REVIEW ARTICLE

Clinical Utility of Cardiac Magnetic Resonance Imaging in Pericardial Diseases

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Abstract: Background: Pericardial diseases are relatively common in clinical practice and encountered in various clinical settings with consequent significant morbidity and mortality. However, the diagnosis as well as management can be complex and challenging, as the clinical presentation is usually non-specific. Therefore, there is an increasing role for Cardiac Magnetic Resonance Imaging (CMR) as an imaging tool to facilitate the diagnosis of pericardial diseases.

Conclusion: Herein we describe conventional and unique CMR approaches to provide an increased non-invasive understanding of the pericardium in health and disease including a novel method to diagnose constrictive pericarditis *via* radio-frequency tissue tagging by defining unique visceral-parietal adherence patterns easily learned by the cardiologist and radiologist.

Keywords: CMR, acute pericarditis, constrictive pericarditis, pericardial diseases, morbidity, mortality.

1. INTRODUCTION: IMAGE ACQUISITION MODALITIES

No doubt much has been written over the years about pericardial disease by all non-invasive modalities so the natural question ensues, “What else is there to say?” Fortunately, in the realm of Cardiac Magnetic Resonance Imaging (CMR), much has been confirmed and even more has been added to the already impressive armamentarium to this approach to elucidate the contemporary contributions of this often complex and multifaceted disease. In this review, we will remind the reader of the classic uses of CMR for this entity while interleaving more recent approaches that dovetail with clinical validations. These more recent findings utilize novel sequences and applications incorporating both contrast and non-contrast techniques. Most intriguing is the recent ability of CMR to incorporate both physiology and ‘virtual histology’ strategies to delineate extraordinarily accurate diagnoses of constriction and pericarditis using pericardial deformation and acute edema techniques, respectively.

The reader will be introduced to the CMR sequence followed by its clinical application and where appropriate, the corroborative evidence.

2. CARDIAC MAGNETIC RESONANCE IMAGING SEQUENCES

The five CMR sequences of clinical significance include spin-echo imaging, T1 weighted contrast-enhanced imaging,

balanced Steady-State Free Precession (SSFP), MR tagging and flow velocity encoding (phase contrast).

When used appropriately, the images acquired from CMR assist in quick, single modality identification and characterization of anatomically complex pericardial diseases.

1. Spin echo imaging/ double inversion recovery sequence. It is a dark blood technique in which the signal from the blood is nulled [1]. It uses blood as a contrast to delineate the cardiac anatomy in T1 weighted images and allows depiction of pericardial fluid and edema during T2 acquisition:
 - a. Black-blood T1-weighted spin-echo MR imaging performed with a fast segmented sequence is the best approach for morphologic study of the pericardium [2].
 - b. T2-weighted spin-echo imaging, preferably performed by using a short-tau inversion-recovery sequence (triple inversion sequence) enables depiction of pericardial fluid and/or edema of the pericardial layers in patients with inflammatory pericarditis [3].
2. Late Gadolinium Enhanced (LGE) is T1 weighted contrast-enhanced and/or late contrast-enhanced imaging following intravenous administration of a paramagnetic contrast agent (Gadolinium). It is useful when evaluating pericardial masses or inflammatory pericarditis Table 2. It is also used to depict concomitant myocardial pathologic conditions and is of great value in depicting persistent chronic inflammation in patients with constrictive pericarditis [4, 5].
3. Balanced Steady-State Free Precession (SSFP). It provides high spatial and temporal resolution of cine CMR

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Fig. (1). Steady-State Free Precession (SSFP) sequence of 4-chamber (left) and short axis (right) view of the heart with mostly focal lateral wall large pericardial effusion (yellow arrow). The parietal and visceral pericardium's (red arrows) are separated with the pericardial fluids. Epicardial fat (blue arrow head). (The color version of the figure is available in the electronic copy of the article)

imaging. It is applied to evaluate the rigidity of the pericardial layers in patients with constrictive pericarditis and physiological impact of pericardial diseases [6]. It is the workhorse imaging sequence.

- MR radio-frequency tissue-tagging techniques are invaluable in the detection of both fibrotic adhesion of pericardial layers and myocardial involvement in constrictive pericarditis [7].
- Phase velocity mapping is an invaluable technique to non-invasively measure and depict blood flow while accurately quantitating blood flow velocities. Regarding the pericardium, it is helpful for understanding the impact on valvular and non-valvular pathologies, constrictive physiology (restrictive vs. non-restrictive) and for evaluation of caval flow and, on occasionally time-volume curves.

