



Role of Cardiac MRI and CT in Pericarditis and Pericardial Constriction

REVIEW

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ABSTRACT

Pericardial diseases span a spectrum from acute and recurrent pericarditis to chronic constrictive pericarditis. While echocardiography remains the first-line test in the evaluation of patients with suspected pericardial disease, advanced cardiac imaging with cardiac magnetic resonance and computed tomography have become complementary and often essential in refining diagnosis and guiding management in many patients. The recent Concise Clinical Guidance Statement from the American College of Cardiology as well as updated European Society of Cardiology guidelines have given new emphasis and recommendations on the use of advanced imaging in both pericarditis and pericardial constriction. This review summarizes the techniques, indications, and recent studies on the use of advanced cardiac imaging in pericarditis and pericardial constriction.

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OVERVIEW OF PERICARDITIS AND PERICARDIAL CONSTRICTION

Acute pericarditis is an inflammatory disorder involving the visceral and parietal layers of the pericardium, with diverse causes and clinical courses of resolution or progression.^{1,2} While acute pericarditis represents about 5% of emergency department admissions for chest pain, delays in diagnosis can occur when emergent causes of chest pain, such as cardiac ischemia and pulmonary embolism, are over-emphasized in the workup. In previous clinical guidelines, a diagnosis of pericarditis was established in the presence of two out of four key criteria: (1) pleuritic and positional chest pain, (2) pericardial friction rub on exam, (3) electrocardiogram (ECG) changes (widespread ST segment elevation, PR depression), and (4) the presence of pericardial effusion on echocardiography.³ However, the sensitivity of ECG and echocardiographic findings is suboptimal, and 30% to 40% of patients with acute pericarditis may not have typical ECG changes or a pericardial effusion. In these cases, further supportive evidence must be sought, such as elevated inflammatory markers (C-reactive protein and erythrocyte sedimentation rate) and neutrophilic leukocytosis. Even so, the disease pathogenesis may involve various mechanisms, and inflammatory markers may be normal in about a quarter of patients.⁴ Given these limitations and progress in imaging techniques, cardiac magnetic resonance (CMR)—which directly evaluates pericardial thickening and inflammation—has revolutionized the care of patients with pericarditis and recently became a “fifth criterion” in its diagnosis (Figure 1).²

Constrictive pericarditis is an uncommon and frequently missed condition where a rigid and thickened pericardium impairs diastolic ventricular filling, leading to venous congestion, anasarca, recurrent pleural effusions and ascites. It often presents as a heart failure syndrome with predominant right heart failure symptoms.

Echocardiography is the first-line test for evaluation of constriction. Established echocardiographic criteria are often sufficient for making the diagnosis when done in a comprehensive fashion and interpreted with caution and expertise.⁵ Cardiac catheterization is used depending on case complexity and level of certainty after clinical and echocardiographic assessment. However, patients with suspected constriction who have coexisting restrictive cardiomyopathy, right ventricular dysfunction, tricuspid regurgitation, or confounding factors can present a challenge in the confirmation of pericardial constriction solely by echocardiography. As a result, CMR and cardiac computed tomography (CT) have also become useful and often necessary tests to confirm the diagnosis in uncertain or difficult cases, to define reversibility of constriction, and to aid in operative planning.

CMR TECHNIQUES AND OUTCOME STUDIES IN PERICARDIAL DISEASE

CMR provides a comprehensive evaluation of the pericardium, including pericardial thickening, pericardial masses, evaluation of pericardial effusion size, hemodynamic impact, and likely nature (exudative, transudative, hemorrhagic). Perhaps more importantly, CMR evaluation of pericardial tissue characteristics including edema, inflammation, and fibrosis has elevated its role in pericarditis care. In the recent Concise Clinical Guidance (CCG) document from the American College of Cardiology,² the diagnosis of pericarditis requires pleuritic chest pain or equivalent as a key criterion combined with the aforementioned classic criteria, but CMR-based late gadolinium enhancement (LGE) is now a key criterion that can be used to confirm or refute the diagnosis, particularly in uncertain cases. The CCG recommends CMR for recurrent

