Curr Cardiol Rep (2015) 17: 24
DOI 10.1007/s11886-015-0577-9

PERICARDIAL DISEASE (AL KLEIN, SECTION EDITOR)

Multimodality Imaging of Pericardial Disease

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Published online: 15 March 2015

Abstract The emergence of multimodality imaging of pericardial diseases has improved diagnosis and management. In acute pericarditis, echocardiography is the first-line test, but cardiac magnetic resonance (CMR) may be beneficial in patients who fail to respond to therapy. An increased T2 short-tau inversion recovery time (STIR) suggests pericardial edema, and increased late gadolinium enhancement (LGE) suggests organizing pericarditis. Computed tomography (CT) can be helpful in procedural planning, either to guide percutaneous drainage of an effusion or to assess calcification and the location of vascular structures before pericardiectomy. On echocardiography, a respiratory septal shift in combination with either a preserved medial e' velocity or prominent expiratory diastolic hepatic vein flow reversal performs well in diagnosing constrictive pericarditis. These patients also have decreased regional longitudinal strain in the anterolateral and right ventricular free walls, presumably related to pericardial to myocardial tethering. Finally, prominent LGE may identify patients with constrictive pericarditis who improve with anti-inflammatory therapy.

Keywords Acute pericarditis · Recurrent pericarditis · Pericardial effusion · Constrictive pericarditis · Echocardiography · Computed tomography · Cardiac magnetic resonance imaging

This article is part of the Topical Collection on *Pericardial Disease*

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Introduction

Over the past few decades, research in cardiovascular medicine has focused on coronary and valvular heart disease with great advances in patient diagnosis and management. By comparison, significantly less progress has been made in pericardial disease, even though pericardial diseases have significant morbidity and mortality [1, 2]. In recent years, however, pericardial pathology has garnered more attention, in part related to the increasing role of multimodality imaging to aid in diagnosis and guide management [3••]. In this review, we will address three questions that are central in the assessment of a patient with known or suspected pericardial disease:

1. What echocardiographic features are most predictive of specific pericardial pathologies?
2. Which patients with a complete echocardiographic assessment should subsequently undergo a computed tomography (CT) or cardiac magnetic resonance (CMR) study?
3. How might the results of a CT or CMR study influence subsequent management?

For the first question, we will focus on recent diagnostic criteria for constrictive pericarditis [4••] and the emerging role of left ventricular mechanics [5, 6••]. The second question is framed by addressing clinical conditions that should prompt ongoing concern and by highlighting anatomic or hemodynamic questions that may remain unanswered after echocardiography. Finally, we will underscore how CT results can guide invasive management of pericardial diseases and will also emphasize how pericardial late gadolinium enhancement (LGE) on CMR can direct anti-inflammatory therapy [7••].

Acute Pericarditis

Acute pericarditis has been estimated to account for 5 % of emergency department visits for non-myocardial infarction chest pain [8]. However, the true incidence is unknown, and may be underestimated, since there is no established gold standard for diagnosis. Instead, acute pericarditis remains a clinical diagnosis with cardinal features including typical positional and pleuritic chest pain, a pericardial friction rub, typical ST elevations, and a pericardial effusion [9, 10].

All patients with suspected acute pericarditis should have an echocardiogram [11]. In the correct clinical setting, a transthoracic echocardiogram with a pericardial effusion is diagnostic of acute pericarditis, though the most common finding is a normal study [12–14]. Furthermore, echocardiography evaluates other causes of chest pain, and an expeditious assessment could forego an unnecessary invasive coronary angiogram as these patients may be suspected of having an ST-segment elevation myocardial infarction [15]. Echocardiography also allows for rapid risk stratification; tamponade physiology is present in 3 % of patients, and 5 % may have segmental wall motion abnormalities suggesting myopericarditis [3••]. Finally, echocardiography may identify stable patients who nonetheless require closer follow-up. Such patients may have evidence of early constriction including an abnormal septal bounce and increased pericardial brightness or thickening.

Conversely, CT provides limited additional value in the initial assessment of non-traumatic acute pericarditis. Nevertheless, a CT scan is often obtained to evaluate other pathologies, and ancillary findings are common [16]. A non-calcified thickened pericardium may indicate acute pericarditis. Occasionally, iodinated contrast will enhance the pericardium, indicating active inflammation [17]. A pericardial effusion may also be present, and the attenuation value of the fluid may aid in diagnosis. Transudative fluid has <10 Hounsfield units, exudative fluid typically has 20–60 Hounsfield units, and hemopericardium usually has >60 Hounsfield units [18]. With the increased use of CT scanning in the emergency department, cardiovascular imagers should be aware of the implications of these findings, even though the CT may have been obtained for a different indication.

