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CASE REPORT

INTERMEDIATE

CLINICAL CASE

Extracardiac Mass After Pericardiectomy

Mediastinal Fat Replacement Secondary to Progressive Constrictive Pericarditis

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ABSTRACT

We describe an extracardiac mass in a 72-year-old man with a history of pericardiectomy for constrictive pericarditis. Imaging studies revealed that progressive shrinkage of the residual pericardium compressed mitral valve anteriority, and fat replacement of the secondarily enlarged posterior space of the atrioventricular groove appeared as a space-occupying lesion. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2021;3:339-44) © 2021 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

A 72-year-old man was referred to our hospital for further examination of a suspected extracardiac mass, which was incidentally detected on annual follow-up transthoracic echocardiography (TTE) after cardiac surgery (**Figure 1A**).

MEDICAL HISTORY

The patient had a history of hypertension, chronic kidney disease, and coronary artery stenting for myocardial infarction. Additionally, he had also undergone coronary artery bypass grafting (CABG) for triple-vessel disease 19 years previously. During the post-surgical period, he received short-term glucocorticoid therapy to manage the inflammation and pericardial effusion caused by post-pericardiotomy syndrome; however, the effusion did not resolve spontaneously. The patient underwent pericardiectomy 1 year after CABG for constrictive pericarditis (CP), when he developed right-sided heart failure attributed to CP. The condition was diagnosed by the appearance of pericardial thickening on computed tomographic (CT) imaging, elevated right atrial pressure, and dip-plateau configuration of right ventricular pressure after pericardiocentesis. There was no recurrence of pericardial effusion or inflammation after pericardiectomy.

LEARNING OBJECTIVES

- To make a differential diagnosis of an extracardiac mass by multimodality imaging.
- To understand the possible clinical course of CP after pericardiectomy.

INVESTIGATIONS

The patient had no symptoms at presentation. His vital signs were as follows: body temperature 35.8°C (96.44°F), heart rate 79 beats/min, blood pressure 127/95 mm Hg, respiratory rate 16 breaths/min, and oxygen saturation 98% on ambient air. On