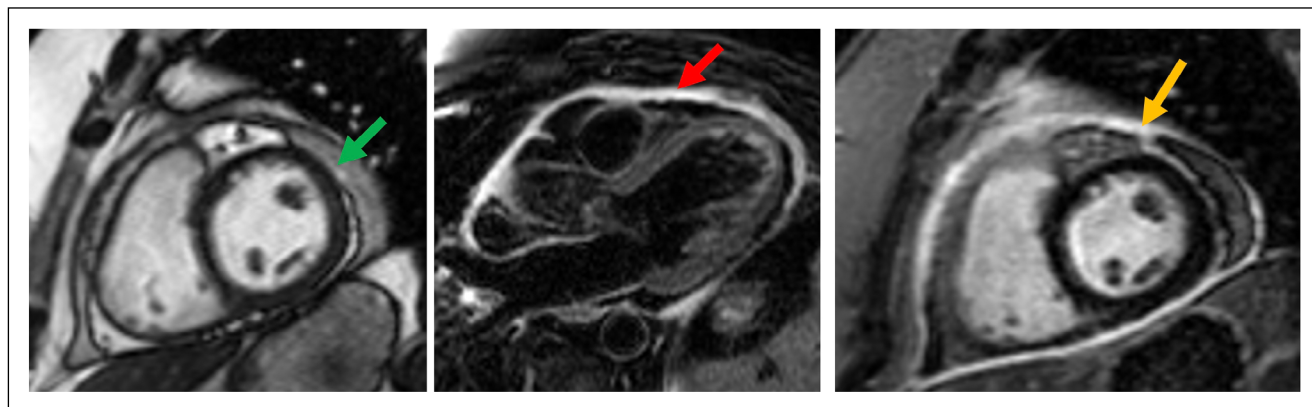


Figure 1 Case example of a 32-year-old man with acute pericarditis. Cine-CMR (left panel) shows a pericardial effusion laterally with heterogeneous signal suggesting an exudative effusion. The middle panel shows T2-weighted imaging with fat suppression showing hyperintensity of the pericardium (red arrow). The right-sided panel shows LGE imaging demonstrating circumferential pericardial LGE (orange arrow). CMR: cardiac magnetic imaging; LGE: late gadolinium enhancement

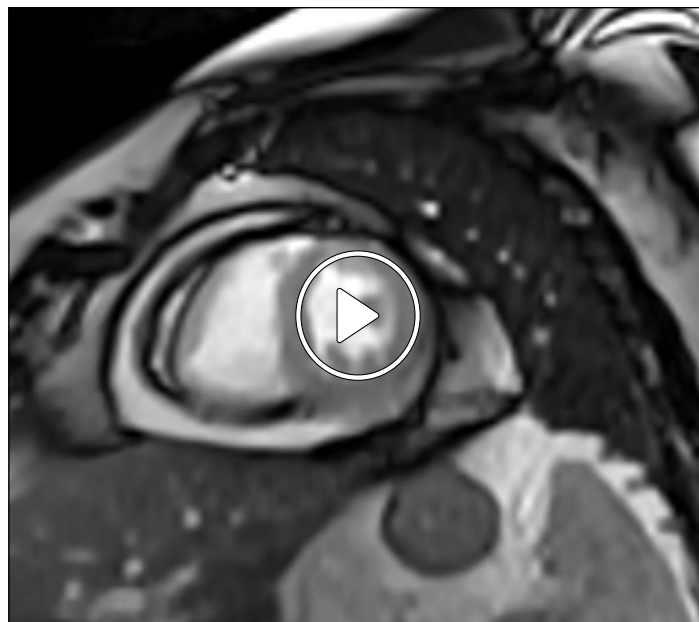
or incessant pericarditis and is considered reasonable in acute pericarditis. CMR also provides an added benefit of assessing presence and degree of coexisting myocarditis.

The CMR protocol to assess pericardial disease can be tailored depending on the assessment goals (ie, pericarditis versus pericardial constriction) but is generally similar for most patients with pericardial disease. The protocol begins with cine imaging using steady-state free precession (SSFP) sequences to assess chamber size, function, and pericardial effusion size and impact. The features of the effusion on cine imaging can suggest whether it's transudative (bright homogenous signal) versus exudative or hemorrhagic (less bright heterogenous signal with blood/fibrinous products in the pericardial space as well as adhesions). A diastolic septal bounce and conical/tubular deformity of the ventricles also aid in diagnosing constrictive pericarditis or constrictive physiology. Next, free breathing real-time cine-CMR is performed, typically in a short-axis slice also demonstrating the diaphragm, to assess for respirophasic paradoxical septal motion and enhanced ventricular interdependence with breathing (Video 1). The sequence is important in both acute pericarditis (to assess for concomitant constrictive physiology), and in the evaluation of constrictive pericarditis patients. Tethering of the myocardium against a thickened/inflamed pericardium can be evaluated as well.

The protocol is usually followed by anatomical assessment using T1- and T2-weighted turbo spin echo sequences (with fat suppression in T2-weighted sequences) in standard long- and short-axis cardiac views to define

pericardial tissue and pericardial fluid characteristics. A normal pericardium is seen as a thin, hypointense curvilinear structure outlined by hyperintense mediastinal and epicardial fat, with normal thickness of < 2 mm. The pericardium may not be seen with standard imaging in some segments if it has normal thickness and directly overlies the epicardium without the presence of epicardial fat. The true normal thickness of the pericardium is approximately 1 mm, which is often below the standard spatial resolution of CMR studies, whereas abnormal thickening is usually defined as > 3 to 4 mm (> 4 mm is definite thickening). Measurement of pericardial thickness is done on spin echo sequences, and reporting pericardial thickening should incorporate the entirety of the pericardium (usually on an axial stack of images) as it could be varied or localized. Care should be taken to avoid overestimating the pericardial thickness on CMR, which may occur with motion of pericardial layers, chemical shift artifact at the interface of fat and fluid (if measured on SSFP sequences), and relatively lower spatial resolution.⁶ Additional caution is needed because pericardial calcification can be missed on CMR since calcifications do not develop a signal and appear as hypodense areas within the pericardium; therefore, assessment of calcification is best done on CT.

