Acute Isolated Viral Pericarditis With Rapid Progression to Constrictive Pericardial Disease

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A previously healthy 42-year-old man presented with fatigue and progressive dyspnea 6 weeks after an episode of massive diarrhea that required supportive intravenous therapy.

A chest x-ray showed bilateral pleural effusion and echocardiography demonstrated extensive pericardial effusion, whereas global systolic right and left ventricular function appeared preserved (Figure 1A).

The patient was referred to cardiac magnetic resonance imaging for suspected inflammatory involvement of the myocardium; cardiac magnetic resonance imaging demonstrated acute inflammation confined to the visceral and parietal pericardial layers (Figure 2A through 2D). Extensive laboratory testing for tuberculosis, autoimmune, and systemic diseases was inconspicuous except for an elevated coxsackie A virus IgM serum titer (1:640). Immunohistologic examinations of right-sided endomyocardial biopsy samples revealed no inflammatory reaction of the myocardium. After successful needle pericardiocentesis and medical treatment with ibuprofen, the patient recovered rapidly and was discharged home in a symptom-free state.

Figure 1. Echocardiographic still frames of a four-chamber view at endsystole. (A) During initial presentation, extensive pericardial effusion (white arrow) and a “swinging heart” pattern were seen. (B) Six months later, paradoxical motion of the interventricular septum and an echo-intense pericardium were found (white arrowheads).

Figure 2. Cardiac MR imaging in short-axis orientation. Images labeled (A–D) were acquired at first presentation; (E–H) during repeat cardiac MR imaging six months later. (A, E) cine imaging (SSFP sequence) at endsystole. (B, F) T1-weighted blackblood imaging with fat saturation prepulse (SPIR). (C, G) T2-weighted blackblood imaging. (D, H) delayed enhancement imaging (intravenous dosage of Gad-DTPA 0.2 mmol/kg, inversion recovery delay 200 ms, delayed time 12 minutes). (A–D) Initially, pericardial effusion was demonstrated. Pericardial layers were of normal thickness; however, postcontrast signal intensity of both layers increased significantly (D). (E–H) At follow-up, an extremely thickened pericardium was found (white arrowheads); note the bright signal streak between the two pericardial layers on T2-weighted imaging (G) indicating the subacute inflammatory reaction with ongoing fibrous organization of exsudate.

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Six months later, the patient presented with 3 weeks of progressive dyspnea on exertion (New York Heart Association class II to III). Repeat echocardiography showed no recurrent pericardial effusion but an elevated right-sided pressure (estimated right ventricular pressure of 40 mm Hg) and an echo-intense pericardium were found (Figure 1B). In addition, repeat cardiac magnetic resonance imaging depicted the presence of a massively thickened pericardium (>14 mm) most prominently in the inferior segments of the right and left ventricle (Figure 2E through 2H). The signal intensity pattern on T1/T2-weighted images and delayed enhancement imaging was consistent with a subacute inflammation of both pericardial layers and indicative of an ongoing process of fibrinous organization.

Subsequent cardiac catheterization proved the presence of the typical hemodynamic pattern commonly found in constrictive pericardial disease with an elevated left and right ventricular enddiastolic pressure and “square root sign” of ventricular pressure tracings (Figure 3A); in addition prominent x- and y-descents were recorded in the right atrium (Figure 3B). Consequently, total pericardiectomy was performed; our patient recovered uneventfully thereafter and remained symptom-free at 1-year follow-up (Figure 4).

Cardiac magnetic resonance imaging provided clear visibility of pericardial structures and together with its capability for tissue characterization facilitated accurate detection of inflammatory disease states. Thus, it may prove useful as a noninvasive diagnostic tool to monitor the effectiveness of anti-inflammatory drug treatment.

**Disclosures**

None.
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