

[CASE REPORT]

Cardiac Magnetic Resonance Imaging of Very Late Intrapericardial Hematoma 8 Years after Coronary Artery Bypass Grafting

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Abstract:

A 55-year-old man presented with dyspnea, edema, and appetite loss. He had undergone coronary artery bypass grafting 8 years previously. He had jugular venous distention and Kussmaul's sign. Contrast-enhanced cardiac magnetic resonance imaging (CMRI) demonstrated an intrapericardial mass compressing the right ventricular (RV) cavity. T1- and T2-weighted black-blood images showed a mass with heterogeneous high signal intensity and a thick and dark rim. The mass was considered to be a chronic hematoma. After pericardiectomy with surgical removal of the hematoma, CMRI showed the marked improvement of the RV function. Late intrapericardial hematoma is rare and CMRI is useful for making a differential diagnosis.

Key words: intrapericardial hematoma, coronary artery bypass grafting, cardiac magnetic resonance imaging

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Introduction

An intrapericardial hematoma is a known complication of cardiac surgery, which usually occurs in the early postoperative period and which generally presents with acute hemodynamic compromise (1). However, on rare occasions, it occurs in the very late stage after surgery as a chronic expanding hematoma; this was first described in 1980 (2), as a lesion that persists and increases in size, and which lasts for more than 1 month after the initial hemorrhage.

We herein present a case involving a very late intrapericardial hematoma that occurred 8 years after coronary artery bypass grafting (CABG) and which caused heart failure. The hematoma was identified using cardiac magnetic resonance imaging (CMRI).

Case Report

A 55-year-old man presented with dyspnea, leg edema, and appetite loss. His medical history included hypertension and diabetes mellitus, and he had undergone CABG 8 years previously. His blood pressure was normal but his heart rate was increased (98 beats per minute). His heart sounds were normal with no murmur. Moderate leg edema was noted in both limbs. He had jugular venous distention and Kussmaul's sign. The patient's laboratory data showed the slight elevation of his brain natriuretic peptide and transaminase levels. Transthoracic echocardiography (TTE) revealed a preserved left ventricular (LV) function and a mass compressing the right ventricle (RV). Emergency contrast-enhanced computed tomography (CT) showed a non-contrasted mass (5.0 cm×5.0 cm×6.0 cm) compressing the

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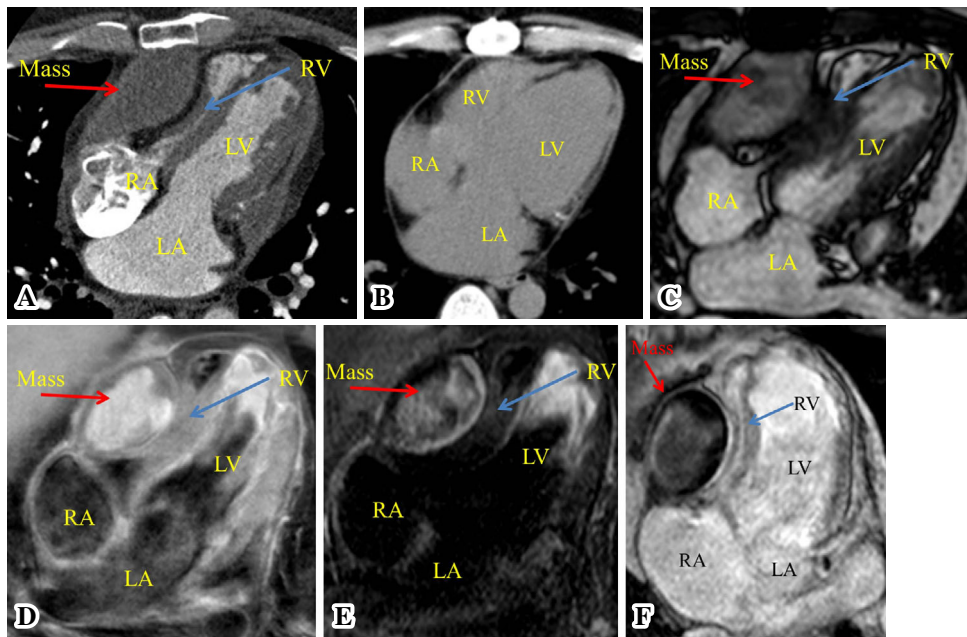


Figure 1. (A) The arrow indicates a mass compressing the right ventricle. The right ventricular cavity was collapsed. (B) Computed tomography performed 12 months previously showed no mass formation. (C-F) Cine magnetic resonance imaging (C) demonstrated an intrapericardial mass measuring 5.0 cm×3.3 cm×7.0 cm in size, lateral to the right ventricle and compressing the right ventricular cavity. T1-weighted (D) and T2-weighted (E) black-blood images showed a mass with heterogeneous high signal intensity and a thick and dark rim. No late gadolinium enhancement was observed in the mass (F).

