wall. **e** Short-axis view on T2 sequences with isointense signal from pericardium (*arrow*) demonstrating resolution of edema of the pericardium after treatment. **f** Absence of enhancement of the pericardium (*arrow*) on late gadolinium images signifying resolution of the inflammation

Moreover, depending on the underlying etiology, analysis of the pericardial fluid can reveal it to be a transudative effusion, an exudative effusion, hemopericardium, chylopericardium, or pyopericardium. It is recommended that cardiac imaging be pursued when there is suspicion of an effusion to make a definitive diagnosis [16].

Echo remains the initial test of choice when diagnosing and assessing a pericardial effusion [28] (Table 1). Pericardial effusions appear as an echo-free space between the two layers of the pericardium on M-mode or 2D echocardiography. In the parasternal long-axis views of the heart, fluid anterior to the aorta is pericardial in nature while fluid posterior to the aorta is pleural in nature. Echo can semi-quantify the amount of fluid by measuring the separation of the two layers of the pericardium in end diastole. When fluid is only seen in systole, it is considered trivial in size. Less than 1 cm, labeled a small effusion, corresponds to 50–300 ml. One to two centimeters of pericardial fluid corresponds to 500 cc (moderate in size) while more than 2 cm (large) generally exceeds 700 cc, the extreme of which would be a swinging heart [29, 30]. Qualitative assessment of the effusion is more difficult. Echogenic densities, fibrin stands, septations, and adhesions suggest an exudative effusion while the presence of a thrombus suggests a hemopericardium [16] (Fig. 2a).

CT and MRI have the added advantage of not only diagnosing a pericardial effusion but also accurately localizing and characterizing the fluid. This is particularly useful when there is suspicion of a loculated effusion, pericardial mass, or a thrombus which is often incompletely evaluated by transthoracic echocardiography [31]. Aside from offering ancillary data such as mediastinal adenopathy or presence of infection, cardiac gated CT can characterize the effusion by measuring its level of attenuation. Transudative effusions have a CT attenuation similar to that of water, less than 10 Hounsfield units (Hu) [1, 32, 33]. Attenuation above 10 Hu suggests an exudate with high protein content. More specifically, CT attenuations ranging from 20 to 60 Hu suggest a purulent, malignant, or myxedematous exudative effusion and attenuation greater than 60 Hu is best correlated with a hemorrhagic effusion [17, 34]. CMR, similar to CT, can quantify, localize, and characterize the fluid. Quantitative evaluation of the effusion can be determined using multi-slice volumetric quantification similar to chamber volumes [7]. In addition, fat-suppression sequences can be utilized to delineate epicardial fat confirming