3. NORMAL PERICARDIUM- ANATOMIC DESCRIPTION AND CARDIAC MAGNETIC RESONANCE IMAGING FINDINGS

The normal pericardium has two distinct layers, a fibrous pericardium and a serous inner layer. The serous layer is a closed sac with the visceral component lining the epicardium and the parietal component lining the fibrous outer layer. The inner visceral layer encloses the heart and is continuous with the outer parietal layer at regions called pericardial reflections. It is the visceral layer of pericardium and not the parietal layer that constricts the heart and it is the layer that undergoes pericardiectomy. Between the visceral pericardium and the myocardium is a variable amount of epicardial adipose tissue [8]. Pericardial fluid is located between the visceral and parietal pericardium. On SSFP cine CMR that exhibits T1/T2 weighing, simple effusions appear bright with the same or even higher image intensity than epicardial fat (9) (Fig. 1).

Only the parietal part of the normal pericardium is sufficiently thick to be visualized by CMR as the inner visceral layer is a thin, monolayer structure [9, 10]. The fibrous pericardial tissue has low-intensity signal on MR images due to long longitudinal relaxation time (T1), short transverse relaxation time (T2) [11], and low spin density [12].

Normal pericardium appears as a thin curvilinear structure that follows the myocardial contours and is surrounded by a variable amount of pericardial fat tissue. It is best visualized along the right ventricle due to the sparseness of pericardial fat tissue. It is often difficult to discern the pericardium along the inferolateral wall due to the presence of pulmonary parenchyma [13]. The average pericardial width as measured on CMR images is 1.9 ± 0.6 mm during systole in normal individuals, whereas pathological pericardium by convention, measures >3 mm [10].

4. CARDIAC MAGNETIC RESONANCE IMAGING FOR ACUTE PERICARDITIS

Acute pericarditis patients tend to have a subclinical course as its prevalence was found to be 1% in autopsy studies [14]. The etiology in majority of the cases (80-85%) in the developed world is idiopathic and assumed to be viral [15]. Histologically, the inflamed pericardial layers are composed of a highly vascularized granulation tissue with fibrin deposition that may cause fibrinous adhesion of the pericardial layers [5].

The diagnosis of acute pericarditis is based on the presence of: 1. Typical chest pain, 2. Pericardial rub, 3. Typical EKG changes, 4. New or worsening pericardial effusion, 5. Elevated C-reactive protein or sedimentation rate, [8]. In equivocal cases, cardiac imaging would be necessary to establish the diagnosis [16]. CMR imaging has excellent tissue characterization evaluation of inflammation [8] Table 1. T2-weighted images and LGE are used to identify pericardial edema and inflammation [16] (Figs. 2 and 3). Therefore, CMR can and often is used in individuals who have limited windows using Transthoracic Echocardiogram (TTE) or in equivocal cases where there is high suspicion for acute pericarditis and the patient does not meet the criteria mentioned above.

In the acute and subacute forms of pericarditis, the thickened or non-thickened pericardium typically has high signal intensity on T2 weighted spin echo (triple inversion) images [13]. Enhancement of thickened pericardium on T1-weighted (T1W) spin echo images or LGE images after the administration of gadolinium-based contrast media confirms active inflammation [5, 13, 17-19] (Fig. 3). Sensitivity for LGE detection of pericardial inflammation, extraordinarily high,

Table 1. Comparison of CMR findings between acute pericarditis, constriction, perhaps restrictive cardiomyopathy and pericardial effusion.

	Acute Pericarditis	Constrictive Pericarditis	Restrictive Cardio-myopathy	Pericardial Effusion
T1W	Enhancement of thickened pericardium	Fibrotic and/or calcified pericardium has low signal intensity unless there is residual inflammation	Normal pericardial thickness and signal	Transudate: low intensity signal Exudative: High intensity signal
T2W	High intensity signal in the pericardial tissue	Fibrotic and/or calcified pericardium has low signal intensity unless there is residual inflammation	Normal pericardial thickness and signal	Transudate: High intensity signal Exudate: low intensity signal
LGE	High intensity signal	No LGE unless there is residual inflammation	Variable depending on the underlying disease but should have LGE in the pericardium	LGE in the case of acute pericarditis
RF tagging		Loss of the normal slippage of the outer pericardium over the epicardial surface during the cardiac cycle	Normal	
SSFP	Thickened pericardial layers (>4 mm), variable amount of pericardial fluid, septal bounce may occur due to decreased pericardial compliance	May have thickened pericardial layers (>4 mm), Septal bounce and respiratory variation in septal excursion	Normal pericardial thickness < 3mm	Pericardial width> 4mm regarded as abnormal amount of fluid
Phase encoding velocimetry	No specific findings unless there is pericardial effusion associated with tamponade physiology	Restrictive filling pattern of RV and LV diastolic filling; >25% fall in mitral inflow velocity and >40% increase in tricuspid velocity in the first beat after inspiration; opposite changes in expiration	May have restrictive filling pattern but no respiratory variation of flow across the mitral and tricuspid valve	In the case of tamponade Restrictive filling pattern of RV and LV diastolic filling; >25% fall in mitral inflow velocity and >40% increase in tricuspid velocity in the first beat after inspiration; opposite changes in expiration

Table 2. CMR features of different pericardial masses.