Unlike CT, CMR may have a directed role in carefully selected patients with known or suspected acute pericarditis (Fig. 1). CMR can complement clinical, laboratory, and echocardiographic data while also providing insight into the acuity and magnitude of active inflammation. Cine steady-state free precession sequences allow for regional assessment of the left and right ventricles. T1-weighted sequences with black blood preparation assess the extent of a pericardial effusion, and pericardial thickness can be measured. A thickened non-calcified pericardium suggests acute pericarditis [19]. Acuity is also assessed on a T2W short-tau inversion recovery sequence (STIR), though assessment may be limited in the presence of a pericardial effusion [20, 21]. An increased T2 STIR signal suggests an acute process with ongoing edema and neovascularization [22]. In addition, pericardial late gadolinium enhancement (LGE) indicates continued inflammation. On histology, LGE represents abnormal vascular permeability and fibroblast proliferation [23•]. Therefore, in a patient with poor prognostic signs including fever or a failure to respond promptly to standard therapy with NSAIDs and colchicine [24], prominent pericardial LGE may support the addition of corticosteroids (triple anti-inflammatory therapy) and a longer course of intense medical therapy.

Chronic and Recurrent Pericarditis

Chronic pericarditis is defined by a duration of symptoms greater than 3 months, and recurrent pericarditis is divided into intermittent and incessant forms [9]. The former is defined arbitrarily as a symptom-free interval of at least 6 weeks whereas the latter refers to symptom recurrence in less than 6 weeks upon drug weaning or discontinuation. Recurrent pericarditis occurs in about 15–30 % of patients after an episode of acute pericarditis, and approximately 50 % of patients with an initial recurrence will have subsequent episodes [25, 26, 27•].

As with acute pericarditis, transthoracic echocardiography is the initial imaging test. Echocardiographic findings are similar to acute pericarditis, though a more careful diastolic assessment with use of a respirometer is necessary as patients may transition to constrictive pericarditis. Likewise, the indications for CMR are similar, though the threshold may be lower than in acute pericarditis for two reasons (Fig. 1). For one, by definition, patients with chronic or recurrent pericarditis have not achieved adequate symptom relief, and the magnitude of pericardial LGE enhancement may inform further anti-inflammatory therapy. Second, some patients may have atypical features and a lack of objective findings including inflammatory markers that are not elevated. In this setting, CMR has an excellent negative predictive value, and a normal study may prompt the clinician to pursue other etiologies for the patient's symptoms.

Pericardial Effusion and Tamponade

Pericardial fluid can accumulate with diseases that directly affect the pericardium, such as acute pericarditis, or in general medical conditions including hypothyroidism, end-stage renal disease, and malignancy [3••]. When pericardial effusion is suspected, echocardiography is the first-line procedure and is nearly 100 % accurate. On M-mode echocardiography, a separation of the parietal pericardium and epicardium only in