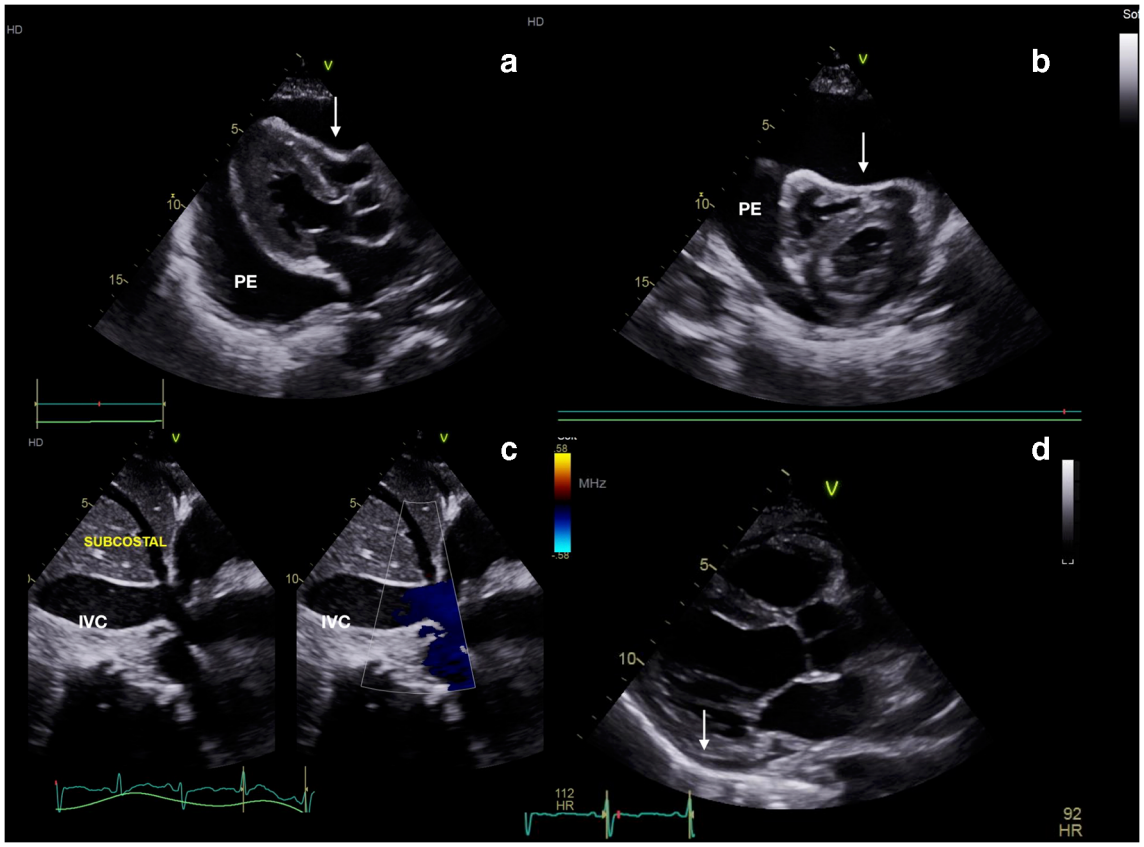

Fig. 2 **a** Apical four-chamber view on 2D echocardiography with large circumferential pericardial effusion (PE) and right ventricular chamber collapse (*arrow*). **b** Parasternal short-axis view on 2D echocardiogram with right ventricular chamber collapse (*arrow*). **c** Plethoric IVC seen on subxyphoid views. **d** Near resolution of the pericardial fluid after therapeutic pericardiocentesis and residual echo bright posterior pericardium (*arrow*)

the presence of the effusion. Qualitative assessment is achieved when comparing T1-weighed spin-echo images with T2 spin-echo CMR images. Transudative effusions are dark in the T1-weighted sequences and bright in T2-weighted sequences. In contrast, exudative effusions vary depending on their composition. Proteinaceous effusions will be bright in T1-weighted sequences and dark in T2-weighted sequences. An exudative effusion will be medium intensity in T1 and medium to bright in T2-weighted sequences. Hemorrhagic effusions will be bright in both sequences, but can evolve to a chronic hematoma with low signal intensity and dark foci signifying hemosiderin deposition surrounded by a dark peripheral rim [2]. Lastly, CT and MRI can offer guidance when a pericardiocentesis is entertained as a therapeutic option to ensure feasibility and determine the safest trajectory when not feasible using an echo-guided approach.

Cardiac Tamponade

Tamponade is a life-threatening condition characterized by fluid accumulation in the pericardial sac which compresses the cardiac chambers and inhibits diastolic filling. Systemic and venous pressures rise in order to maintain cardiac output. When this compensatory response fails, preload is impaired and cardiac output drops [1]. While cardiac tamponade is a clinical diagnosis, transthoracic echocardiogram is the initial modality of choice to corroborate clinical findings (Table 1). The most notable findings include cavitary collapse of the right atrium and right ventricle in early systole and early diastole respectively, hepatic vein systolic flow reversal on expiration, and a plethoric inferior vena cava (IVC) defined as an IVC diameter greater than 2.1 cm and associated with less than 50% reduction in its diameter with inspiration [3, 35] (Fig. 2b, c). With a fixed intrapericardial volume and increased pressures, one side of the heart fills at the expense of the other causing a characteristic respiro-phasic septal shift as well as mitral and tricuspid respiratory inflow variation that can be quantified [36]. Various percentages of variation have been suggested; however, a mitral inflow variation greater than 30% and a tricuspid inflow variation greater than 60% seem to correlate best with tamponade physiology [37, 38]. Once the diagnosis of cardiac tamponade is established both clinically and supported with echocardiography, there is no role for additional imaging [1]. Transesophageal

echocardiography (TEE) plays a more central role in the post-operative setting when trying to identify intrapericardial clots or loculated effusions causing localized tamponade [39].