	Primary Benign Pericardial Tumors					Primary Malignant Pericardial Tumors		Secondary Malignant Pericardial Tumors			
	Tertatoma	Lipoma	Fibroma	Hemangioma	Lymphangioma	Mesothelioma	Angiosarcoma	Lymphoma	Melanoma	Lung carcinoma	Breast Carcinoma
T1weighted	-	Isointense	Low intensity	Heterogeneous Iso- or Hypointense	Hyperintense	Isointense	High intensity	Low intensity	High intensity signal	Low intensity	Low intensity
T2 weighted	-	Heterogeneous	Low intensity	Heterogeneous hyperintense	Hyperintense	Heterogeneous	High intensity	High intensity signal	High intensity signal	High intensity signal	High intensity signal
Fat suppression technique	-	Signal reduction	-	-	-	-	-	-	-	-	-
Invading the myocardium	-	-	-	-	Does not invade	-	-	Does not invade	Invade	Does not invade	Does not invade
Pericardial effusion	-	-	-	-	-	-	-	Usually	Usually	Usually	Usually
LGE	-	Heterogeneous	-	Heterogeneous enhancement	Heterogeneous	Heterogeneous	Sunray appearance	-	-	-	-

Fig. (2). Left- T2 weighted edema (triple inversion) sequence of the short-axis of the cardiac muscle showing hyperintense signal from the pericardium suggesting high water content (edema, acute inflammatory process) (Yellow arrows). The epicardial fat is appears dark on this sequence (red arrow head) and the blood signal is suppressed (Green circle). Right- triple inversion recovery edema (T2 weighted images) showing hyperintense signal from the parietal (thick yellow arrow) and visceral pericardium (thin yellow arrow) over the RV anterior wall and the apex. There is moderate pericardial effusion (red arrow). Pleural effusion also seen (arrow head). This is a unique sequence to judge the acuity of the pericarditis (see text). (The color version of the figure is available in the electronic copy of the article)

Fig. (3). Late gadolinium enhancement of short-axis of the heart showing mild hyperenhancement of the parietal and visceral pericardium (arrows) with pericardial effusion in between (arrow head).

reported to range from 94% to 100% [5, 20]. The use of gadolinium contrast during T2-weighted images with LGE imaging offers an additional method of detecting abnormal redistribution of water into the pericardial interstitium [21]. On the other hand, chronic fibrotic pericarditis, characterized by avascular pericardial layers with an abundance of collagen fibers and fibroblasts in the absence of vascularized granulation tissue, demonstrates pericardial thickening without enhancement [5].

In a recent expert analysis [21] the following groups of patients seem most likely to benefit from CMR imaging for pericarditis:

1. Patients with persistent chest pain after a negative ischemic evaluation.
2. Patients with non-ischemic chest pain suspicious for pericarditis, but no detectable effusion on 2-D echocardiography.
3. Patients with non-ischemic chest pain suspicious for pericarditis, but a poor quality or non-diagnostic 2-D echo study.

4. Patients with recurrent chest pain after a negative ischemic evaluation and a negative "non-cardiac" chest pain evaluation.
5. Younger patients with atypical chest discomfort and a low likelihood of coronary atherosclerotic disease.

This was based on data that have been recently published by Boniface *et al.* [22]. In that study a series of chest pain patients for evaluation of pericarditis by CMR using T2 weighted and LGE sequences. Evaluating the 44 patients in that study with documented acute pericarditis by CMR, 41% had not met current criteria for a diagnosis of pericarditis based on physical exam, EKG and TTE. Furthermore, 66% of these patients with CMR documented pericarditis had a small pericardial effusion on their CMR study, which was missed on a 2-D echo study. Thus, heightened clinical suspicion should direct appropriate imaging towards CMR and, as well will see, both diagnostic and prognostic value.

Boniface *et al.* [23] demonstrated that in 20% of the patients with persistent chest pain despite negative ischemic evaluation, CMR revealed evidence of pericarditis, undetected by standard diagnostic criteria. This diagnostic value for suspected acute pericarditis using CMR in patients with chest pain was highest among patients under age 40, and much less likely in patients over age 60 [21]. These data suggest that a substantial portion of patients with chest pain have pericarditis detectable by CMR imaging, but not by currently accepted diagnostic criteria.

Acute pericarditis is often accompanied by some degree of myocarditis, presumably as they share common etiologic agents; mainly cardiotropic viruses [15]. Recognition of associated myocarditis may be clinically relevant, and is a negative prognostic predictor in patients with pericarditis, often requiring hospitalization and a full etiologic search [24]. Myocardial involvement can be confirmed in the presence of elevated cardiac enzymes level and the presence of transient regional and global wall motion abnormalities [25, 26]. While widespread ST segment elevation is among the criteria for the diagnosis of acute pericarditis [14]. In our CMR experience, it is patients who have myocardial involvement in the setting of acute pericarditis who may manifest with diffuse ST segment elevation [27].

