Constrictive Pericarditis Caused by Calcification and Organized Hematoma 30 Years After Cardiac Surgery

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A 54-year-old man, who had undergone atrial septal defect (ASD) closure 30 years previously, was admitted for exertional dyspnea and chest oppression. He presented with right pleural effusion and hepatomegaly. Hemodynamic characteristics were consistent with constrictive pericarditis caused by multiple cystic lesions anterior to the main pulmonary artery and right ventricle, and severe calcification over the posterior and diaphragmatic sides of the heart. Magnetic resonance imaging was useful for differential diagnosis of the cystic mass and at surgery, it was revealed that the cystic lesions were old hematoma without cells. Pericardiectomy and removal of the calcification were performed safely using an ultrasonic scalpel, without cardiopulmonary bypass, resulting in hemodynamic improvement and relief of his symptoms. (Circ J 2002; 66: 610–612)

Key Words: Calcification; Constrictive pericarditis; Magnetic resonance imaging; Hematoma; Ultrasonic scalpel

The dilated hepatic vein (Fig 1), and an echolucent mass anterior to the main pulmonary artery (PA) and RV. On a computed tomography (CT) chest scan, the mass was revealed to be multiple cystic lesions compressing the PA and RV outflow (Fig 2). CT also showed right pleural effusion and a heavily calcified mass over the posterior and diaphragmatic sides of the heart. From the T2-weighted magnetic resonance imaging (MRI) scans, the cystic mass anterior to the PA seemed to contain blood, protein and viscous components (Fig 3). A gallium scintigram did not show accumulation of the agent around the heart. Cardiac catheterization revealed elevated end-diastolic pressures in the right atrium and both ventricles (22, 20, and 27 mmHg, respectively). The pressures were: RA 25/22 mmHg; RV 40/16 mmHg with an early diastolic dip; PA 41/23 mmHg; mean pulmonary wedge, 24 mmHg; and left ventricle, 119/14 mmHg. The thermodilution cardiac index was 2.8 L·min⁻¹·m⁻².

Fig 1. Transthoracic echocardiograms before (I) and after (II) the surgery. Note that the inferior vena cava (IVC) was dilated up to 22 mm and there were prominent diastolic flow reversals in the dilated hepatic vein before surgery (I). After surgery (II), the diameter of the IVC decreased to 15 mm and the diastolic flow reversals in the hepatic vein became less prominent.
Based on a diagnosis of chronic constrictive pericarditis, surgery was performed to remove both the multiple cystic mass and the heavily calcified lesions. After a median sternotomy along the previous skin incision, the closed pericardium was opened. The cystic mass over the main PA ruptured, overflowing thick, semifluid, chocolate-like material, presumably old coagulated blood. Other cystic lesions with separating capsules over the anterior surface of the heart were also removed. Pathological evaluation of the mass was consistent with an organized hematoma without cells. Subsequently, the adhered pericardium was dissected from the surface of the RA, superior vena cava, and RV with the aid of an ultrasonic scalpel (Harmonic Scalpel; Ethicon Endo-Surgery, CVG, Cincinnati, OH, USA). We removed as much of the calcified lesions on the diaphragmatic surface, which were cement-like, as was possible without using cardiopulmonary bypass.

The postoperative course was uneventful. The chest CT scan revealed that the cystic mass had gone and less calcification around the heart (Fig 2). The echocardiographic examination showed that the diameter of the IVC had decreased (from 22 mm to 15 mm) and there were less prominent diastolic flow reversals in the hepatic vein (Fig 1). The mean RA pressure decreased to 18 mmHg and the early diastolic ‘dip’ of the RV pressure disappeared. Laboratory data before discharge showed a decrease in total bilirubin to 0.5 mg/dl and lactate dehydrogenase to 290 IU/L.

**Discussion**

The most recent report from the Mayo Clinic revealed that the 3 most common identifiable causes of constrictive pericarditis are cardiac surgery (18%), pericarditis (16%), and mediastinal irradiation (13%), although 33% of cases were idiopathic. There was a trend toward open heart surgery and irradiation as important causes because of the declining incidence of tuberculous pericarditis. The first interesting point about the present case of constrictive pericarditis is that the patient presented 30 years after cardiac surgery, and the second interesting point is that it was induced by the mixed pathology of hemopericardium and calcification. There have been other case reports of constrictive pericarditis caused by hemopericardium triggered by blunt trauma or pericardiocentesis, but the present patient did not have a history of chest trauma, and the hematoma was considered to be secondary to the previous surgery. Although the diagnosing the calcification was easy, it was difficult to qualitatively identify the multiple cystic lesions over the PA before surgery. MRI was the most useful among the several diagnostic modalities we tried, including CT and echocardiography. Others have also stressed the utility of MRI for assessing intrapericardial hematoma causing hemodynamically significant cardiac compression.

As a diagnostic modality, Doppler echocardiography is quite easy and useful for defining the hemodynamic condition of the patient with constrictive pericarditis, compared with catheterization, which is invasive and influenced by examination conditions. In particular, the expiratory flow reversals in the dilated hepatic vein (Fig 2) are considered to be of equal importance to catheterization in evaluating the hemodynamic effect of constrictive pericarditis.

At surgery, we used the Harmonic Scalpel to dissect the adhered pericardium. It was so safe and effective for pericardiectomy that we could remove the calcified lesions without the need for cardiopulmonary bypass, resulting in hemodynamic improvement. The Harmonic Scalpel has 2 cutting mechanisms: longitudinal vibration enabling the scalpel to incise tissues, and cavitation disrupting low-density tissues and causing tissue planes to separate. The latter mechanism facilitates the dissection planes of adhered pericardium and avoids damage to adjacent vital structures such as the RA.

**References**

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