RV, and revealed that the cavity of the RV had collapsed (Fig. 1A). Right-sided pleural effusion, ascites, and a dilated inferior vena cava were found. There were no signs of malignancy. However, TTE and CT could not accurately confirm whether the mass was located within the pericardial cavity. In addition, CT performed 12 months previously showed no mass formation (Fig. 1B). To further elucidate the etiology and the accurate location of the mass, we performed contrast-enhanced CMRI. Cine MRI demonstrated an intrapericardial mass measuring 5.0 cm×3.3 cm×7.0 cm, lateral to the RV, and compressing the RV cavity. T1- and T2-weighted black-blood images showed a mass with heterogeneous high signal intensity and a thick and dark rim (Cine MRI, Fig. 1C; T1-weighted CMRI, Fig. 1D; T2-weighted CMRI, Fig. 1E). No late gadolinium enhancement (LGE) was observed in the mass (Fig. 1F). The mass was considered to be a chronic hematoma. Coronary angiography excluded perforation of the graft, and local or systemic inflammation was excluded based on the laboratory data and MRI findings. Bacterial and tuberculous cultures of the pericardial effusion were both negative. Cardiac catheterization revealed a central venous pressure (CVP) of 22 mmHg, and a dip and plateau pattern of pressure in the RV. The mass was diagnosed as a very late intrapericardial hematoma and pericardiectomy with the surgical removal of the hematoma was performed. Median sternotomy showed that the hematoma was located on the surface of the heart (Fig. 2A). The hematoma was completely removed but the bleeding source

was not detected. After pericardiectomy, the CVP improved from 22 mmHg to 6 mmHg. The removed hematoma was composed of clotted blood with fibrosis and organization (Fig. 2B). The excised pericardium was organized and hyalinized (Fig. 2C). Focal infiltration of lymphocytes and plasma cells with signs of angiogenesis and hemosiderosis were observed (Fig. 2D). A pathological examination showed a hematoma without infectious or neoplastic changes. CMRI showed the marked improvement of the RV function at 1 month after the procedure (Fig. 2E).

Discussion

We reported the case of a patient with a very late intrapericardial hematoma that occurred 8 years after CABG and which caused heart failure. Because a CT scan performed 12 months previously showed no mass formation and because the symptoms had occurred for 6 months, it was assumed that the hematoma had gradually formed within the year since the previous examination. Thus, the hematoma was considered to be unrelated to the CABG procedure. Although some cases of chronic expanding hematoma have been reported (3-5), the mechanism underlying their formation is still unclear. One possible theory is that the products of the cellular breakdown of leukocytes, erythrocytes, hemoglobin, platelets, and fibrin inside the clot cause a fibroblastic reaction (6). Continued inflammation increases the permeability of the vascular wall resulting in the bleeding of