5. CARDIAC MAGNETIC RESONANCE IMAGING FOR CONSTRICTIVE PERICARDITIS

Constrictive pericarditis reflects a condition in which the compliance of the pericardium is decreased, which may result in impaired ventricular filling, severe diastolic dysfunction and right heart failure [28] and if not identified, progressive indolent death. Although subacute and even acute forms of pericardial constriction have been described, this disease usually clinically manifests years after an initial pathologic trigger. The spectrum of causes of pericardial constriction has shifted over time from infectious causes (particularly tuberculosis) to post-irradiation and postoperative forms, which have now become the most frequent causes of the disease [29, 30].

The diagnosis of constrictive pericarditis remains challenging. TTE remains the initial imaging test and can be sufficient to make the diagnosis especially when history, physical exam and blood work are suggestive of constrictive pericarditis. On the basis of its' ability for comprehensive morphologic assessment of the heart as well as the pericardium and hemodynamic characteristics, in our opinion, CMR should be reserved to 1. Those situations in which echocardiographic findings are equivocal; 2. In patients with increased inflammatory biomarkers or a short duration of constrictive symptoms (<3 months, usually); 3. Chronic undiagnosed constrictive symptoms and/or physical exam); 4. When the diagnosis remains equivocal by conventional invasive or non-invasive testing and 5. When coexisting myocardial disease is suspected, or comprehensive pericardial as well as cardiovascular anatomy is warranted for subsequent management decisions.

When used, CMR can assess the extent of pericardial inflammation. If the pericardial inflammation is intense, a trial of anti-inflammatory agents should be considered before pericardiectomy and has been shown to be remarkably valuable in reducing need for surgical pericardial stripping [4, 16, 19]. In addition CMR imaging offers an almost complete appreciation of constrictive pericarditis, with exception of demonstration of pericardial calcifications. First, all other causes of right heart failure (pulmonary hypertension, severe tricuspid insufficiency, myocardial infarction) can be excluded. Second, it allows determination whether the pericardium is causing constriction, thereby impeding cardiac fill-

ing. Third, it helps determine the optimal treatment (pericardial stripping vs. medical treatment) [18].

Encasement of the heart by a noncompliant, rigid pericardium leads to (1) dissociation between intracardiac and intrathoracic pressure, which isolates the heart from normal respiratory changes in intrathoracic pressure; (2) increased ventricular coupling; and (3) increased cardiac filling pressures with pressure equalization in all four cardiac chambers.

5.1. Morphology-non Compliant, Rigid Pericardium

A new description of specific forms of constrictive syndromes has been introduced: 1. Transient constrictive pericarditis, 2. Effusive-constrictive pericarditis 3. Chronic constrictive pericarditis [31]. Distinguishing transient vs. chronic constrictive pericarditis is crucial as patients with transient constrictive pericarditis may improve with conservative management using non-steroidal inflammatory drugs and colchicine [31]. CMR (and occasionally ^{18}F -labeled fluorodeoxyglucose PET/CT) are the only imaging modalities that have that distinction capability, which impart a diagnostic and therapeutic function to CMR [31, 32]. In chronic constrictive pericarditis there is prominent enhancement following contrast material administration, whereas acute pericardial enhancement may also be suggestive of a transient constrictive pericarditis phenomenon responsive to pharmacologic intervention [4, 5, 19] (Fig. 4). Differentiation between pericardial thickening and effusion is usually straightforward on CMR images.

The chronically thickened fibrotic and/or calcified pericardium has low signal intensity on T1-weighted (Fig. 5) and T2-weighted spin-echo MR images and at SSFP cine imaging (Fig. 6). Although pericardial thickness is traditionally used as an important criterion for constrictive pericarditis (pericardial thickness ≤ 3 mm is normal, >4 mm is suggestive of pericardial constriction in patients with the appropriate clinical presentation, and >5 -6 mm is highly specific for constriction [33-35]), this concept has been increasingly challenged. Two studies have found that pericardial thickness in transient constrictive pericarditis was significantly higher (>4 mm) than pericardial thickness in chronic constrictive pericarditis (<2 -3 mm) [4, 19]. Those two studies have further underscored the importance this new classification of constrictive pericarditis syndromes and the value of

Fig. (4). Late gadolinium enhancement sequence of a short-axis view (left) and four-chamber view (right) of the cardiac muscle shows hyperenhancement of the pericardium (arrows). This post-contrast sequence conveys the chronicity of the pericarditis and the subsequent fibrotic conversion.

Fig. (5). Black blood T1 weighted (Double inversion recovery) showing thick pericardium in a patient with pericarditis. Blue arrow indicates the thickened pericardium. Yellow arrow indicates epicardial fat. Red arrow indicates pericardial fat. (The color version of the figure is available in the electronic copy of the article).

Fig. (6). Steady-State Free Precession (SSFP) showing very thick pericardium in a patient with pericarditis. Left; 3-chamber and right; short-axis views.

contrast-enhanced MR imaging in depicting residual inflammation in patients with constrictive pericarditis [4, 19]. It should also be noted that the degree of pericardial thickening is only weakly related to the degree of cardiac constriction [14].

