

Assessment of Pericardial Diseases and Cardiac Masses with Cardiovascular Magnetic Resonance

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Abstract

Imaging plays an important diagnostic and prognostic role in the assessment of pericardial diseases and cardiac tumors and in differentiating these conditions from other cardiac and noncardiac diseases. A number of imaging modalities are available for this task; each has advantages and limitation. Cardiovascular magnetic resonance (CMR) is a highly versatile imaging modality that provides detailed anatomical information, tissue characterization, cardiac function assessment, and evaluation of the impact of these conditions on hemodynamics. In this review we focus on the current state-of-the-art application of cardiovascular magnetic resonance in assessing pericardial diseases and cardiac tumors. (Prog Cardiovasc Dis 2011;54:305-319)
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Advanced imaging modalities have greatly facilitated the diagnosis of pericardial diseases and the detection of cardiac masses. An integrated, multimodality assessment is often necessary to provide all the clinicians with complete information. This review will highlight the role that cardiac magnetic resonance (CMR) plays in such clinical scenarios.

Special considerations on pericardial imaging

CMR is a very well suited and highly versatile imaging modality to visualize fine structures such as the pericardium¹ or to provide accurate evaluation of a mass and tissue characterization. In addition, it provides excellent assessment of cardiac function and hemodynamics.

There are, however, some technical issues that can compromise accurate image collection; these need to be

understood and considered when images are acquired. It is important that the imaging personnel know in advance those details of pericardial disease that are being looked for. The presence of epicardial and pericardial fat in CMR imaging is advantageous, as fat gives bright signal intensity on T1- and T2-weighted images, in contrast to the low signal intensity arising from pericardium.² Sometimes the pericardium can be obscured by the chemical shift or cancellation artifacts; these need to be recognized. A technicality applicable to CMR is that the slice thickness of routine imaging is 7 mm as it is usually optimized for myocardial and not pericardial imaging. The normal pericardial thickness reported on CMR is up to 4 mm.³ These factors can contribute to some partial volume effect. Several CMR sequences are used to image the pericardium. On turbo-spin echo (TSE) images the pericardium gives a relatively low intensity signal,⁴ in contrast with the high signal intensity given by the surrounding fat, on both T1- and T2-weighted imaging. Pericardium is also seen as low signal intensity on the steady-state free precession (SSFP) sequences. In addition, it is more easily distinguished at end-systole during cine imaging. Cine imaging also allows assessment of the pericardial mobility. In acute inflammation, the pericardium will appear very bright, due to a high

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Abbreviations and Acronyms

CMR = cardiac magnetic resonance
CT = computed tomography
IVRT = isovolumic relaxation time
MSCT = multislice computed tomography
SSFP = steady-state free precession
TSE = turbo spin echo

amount of water, therefore giving a persistent high signal on T2-weighted imaging. If doubt persists, the pericardium can be “separated” by the surrounding fat by applying a fat saturation pre-pulse in addition to T2-weighted imaging (short tau inversion recovery). An inflamed pericardium will also enhance

with administration of gadolinium contrast, appearing very bright on early and late gadolinium-enhanced images.^{5,6} Real-time free breathing cine loops will demonstrate abnormal motion of the right ventricle free wall or the interventricular septum, which enhances with breathing.⁷ Finally, tagging can be applied to the pericardium to assess its mobility.⁸

Assessment of the pericardium**Brief review of anatomy and physiology**

As a thin, elastic structure surrounding the heart, the pericardium has an important contribution to cardiac physiology, governing the diastolic interaction between the 2 ventricles. When diseased, the restraining effect of the pericardial apparatus can lead to significant morbidity and culminate into life-threatening situations. The 2 serosal

Table 1

Etiology of pericardial disease

1. Congenital abnormalities
a. Partial or complete absence of the pericardium
b. Pericardial cysts
c. Pericardial diverticulum
2. Infections
a. Viral (Echo, Coxsackie, adenovirus)
b. Bacterial: <i>Mycobacterium tuberculosis</i> , gram-positive cocci
3. Postcardiac surgery
4. Post-radiotherapy
5. Trauma
6. Post-myocardial infarction
7. Connective tissue diseases: SLE, rheumatoid arthritis
8. Granulomatous disorders: sarcoidosis
9. Malignant conditions
a. Primary
b. Secondary
10. Metabolic: uremia
11. Idiopathic

layers (with a virtual space in between, accommodating 10–50 mL of fluid) and the fibrous pericardium taken together have a thickness between 0.5 and 1 mm.⁹ The pericardium extends up 3 to 4 cm onto the great vessels and at this level it reflects as the transverse sinus. Behind the atria, at the level of the great veins it reflects as the oblique sinus.^{9,10} The pericardium anchors the heart in the thorax onto the diaphragm inferiorly, the spine posterior, and the sternum anterior. Apart from the anchoring function, it also provides an important mechanical function, facilitating the heart motion within the pericardial sac in the presence of the normal pericardial fluid.¹¹ This range of motion is relatively limited, a fact that contributes to the phenomenon of ventricular interdependence.

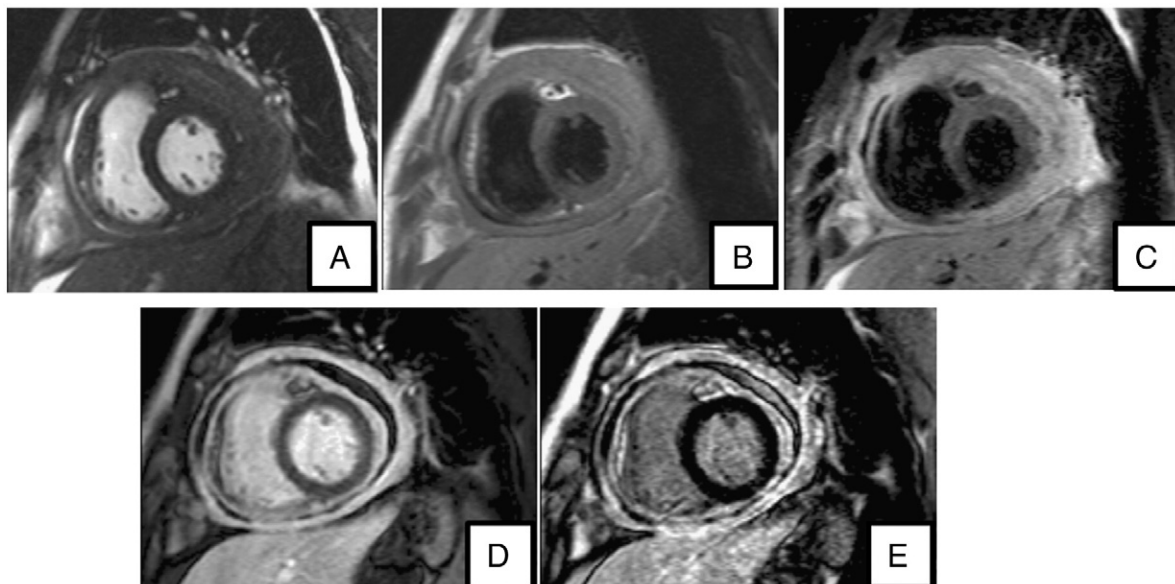


Fig 1. Serial short axis view of SSFP, T1W-TSE, T2W-TSE with fat suppression, early and late after gadolinium administration (A to E, respectively), demonstrating thickening of both pericardial layers with a small pericardial effusion. Note that pericardium enhances with contrast.

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