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ABBREVIATIONS AND ACRONYMS

CABG = coronary artery bypass grafting

CP = constrictive pericarditis

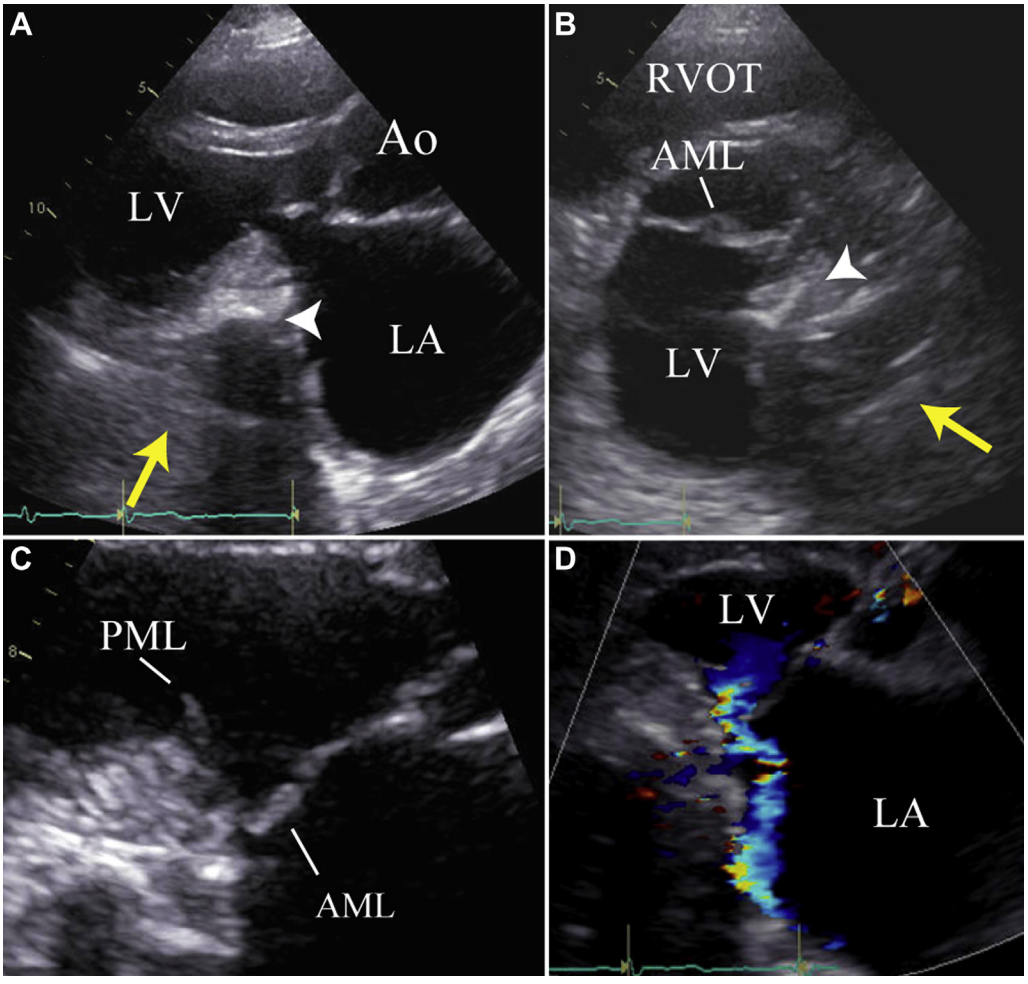
CT = computed tomographic

MRI = magnetic resonance imaging

TTE = transthoracic echocardiography

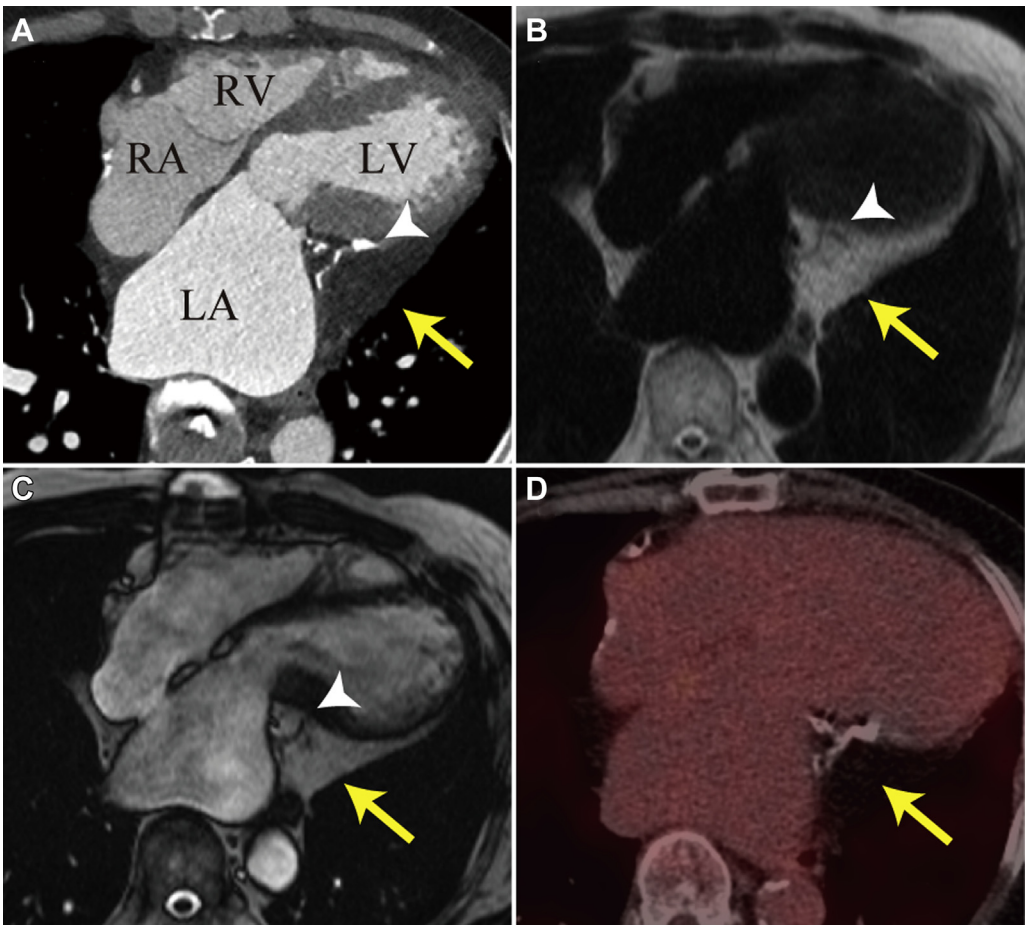
physical examination, jugular venous pulsations were not elevated, and slight pitting edema was seen on his left tibia, which had persisted since the saphenous vein graft harvest for CABG. The brain natriuretic peptide level was 90.6 pg/ml (<18.4 pg/ml), and no pleural effusion was seen on chest radiography. TTE revealed a reduced left ventricular ejection fraction of 45% and enlargement of both atria. The inferior vena cava was 27 mm in diameter, with more than 50% change with respiration. The left atrioventricular groove had a dent deformity indicative of a 40 × 60 mm wedge-shaped mass with massive calcification, as if compressing the heart (**Figures 1A and 1B**, Videos 1 to 4). A narrowed mitral annulus due to an anterior shift of the posterior mitral annulus and narrowing of mitral inflow was identified, where the posterior mitral leaflet tethered toward the left ventricular apex and the anterior mitral leaflet coaptated with the protruding annulus, creating mild mitral regurgitation (**Figures 1C and 1D**). The mean pressure gradient of transmitral flow by continuous-wave Doppler examination was 3.1 mm Hg, suggesting mild mitral stenosis.