5.2. Dissociation Between Intracardiac and Intrathoracic Pressure

The pericardium functions as a pressure transducer between the pleural spaces and cardiac chambers. In normal conditions, the intra-thoracic respiratory changes are directly transmitted to the cardiac chambers. In patients with constrictive pericarditis, the pulmonary capillary wedge pressure is influenced by the inspiratory fall in intra-thoracic pressure, while the left ventricular pressure is shielded from respiratory pressure variations by the pericardial scar. Thus, inspiration lowers the pulmonary capillary wedge pressure, and presumably left atrial pressure, but not left ventricular diastolic pressure, thereby decreasing the pressure gradient for ventricular filling. The less favorable filling pressure gradient during inspiration explains the decline in filling velocity [36].

Although the hemodynamic consequences are assessed in clinical practice by means of echocardiography and cardiac catheterization, CMR imaging has great potential, because information regarding pericardial-cardiac morphology and tissue characteristics can be merged with functional hemo-

dynamic information [14, 36, 37]. Phase-contrast CMR imaging of the tricuspid valve inflow shows a restrictive filling pattern of enhanced early filling and decreased or absent late filling, depending on the degree of pericardial constriction and increased filling pressures. Also, flow in the inferior vena cava shows restrictive physiology with diminished or absent forward—or even reversed—systolic flow, increased early diastolic forward flow, and late reversed flow. Constrictive pericarditis, in contrast to restrictive cardiomyopathy, is typically characterized by a strong respiratory-related variation in cardiac filling (*i.e.*, enhanced right ventricle filling on inspiration, enhanced left ventricle filling on expiration). Real-time phase-contrast CMR imaging is an attractive alternative to Doppler echocardiography to assess the effects of respiration on cardiac filling [38] (Fig. 7).

5.3. Increased Ventricular Coupling

In patients with constrictive pericarditis, total cardiac volume is fixed by the noncompliant pericardium. The septum is not involved and can therefore bulge toward the left ventricle when left ventricular volume is less than that on the right. As a result, ventricular interdependence is greatly enhanced at early diastolic filling [39-42].

Reciprocal changes occur in the velocity of right ventricular filling [36, 43]. These changes are mediated by the ventricular septum, not by increased systemic venous return. Abnormalities are most pronounced in the basal septum,

Fig. (7). Real time CMR imaging. Short-axis of LV and RV demonstrating the septal wall motion (Septal bounce) with interventricular interdependence. During inspiration (left) there is a decrease in intrathoracic pressure with consequent increase in IVC/SCV flow into the RA/RV tending to drive septal forces leftward that are reversed in expiration (right) due to the restricted pericardium and the pulmonary veins being outside the pericardium. With exaggerated real-time CMR images, this finding is nearly pathognomonic for constrictive pericarditis. Note the not subtle migration of the septum with no effective change in total ventricular volume.

Fig. (8). Radio-frequency tissue-tagging sequence demonstrating the lack of slippage between the parietal and visceral pericardium (adhesion) along the right ventricular base (arrows). The systolic frames have not revealed clear slippage planes between the visceral and parietal pericardium as evidenced by the distortion of the otherwise cuboidal tag interspaces diagnostic and pathognomonic for focal constrictive anatomy.

leading to an S-like septal motion on a horizontal long-axis view.

The clinical potential of novel high temporally resolved Real-Time cine SSFP sequences to study the effects of free breathing on ventricular interdependence was recently studied [6]. Patients with constrictive pericarditis demonstrate the typical respiratory pattern of septal abnormalities, while those with restrictive cardiomyopathy showed a pattern similar to that of healthy volunteers. Quantification of the total septal excursion between inspiration and expiration was very helpful in differentiating between constrictive pericarditis and restrictive cardiomyopathy [44]. Moreover, patients with inflammatory pericarditis also often show increased septal excursion, most likely related to the decreased compliance of the inflamed pericardial layers. The added value of real-time imaging during free breathing to evaluate the hemodynamic effect of the pericardium on cardiac filling has become an essential part of CMR examination [26, 45].

More recently Radio-frequency (RF) tissue tagging has been suggested to possess certain morphological characteristics that naturally lend themselves toward distinction of vis-

ceral-partial attributes [46]. RF tissue tagging is an imaging technique that can image motion via spatially modulating the degree of magnetization prior to imaging following the intrepid initial observation of Axel *et al.* [47]. CMR utilizes RF tissue-tagging to facilitate identification of constrictive pericarditis positive patients by defining visceral-parietal adherence patterns [7]. Whereas in normal pericardium, tag lines rapidly become discontinuous during the cardiac cycle due to shear motion of the inner and outer pericardial layer, persistence of these tag lines is indicative of fibrotic fusion. We have recently shown 100% agreement between cardiac magnetic resonance *via* RF tissue tagging–defined constrictive pericarditis positivity and postsurgical findings [48] (Fig. 8) which may become the new ‘gold standard’ when further validated Table 3.

