Constrictive pericarditis 20 years after surgical aortic valve replacement

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A 76-year-old man presented with a 2-month history of dyspnea and anorexia. The patient underwent surgical aortic valve replacement 20 years before the presentation. He had a history of smoking but no history of asbestosis exposure. He was afebrile, with a blood pressure of 132/103 mmHg, pulse rate of 70 beats per minute, respiratory rate of 20 per minute, and oxygen saturation of 88% on room air. On physical examination, marked jugular venous distention, bilateral edema in the legs, and pericardial knock were present. Laboratory findings showed increased creatinine (1.23 mg/dL) and normal blood urea nitrogen (18.2 mg/dL). Chest radiography showed bilateral dullness in the costophrenic angles. Electrocardiography showed atrial fibrillation with low-voltage QRS complexes in the limb leads. Transthoracic echocardiography demonstrated left ventricular diastolic dysfunction and pericardial thickening but no interventricular septal thickening or brightening. Left ventricle ejection fraction was preserved (66%). Chest computed tomography revealed a thickened and calcified pericardium, mechanical valve, and bilateral pleural effusion (Figure 1). Thoracentesis was performed, and the pleural effusion showed a transudative pattern. Cytologic examination revealed no malignancy. All cultures were negative for bacteria and fungi. Acid-fast staining, mycobacterial culture, interferon-gamma release assay (IGRA), and polymerase chain reaction (PCR) test of sputum and pleural fluid were all negative for Mycobacterium tuberculosis. Assays for antinuclear antibodies, antineutrophilic cytoplasmic antibodies, serum angiotensin-converting enzyme levels, and immunoglobulin G4 (IgG4) levels were all within the normal range. Right heart catheterization showed a prominent “y” descent, known as Friedreich's sign, with a dip-and-plateau configuration (Figure 2). These findings were consistent with constrictive pericarditis (CP) or restrictive cardiomyopathy. Since increased pericardial thickening and calcification strongly suggested CP, we clinically diagnosed CP as a complication of cardiac surgery. The patient developed dyspnea...
equivalent to New York Heart Association Class III and was therefore referred for a pericardectomy. Nevertheless, his condition was considered inoperative because of an increased operative mortality risk. He was thus treated with diuretics, which improved his symptoms. He remained asymptomatic at a 1-year follow-up.

The etiology of CP varies widely, and tuberculosis is now recognized as a rare cause in developed countries. However, in Japan, case reports of CP secondary to tuberculosis have been reported. In our current case, a negative staining of sputum and pleural effusion, cultures, PCR, and IGRA excluded tuberculosis. In addition, we ruled out postradiotherapy, malignancy, trauma, asbestosis, sarcoidosis, uremic pericarditis, connective tissue disorder, and systemic IgG4-related disease, based on the history and additional test results. Therefore, in this case, CP is a late complication of cardiac surgery. Generally, the incidence of CP after cardiac surgery is relatively low (range, 0.2%–2.4%), and the timing of the onset varies between 1 month and 204 months after surgery. We experienced a rare case with CP, which occurred 20 years after surgery. CP should be considered in patients with dyspnea, anorexia, and refractory fluid retention after cardiac surgery. History taking and physical examination are important clues for the diagnosis of CP.

CONFLICT OF INTEREST
The authors have stated explicitly that there are no conflicts of interest in connection with this article.

CONSENT FOR CASE REPORT
Patient consent has been obtained.

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