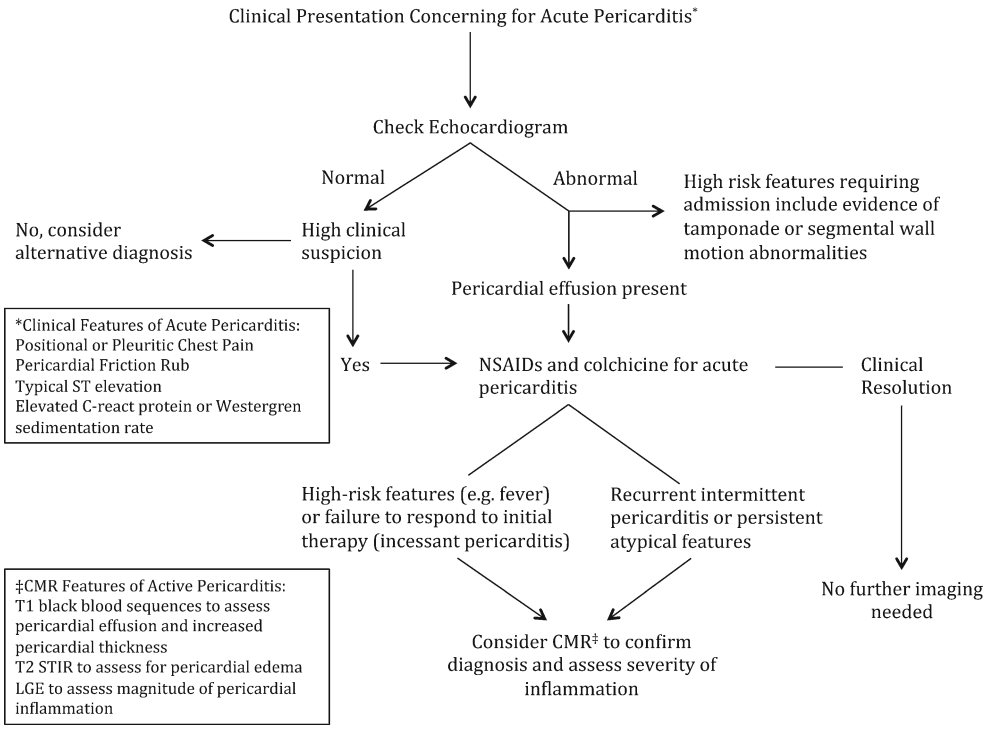


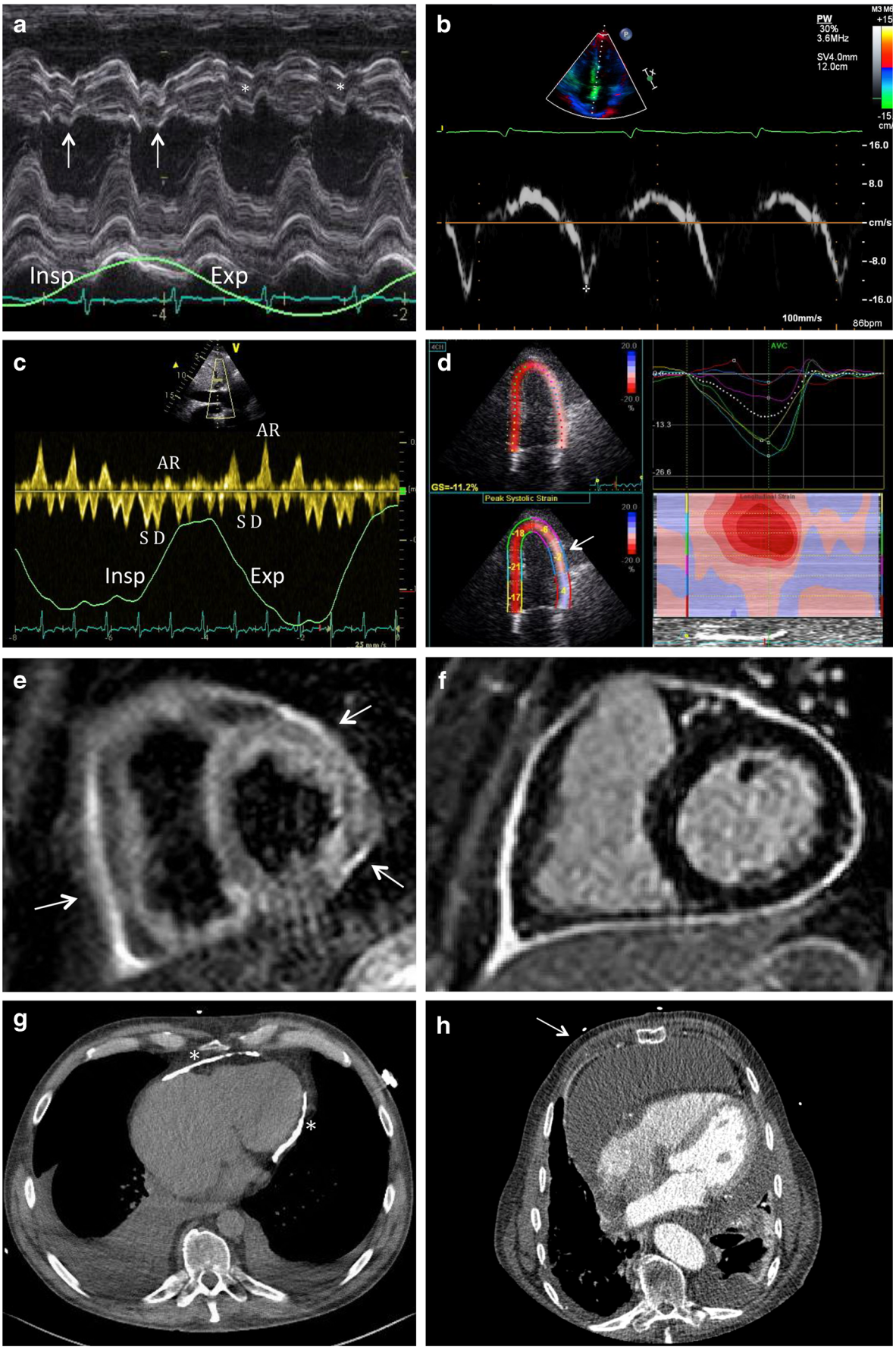
Fig. 1 Simplified approach for imaging of suspected acute or recurrent pericarditis. *CMR* cardiac magnetic resonance, *STIR* short-tau inversion recovery time, *LGE* late gadolinium enhancement