FIGURE 1 Transthoracic Echocardiography



(A, C, D) Parasternal long-axis view. **(B)** Short-axis view at mitral annular level. Note the deformity of mitral annulus with the wedge-shaped mass **(yellow arrows)** and calcification with acoustic shadow underneath the mitral annulus **(arrowheads)**. **(C)** Lost coaptation of the mitral valve due to annular deformity. **(D)** Color Doppler of the mitral valve in systole shows mild mitral regurgitation. See Videos 1 to 4.
AML = anterior mitral leaflet; Ao = aorta; LA = left atrium; LV = left ventricle; PML = posterior mitral leaflet; RVOT = right ventricular outflow tract.

FIGURE 2 **Multimodality Imaging Studies**

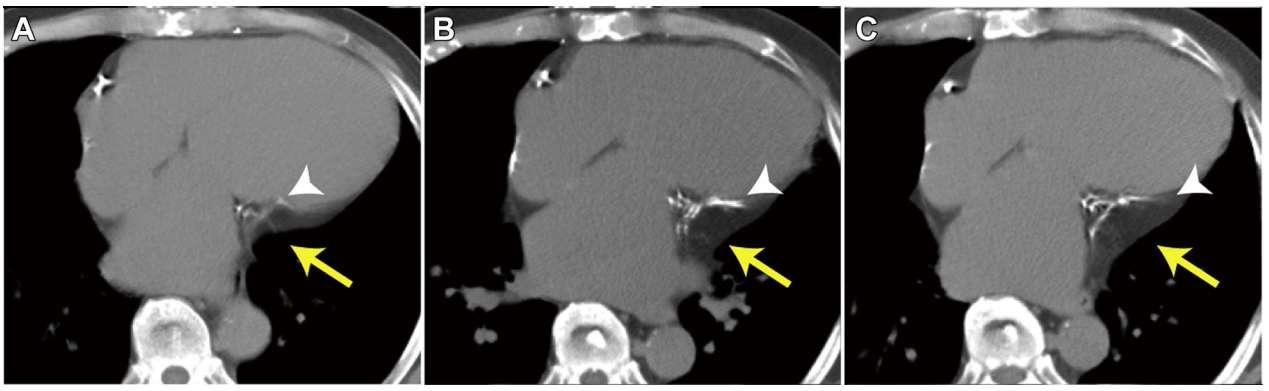


(A) Axial view on contrast-enhanced computed tomography (CT). See Video 5. **(B)** T1-weighted magnetic resonance imaging. **(C)** T2-weighted magnetic resonance imaging. **(D)** Fluorine-18-fluorodeoxyglucose (FDG) positron emission tomography/CT. Fat signal tissue occupies the posterior space of the atrioventricular groove **(yellow arrows)**, which continues until the normal mediastinal fat without border with no contrast enhancement or FDG uptake. Calcified pericardium lateral to the posterior left ventricle (LV) **(white arrowheads)**. LA = left atrium; RA = right atrium; RV = right ventricle.