The black blood sequences are also used to define the content of pericardial fluid when present. Transudative effusions typically demonstrate low T1 and high T2 signal, whereas exudative effusions show intermediate T1 signal. Hemorrhagic effusions are characterized by high T1 signal depending on the acuity and a heterogeneous



Video 1 Real-time free breathing short-axis cine cardiac magnetic resonance shows respirophasic motion of the interventricular septum and enhanced ventricular interdependence. Note the shift of the septum towards the left ventricle during inspiration; see also at <https://vimeo.com/1161598636/ca19594af5>.

appearance. Despite these features, overlap exists, and fluid analysis remains the reference standard. T2-STIR (short tau inversion recovery) sequences are traditionally used to assess pericardial edema, and they vary in their findings depending on the acuity of pericarditis—which is often abnormal with acute or subacute pericarditis but can be normal in chronic or lower grade pericarditis, while pericardial LGE is still present. In our experience, T1- and T2-weighted black blood spin echo and T2-STIR sequences are not always required for a thorough and accurate evaluation. Assessment of pericardial thickness can be done on rapidly acquired axial stacks of black blood single shot acquisitions (forgoing the need for T1-weighted spin echo); T2-weighted imaging suffers from artifacts and can be falsely negative or positive for pericardial inflammation, while pericardial LGE assessment (discussed below) is more reliable and often sufficient to make the diagnosis.

After contrast administration, LGE sequences are a key portion of the exam and are used to evaluate presence, degree, and location of pericardial enhancement. The presence and degree of pericardial LGE correlates with an increased risk of recurrent pericarditis.^{2,7,8} In pericarditis, mesothelial cell desquamation is followed by formation of granulation tissue and neovascularization in the pericardial tissue, and gadolinium contrast is retained in the pericardial tissue. Both the visceral and parietal pericardium can be evaluated in many patients (Figure 2). Resolution of pericarditis can be demonstrated as well on follow-up CMR after medical therapy (Figure 3).

Pericardial LGE can be difficult to distinguish from epicardial and pericardial adipose tissue. Comparison of LGE images against matched SSFP and anatomical sequences is helpful to differentiate the two tissues. In addition, LGE sequences with fat suppression—via the double spectral attenuated inversion recovery technique—have been developed and are now in clinical use.⁹ These sequences are now recommended when available to differentiate pericardial LGE from pericardial fat and increase diagnostic accuracy (Figure 4).

The ACC CCG also recommends a pericardial LGE grading scheme (mild, moderate, and severe) based on evaluating three short-axis slices (basal, mid, apical), preferably with fat suppression. Pericardial LGE severity is determined based on the circumferential extent of pericardial LGE, the thickness of pericardial LGE, and the number of slices affected. Severe pericardial LGE is defined as > 50% circumferential LGE, presence of LGE in all three short-axis slices, and LGE thickness > 3 mm. This grading scheme to correlate with the degree of inflammation (CRP levels) and time to recurrence (eg, mild LGE had fewer recurrences) was used in a substudy of the RHAPSODY (Study to Assess the Efficacy and Safety of Rilonacept Treatment in Participants With Recurrent Pericarditis) trial.¹⁰ Caution should be implemented as well in cases of minimal LGE and non-convincing symptoms to prevent over-diagnosis of pericarditis. Mild pericardial LGE is a frequent finding after cardiac surgery and may be present for several years but not associated with clinical pericarditis.^{11,12}

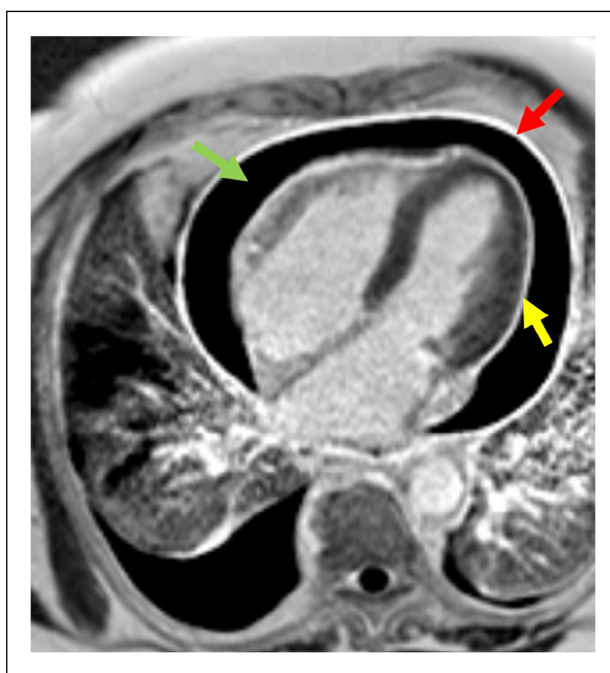


Figure 2 Late gadolinium enhancement (LGE) cardiac magnetic resonance image of a patient with acute pericarditis (phase sensitive inversion recovery sequence). Note the moderate circumferential pericardial effusion (green arrow, jet black pericardial space), and circumferential pericardial LGE involving both the visceral (yellow arrow) and parietal pericardium (red arrow).