6. CARDIAC MAGNETIC RESONANCE IMAGING OF PERICARDIAL EFFUSION

Pericardial effusion may occur in patients with heart failure, renal and liver insufficiency, inflammation, infection, neoplastic disease, trauma, and myocardial infarction [14]. Cardiac imaging is used to confirm the presence, severity,

Table 3. CMR features that could influence the management of constrictive pericarditis.

	LGE	No LGE
Pericardial thickness > 4mm	Colchicine and NSAIDs similar to acute pericarditis	Unlikely to benefit from Colchicine and NSAIDs
Pericardial thickness ≤ 4 mm	Suggest longer duration of colchicine and NSAIDs (> 6months)	Pericardiectomy

and extent of fluid, to characterize the nature of fluid (transudates vs. exudates); to rule out pericardial inflammation, to determine the hemodynamic effect on the heart, and ultimately to guide pericardiocentesis.

Although Transthoracic Echocardiography (TTE) is the preferred first-line modality for this evaluation due to its' widely recognized cost effectiveness, bedside availability, widely availability and comprehensive assessment of both anatomy and physiology. However, CMR imaging can provide incremental information to the information obtained from TTE [8]. Incremental information provided by CMR includes: 1. Quantification and localization of the pericardial fluid. 2. Differentiation between exudative and transudative pericardial effusion and 3. Assessment of the pericardial layers while determining the etiology of acute pericarditis.

CMR allows quantification and localization of the pericardial fluid (Fig. 9). For instance, a pericardial space anterior to the right ventricle that is greater than 5 mm corresponds to a moderate effusion of 100-500 mL of fluid [18, 49]. This is particularly helpful to assess response to treatment, as it delineates the distribution and amount of pericardial fluid more precisely than echocardiography [18, 50].

Fig. (9). Steady-state Free Precession (SSFP) sequence of short-axis view of the heart with moderate sized nearly constrictive pericardial effusion (Red arrow). The parietal and visceral pericardiums (yellow arrows) are separated with the pericardial fluids. (The color version of the figure is available in the electronic copy of the article).

Characterization of pericardial fluid can, to some extent, be achieved by measuring signal intensity on MR images. Transudates typically manifest with low signal intensity on T1-weighted MR images and with high signal intensity on

T2-weighted images. Exudates, having high protein and cell content, increase the rate of T1 relaxation (higher signal intensity) and shorten the rate of T2 relaxation (lower signal intensity). Hemopericardium can be suspected in patients who have previously undergone aortic or cardiac surgery or who have a history of trauma or neoplastic disease and is characterized by high signal intensity on T1-weighted images and inhomogeneous low signal intensity on cine SSFP images. Because of motion artifacts, however, precise pericardial fluid characterization is not always feasible. Bright-blood dynamic cine MR imaging often allows a better appreciation of the intrapericardial contents, such as the visualization of fibrinous strands or of the presence of coagulated blood [18].

CMR also enables accurate depiction of the pericardial layers, with assessment of thickness and composition. As such, MR imaging allow the differentiation of simple pericardial effusions from inflammatory effusive pericarditis or malignant pericardial diseases [51].

7. CARDIAC MAGNETIC RESONANCE IMAGING IN CONGENITAL PERICARDIAL LESIONS

Congenital anomalies of the pericardium include pericardial cysts, diverticula, and an absence of pericardium.

7.1. Pericardial Cyst and Diverticulum

Pericardial cysts are rare mediastinal masses that have an incidence of 0.01% and are most often located in the right cardiophrenic angle [13, 52]. They represent up to 6% of mediastinal masses [31]. Fusion abnormalities of the mesenchymal lacunae result in the formation of both pericardial cysts and diverticula with cysts being three times more common than diverticula [53].

On CMR imaging, pericardial cysts are seen as homogeneous, thin walled, well-outlined masses with characteristic low to intermediate signal intensity on T1-weighted sequences and high intensity on T2-weighted sequences [49, 53]. However, cysts with a hemorrhagic component or elevated protein levels may show medium or high signal intensity on T1-weighted sequences [13]. Pericardial cysts characteristically do not enhance with administration of gadolinium [54] (Fig. 10a, b, c, d).

Some key differences between pericardial cysts and diverticula exist. In contrast to cysts, diverticula may vary in size with changes in body positions and during respiratory cycle [53]. Furthermore, cysts do not communicate with the pericardial space, whereas diverticula will [31].

Care must be taken to distinguish between inflammatory pseudocysts, encapsulated and loculated pericardial effusions

Fig. (10). **a.** Late gadolinium enhancement shows no uptake of the pericardial cyst behind the right atrium (Blue arrow) (upper left). **b.** Triple inversion recovery (T2) demonstrates a hyperintense signal from the retro-right atrial pericardial cyst (Arrow) (upper right). This is a classical area in the right costophrenic angle for a benign pericardial cyst. **c** and **d** shows SSFP of the pericardial cyst in the coronal and axial position depicting their typical location in the right costophrenic angle (90% in right costophrenic angle vs. 10% in left costophrenic angle).

caused by rheumatic disorders, bacterial infection, trauma, or cardiac surgery and congenital pericardial cysts [31].