The limited role of cardiac gated CT is in the presence of an acute aortic dissection complicated by hemopericardium, or tamponade from a loculated effusion unidentifiable by echocardiography. Indirect findings of cardiac tamponade on cardiac CT include a "flattened heart" from compression as well as a dilated SVC (superior vena cava) with a diameter similar to the adjacent thoracic aorta dimensions) and dilated IVC (with dimensions twice that of the adjacent descending aorta). Evidence of increased right heart pressures as suggested by reflux of contrast material into the IVC and azygous vein, and dilated renal and hepatic veins, are also an indirect sign [40, 41]. Real-time gated cardiac CT and CMR are also able to demonstrate the same respiratory variations seen by echo including the septal shift and chamber collapse; however, this is rarely, if ever, clinically indicated [2].

Constrictive Pericarditis (Echo, CT, and MRI)

Chronic inflammation of the pericardium can lead to a thick non-compliant, fibrotic, and/or calcified pericardium which limits the diastolic filling of the ventricles. The restrictive nature of the pericardium causes a rapid early diastolic filling followed by an abrupt stop. As this disease progresses, the systemic venous pressures continue to rise to systemic venous congestion and symptoms of right heart failure ensue. Moreover, the constricting pericardium insulates the heart from the respiratory intrathoracic pressure changes resulting in less atrial filling and less trans-mitral inflow on inspiration. Similar to tamponade physiology, with a fixed combined volume of the right and left ventricles and reduced left-sided filling during inspiration, the septum shifts towards the left ventricle. This phenomenon, known as ventricular interdependence, is a characteristic finding in constrictive pericarditis [42, 43]. While the most common cause is idiopathic, constrictive physiology has been described post cardiac surgery, post radiation therapy, or pericardial calcifications resulting from tuberculosis [44]. The diagnosis of constrictive pericarditis is often suggested during a transthoracic echocardiogram; however, a more definitive diagnosis is often made with additional imaging such as CT or CMR. Ancillary imaging can also evaluate the presence of pericardial inflammation and myocardial involvement as well as assess the cardiovascular anatomy in view of a future intervention [45].

Echo remains the initial imaging modality of choice when suspecting constrictive physiology [46] (Table 1). 2D echocardiography can identify a thickened or even calcified pericardium and bi-atrial enlargement, suggesting increased filling pressures from impaired filling. Ventricular interdependence can be objectified using various indices including the presence of a septal bounce, respiro-phasic shifting of the septum, and respiratory variation of the mitral/tricuspid inflows upon Doppler interrogation. Also, characteristics of constrictive physiology are the increased medial mitral annular velocities ("annulus reversus") and the reversed linear relation of the E/e' ratio to LA pressure ("annulus paradoxus") [36, 47] (Fig. 3a, b). Finally, increased venous congestion leads to a plethoric IVC and hepatic vein flow reversal with expiration [46, 48, 49] (Fig. 3c). One of the newer techniques, myocardial strain analysis, shows reduced circumferential strain, reduced early diastolic twisting, and LV torsion while global longitudinal strain is often preserved [50]. When interrogating regional strain, however, Kusunose et al. were able to demonstrate decreased LV anterolateral wall and right ventricular free wall strain with preserved septal strain ("strain reversus") best explained by myocardial tethering which is a common complication of constrictive pericarditis (CP) [45].