systole represents a trivial pericardial effusion whereas separation in systole and diastole is associated with effusions greater than 50 mL [28]. With two-dimensional echocardiography, pericardial effusions are usually semi-quantified at end diastole as small (<1 cm), moderate (1–2 cm), large (>2 cm), or very large (>2.5 cm) [29]. Pericardial fluid may appear loculated or have features consistent with exudate or a clot. Epicardial fat can be difficult to distinguish from organized pericardial fluid but generally is brighter than the myocardium and moves with the heart. In contrast, pericardial fluid is generally motionless and echolucent. CT and CMR can aid in localization and characterization of pericardial fluid, but these modalities are rarely primarily obtained to assess a pericardial effusion [30]. Instead, the pericardial effusion is often an incidental finding [31]. However, in a stable patient with a loculated or complex effusion, CT may inform the percutaneous approach or may indicate that surgical drainage is preferable (Fig. 2).

Cardiac tamponade is characterized by elevated and equal intracardiac diastolic pressures due to impaired chamber filling with subsequent embarrassment of cardiac output [32]. Given its life-threatening nature, emergent echocardiography should be obtained when cardiac tamponade is suspected. The most important echocardiographic findings include the presence of a pericardial effusion, a dilated inferior vena cava, and Doppler evidence of reduced stroke volume [3••]. Diastolic chamber inversion or collapse can also support a diagnosis of cardiac tamponade. Right atrial indentation begins at the onset of the R waves and continues during ventricular systole until right atrial pressure exceeds pericardial pressure. Collapse of the right atrium for more than one third of the cardiac cycle is highly sensitive and specific for tamponade [33]. Similarly, the duration of right ventricular diastolic collapse can also indicate tamponade, but right atrial collapse may be more predictive [34]. Finally, due to ventricular interdependence and an accentuated inspiratory decrease in pulmonary venous to left atrial pressure gradient, significant respiratory variation in mitral and tricuspid inflow can be observed in tamponade [35]. For the peak mitral *E* velocity, the first beat of inspiration typically decreases >30 % whereas the peak tricuspid *E* velocity typically decreases >60 % with the first beat of expiration [36]. While these features can corroborate a diagnosis of cardiac tamponade, the diagnosis is defined by a reduced stroke volume and elevated central venous pressures in the presence of a pericardial effusion.

Constrictive Pericarditis

Constrictive pericarditis is often difficult to diagnose, especially in cases of mixed constriction and restriction as in radiation heart disease. In the developed world, constrictive pericarditis is most often idiopathic or viral, though cases attributed to cardiac surgery are increasing [36, 37]. As in cardiac tamponade, constrictive pericarditis is characterized by noncompliant cardiac chambers and prominent respiratory variation in cardiac filling. Unlike cardiac tamponade, ventricular filling in early diastole is increased in constrictive pericarditis as the inelastic pericardium does not impose restraint until mid-diastole.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◀ **Fig. 2** In patients with constrictive pericarditis (**a**–**d**), the M mode of the ventricular septum demonstrates a septal notch and movement of the ventricular septum toward the left ventricle with inspiration (*arrows*) and toward the right ventricle with expiration (*asterisk*) (*Insp* inspiration, *Exp* expiration) (**a**). Medial mitral annular *e′* velocity is preserved or increased (**b**). Late diastolic flow reversal in hepatic veins is prominent upon expiration (*S* systole, *D* diastole, *AR* atrial reversal) (**c**), and global longitudinal strain is decreased in the anterolateral wall (*arrow*) (**d**). In patients with pericarditis, an increased pericardial T2 STIR signal (*arrows*) (**e**) suggests edema, and the intense circumferential LGE in this patient indicates ongoing neovascularization and fibroblast proliferation (**f**). These findings indicate active inflammation while pericardial calcification on CT (*asterisk*) usually represents the end stage of fibrosis (**g**). CT can also inform percutaneous drainage of a loculated or complex pericardial effusion as in this example where CT results were used to guide a right parasternal approach (*arrow*) (**h**). CT, computed tomography; STIR, short-tau inversion recovery; LGE, late gadolinium enhancement