7.2. Congenital Absence of Pericardium

Absence of pericardium is a rare but often confusing congenital heart disease that is classified into five subtypes including complete absence of the pericardium, complete left or right-sided absence, and partial left or right-sided absence. Complete absence of left side is the most common subtype [24, 55, 56]. Various complications including sudden cardiac death have been reported from absence of pericardium [57]. Deficiencies of pericardium can be difficult to diagnose as normal pericardium is <2mm in width and may not be well visualized [57] with echocardiography, impossible via cardiac catheterization and nuclear techniques and can be anatomically suspected by CT. CMR is the modality of choice.

The diagnosis can be made using MR imaging using specific protocols. It is proposed to use dark blood T1-weighted whole heart stack in two planes, axial and sagittal or coronal, cine SSFP in standard long-axis views, whole heart short-axis and axial stacks and real-time cine imaging to evaluate for paradoxical septal motion with a slice thickness of 4-6 mm [57]. Diagnostic findings include an absence of the pericardial layer, marked and extreme levorotation of the heart in the absence of dilation, interposition of lung tissue in the anterior space between aorta and pulmonary artery or between the diaphragm and the base of the heart and presence of subepicardial myocardial crease due to external pressure from a foramen type pericardial defect [8] (Fig. 11).

CMR imaging also has the potential to identify high-risk features that would suggest the risk of sudden cardiac death

including left ventricular myocardial crease or hinge point, inducible ischemia on stress perfusion imaging and evidence of left atrial appendage herniation [57].

Fig. (11). Black-blood T1-weighted spin-echo MR imaging performed on asymptomatic 21 year old male who had levoposition of the heart in the posteroanterior projection of a CXR. He was found to have congenital absence of entire pericardium.

8. PERICARDIAL TUMORS

Pericardial and cardiac tumors are a rare entity with an incidence of up to 0.03% based on previous autopsy series [58]. The tumors may be classified as benign or malignant, primary or secondary based on origin or using a combination

of these terms [53]. Primary tumors of the pericardium occur much less frequently than secondary tumors [18].

Patients with pericardial tumors may present with a myriad of clinical symptoms including chest pain, shortness of breath, palpitations and physical findings which may be concerning for acute pericarditis or pericardial tamponade [59]. These patients may appropriately undergo initial evaluation with use of TTE, which may further dictate a need for cross-sectional imaging to further characterize these lesions [8].

Primary pericardial tumors may be benign or malignant. Benign primary tumors can be found in both the parietal and visceral pericardium as discrete pedunculated or sessile masses. These include soft tissue tumors such as teratomas, lipomas, fibromas, hemangiomas, and lymphangiomas. The most common primary malignant tumors of the pericardium are malignant mesothelioma and angiosarcoma [8]. CMR can not only precisely outline anatomical relations of tumors but also accurately characterize some primary soft-tissue tumors like lipomas, liposarcomas, fibromas, and angiosarcomas secondary to their characteristic features [18, 53].

Lipomas are isointense on T1 weighted sequences, heterogeneous on T2 weighted and demonstrate a heterogeneous enhancement using gadolinium contrast [60]. Lipomas also demonstrate signal reduction when using fat suppression techniques. Fibromas characteristically lack complex vascularization, which is inherent of angiosarcomas. Thus, fibromas with hypo-intense signals on T1 and T2 weighted images, lack the heterogeneous medium and high signal intensity on T1 and T2-weighted sequences and heterogeneous “sunray” gadolinium enhanced appearance of angiosarcomas [53]. Mesotheliomas appear homogeneously isointense on T1-weighted images, heterogeneous on T2-weighted images due

to focal areas of necrosis and enhance with gadolinium contrast administration [61].

Lymphomas, melanomas, lung carcinoma and breast carcinoma are some secondary tumors of the pericardium. Metastatic lesions to the pericardium are often associated with serosanguinous appearing effusions. Apart from malignant melanoma, which classically involves the myocardium, most secondary tumors of pericardium do not invade the myocardium [13, 62] (Fig. 12).

Secondary-malignant pericardial tumors have low signal intensity signals on T1 weighted imaging like the myocardium and increased signal on T2 weighted imaging [8]. An exception to low T1 signal intensity is malignant melanoma which shows a high-intensity signal on T1 weighted imaging due to melanin binding metallic compounds [8, 53].

CMR can also be used to further differentiate hematomas from tumors. Furthermore, diffuse and heterogeneous uptake of gadolinium may identify areas of tumor necrosis or active growth [53]. Hemorrhagic pericardial effusions, which are disproportionately large to the size of the tumor mass, are common in pericardial metastases and usually demonstrate high signal intensity on T1-weighted images [8].