Cardiac-gated CT (CCT) is the most accurate method for evaluating pericardial thickness which has been previously described to vary between 4 and 20 mm in the presence of inflammation or constrictive physiology [3]. CCT is also the most accurate method in evaluating the presence of calcium (seen in 50% of cases) including the degree and extension [51] (Fig. 3d). Due to the constrictive nature of the pericardium, there is often a tubular deformation of the ventricles associated with bi-atrial enlargement along with straightening of the interventricular septum. Dilatation of the IVC and hepatic veins along with the presence of hepato-splenomegaly, ascites, and pleural effusions suggests impaired atrial filling [3]. The aforementioned findings in the presence of a thickened pericardium are practically diagnostic of CP [52]. Moreover, failure of the adjacent regional pulmonary structure to pulsate during the cardiac cycle in the presence of a thickened pericardium strongly suggests CP [16]. Lastly, CT also has the added advantage of offering additional information to a surgeon preparing for a pericardiectomy. Identifying features associated with a high risk of bleeding such as proximity of bypass grafts, the location of both the ascending aorta and innominate arteries, and the position of the right ventricle in relation to the back of the sternum are of particular importance [1].

To some, CMR is the diagnostic modality of choice for constrictive pericarditis [53]. Much like cardiac-gated CT, CMR can assess the thickness of the pericardium, the presence of a pericardial effusion and demonstrate the tubular shape deformation of the RV due to the constricting pericardium (Fig. 3e) [54]. CMR's increased temporal resolution permits a proper evaluation of cardiac function and hemodynamics. Abrupt cessation of diastolic filling of the ventricles is readily seen on steady-state free precession images, and an abnormal diastolic septal motion can be identified with real-time free breathing sequences (sensitivity 81%; specificity 100%) [55, 56]. Real-time phase-contrast sequences acquired over 10 s during unrestricted breathing can identify a respiratory