The hemodynamic effects of constriction are often manifest on echocardiography (Fig. 2). The M mode of the ventricular septum with a respirometer can highlight ventricular interdependence. In a patient with constrictive pericarditis, the ventricular septum will move toward the left ventricle with inspiration and toward the right ventricle with expiration [38]. In addition, there will be a diastolic septal bounce, or a septal shudder, which is seen as a septal notch on the M-mode tracing. In addition, respiratory changes in ventricular diastolic filling demonstrate this pathophysiology. The mitral inflow velocity usually decreases by at least 25 %, and the tricuspid inflow velocity usually increases by at least 40 % with inspiration [39, 40]. Doppler profiles of the hepatic veins can further highlight the dissociation between intracardiac and intrathoracic pressures. In constrictive pericarditis, hepatic vein diastolic flow reversal is prominent upon expiration. Finally, tissue Doppler imaging can also aid in distinguishing constrictive pericarditis from restrictive cardiomyopathy. In normal patients, the medial mitral annulus *e′* velocity is typically less than the lateral *e′* velocity. However, in constrictive pericarditis, the medial *e′* velocity increases as the disease worsens. Therefore, the medial *e′* velocity can be less than the lateral *e′* velocity, an observation termed "annulus reversus" [41, 42].

While these echocardiographic features have all been associated with constrictive pericarditis in isolation, their possible hierarchical relationship had not been evaluated until recently. By comparing patients with surgically confirmed constrictive pericarditis to patients with restrictive cardiomyopathy or severe tricuspid regurgitation, multiple echocardiographic parameters were evaluated [4••]. On multivariable analysis, the respiratory ventricular septal shift, medial mitral *e′* velocity, and hepatic expiratory diastolic reversal ratio were independently associated with constrictive pericarditis. In evaluating optimal diagnostic characteristics, a ventricular septal shift combined with either a medial $e' \geq 9$ cm/s or hepatic expiratory diastolic reversal to forward velocity ratio of ≥ 0.79 performed best with a sensitivity of 87 % and a specificity of 91 %. If all factors were present, specificity increased to 97 % at the expense of a decrease in sensitivity to 64 % [4••]. However, the patients with constrictive pericarditis in this cohort were all referred for surgery which introduces bias and limits generalizability. Also, as in acute pericarditis, a gold standard for the diagnosis of constrictive pericarditis does not exist, so an analysis of sensitivity and specificity is difficult. Nevertheless, these findings provide valuable insight into an organized approach for multiple parametric testing in constrictive pericarditis.

With the emergence of two-dimensional speckle tracking echocardiography, patterns of ventricular mechanics in constrictive pericarditis have also been described [5, 6••]. In general, circumferential strain, torsion, and early diastolic twisting are decreased in constrictive pericarditis. In contrast, global longitudinal strain is usually preserved. Patients with restrictive cardiomyopathy can demonstrate the converse pattern: global longitudinal strain is decreased while circumferential strain and early diastolic untwisting are preserved [5].

More recently, regional differences in strain have been observed [6••]. In constrictive pericarditis, pericardial tethering can involve the left and right ventricular free walls. This pericardial to myocardial tethering can reduce longitudinal deformation with preserved deformation of the interventricular septum [43]. In a study including patients with constrictive pericarditis, restrictive cardiomyopathy, and normal controls, patients with constrictive pericarditis had decreased left ventricular anterolateral wall and right ventricular free wall strain while septal wall strain was preserved [6••]. Moreover, longitudinal strain in these segments improved with pericardiectomy, supporting the hypothesis that tethering caused impaired deformation of these walls. Even though this study underscores the pathophysiology of how constrictive pericarditis leads to diastolic dysfunction and heart failure, an integrated diagnostic approach incorporating the M mode, Doppler, and two-dimensional speckle tracking is needed to determine the incremental value of two-dimensional speckle tracking.

