Case Report

A case of right ventricular diastolic dysfunction due to a large hematoma posterior to the left ventricle

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ABSTRACT

We report a case of right ventricular (RV) diastolic dysfunction due to a large hematoma posterior to the left ventricle (LV) after cardiology surgery. An 80-year-old woman underwent cardiology surgery, After surgery, her physical findings revealed right heart failure. Localized hematoma posterior to the pericardial space and the RV compression to the sternum were shown by computed tomography. Transthoracic Doppler echocardiography demonstrated restrictive physiology of the RV although there was no evidence of constrictive pericarditis. These findings suggest that RV diastolic dysfunction could have occurred due to the hematoma posterior to the LV. Since pleural effusion had persisted despite medical therapy, the hematoma was removed surgically. Soon after surgery, dyspnea and pretibial edema were diminished; bilateral pleural effusion dramatically disappeared. RV diastolic dysfunction estimated by echocardiography was improved and RV compression disappeared.

We speculate that there are two physiological mechanisms for the RV compression: (1) the localized hematoma elevated the intrapericardial pressure and (2) the hematoma shifted the entire heart to the sternum. In conclusion, this is the first case report of RV diastolic dysfunction due to large hematoma posterior to the LV.

<Learning objective: Localized hematoma posterior to the left ventricle can be a cause of right ventricular compression that leads to onset of severe right ventricular diastolic dysfunction.> © 2015 Japanese College of Cardiology. Published by Elsevier Ltd. All rights reserved.

Introduction

Cardiac tamponade is a well-recognized early and late complication of cardiac surgery. However, right ventricular (RV) compression due to a localized hematoma in the posterior pericardial space has been rarely reported. We present a case of RV diastolic dysfunction which was caused by a localized hematoma posterior to the left ventricle (LV) after elective aortic valve replacement and coronary artery bypass graft surgery.

Case report

An 80-year-old woman with pretibial edema was referred to our hospital. She was diagnosed with severe aortic stenosis and multivessel coronary artery disease, and she underwent elective aortic valve replacement and coronary artery bypass graft surgery. Manual positioning of the heart for the distal anastomosis of the circumflex branch caused epicardial bleeding posterior to the LV, and optimal hemostasis was confirmed after using absorbable hemostat, SURGICEL® NU-KNIT (Ethicon, Somerville, NJ, USA). Her postoperative course was uneventful and she was discharged on postoperative day 23.

On postoperative day 46, she was readmitted because of dyspnea on exertion. She had findings of low cardiac output including tachycardia with 113 beats per minute and renal
(A) Preoperative 2-dimensional transthoracic echocardiography. Long-axis view showed hematoma posterior to the left ventricle (yellow arrows). (B) Pulse-wave Doppler recording (apical window) at the level of the open leaflet tips. Right ventricular inflow pattern was a restrictive pattern. (C) Continuous-wave Doppler recording at the pulmonary artery. Antegrade late diastolic flow in pulmonary artery existed (yellow arrow). (D) Tricuspid annular tissue Doppler recording. Tricuspid annular e' velocity was 8 cm/s. (E) Respiratory variation in tricuspid inflow velocity. Inspiratory early (E) inflow velocity increased and expiratory early (E) inflow velocity decreased. Exp indicates expiration; and insp indicates inspiration. (F) Pulse-wave Doppler recording (apical window) at the level of the open leaflet tips. Left ventricular inflow pattern was abnormal. (G) Respiratory variation in mitral inflow velocity. Respiratory variation was not observed. (H) Midventricular septal M-mode recording (parasternal long axis). Respiration-related ventricular septal shift was not seen. (I) Medial mitral annular tissue Doppler recording. Medial mitral annular e' velocity was reduced (4.5 cm/s). (J) Pulse-wave Doppler recording (subcostal window) within the hepatic vein. Prominent hepatic vein expiratory diastolic flow reversals were not seen.
dysfunction with creatinine level of 2.2 mg/dl, a jugular venous distention, and a pretibial edema. Chest X-ray showed bilateral pleural effusion. Transthoracic echocardiography showed hematoma posterior to the LV (Fig. 1A), restrictive RV inflow pattern (Fig. 1B), and antegrade late diastolic flow in pulmonary artery (Fig. 1C). Additionally, tricuspid annular e' was 8 cm/s (Fig. 1D), respiratory variation in tricuspid inflow velocity (Fig. 1E) was observed, and expiratory and inspiratory diameters of the inferior vena cava (IVC) were 17 mm and 12 mm, respectively. LV inflow pattern was abnormal (Fig. 1F) and respiratory variation in mitral inflow velocity was not observed (Fig. 1G). Chest computed tomography (CT) showed localized hematoma posterior to the pericardial space and the RV compressed to the sternum (Fig. 2A). These findings indicated that RV diastolic dysfunction could be caused by the hematoma posterior to the LV. Since pleural effusion and renal dysfunction persisted despite medical therapy, the hematoma was removed surgically. The pericardial space was approached with a left anterior thoracotomy, and a large amount of capsulated thrombus was evacuated from the posterior pericardial space. The posterolateral pericardium was found to be thickened.
and surgically removed. At the same time, the anterior RV was freed from the sternum. After surgery, chest CT revealed regression of RV compression (Fig. 2B) and also, dyspnea, tachycardia, jugular venous distention, and pretilial edema were improved, serum creatinine level was lowered to 1.6 mg/dl, and bilateral pleural effusion dramatically disappeared. Four months later, echocardiography showed no pericardial hematoma (Fig. 3A) and abnormal RV inflow pattern (Fig. 3B). Antegrade late diastolic flow in pulmonary artery (Fig. 3C) was absent, tricuspid annular e' was 9 cm/s (Fig. 3D), respiratory variation in tricuspid inflow velocity was reduced (59% → 25%) (Fig. 3E), and expiratory and inspiratory diameters of the IVC were 19 mm and 9 mm, respectively.

Discussion

This case suggests that localized large hematoma posterior to the LV could be a cause of RV compression that leads to onset of severe RV diastolic dysfunction. Beppu et al. reported that accumulating pericardial coagula was localized in the right side of the heart, and directly deformed the right atrial and ventricular chambers in 12 cases [1]. Some previous studies reported that a localized effusion or hematoma directly compressed the selective chamber in contact with it, and as a result caused regional cardiac tamponade [2–5]. However, our case was different from these previous cases, a localized hematoma posterior to the LV did not directly compress the LV. Welch et al. reported that respiration-related ventricular septal shift, preserved or increased medial mitral annular e' velocity (e' ≥ 9 cm/s), and prominent hepatic vein expiratory diastolic flow reversals are independently associated with the diagnosis of constrictive pericarditis [6]. In our case, respiration-related ventricular septal shift was not seen (Fig. 1H), medial mitral annular e’ velocity decreased (4.6 cm/s) (Fig. 1I), and prominent hepatic vein expiratory diastolic flow reversals were not seen (Fig. 1J). Based on these findings, right heart failure in this case could not have been caused by constrictive pericarditis. Thus, we speculate that there are two physiological mechanisms for the RV compression: (1) the localized hematoma elevated the intrapericardial pressure, consistent with the previous paper by Beppu et al. [1], (2) the hematoma shifted the entire heart to the sternum. In conclusion, this is the first case, to the best of our knowledge, of RV diastolic dysfunction due to a large hematoma posterior to the LV.

Conflicts of interest

There are no conflicts of interest to declare.

References