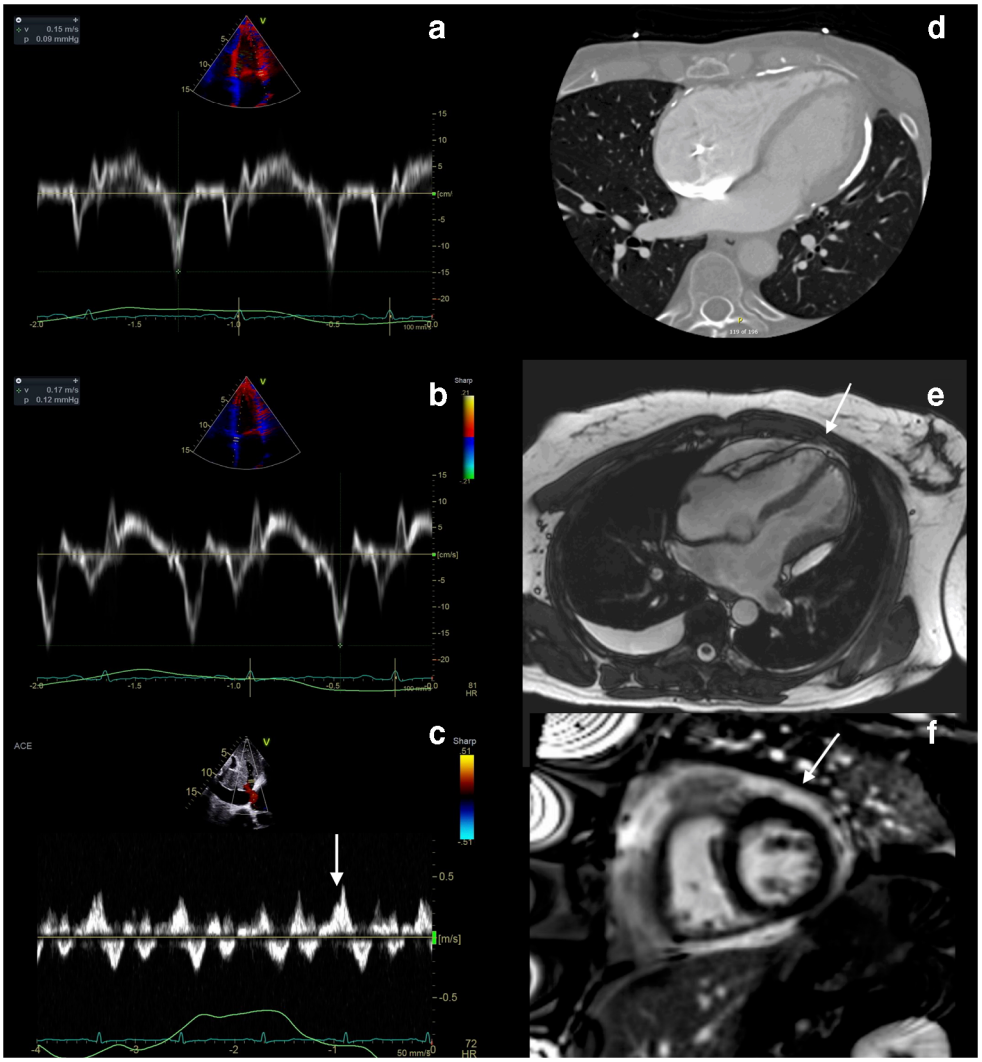

Fig. 3 52-year-old woman with calcific constrictive pericarditis. **a** Tissue Doppler velocity of the lateral mitral annulus and **b** tissue Doppler velocity of the medial mitral annulus demonstrating *annulus reversus*. **c** Pulsed wave Doppler sampling of the hepatic veins demonstrating expiratory end-diastolic flow reversal on expiration. **d** Coronary CT with pericardial calcifications along the anterolateral wall of the left ventricle and the free wall of the right ventricle. **e** Four-chamber view of the heart on cardiac MRI with mild pericardial thickening and pericardial fluid (*arrow*). **f** Delayed gadolinium enhancement of the pericardium on cardiac MRI (*arrow*)

variation exceeding 25% in the transmittal valve flow and respiratory variation exceeding 45% across the tricuspid valve further confirming ventricular interdependence [3]. CMR tagging sequences, which apply a grid-like pattern over cine images, can quantify grid deformation over time and can help tease out pericardial-myocardial tethering, a common finding in CP [57]. What sets CMR apart from the other modalities is its ability to characterize the pericardium and determine both the presence and the stage of inflammation. Recall that the presence of LGE in the pericardium signifies continued fibroblast proliferation and neo-revascularization and an increased T2 STIR signal represents edema (Fig. 3f) [20]. Therefore, an increased signal on a T2 STIR with prominent LGE represents an acutely inflamed pericardium. A normal T2 STIR signal with an increased LGE suggests a subacute phase of inflammation with resolution of edema. Absence of both LGE and T2 signal represents the chronic phase in which there is no longer any active inflammation [26]. This is a particularly important clinical tool, as the presence of ongoing inflammation has been found to be the best predictor of reversibility with anti-inflammatory treatment. Active inflammation of the pericardium also has prognostic value in predicting clinical remission [56, 58]. Finally, CMR can contribute to the perioperative planning. The presence of extensive myocardial fibrosis and/or atrophy increases the associated mortality rate for pericardiectomy and should be taken into consideration when evaluating the various therapeutic strategies [59].

Effusive Constrictive Pericarditis

Effusive-constrictive pericarditis is an uncommon form of pericardial disease and should be suspected in patients who present with pericardial tamponade and fail to improve after removal of the pericardial fluid. This disease occurs when there is pericardial fluid accumulation between a thickened, edematous, or fibrotic parietal and visceral pericardium. After the pericardial fluid is removed, the cardiac chambers remain

constricted within the thickened pericardium and demonstrate elevated RA, end-diastolic ventricular pressures, a dip and plateau ventricular waveform, and respiratory ventricular interdependence. Not surprisingly, the diagnosis was traditionally established on cardiac catheterization with a failure of right atrial pressures to drop to more than or equal to 50% after pericardiocentesis [60]. Now more than ever, the diagnosis is made by non-invasive imaging. Echocardiography performed shortly after the pericardiocentesis may demonstrate early signs of constrictive physiology and is therefore recommended as the initial modality when suspecting this pathology. Effusive constrictive pericarditis can be viewed as a spectrum from an acute pericarditis and tamponade phase to a more chronic constrictive physiology phase, and the echocardiographers' interpretation of the images should reflect this evolution. The primary role of additional imaging with CT or CMR is to identify pericardial thickening or inflammation as this holds important prognostic information. More specifically, late gadolinium enhancement of the pericardium on CMR has been shown to predict reversibility of the inflammatory process and reversibility of the constrictive physiology. The late enhancement can also be followed clinically to ascertain response/adequate therapy [30, 61, 62, 63••].

