Pericardial hematoma after cardiac surgery: An unexpected cause of constrictive pericarditis

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Introduction

Constricitive pericarditis (CP) is a form of severe diastolic dysfunction secondary to noncompliant, diseased pericardium. It is a rare complication in patients who have undergone cardiac surgery, with reported rates of 0.2–0.4% (1). It is still hard to identify the disease, because the right heart failure signs can be the sole presenting symptom. Besides, the timing of the presentation ranges from 1 month to 204 months following surgery (2). Here, we described a patient who has been diagnosed with CP 7 years following his cardiac surgery.

Case Report

A 60-year-old male patient was admitted to the hospital because of progressive dyspnea on exertion and right-sided heart failure symptoms. He had a history of mechanical aortic valve replacement and coronary artery bypass grafting in 2013. He had been suffering from fatigue and dyspnea since 2016. Thorax computerized tomography (CT) and echocardiographies carried out during the postoperative follow-up displayed organized hematoma at the posterior side of the left atrium and ventricle. Patient also suffered from weight loss and lower extremity edema with abdominal distention upon presentation. He had severe dyspnea and fatigue on mild exertion as well as loss of appetite, especially in the last year. Physical examination revealed moderate pretibial edema with marked jugular distention. Hepatomegaly as well as ascites is noted. Laboratory findings showed normal hepatic function with moderately elevated bilirubin levels and an INR level of 2.6 under warfarin treatment. He had normal renal functions and normal hemogram levels except the low platelet levels ($93x10^{3}/\mu$ L). Both the sedimentation rate and the C-reactive protein level were slightly elevated, and the NT-proBNP level was 816 pg/mL.

His echocardiography showed biatrial dilatation with preserved ventricular function. A heterogeneous mass, consistent with organized hematoma with a size of 4.8 x 6 cm, was observed behind the left atrium and left ventricle (Video 1). The pericardium was slightly thickened (4 mm) with calcific areas around the hematoma. Mitral inflow pulsed-wave Doppler exhibited a restrictive pattern with 25% inspiratory decrease in early diastolic velocity (Fig. 1a). Septal bounce was observed, and vena cava inferior was plethoric with a diameter of 2.7 cm. Expiratory diastolic flow reversal was noted in the dilated hepatic veins (Fig. 1b). Tissue Doppler of medial mitral annulus showed an early diastolic velocity of 12 m/sec, which is equal to the velocity of lateral annulus. Parameters were consistent with the CP diagnosis. Thorax CT was performed to further identify the pericardial mass, and it revealed a 10x8x5 cm hypodense lesion with focal calcifications (Fig. 2a). Both size and structure were described to be similar to the hematoma reported on the thorax CT 4 years ago. Nevertheless, hepatic contour irregularity and parenchymal heterogeneity are newly diagnosed in the recent imaging. Hepatic ultrasonography also supported the diagnosis

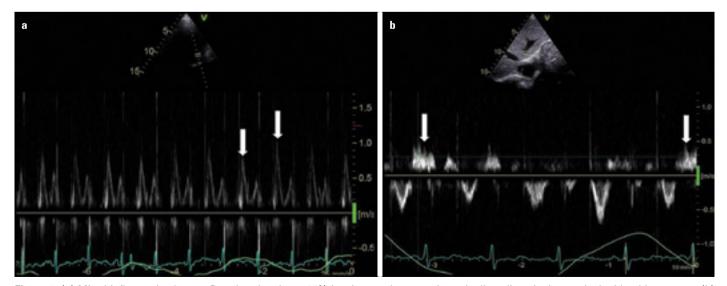


Figure 1. (a) Mitral inflow pulsed-wave Doppler showing a 25% inspiratory decrease in early diastolic velocity, marked with white arrows. (b) Expiratory diastolic flow reversal was seen in dilated hepatic veins, marked with white arrows (light green tracing previously correlates to simultaneous respirometer; downward deflection marks the beginning of expiration)

of liver cirrhosis, and it was found to be Child-Pugh class A by gastroenterology and described to be at the reversible stage.

To validate our diagnosis of CP, cardiac cathetherization was performed, since it was the gold standard technique, and highrisk reoperation was the other option. Coronary angiography showed no occluded arteries, and right-sided cathetherization displayed a diastolic dip plateau phase and right atrial Y wave, both validating our diagnosis (Fig. 2b). The patient was scheduled for pericardiectomy.

Discussion

Constrictive pericarditis is a rare complication following cardiac surgery and is rarely reported in the literature (3). The disease is characterized by the encasement of the heart in rigid pericardium because of fibrosis and adhesions. Intraoperative irritation of the pericardial layers was a predisposing factor (4). Blood in pericardial space induced fibrosis in the impaired serosal surface, and warfarin use and normal left ventricular ejection fraction were correlated with CP (5). A low threshold should be kept for pericardial drainage postoperatively, especially in anticoagulated patients (2). In our case, the patient had not been followed up for hematoma, as it was considered not to lead to hemodynamic instability and is thought to be resorbed through time. However, the patient had the risk factors for postoperative CP, and probably the clinic had been progressing for the last 4 years, leading to cardiac cirrhosis. Since complete surgical pericardiectomy is the only definitive treatment, the patient was led to cardiovascular surgery (6).

Conclusion

Postoperative pericardial bleeding as well as pooling of blood is a risk factor for future CP, especially in patients under

warfarin treatment and normal ejection fraction. Diagnosis is difficult and requires a high index of clinical suspicion.

Informed consent: Informed consent was signed and given by the patient.

Video 1. The video shows the mass posterior to the left atrium and left ventricle, compatible with intrapericardial hematoma.

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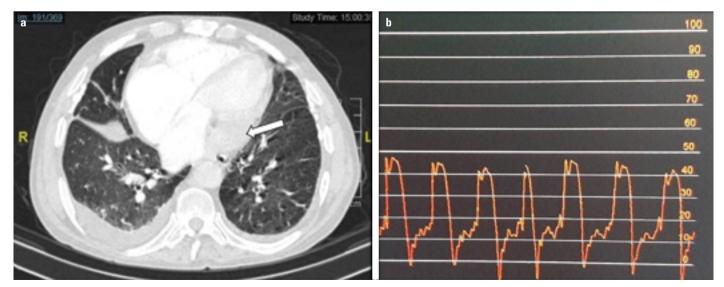


Figure 2. (a) Intrapericardial hypodense mass, described to be consistent with hematoma, posterior to the left atrium and left ventricle and partially obstructing the left atrium (marked with white arrow). (b) Diastolic dip plateau phase recording from the right ventricle during right-sided cathetherization