Importance of Cardiac Magnetic Resonance in a Patient With Crohn’s Disease–Associated Constrictive Pericarditis

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A 43-year-old woman with a 19-year history of Crohn’s Disease presented to the emergency department with a 4-day history of chest pain radiating to the posterior neck and trapezius muscle. The pain was sharp, worse in the supine position, and pleuritic in nature. She denied any infectious symptoms or preceding viral illness. Her Crohn’s Disease was active with a recent increase in bowel symptoms. She also had a new 2x2 cm violaceous plaque with rolled borders on the left inner thigh with a central hemorrhagic ulcer. Methotrexate had been stopped 8 weeks earlier. Her past medical history was significant for pyoderma gangrenosum. Her medications included infliximab, mirtazapine, and lansoprazole.

Vital signs showed a heart rate of 68 beats/min, blood pressure of 93/65 mm Hg, respiratory rate of 22 breaths/min, body temperature of 38°C, and normal pulse oximetry on room air. Physical examination was remarkable for prominent x and y descents in the jugular venous waveform. The jugular venous pressure was 3 cm above the sternal angle. There was no clinical pulsus paradoxus, Kussmaul sign, or pericardial knock.

ECG showed normal sinus rhythm with borderline low voltage. There was no ST elevation. Troponin I was within normal limits; however, inflammatory markers were elevated with C-reactive protein of 139 mg/L (normal value <10 mg/L). A computed tomography pulmonary angiogram to rule out pulmonary emboli showed a pericardial effusion. Because of concern about possible myopericarditis, cardiac magnetic resonance imaging was performed. The pericardium was markedly thickened to 7 mm with a small pericardial effusion. There was no evidence of interventricular dependence with septal shift during free breathing. T2-weighted triple inversion recovery sequences showed normal signal in the pericardium, consistent with resolved inflammation. The respiratory-related septal shift and dilated inferior vena cava could not be demonstrated, suggesting resolution of constrictive physiology (Figure 1).

Causes for pericarditis were entertained. Drug-induced lupus was unlikely because antinuclear antibody, anti–double-stranded DNA, and anthistone antibody levels were all within normal range. A tuberculosis skin test was nonreactive. A computed tomography abdomen/pelvis scan showed several thickened ileal loops adherent to the anterior abdominal wall in the left lower quadrant. Subsequent colonoscopy confirmed mild to moderate inflammation and ulceration at the proximal portion of the neoterminal ileum. Biopsies showed inflammation with focal active ileitis. The thigh lesion was confirmed to be recurrent pyoderma gangrenosum.

On the basis of these findings, active Crohn’s Disease with bowel, skin, and heart involvement was considered the likely cause of the pericarditis. She was treated with intravenous methylprednisolone followed by oral prednisone. Colchicine 0.6 mg twice daily was used for pericarditis. There was rapid normalization of her C-reactive protein, and her pain gradually improved over the next couple of weeks. A follow-up cardiac magnetic resonance study 5 weeks later showed marked improvement. The pericardium was near-normal thickness (3–4 mm) with no pericardial or pleural effusion. T2-weighted triple inversion recovery sequences showed normal signal in the pericardium, consistent with resolved inflammation. The respiratory-related septal shift and dilated inferior vena cava could not be demonstrated, suggesting resolution of constrictive physiology (Figure 2).

Discussion

Constrictive pericarditis is characterized by pathological constraint of the heart chambers attributable to inflammation and fibrosis of the pericardium, resulting in impaired filling and progressive heart failure. Although the diagnosis of this condition is often late, a reversible effusive constrictive phase precedes late fibrosis. When recognized late into the fibrotic constrictive phase, treatment requires pericardiectomy, which is associated with a relatively high morbidity and mortality despite the advances in surgical techniques. Hence, early diagnosis and treatment in the inflammatory effusive phase may mitigate fibrosis and irreversible constrictive physiology. The present case highlights the role of T2-weighted imaging in identifying the acute, inflammatory phase, which may predict a response to medical treatment.

Similar to our case, a pilot study by Feng et al found that anti-inflammatory therapy is associated with a reduction of late gadolinium enhancement, reduction in pericardial thickness, lower markers of systemic inflammation, and relief of constrictive physiology.
This case highlights the rare association of pericarditis with inflammatory bowel disease. T2-weighted pericardial imaging can be helpful in the evaluation of pericardial disease by cardiac magnetic resonance and may be predictive of response to medical treatment. These results should be confirmed in a larger study or case series.

Disclosures
None.

References

Figure 1. Cardiac magnetic resonance at initial presentation. A, Left ventricular short-axis view: dark blood T2-weighted images showing marked thickening and edema of the pericardium (arrow). B, Left ventricular short-axis view: late gadolinium enhancement imaging demonstrating pericardial thickening and enhancement (arrow).

Figure 2. Cardiac magnetic resonance at 5-week follow-up. A, Left ventricular short-axis view: dark blood T2-weighted images showing resolution of pericardial edema. B, Left ventricular short-axis view: late gadolinium enhancement demonstrating no pericardial enhancement and near-normal thickness.
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