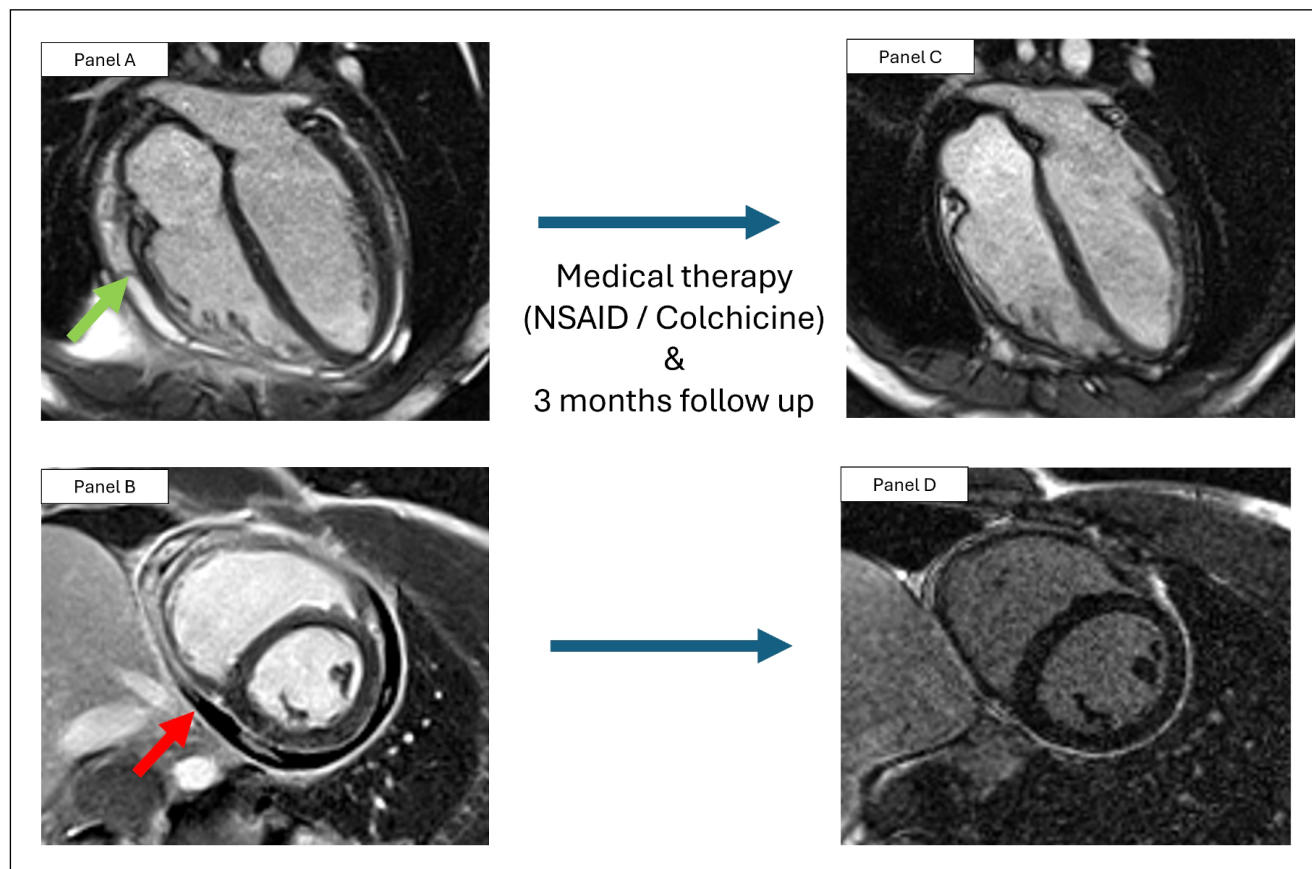


Figure 3 Case example of a 38-year-old man with acute pericarditis. Still image of cine cardiac magnetic resonance (CMR) shows a small circumferential pericardial effusion with adhesions (green arrow, panel A), thickened pericardium, and pericardial late gadolinium enhancement (LGE) (panel B). After medical therapy with nonsteroidal anti-inflammatory drugs and colchicine, follow up CMR 3 months later shows resolution of pericardial thickening and the pericardial effusion (panel C), with mild residual pericardial LGE (panel D).