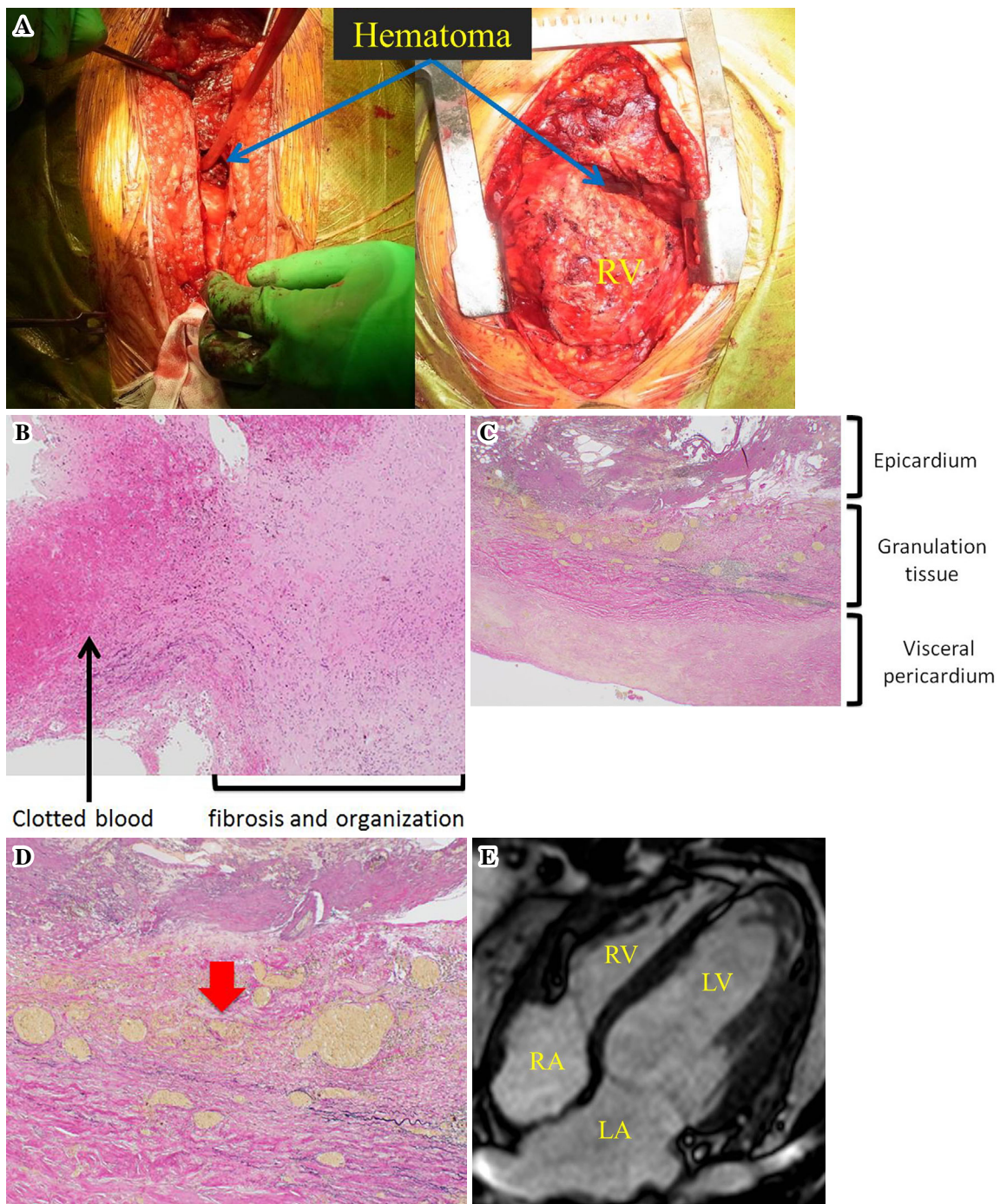


Figure 2. (A) The hematoma was located on the surface of the heart and was easily aspirated. (B) The microscopic findings of the removed hematoma showed that it was composed of clotted blood (arrow) with fibrosis and organization. (C) The pathological findings of the excised pericardium included organization, hyalinization, and the focal infiltration of lymphocytes and plasma cells. (D) The arrow indicates plasma cells with signs of angiogenesis and hemosiderosis. (E) The marked improvement of the right ventricular function was observed.

the damaged capillaries beneath the fibrous capsule. In this case, although we could not detect the bleeding source macroscopically, a pathological examination showed angiogenesis and hemosiderosis in the pericardium. We assumed that this angiogenesis of the inflammation site in the pericardium was the cause of the hematoma.

It is difficult to differentiate chronically expanding hematomas from malignant tumors because of their gradual in-

crease in size. CMRI is preferred to TTE and CT because MRI can clearly show the tumor and the detailed inner fluid characteristics of the mass better than TTE and CT (7). CMRI has been reported to be useful for evaluating LV and RV function, for differentiating between cardiomyopathies, and for distinguishing between malignant and benign cardiac masses (8-11). In the present case, CMRI clearly showed a mass in the intrapericardium and helped to diagnose the in-

trapericardial hematoma via the different intensities of T1- and T2-weighted imaging and the absence of LGE in the mass. Moreover, CMRI was employed, both preoperatively and postoperatively, to assess the hemodynamics. The patient has been stable since the surgery and will be followed up with CMRI.

The authors state that they have no Conflict of Interest (COI).

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