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LETTER TO EDITOR

What if the Ventricle was Innocent for Once? The Infrequent Case of a Guilty Atrium

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Abbreviations

ECG: Electrocardiogram; EF: Ejection Fraction; GLA: Giant Left Atrium; LA: Left Atrial/Atrium; LV: Left Ventricle/Ventricular; RA: Right Atrium; RV: Right Ventricle; TTE: Transthoracic Echocardiography

Dear Editor,

Giant left atrium (GLA) is a rare disease (reported incidence of 0.3%), generally occurs in the mitral valve disease, chronic atrial fibrillation, in patent ductus arteriosus and ventricular septal defect [1,2] and correlated symptoms as dyspnea and dysphagia are a specific [3].

The transthoracic echocardiography (TTE) is the most sensitive imaging technique to diagnose such a rare case, requiring an atrial anteroposterior diameter greater than 80 mm [4] measured in the parasternal long axis view.

An 82-year-old man with chronic atrial fibrillation was admitted to our department for progressive dyspnea, III NYHA class. Almost 10 years ago, he underwent replacing of mitral valve with a mechanical prosthesis CarboMedics size 31 (Sorin Group - Milan, Italy) due to severe mitral valve insufficiency and plastic of tricuspid valve due to moderate - severe insufficiency.

The post-operative TTE showed normal size and wall motion of the left ventricle (LV), mechanical mitral valve prosthesis without paraprosthetic regurgitation, ejection fraction (EF) of 55% and dilated left atrium (LA) with an anteroposterior diameter of 61 mm. The patient has had a follow-up every six month for the first year and then every year, in which echocardiographic assessments were stable.

At admission, he had normal hemodynamic parameters. The electrocardiogram (ECG) showed bradyarrhythmia for chronic atrial fibrillation with heart rate of 55 bpm.

Although the disabling dyspnea, TTE showed a normal LV segmental kinesis, but a surprising abnormal LA dilatation with a significant LV compression by the huge LA (Figure 1A and Figure 1B). The estimated LA volume was between 822.6 and 1035.1 ml with an anteroposterior diameter between 141.08 and 149.59 mm (Figure 1C and Figure 1D). Impossible to estimate correctly EF. Absence of pericardial effusion. Systolic pulmonary artery pressure was 40 mmHg. There was also right atrium (RA) dilatation (Figure 2).

Literature suggests that LA dilatation occurs in response to pressure overload resulting from fibrosis and/or calcification of the LA, increased LA afterload, in the setting of mitral valve disease or LV dysfunction [5], but in our case LV dysfunction is a consequence of crushing by huge LA. The literature does not contain case report in this regard about the GLA that compresses the LV, nor a LA anteroposterior diameter > 130 mm [6].

Left atrial plication, among other surgical techniques to reduce LA dimensions, seems the most suitable, although some surgeons established that the mitral valve