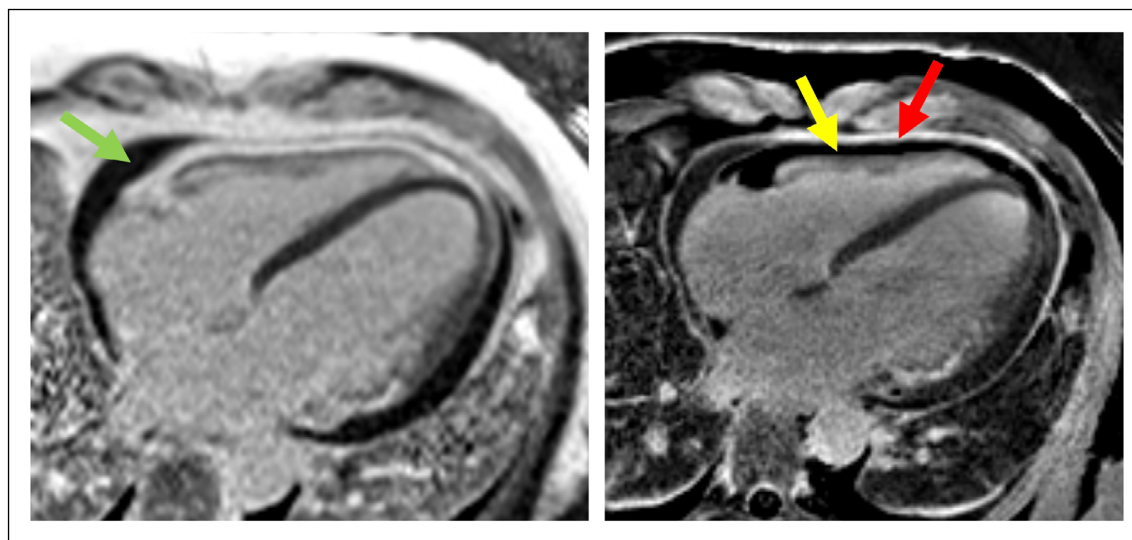


Figure 4 Case example of a 45-year-old patient with pericarditis undergoing standard late gadolinium enhancement (LGE) with phase sensitivity inversion recovery (left panel) and double spectral attenuated inversion recovery (DSPAIR, right panel). The patient has a small pericardial effusion (green arrow) and high signal intensity on the epicardial and the pericardial surface, increasing the difficulty of determining presence of pericardial LGE versus pericardial fat or both. Use of DSPAIR suppresses epicardial fat on the anterior right ventricular wall (yellow arrow) and persistent high signal intensity of the parietal pericardium is present (red arrow) confirming active pericardial inflammation.

Evaluation of the resolution versus persistence of pericardial LGE after treatment can also be considered in patients with recurrent pericarditis or persistent symptoms to guide the duration of therapy. However, in patients with established relapsing pericarditis, even the absence of pericardial LGE after treatment with IL-1 inhibition (riloncept) did not predict the absence of pericarditis recurrence. In a small substudy of the RHAPSODY trial, patients experienced a recurrence of pericarditis after stopping treatment despite normal CMR findings prior to cessation of treatment.¹³

In suspected or established constrictive pericarditis, one study of 42 patients with surgically confirmed constrictive pericarditis found six CMR findings to have high accuracy of confirming constriction, including pericardial thickness, respirophasic septal shift, diastolic bounce, pathological coupling (respirophasic abnormalities in ventricular filling), left ventricular area change, and eccentricity index (inspiratory-expiratory). Presence of pericardial LGE is commonly seen in transient constriction, effusive-constrictive pericarditis, and earlier stages of constrictive pericarditis. The presence of pericardial LGE is associated with fibroblast proliferation and chronic inflammation in pericardial tissue after pericardiectomy,¹⁴ and it indicates the potential of reversing constriction with anti-inflammatory therapy (and sparing the patient from undergoing pericardiectomy). In a study of 29 patients with constrictive pericarditis who underwent CMR and anti-inflammatory therapy, 14 had resolution of constriction; of these, 93% showed moderate or severe pericardial LGE. Another study yielded similar findings, with the extent of pericardial LGE predicting response to medical therapy in pericardial constriction.¹⁵ Early institution of therapy is likely a key factor, and patients may progress to needing pericardiectomy if not diagnosed and treated promptly, even with medical therapy.

CARDIAC CT TECHNIQUE AND USE IN PERICARDIAL DISEASE

Cardiac CT is not primarily used to diagnose acute pericarditis; however, it is frequently used in the emergency department in patients with acute chest pain. ECG-gated cardiac CT improves evaluation of pericardium and demonstrates pericardial thickening, pericardial effusion, and occasionally pericardial enhancement (on delayed acquisitions) in patients with pericarditis. Assessing pericardial thickness on cardiac CT benefits particularly from its high spatial resolution. The attenuation pattern of the effusion on noncontrast CT may provide clues to the etiology, with high attenuation pericardial fluid suggesting hemorrhagic or proteinaceous fluid. In addition, patients

with pericarditis can have new bilateral small pleural effusions that can support the diagnosis (pleuropericarditis). Cardiac CT is often useful as a secondary test in identifying concomitant malignancy and pericardial tumors or pericardial nodularity in metastatic disease, and it is helpful in cases where differentiation of pleural versus pericardial effusion is challenging with echocardiography (such as in postoperative patients with localized effusions).