While an assessment for constrictive pericarditis may be complete in many patients after a thorough clinical assessment and comprehensive echocardiogram, select patients may benefit from a CT or CMR (Fig. 3). This decision to pursue advanced imaging should focus on whether a specific anatomic or hemodynamic question remains unanswered. Presently, CT is primarily used in preoperative planning for patients with prior cardiac surgery or radiation heart disease. CT can precisely assess the magnitude of calcification and location of critical vascular structures [44]. Pericardial calcification on CT can represent the end stage of fibrosis and inflammation in constrictive pericarditis, but many patients with calcification may not have constrictive physiology. Furthermore, CT provides an excellent assessment of pericardial thickness, but patients can have normal pericardial thickness and still have constriction [45].

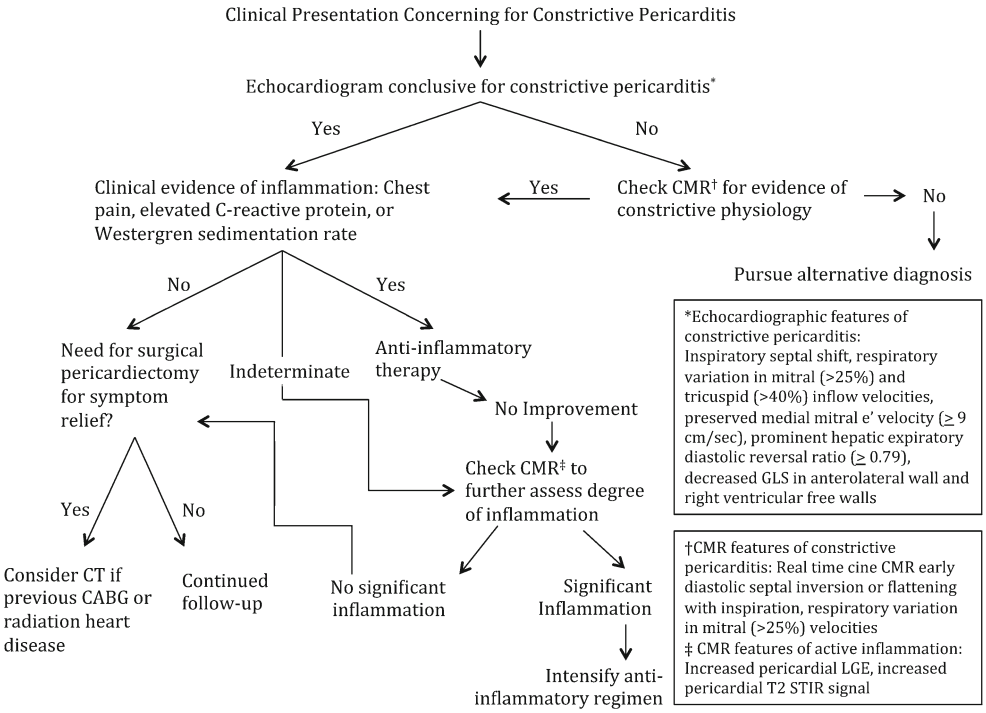

Fig. 3 Simplified imaging approach for suspected constrictive pericarditis. *CMR* cardiac magnetic resonance, *GLS* global longitudinal strain, *LGE* late gadolinium enhancement, *STIR* short-tau inversion recovery time, *CT* computed tomography, *CABG* coronary artery bypass grafting

These limitations underscore that CT primarily provides a morphologic assessment while constrictive pericarditis is a clinical and hemodynamic diagnosis. In this regard, CMR can provide additional insight. Similar to echocardiography, respiratory variation in transvalvular flow with CMR can assess for ventricular interdependence. In a small study with real-time phase contrast CMR in spontaneously breathing patients, respiratory variation greater than 25 % across the mitral valve performed perfectly in diagnosing constrictive pericarditis [46•]. However, generalizability is tempered by the small size of the study.

Real-time cine CMR also allows assessment of respiratory variation in ventricular septal motion. In a single-center study, ventricular septal position was assessed in the short axis during operator-guided deep respiration [47]. At the onset of inspiration, all patients with constrictive pericarditis had early diastolic septal inversion or flattening. Again, this study is limited given the small size. However, in patients with equivocal echocardiographic findings, evidence of significant respiratory variation in transvalvular flow or inspiratory septal abnormalities can confirm constrictive pericarditis.