Subsequent CT imaging showed homogeneous fat-density tissue with a localized calcified layer on the outer side, which was adjacent to the left atrioventricular groove (**Figure 2A**, Video 5). This lesion was confirmed as a high-intensity area on both T1-weighted and T2-weighted magnetic resonance imaging (MRI), which was compatible with the fat-containing tissue (**Figures 2B and 2C**). Additional multiplanar reconstruction of the MRI revealed sequential continuation of the tissue into the surrounding pericardial fat, without a clear boundary. This lesion showed no contrast enhancement, no mass effect on the pleural cavity, and no fluorodeoxyglucose uptake (**Figure 2D**).

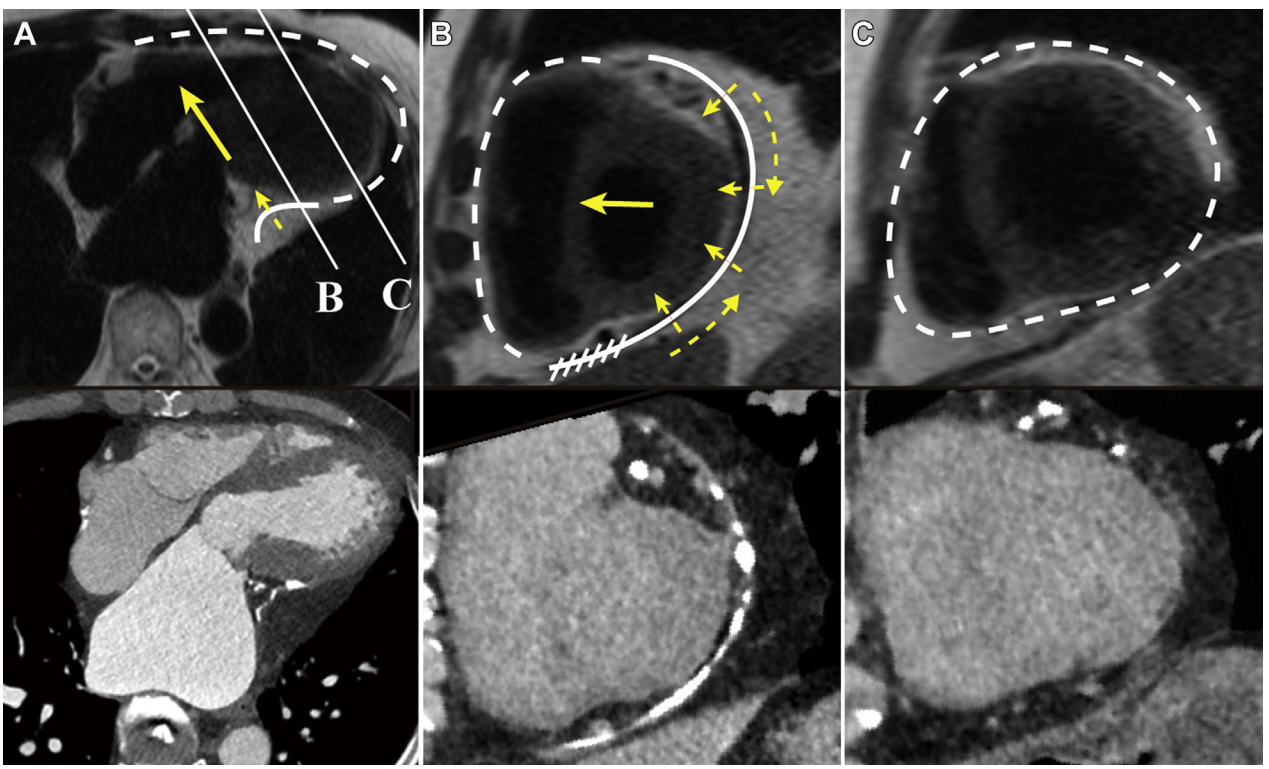
We further referred to the previous CT images. Current observations were similar to the CT images taken 4 years earlier (14 years after pericardiectomy), but CT images taken 10 years earlier (8 years after pericardiectomy) showed almost no deformity of the mitral annulus and less calcification and enlarged posterior space of the atrioventricular groove (**Figure 3**). Location of the wide stripe calcification matched the residual pericardium, which was identical around the left atrioventricular groove. Here, the strong tissue adhesion and bypass grafts prevented it from being resected during the preceding surgery, and the infracardiac diaphragmatic surface was particularly immobile on cine MRI (Video 6).