On cardiac CT, pericardial effusion size is assessed by measuring the maximal thickness of pericardial fluid on axial or reformatted images. Effusions are commonly categorized as small when the fluid thickness is less than 10 mm, moderate when measuring 10 mm to 20 mm, and large when greater than 20 mm, albeit this classification does not account for heart size (large versus small) and thus it may not correlate well with effusion size (in mL) as a result. CT allows accurate evaluation of effusion distribution, distinguishing circumferential from loculated collections, and can identify regional predominance. Although CT provides precise anatomic assessment of effusion size, it does not directly assess hemodynamic significance, which should be evaluated clinically in conjunction with echocardiography and/or catheterization. In addition, care should be taken to account for the cardiac phase selected for scanning. In systolic imaging, the effusion may appear larger as it is traditionally measured at end diastole. Motion artifacts or measuring pericardial thickness on non-ECG gated scans may lead to overestimation of pericardial thickness.

The attenuation pattern of the effusion on non-contrast CT may provide clues to the etiology. Low-attenuation effusions (0-20 HU) are typically transudative, whereas intermediate attenuation (20-50 HU) suggests exudative fluid. High-attenuation effusions (> 50 HU) raise concern for hemorrhage, and very high HU suggests pericardial leakage of contrast (eg, ruptured dissection or bleeding). For a chylous effusion, the HU is low at -60 to -80.² In a retrospective study, CT findings of pericardial thickening or enhancement had a sensitivity of 54% to 59% and a specificity of 91% to 96% for pericarditis, thus they can be helpful to rule in pericarditis.¹⁶

In patients with suspected or established constrictive pericarditis, CT is particularly valuable for delineating the extent and distribution of pericardial thickening and calcification (Figure 5), thereby aiding diagnostic assessment and facilitating preprocedural planning prior to pericardiectomy. Retrospective gating on high temporal resolution scanners (such as dual source scanners) can help in assessing ventricular interdependence, although this is better evaluated with the other non-radiation-based modalities. Ancillary findings such as pleural effusions, conical deformity of the ventricles, and ascites all aid in confirming the diagnosis of constriction.

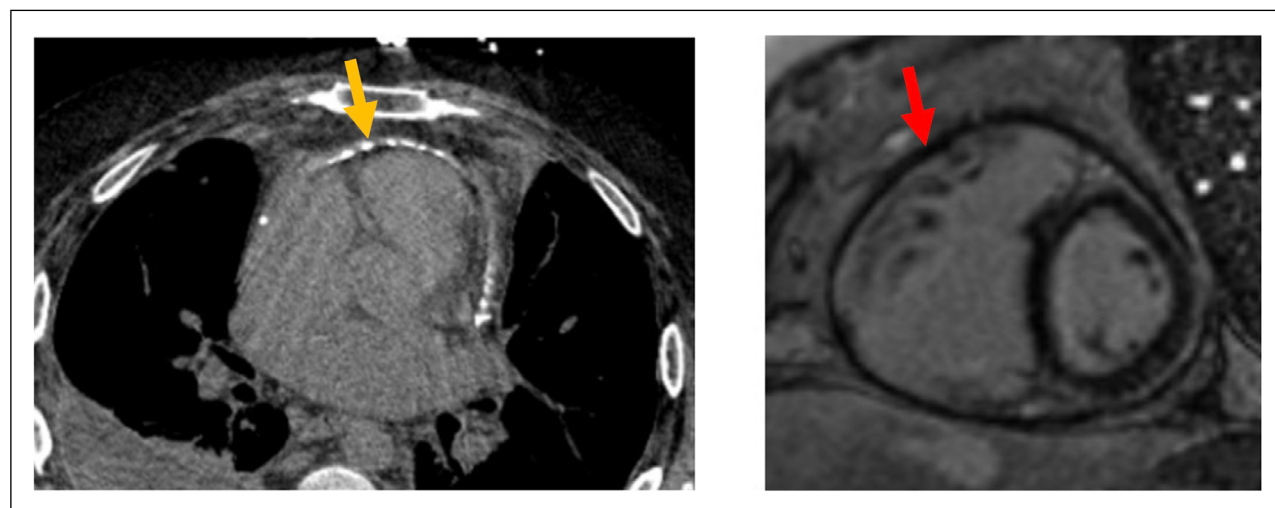


Figure 5 Example case of a patient with advanced end-stage calcific constrictive pericarditis. Note the pericardial calcifications on computed tomography (left panel, orange arrow), pericardial thickening, and absence of late gadolinium enhancement (LGE) on cardiac magnetic resonance (CMR, right panel, red arrow). Care should be applied when interpreting CMR as the lack of pericardial LGE does not exclude a pericardial pathology.