Transient Constriction

Transient constrictive pericarditis is a form of "temporary" constrictive physiology, frequently resulting in permanent resolution either spontaneously or with medication. After the initial insult, often of viral nature, there is pericardial inflammation, pericardial edema, and fibrin deposition. The inflamed pericardium is less compliant resulting in constrictive physiology [64]. Clinical experience has suggested that this entity may be an early reversible and treatable stage on the spectrum leading to eventual chronic constrictive pericarditis and therefore needs to be recognized. Echocardiography remains the initial imaging modality of choice when suspecting transient constrictive pericarditis. Findings consistent with acute pericardial inflammation such as a pericardial effusion and/or thickening of the pericardium along with early signs of constrictive physiology previously mentioned are suggestive of transient constriction. The addition of CMR, currently endorsed by the European Society of Cardiology (ESC), remains a pivotal investigation to identify active pericardial inflammation in addition to corroborating the various elements of constrictive physiology previously described [65]. Similar to constrictive pericarditis and effusive constrictive pericarditis wherein pericardial enhancement predicts response to therapy, a novel technique using Positron emission tomography imaging with fluorodeoxyglucose as a tracer can successfully predict response to steroid therapy and may find its role in the pericardial imaging arsenal in the future [66••].

Pericardial Masses (Echo, CT, and MRI)

Pericardial masses are uncommon and can sometimes go unnoticed until the underlying disease is advanced [1]. They can present as primary benign tumors or primary malignant tumors or secondary metastatic tumors. The most common primary benign lesion in order of frequency includes cysts and lipomas, teratomas, fibromas followed by hemangioma and lymphangiomas. Amongst the primary malignant tumors, melanoma is the most common and the only one found to invade the myocardium [67]. Others include sarcomas and lymphomas [3]. Secondary metastatic tumors commonly found in the pericardium include breast and lung cancer followed by renal cell carcinoma, lymphomas, and melanomas [68]. They have been found to spread by hematogenous spread, lymphatic spread, or direct invasion [69]. Although cytology from pericardiocentesis may yield a diagnosis in secondary malignant tumors, complete resection of the pericardial masses remains the treatment of choice as well as the gold standard for histopathological diagnosis of both primary benign and primary malignant pericardial tumors [16, 70].

Echocardiography is often an initial modality when identifying the presence of a pericardial mass; however, its ability to characterize, differentiate, or evaluate adjacent structures involved is limited [2] (Table 1). Masses on echocardiography can appear nodular with or without a thickened pericardium. They are often non-mobile with a homogenous or heterogenous appearance. Otherwise, echocardiography allows for evaluation of the presence of an effusion and to assess for any hemodynamic involvement caused from either the tumor and/or the effusion. Further characterization usually requires ancillary imaging with cardiac gated CT and/or CMR. CT can better define the mass's dimensions, vascularity, and tumor spread. Moreover, invasion of the adjacent structures can be evaluated [2]. CT has the added advantage of being able to evaluate other structures in the chest wall or lungs including mediastinal lymphadenopathy. CMR is the test of choice when attempting to characterize a pericardial mass [70]. The images can be suggestive of a particular etiology based on T1-weighted sequences, T2-weighted sequences, and fat suppression as well as early and late contrast enhancement sequences. Pericardial fibromas will appear well-defined and encapsulated and will demonstrate low signal intensity on T2-weighted images due to its fibrous content. Liposarcoma will have a high T1 signal intensity because of its fatty composition, and our ability to suppress the fat using suppression techniques confirms the diagnosis. Malignant tumors will often be isointense to the myocardium with T1-weighted sequences with the exception of melanoma which has a high signal intensity due to the paramagnetic materials bound by melanin [70]. Other features suggestive of malignant tumors are irregular borders and the presence of a loculated pericardial effusion. Malignant tumors will frequently enhance on early