FIGURE 3 Time Course of Computed Tomographic Imaging



Axial view on computed tomography: **(A)** 10 years previously, **(B)** 4 years previously, and **(C)** at the time the mass was detected. Constriction of the calcified pericardium **(white arrowheads)** and enlargement of the posterior space of atrioventricular groove **(yellow arrows)** progress between **A** and **B**. See Video 6.

FIGURE 4 Schematic Explanation of the Hypothesis of the Disease Course



(A) Axial view. **(B, C)** Short-axis view at base and middle of the left ventricular (LV) level of the T1-weighted magnetic resonance image and computed tomography. Calcified unresected pericardium **(solid white line)**, adhered to the bottom **(striped area)**, shifts toward the anterior thorax **(yellow arrows)** with progressive shrinkage **(yellow dotted arrows)** creating posterior space. No LV deformity and posterior space enlargement was seen at the midventricular level without pericardium **(white dotted lines)**.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of a homogeneous fat-containing mass-like lesion in the atrioventricular groove included neoplasms.

MANAGEMENT

The homogeneous fat-containing tissue without a clearly demarcated boundary, no contrast enhancement or fluorodeoxyglucose uptake, and no change in appearance for 4 years supported the exclusion of neoplasms.

In this case, a history of pericardiectomy and progressive calcification, consistent with the residual pericardium, was a notable finding. Hence, we hypothesized that post-operative adhesions and progressive degeneration of the shrunken residual pericardium accompanied by concomitant immobility of its supradiaphragmatic portion compressed the heart to the anterior chest wall and contributed to the anterior shift of the posterior mitral annulus. Following this, mediastinal fat tissue occupying the secondary space created in the posterior space of the atrioventricular groove appeared as a mass-like lesion (**Figure 4**).

The patient presented with mild mitral stenosis that did not require intervention, and his chronic heart failure was managed with medication. The patient was kept under regular observation with an annual TTE.

DISCUSSION

Progressive constriction of the residual pericardium might result in mitral deformity and secondary mitral stenosis and regurgitation, as well as the creation of an enlarged posterior epicardial space, with fat compensation mimicking a space-occupying lesion as seen in the present case. Sequential change seen on CT imaging and multimodality imaging findings were helpful for identification of the probable cause.

Generally, the pathophysiological response of the pericardium caused by infectious and noninfectious underlying conditions is suggested to be the main contributor to the progression of pericarditis (1). Among various etiologies, preceding CABG was a presumed cause in this patient. The incidence of CP after cardiac surgery ranges from 0.2% to 2.4%, and the period between surgery and the appearance of symptoms related to CP varies from 1 to 8 months (2,3). Early post-operative pericardial effusion that is not drained is reported to be the risk factor for the development of CP, and the residual blood in the pericardium after surgery leading to inflammation is the probable mechanism (3). However, other potential mechanisms are also indicated by the clinical course in this case. These include continuous pericardial effusion after primary surgery and the progress of the residual pericardial constriction asymptomatically in the chronic phase.

There are insufficient data regarding patient factors or specific mechanisms related to recurrent CP, and incomplete resection of the pericardium during initial surgery is suggested as a potential factor (4). Pericardiectomy is an established standard therapy for patients with CP. Although the extent of decortication to achieve better long-term outcomes is debatable, resection of the pericardium as far as possible before consequent irrevocable cardiac damage is generally recommended (5–7). In the present case, although there was a distinct mitral annular deformity, the adverse effects on hemodynamic status following the preceding pericardiectomy were limited. Progressive constriction of the residual pericardium is considered the probable pathophysiology for arriving at a diagnosis.

FOLLOW-UP

The patient developed no symptoms, and the mitral annular deformity and severity of mitral stenosis assessed on TTE have remained unchanged for 3 years.

CONCLUSIONS

This was a rare case of chronic progressive constriction of the residual pericardium following pericardiectomy, which resulted in secondary mitral deformity and an apparent space-occupying lesion. This case suggests the relevance of optimal pericardial reduction during the first surgery and highlights the importance of following serial multimodality imaging follow-up for ensuring residual pericardium constriction.

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KEY WORDS cardiac magnetic resonance, computed tomography, constrictive, imaging, mitral valve, post-operative, ultrasound

APPENDIX For supplemental videos, please see the online version of this paper.