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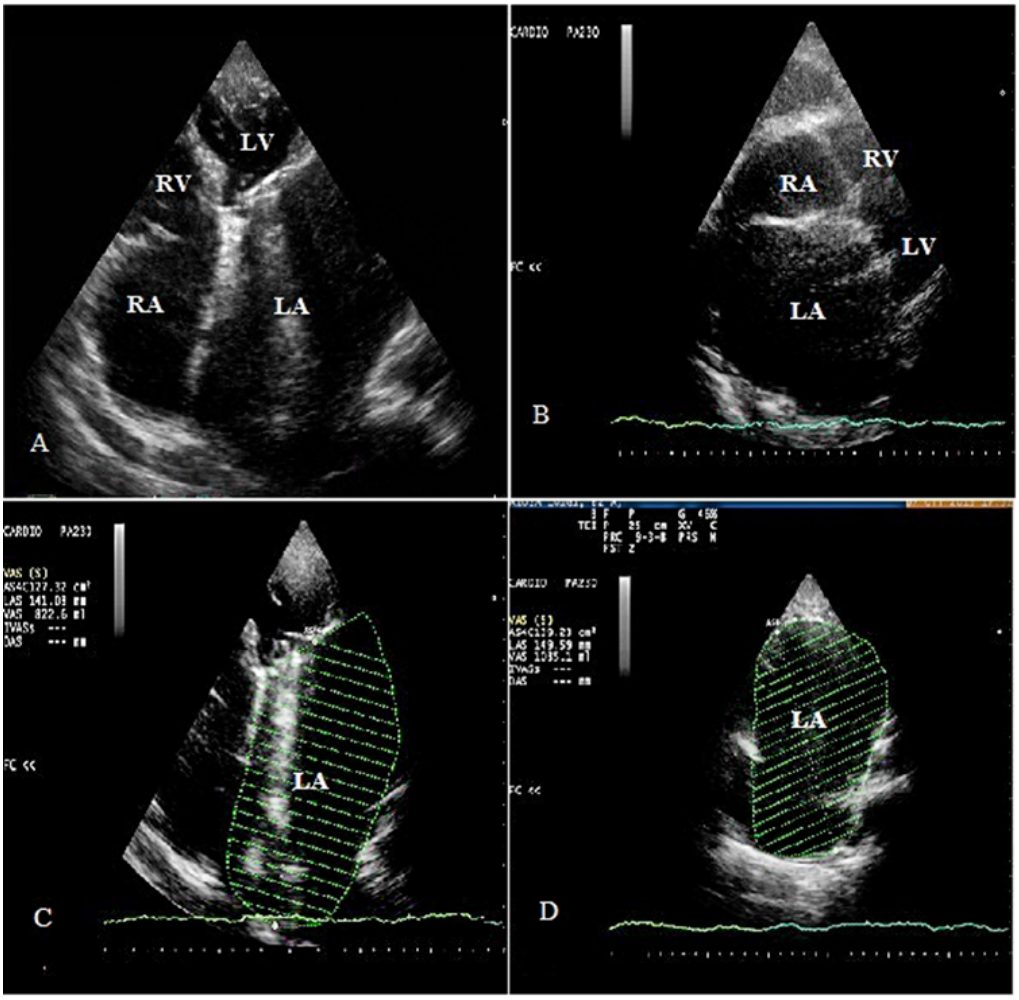

Figure 1: A) Apical four-chamber view of the LV compressed by the huge LA and mechanical mitral valve prosthesis; B) Subcostal four-chamber view of the enormous LA; absence of pericardial effusion; C,D) Apical four-chamber view of the LA dimensions.

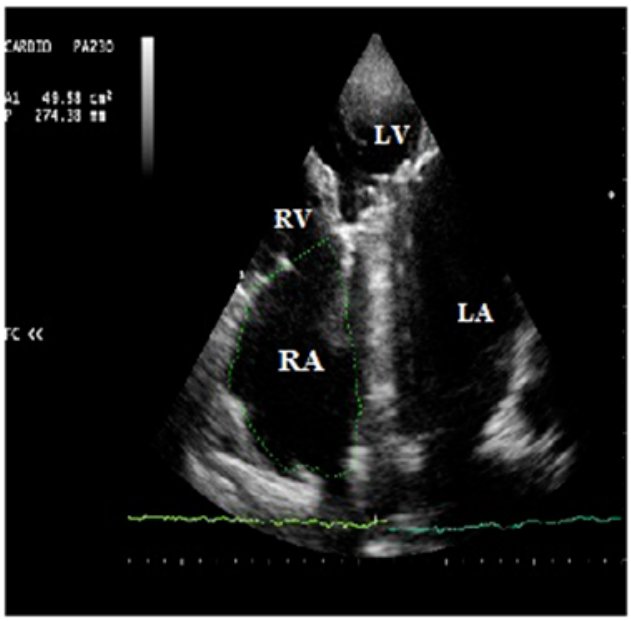

Figure 2: Apical four-chamber view: Highlights increased size of RA.

surgery alone will result in the reduction of left atrial size. Nevertheless, the rheumatic and irreversible process causes loss of tone by the elastic fibers of the tissue; Thus, the left atrial enlargement is beyond repair [7]. Atrial fibrillation also may be a possible cause of atrial enlargement since experimental animal studies have documented that it induces electrical, contractile and structural remodeling [8].

The normal wall motion of the LV suggests that our patient is affected by an atypical dyspnea because it is not induced by LV diastolic dysfunction [9]. First reported in literature, the enormous atrium of our patient (anteroposterior diameter between 141.08 and 149.59 mm) plays an important role in determining the ingravescent dyspnea: The GLA crushes the LV that involves incomplete and inadequate ventricular filling. The LV compression probably induces not only diastolic dysfunction but also systolic one, since it was difficult to estimate the effective FE just because the LV is crushed.

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