POTENTIAL ROLE OF POSITRON EMISSION TOMOGRAPHY

^{18}F -fluorodeoxyglucose positron emission tomography with CT (FDG-PET/CT), which detects inflammatory activity, can be considered in patients who cannot have CMR or at institutions lacking CMR equipment or expertise.^{2,17} High pericardial uptake of FDG on PET has been demonstrated in patients with pericardial inflammation (Figure 6), and thus it may aid in the diagnosis of pericarditis and the differentiation of reversible inflammatory constrictive pericarditis from chronic fibrotic forms. It also enables identification of other foci of inflammation and potential malignancy. FDG uptake reflects increased glucose metabolism in activated macrophages and requires a high-fat low-carbohydrate preparatory diet since physiologic myocardial uptake can obscure pericardial signals. These studies are more demanding than perfusion studies, and specialized expertise in high-volume centers, with adequate preparation, are required to achieve high-quality diagnostic imaging. In a smaller prospective study of 16 patients with constrictive pericarditis (50% with pericardial tuberculosis) who underwent FDG-PET pre and post medical therapy with steroids, pericardial uptake on FDG-PET improved significantly with medical therapy. Presence of baseline pericardial uptake (defined as standard uptake value [SUV_{max}] > 3) predicted response to steroid therapy.¹⁸ The concordance between LGE-CMR and FDG-PET findings is unknown, and prospective studies are needed to further validate the use of FDG-PET and identify optimal use cases.

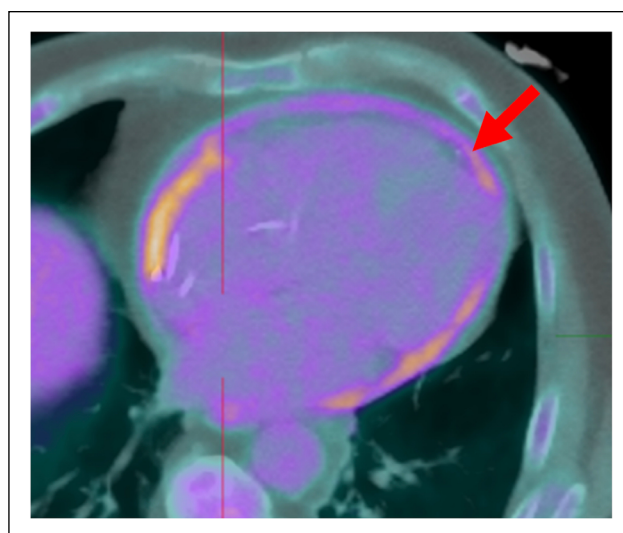


Figure 6 ^{18}F -fluorodeoxyglucose positron emission tomography shows circumferential uptake in the pericardium in a patient with established lupus pericarditis.

SUMMARY

Advanced cardiac imaging with CMR, CT, and potentially PET are important modalities with various strengths and limitations in the evaluation of patients with suspected pericardial disease and are endorsed in the most recent guidelines. In complicated or relapsing pericarditis, CMR has become a cornerstone in the evaluation and management. Further research is needed on how best to implement these modalities to improve the diagnostic yield and accuracy and avoid delays in diagnosis.

KEY POINTS

- Cardiac magnetic resonance and cardiac computed tomography are now recommended in patients with pericarditis and pericardial constriction.
- Use of advanced imaging can aid in clarifying the diagnosis in uncertain patient scenarios, guide clinical management, and identify complications.
- Cardiac positron emission tomography may be used in identifying the presence of pericardial inflammation, but more studies are needed to define its accuracy.

COMPETING INTERESTS

Dr. Shah is a consultant for Kiniksa and conducts research on behalf of Guerbet. The other authors have no competing interests to declare.