Despite the ability to define tumors so precisely, contraindication of gadolinium use in patients with glomerular filtration rates <30ml/min and gating in tachycardia have some challenges faced during MR imaging of pericardial tumors although in our practice, the life-threatening importance of defining pathology, surgical margins and association to adjacent organs while providing surgical roadmaps trump the very low risk of Nephrogenic Sclerosing Fibrosis (NSF). Some gadolinium agents such as MultiHance (gadobenate

Fig. (12). A. Steady-state free precession (SSFP) 2 chamber (upper left) B. Steady-state free precession (SSFP) short-axis (upper right) of the LV showing an epicardial mass (arrow) over the anterior and anterolateral walls of the left ventricle in a 65-year-old male with lymphoma with pericardial involvement. C Black blood double inversion (T1 weighted) (left lower) sequence of the short axis of the heart showing the epicardial mass (lymphoma) as isointense. D (right lower) triple inversion edema (T2 sequence) short-axis of the heart showing slight hyperintensity of the epicardial mass (lymphoma).

Fig. (13). Radio-frequency tissue tagging sequence performed on short-axis LV showing diastolic frame (left) and systolic frame (right) for the 65-year-old male who had pericardial lymphoma with myocardial extension. Notice the absence of separation line (adherence) between the pericardial mass and the LV anterior wall and the continuity of the tag lines with the twist and rotational motion of the LV (between the mass and the pericardium/epicardium). This confirmed the invasive character of the epicardial mass leading to the final surgical opinion that the mass was inoperable. Despite radiation therapy and chemotherapy, the patient passed away 4 months later.

dimeglumine; Bracco, Princeton, NJ) have never had a single episode of NSF serving as a preferential gadolinium agent in such cases.

We also note that CMR may also be used in the characterization of the composition of cardiac masses in the setting of infective endocarditis [63]. Here, contrast-enhancing lesions may identify subendocardial perimyocardial involvement [64]. Cine images may identify floating dynamic masses or vegetation in a manner similar to echocardiography [64]. Complications like abscesses, aneurysms, dissections, or fistulas are identified, and, using multiple sequences, the composition of masses may be characterized comprehensively to distinguish thrombus, infectious vegetation, or even a tumor.

9. FUTURE DIRECTIONS

The advent of rapid gradient-echo MRI was followed by the introduction of ECG-gated gradient-echo sequences that revolutionized CMR studies by initiating and stimulating a large range of new techniques for functional assessments. Current state-of-the-art CMR relies on EKG-synchronized cine acquisitions with balanced SSFP contrast, typically at a magnetic field strength of 1.5 T [65-67]. However recent work at both 1.5T and 3T reports significant potential for real-time CMR of cardiac function [68] and cardiovascular flow [69, 70]. Real-time CMR not only improves patient compliance because of free breathing and eventually shorter examination times, it also offers extended diagnostic opportunities by providing functional information about individual cardiac cycles and access to immediate physiologic responses to stress and exercise. However real-time CMR at 3 T is frequently affected by off-resonance “banding” artifacts due to the magnetic field in homogeneities [71], so there has been proposed a method that employs a highly undersampled radial gradient-echo CMR technique with fully balanced gradients [72] in conjunction with serial image reconstruction by nonlinear inversion. Finally, as shown in limited experience, conversion of surgical constriction into medical management utilizing pharmacological strategies forestalling or negating surgical considerations in concert with CMR is a

tantalizing concept taking hold and we expect CMR to play a pivotal role in this approach.

CONCLUSION

CMR has emerged as an imaging modality that allows visualization and tissue characterization of the pericardium. Pericardium morphology is evaluated using dark-blood T1-weighted fast spin-echo and bright-blood cine SSFP imaging. Dark-blood T2-weighted images with LGE imaging offers an excellent method for diagnosis of acute pericarditis especially when chest pain is of unknown etiology. In addition, CMR using phase contrast and cine SSFP sequences has a great potential for diagnosis of constrictive pericarditis. Recently we showed that CMR *via* RF tissue tagging offers a unique, efficient and effective manner of defining clinically and surgically relevant constrictive pericarditis. Dovetailing the diagnostic and therapeutic opportunities creates and, as yet, soon to be realized capability for merging imaging with outcomes. As shown for many pathologies, CMR has been shown to define insightful mechanisms of disease, heretofore unrecognized leading to more prescient pharmacologic and surgical interventions; intervening when necessary but not intervening when inappropriate. The ‘unmasking’ by CMR, we proposed, now leads the interested clinician to utilize advanced CMR in a relevant clinical manner solving many of the vexing problems in pericardial diseases.

LIST OF ABBREVIATIONS

CMR	=	Cardiac Magnetic Resonance imaging
SSFP	=	Balanced Steady State Free Precession
TIR	=	Triple Inversion Recovery

CONSENT FOR PUBLICATION

Not applicable.

CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

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