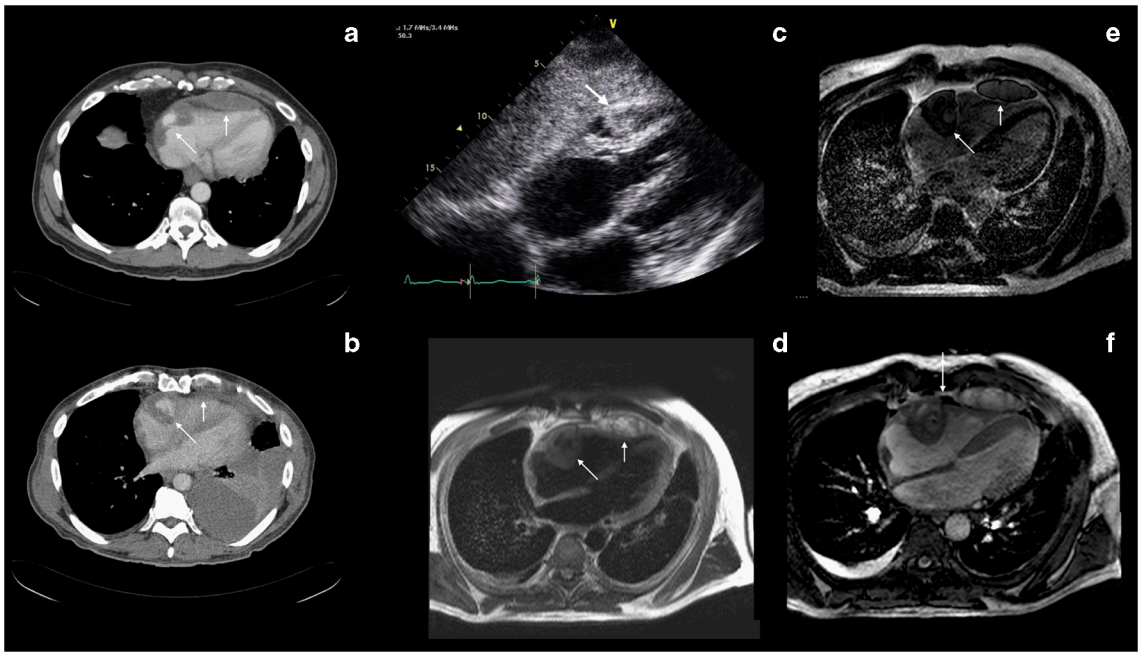

Fig. 4 45M with angiosarcoma of the pericardium. **a**, **b** Axial four-chamber views of the heart on contrast-enhanced CT demonstrating a heterogenous enhancing soft tissue mass in the right atrioventricular groove (*long arrow*) and a heterogenous nodularity on the anterior and diaphragmatic pericardium suggestive of a pericardial mass (*short arrow*). **c** Subxyphoid view on 2D echocardiography showing a hyperechoic, heterogenous mass within the pericardium compressing the RV free wall (*arrow*). **d** T1-weighted sequence on CMR showing a heterogenous mass in the right atrioventricular (AV) groove and extending into the right atrium with intermediate signal intensity (*long arrow*). There is a second mass lesion anterior to the right ventricle free wall with high signal intensity and has the appearance of loculated fluid collection within the pericardium (*short arrow*). **e** T2-weighted sequence of the mass within the AV groove (*long arrow*) and the outer portion of the fluid-like collection within the pericardium (*short arrow*) showing an isointensity with the myocardium. **f** Late enhancement sequences with a mild enhancement of the pericardium (*arrow*) suggestive of pericardial involvement

contrast enhancement due to their increased vascularity. Moreover, late contrast enhancement is typically heterogeneous (Fig. 4) [71]. Thrombus, which is often considered within the differential diagnosis, will usually fail to enhance with all sequences and will have no enhancement on early and delayed contrast enhancement sequences and sequences with a long inversion time. Nevertheless, the gold standard currently remains a histopathological diagnosis [30].