While echocardiography provides the primary hemodynamic assessment in patients with constrictive pericarditis, an assessment for ongoing pericardial inflammation is absent. In this setting, CMR findings are not complementary to echocardiography but independently valuable. As mentioned, LGE on CMR indicates continued fibroblast proliferation and neovascularization [23•], and an increased pericardial signal on T2 STIR sequences is consistent with pericardial edema [22] (Fig. 2). Therefore, CMR findings can inform the stage of inflammation in patients with constrictive pericarditis. A patient with an increased T2 STIR signal and prominent pericardial LGE is acutely inflamed while normal T2 STIR images and increased pericardial LGE suggest that a patient is in a sub-acute phase of the disease. The absence of pericardial LGE and a normal T2 STIR signal indicate a patient who no longer has active inflammation and may therefore not be modifiable with anti-inflammatory therapy.

These differential CMR patterns can thus identify patients with potentially reversible versus those with likely persistent constrictive pericarditis. Reversible patients may respond to anti-inflammatory therapy whereas persistent patients may need a pericardiectomy for relief. In a small study, patients with significant pericardial LGE were indeed more likely to have resolution of constrictive pericarditis compared with those with medical treatment [7••]. Moreover, even for patients who will eventually require pericardiectomy, preoperative treatment of active inflammation may facilitate a better surgical result, though evidence to support this approach is anecdotal.

Conclusions

In recent years, investigations in pericardial diseases have surged, and this increased interest parallels innovations in cardiovascular imaging [3••]. However, all forms of pericarditis and cardiac tamponade are diagnosed clinically, and echocardiography remains the first-line study. In acute pericarditis, a typical presentation coupled with a pericardial effusion is diagnostic. Echocardiography also identifies high-risk patients,

including those with evidence of tamponade, segmental wall motion abnormalities, and early constrictive pericarditis. In constrictive pericarditis, recent echocardiographic data suggests that an inspiratory ventricular septal shift coupled with either a medial *e′* velocity ≥9 cm/s or a hepatic expiratory diastolic reversal ratio ≥0.79 is sensitive and specific [4••]. Also, pericardial to myocardial tethering can result in decreased global longitudinal strain in the lateral wall of the left ventricle and the right ventricular free wall [6••].

Currently, the role of CT in pericardial diseases is limited as CT generally provides an anatomic and not a hemodynamic assessment. Pericardial pathology including effusion, increased thickness, contrast enhancement, and calcification can be observed and should be investigated even if a CT is ordered for another indication. Conversely, a CT can have a major role in procedural planning. For example, CT can help guide a percutaneous approach to a complicated or loculated pericardial effusion. In addition, information from CT including the extent of calcification and the location of major vascular structures can be invaluable in planning prior to surgical pericardiectomy.

Compared to CT, CMR has a more prominent role in pericardial diseases as information is provided regarding inflammation and hemodynamics. In equivocal cases, CMR evidence of greater than 25 % respiratory variation in mitral inflow velocities and early diastolic septal inversion or flattening upon inspiration can help confirm constrictive pericarditis [46•, 47]. Recently, the histology of pericardial LGE has been demonstrated as abnormal vascular permeability and fibroblast proliferation [23•]. Thus, in patients with acute pericarditis who do not respond to initial therapy or patients with recurrent pericarditis who do not have adequate symptom relief, the magnitude of pericardial LGE correlates to the degree of active inflammation and can inform subsequent treatment. Similarly, in patients with constrictive pericarditis, the pattern of CMR abnormalities can inform the stage of disease. Patients with an increased pericardial T2 STIR signal and LGE are acutely inflamed while patients with only increased LGE may be in a sub-acute stage of disease. Both of these patterns suggest potentially reversible constrictive pericarditis. In contrast, patients without increased pericardial T2 STIR or LGE may have persistent constrictive pericarditis and may be less likely to resolve without pericardiectomy [7••]. Despite these advances in multimodality imaging, published studies have been small and randomized trials are lacking. Therefore, research opportunities are ripe within the nascent field of multimodality imaging of pericardial disease.

Compliance with Ethics Guidelines

Conflict of Interest Paul C. Cremer declares that he has no conflict of interest.

Deborah H. Kwon has received consulting fees from Astellas.

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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• Of importance
•• Of major importance

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