Comprehensive Characterization of Constrictive Pericarditis Using Multiparametric CMR

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Constrictive pericarditis continues to be a diagnostic and therapeutic challenge. Even with invasive cardiac catheterization its distinction from restrictive cardiomyopathy, which shares features of impaired ventricular filling, can remain difficult.

Cardiac magnetic resonance (CMR) is uniquely placed to characterize constrictive pericarditis. CMR comprehensively defines pericardial and myocardial anatomy as well as their structural and functional relationship. Indeed, through evaluation of regional myocardial deformation, ventricular interaction and venous return, CMR can accurately assess the physiological consequences of pericardial constriction. Furthermore, CMR provides in vivo insight into pericardial histology. As such, the diagnosis of constrictive pericarditis has become possible using a single, noninvasive investigation.

The ACCF/ACR/SCCT/SCMR/ASNC/NASCI/SCAI/SIR 2006 Appropriateness Criteria for Cardiac Computed Tomography and Cardiac Magnetic Resonance Imaging (1) indicates that CMR is appropriate for evaluation of pericardial conditions, including pericardial constriction. More recently the ACCF/ACR/AHA/NASCI/SCMR 2010 Expert Consensus Document on Cardiovascular Magnetic Resonance (2) recommends that CMR may be used as a noninvasive imaging modality to diagnose patients with suspected pericardial disease. In this paper we present a case that demonstrates the value of multiparametric CMR in the noninvasive assessment of constrictive pericarditis and the subsequent recovery following surgical intervention (Figs. 1, 2, 3, 4, 5, and 6).
Figure 1. Anatomical Characterization

A 51-year-old man initially presented with weight loss, malaise, and a raised jugular venous pressure. He subsequently went on to develop exertional breathlessness and clinical features of venous congestion. (A,B) (Online Videos 1 and 2). Parasternal long-axis and apical 4-chamber echocardiography images are shown. Echocardiography demonstrated an apparent echolucent zone surrounding the heart that was initially mistaken for pericardial effusion and an unsuccessful attempt at pericardiocentesis was made, as well as global impairment of left ventricular systolic function. The patient was referred for cardiac magnetic resonance (CMR) for further investigation. (C) Half Fourier single-shot turbo spin-echo image of the thorax is shown. The wide field-of-view afforded by CMR allowed visualization of a large left-sided pleural effusion, air present in the right pleural space following pleurocentesis, severe pericardial thickening, and deviation of the interventricular septum towards the left ventricle. (D,E,F) (Online Videos 3 and 4) Balanced steady-state free precession (SSFP) 3-chamber, 4-chamber, and mid-ventricular short-axis images demonstrated the pericardium to be markedly thickened (measuring up to 13 mm) normal ventricular wall thickness, biatrial dilation, and global severe impairment of left ventricular systolic function with an ejection fraction of 25%.

Figure 2. Functional Characterization

(A) Mid-ventricular short-axis spatial modulation of magnetization image ([SPAMM] or “tagging”) displayed at end-systole is shown. Widespread pericardial adherence to the myocardium was demonstrated by the failure of myocardial and pericardial gridlines to offset through the cardiac cycle. (B) (Online Video 5) Graphical display of transmural circumferential strain, measured from a mid-ventricular short-axis SPAMM image; x-axis, frame count; y-axis, percentage circumferential strain; colored lines correspond to myocardial segments according to the diagram in the bottom right corner. Peak systolic circumferential strain was markedly reduced in all segments. Furthermore, loss of function showed significant regional heterogeneity, with the lateral wall, where pericardial adherence was particularly evident, showing the greatest reduction in strain (light blue and purple lines). As such, left ventricular dysfunction appeared to result from a combination of impaired segmental function secondary to pericardial adherence and dyssynchrony between segments. (C) Phase-contrast imaging with velocity-encoding of the dilated inferior vena cava; inferior vena cava circled in blue; descending aorta circled in red; with (D) the corresponding graph of inferior vena cava blood flow velocity; x-axis, time from end-diastole in milliseconds; y-axis, blood flow velocity in centimeters per second; positive values correspond to blood flow towards the right atrium. Velocity and duration of diastolic forward flow were both significantly reduced and marked reversal of blood flow was seen in late-diastole. (E,F) (Online Videos 6 and 7) Real-time imaging demonstrated ventricular interdependence, with deviation of the interventricular septum toward the right ventricle during expiration (E) and toward the left ventricle during early inspiration (F).
Figure 3. Tissue Characterization

(A,B) Three-chamber and short-axis phase-sensitive inversion-recovery gradient-echo images taken in the late-phase following gadolinium contrast. Striking enhancement of the serosal layer of the parietal pericardium (which was contiguous with the enhancing fibrous debris within the pericardial space [see Fig. 4] and epicardial fat) and the outer layer of the parietal pericardium was seen. The “body” of the parietal pericardium did not enhance. This enhancement pattern was in keeping with the subsequent histological findings of an inflammatory infiltrate covering the thickened avascular parietal pericardium (see Fig. 4). No myocardial enhancement was seen, indicating myocardial viability and suggesting that there would be an improvement in ventricular function following pericardectomy.

Figure 4. Operative and Histopathological Findings

Based on the clinical features and the cardiac magnetic resonance findings that unequivocally confirmed the presence of constrictive physiology, and demonstrated sufficient distance between the thickened pericardium and the right ventricular free wall to allow successful surgery, the patient underwent pericardectomy. (A) At operation, the parietal pericardium was found to be severely thickened and the visceral pericardium was adherent to epicardial fat. Fibrinous debris was seen throughout the pericardial space. On stripping the pericardium the central venous pressure immediately halved. (B) Histopathological analysis revealed a fibrotic pericardium covered by organizing fibrin with cholesterol clefts and a chronic inflammatory cell infiltrate. No granulomata or evidence of neoplasia were found.

Figure 5. Post-Operative Anatomical Characterization

Six-months after surgery, repeat CMR imaging was performed. (A) Half Fourier single-shot turbo spin-echo images revealed resolution of the pleural effusions. (B,C,D) (Online Videos 8 and 9) SSFP 3-chamber, 4-chamber, and mid-ventricular short-axis images showed there was only a small amount of residual pericardial tissue, a large increase in ventricular volumes, and normalization of left ventricular systolic function, with an ejection fraction of 55%. Abbreviations as in Figure 1.
REFERENCES


APPENDIX

For supplementary videos and their legends, please see the online version of this article.