Congenital Malformations/Absence of the Pericardium

Pericardial cysts and pericardial diverticula are rare, benign congenital malformations. Pericardial diverticula are felt to be the result of an outpouching or herniation through a defect of the parietal pericardium while a cyst may be a remnant of the diverticulum which has lost

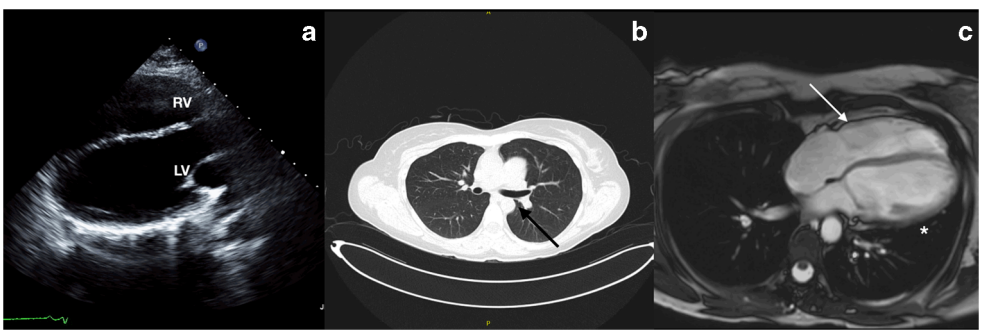

Fig. 5 **a** Parasternal long-axis view of the heart on transthoracic echocardiogram showing abnormal orientation of the heart with an elongated RV. **b** Axial four-chamber views of the heart on contrast-enhanced CT demonstrating a leftward apex/levocardia and interposition of lung between aorta and the pulmonary artery (*arrow*). **c** T1-weighted cine sequence on CMR showing leftward apex/levocardia with partial agenesis of the pericardium (*). There remains a normal portion of the pericardium near the RV free wall (*arrow*). RV = Right ventricle

communication with the pericardium [2, 16, 72]. A pericardial cyst on echocardiography appears as a well-circumscribed, homogenous echolucent structure typically adjacent to the right atrium. Pericardial cysts are better defined on cardiac CT or CMR. On CT, the cyst appears as thin-walled non-enhancing structure with near water attenuation (30–40 HU). When imaging with CMR, cysts will have a low signal intensity with T1-weighted sequences and a high signal intensity with T2-weighted STIR sequences without enhancement on delayed contrast imaging [5, 73]. Diverticula distinguish themselves from cysts if an open communication to the pericardial sac can be identified, usually best ascertained on CT and CMR [74].

Congenital absence of the pericardium is a rare congenital disorder that can occur in isolation in the presence of other congenital pathologies [75]. It can manifest as complete or partial, left sided or right sided, and is often suspected on echocardiogram. Typical findings include an unusual echo window, falsely appearing dilated RV, excessive motion of the heart, and an abnormal septal motion [76]. Although spatial resolution is improved with CT and CMR, the diagnosis can remain a challenge. As such, the diagnosis is established based on indirect findings (Table 1). Leftward cardiac displacement or excessive levorotation is one finding while the presence of lung tissue between the aorta and the pulmonary artery or between the diaphragm and the base of the heart is another [77] (Fig. 5).

Conclusion

Pericardial diseases are a spectrum of pathologies that are commonly encountered and remain a diagnostic conundrum in day-to-day practice. Echo remains the initial modality of choice when imaging the pericardium, but the role of complementary imaging with CCT and CMR is well established. Moreover, new imaging techniques offer important diagnostic and prognostic information that can guide therapy, making their role invaluable in the management of patients with pericardial diseases. Knowing how these modalities complement each other is central to efficient and effective utilization of these imaging techniques.

Compliance with Ethical Standards

Conflict of Interest Michael Chetrit, Bo Xu, and Beni R. Verma declare that they have no conflict of interest. Allan L. Klein has received honoraria from Sobi and a research grant from Kiniksa.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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