Giant Left Atrium in an Octogenarian with a Complex Etiology

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An 89-year-old woman was admitted to the intensive care unit because of progressive dyspnea and chest discomfort. Her medical history was remarkable for heart failure, atrial fibrillation, and surgical excision of left atrial myxoma 15 years ago. She was found to be well postsurgery follow-up visits and has been treated with furosemide every other day and acenocoumarol.

Following a physical examination, distended jugular veins, perioral cyanosis, fine basal crackles, and muffled heart sounds were observed. She exhibited hypotension with a blood pressure of 96/67 mmHg and a heart rate of 65 beats per minute. Furthermore, hepatomegaly, ankle edema, and an elevated N-terminal pro b-type natriuretic peptide level of 4,524 pg/ml were noted.

Two-dimensional echocardiography (2DE) revealed a significantly enlarged left atrium (LA), measuring 80 mm in anteroposterior diameter, with an LA volume index of 300 ml/m^2. The LA reservoir strain was 6.4% and the conduit strain was 6.9% (Supplementary Video 1). Moderate functional atrial mitral regurgitation was observed. The left ventricle exhibited hypertrophic apical segments, with a preserved ejection fraction (63%). Reduced longitudinal strain was more pronounced in the apical segment. Furthermore, restrictive diastolic function with elevated filling pressures (E/Em 18) was observed (Supplementary Video 2, 3). Her right atrium (RA) was dilated with an RA volume index of 125 ml/m^2. Pulmonary hypertension with moderate tricuspid valve regurgitation was observed. Agitated saline 2DE was performed. However, no intracardiac shunts were confirmed (Supplementary Video 4).

Computed tomography was performed for further investigation (Figure 1, 2). Severely dilated atria were confirmed with the LA larger than the RA. Moreover, a small thrombus in the LA appendage and apical hypertrophic cardiomyopathy (AHCM) were confirmed.

Giant left atrium (GLA) is a rare condition. Its precise etiology remains unknown; however, it is strongly associated with rheumatic mitral valve disease.[1] "Non-rheumatic" GLA has been sporadically reported with different etiologies, such as mitral valve prolapse, cleft of anterior mitral valve with ostium primum atrial septal defect, small ventricular septal defect, and other congenital anomalies.[2-4]

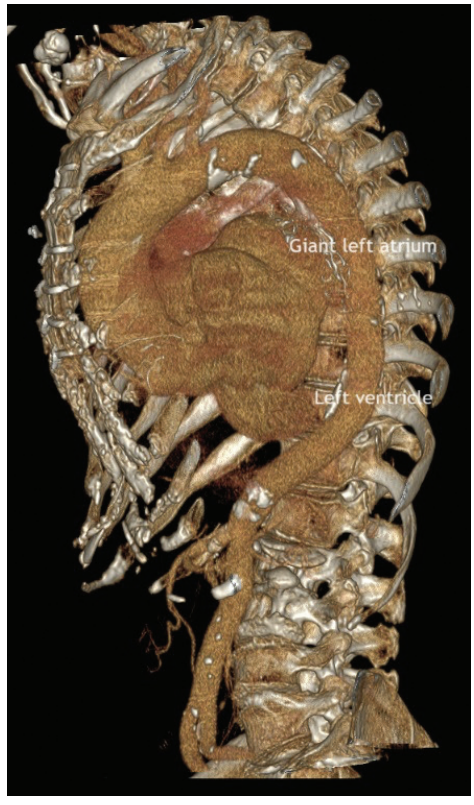

FIG. 1. Computed tomography reconstruction images demonstrating the giant left atrium and small left ventricle.

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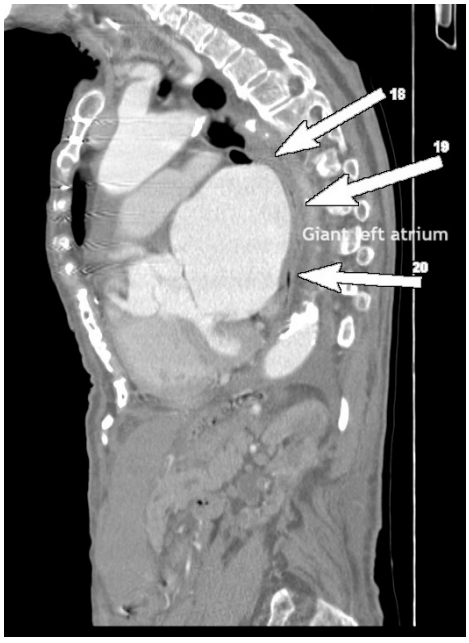

FIG. 2. Computed tomography image with contrast demonstrating giant left atrium and apical hypertrophic cardiomyopathy.

Our case presented a GLA with atrial failure in an octogenarian with AHCM, 15 years after myxoma surgery. The pathophysiology of GLA seems to be complex, involving: 1) hemodynamic response to elevated filling pressures in the context of AHCM; 2) bi-atrial remodeling due to atrial arrhythmia; 3) functional atrial mitral regurgitation; 4) myxoma; and 5) age-related.

Currently, there is no consensus on the management of GLA - whether to opt for surgical reduction of the LA or pursue a conservative strategy while addressing complications, such as atrial failure, thrombogenicity, and arrhythmia.

Following conservative management, the patient showed improvement and was discharged after five days.

Informed Consent: It was obtained.

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