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REFERENCES

1. **Schulz-Menger J, Collini V, Groschel J**, et al. 2025 ESC Guidelines for the management of myocarditis and pericarditis. *Eur Heart J*. 2025 Oct 21;46(40):3952-4041. doi: [10.1093/eurheartj/ehaf192](https://doi.org/10.1093/eurheartj/ehaf192)
2. **Wang TKM, Klein AL, Cremer PC**, et al. 2025 Concise Clinical Guidance: An ACC Expert Consensus Statement on the Diagnosis and Management of Pericarditis: A Report of the American College of Cardiology Solution Set Oversight Committee. *J Am Coll Cardiol*. 2025 Dec 23;86(25):2691-2719. doi: [10.1016/j.jacc.2025.05.023](https://doi.org/10.1016/j.jacc.2025.05.023)
3. **Adler Y, Charron P, Imazio M**, et al. 2015 ESC Guidelines for the diagnosis and management of pericardial diseases: The Task Force for the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC) Endorsed by: The European Association for Cardio-Thoracic Surgery (EACTS). *Eur Heart J*. 2015 Nov 7;36(42):2921-2964. doi: [10.1093/eurheartj/ehv318](https://doi.org/10.1093/eurheartj/ehv318)
4. **Imazio M, Brucato A, Maestroni S**, et al. Prevalence of C-reactive protein elevation and time course of normalization in acute pericarditis: implications for the diagnosis, therapy, and prognosis of pericarditis. *Circulation*. 2011 Mar 15;123(10):1092-7. doi: [10.1161/CIRCULATIONAHA.110.986372](https://doi.org/10.1161/CIRCULATIONAHA.110.986372)
5. **Welch TD, Ling LH, Espinosa RE**, et al. Echocardiographic diagnosis of constrictive pericarditis: Mayo Clinic criteria. *Circ Cardiovasc Imaging*. 2014 May;7(3):526-34. doi: [10.1161/CIRCIMAGING.113.001613](https://doi.org/10.1161/CIRCIMAGING.113.001613)
6. **Bogaert J, Franccone M**. Cardiovascular magnetic resonance in pericardial diseases. *J Cardiovasc Magn Reson*. 2009 May 4;11(1):14. doi: [10.1186/1532-429X-11-14](https://doi.org/10.1186/1532-429X-11-14)
7. **Conte E, Agalbato C, Lauri G**, et al. Cardiac MRI after first episode of acute pericarditis: A pilot study for better identification of high risk patients. *Int J Cardiol*. 2022 May 1;354:63-67. doi: [10.1016/j.ijcard.2022.03.007](https://doi.org/10.1016/j.ijcard.2022.03.007)
8. **Yesilyaprak A, Kumar AK, Agrawal A**, et al. Predicting Long-Term Clinical Outcomes of Patients With Recurrent Pericarditis. *J Am Coll Cardiol*. 2024 Sep 24;84(13):1193-1204. doi: [10.1016/j.jacc.2024.05.072](https://doi.org/10.1016/j.jacc.2024.05.072)
9. **Jenista ER, Jensen CJ, Wendell D**, et al. Double spectral attenuated inversion recovery (DSPAIR)-an efficient fat suppression technique for late gadolinium enhancement at 3 tesla. *NMR Biomed*. 2021 Oct;34(10):e4580. doi: [10.1002/nbm.4580](https://doi.org/10.1002/nbm.4580)
10. **Cremer PC, Lin D, Luis SA**, et al. Pericardial late gadolinium enhancement and time to recurrence: a substudy from RHAPSODY, a phase 3 clinical trial of riloncept in recurrent pericarditis. *Eur Heart J Imaging Methods Pract*. 2023 May 26;1(1):qyad003. doi: [10.1093/ehjimp/qyad003](https://doi.org/10.1093/ehjimp/qyad003)
11. **Al-Kazaz M, Cremer PC**. Pericardial Late Gadolinium Enhancement After Cardiac Surgery: Defining Disease Begins With Understanding Normal. *Circ Cardiovasc Imaging*. 2023 Nov;16(11):e016131. doi: [10.1161/CIRCIMAGING.123.016131](https://doi.org/10.1161/CIRCIMAGING.123.016131)
12. **Pavon AG, Martinez Fernandez R, Arangalage D**, et al. Prevalence of Pericardial Late Gadolinium Enhancement in Patients After Cardiac Surgery:

- Clinical and Histological Correlations. *Circ Cardiovasc Imaging*. 2023 Nov;16(11):e015606. doi: [10.1161/CIRCIMAGING.123.015606](https://doi.org/10.1161/CIRCIMAGING.123.015606)
13. **Cremer PC, Brucato A, Insalaco A**, et al. Longitudinal cardiac magnetic resonance imaging following clinical response to rilonacept and prior to recurrence upon treatment suspension: a RHAPSODY subgroup analysis. *Eur Heart J Cardiovasc Imaging*. 2024 Dec 31;26(1):72-79. doi: [10.1093/ehjci/jeae200](https://doi.org/10.1093/ehjci/jeae200)
 14. **Zurick AO, Bolen MA, Kwon DH**, et al. Pericardial delayed hyperenhancement with CMR imaging in patients with constrictive pericarditis undergoing surgical pericardiectomy: a case series with histopathological correlation. *JACC Cardiovasc Imaging*. 2011 Nov;4(11):1180-91. doi: [10.1016/j.jcmg.2011.08.011](https://doi.org/10.1016/j.jcmg.2011.08.011)
 15. **Cremer PC, Tariq MU, Karwa A**, et al. Quantitative assessment of pericardial delayed hyperenhancement predicts clinical improvement in patients with constrictive pericarditis treated with anti-inflammatory therapy. *Circ Cardiovasc Imaging*. 2015 May;8(5):e003125. doi: [10.1161/CIRCIMAGING.114.003125](https://doi.org/10.1161/CIRCIMAGING.114.003125)
 16. **Hammer MM, Raptis CA, Javidan-Nejad C, Bhalla S**. Accuracy of computed tomography findings in acute pericarditis. *Acta Radiol*. 2014 Dec;55(10):1197-202. doi: [10.1177/0284185113515866](https://doi.org/10.1177/0284185113515866)
 17. **Klein AL, Wang TKM, Cremer PC**, et al. Pericardial Diseases: International Position Statement on New Concepts and Advances in Multimodality Cardiac Imaging. *JACC Cardiovasc Imaging*. 2024 Aug;17(8):937-988. doi: [10.1016/j.jcmg.2024.04.010](https://doi.org/10.1016/j.jcmg.2024.04.010)
 18. **Chang SA, Choi JY, Kim EK**, et al. [¹⁸F]Fluorodeoxyglucose PET/CT Predicts Response to Steroid Therapy in Constrictive Pericarditis. *J Am Coll Cardiol*. 2017 Feb 14;69(6):750-752. doi: [10.1016/j.jacc.2016.11.059](https://doi.org/10.1016/j.jacc.2016.11.059)

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