And if the Ventricle was Innocent?: The Rare Case of the Guilty Atrium

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Abstract

The literature is full of clinical cases related to the giant left atrium (GLA), which is a cause of compression of the left ventricle, giving related symptoms including dyspnea, dysphagia and atrial fibrillation.

In these settings we are presenting though an interesting case report of a patient of 82 years old man with an abnormal enlarged left atrium, who was admitted by our department for aggravated dyspnea.

The transthoracic echocardiogram performed in our department showed a surprisingly dilated left atrium and compressed left ventricle which over time caused dyspnea, with significant images.

Therefore, we conclude that the incomplete and inadequate ventricular filling, causing the diastolic dysfunction, are caused by compression of the left ventricle from the huge left atrium.

Keywords: Giant left atrium; Echocardiogram; Atrial fibrillation

Abbreviations

Introduction

Giant Left Atrium (GLA) is a rare disease (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 aspecific [3].

The echocardiogram is the most sensitive imaging technique to diagnose such a rare finding, requiring an atrial anteroposterior diameter greater than 80 mm associated with dyspnea e dysphagia (Table 1) [4-6].

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Table 1: Criteria of GLA definition

Case Report

A 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 Optiform 31 mm, Sorin (Liva Nova), London, due to severe mitral valve insufficiency and plastic of tricuspid valve due to moderate-severe insufficiency. The post-operative transthoracic echocardiogram (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. This surprising data had never emerged during previous echocardiographic checks, in fact this leads us to believe that there has probably been a rapid increase in the volume of the left atrium.
At admission, he had normal hemodynamic parameters. The electrocardiogram (ECG) showed bradycardia for chronic atrial fibrillation with heart rate of 55 bpm.

Although the disabling dyspnea, TTE showed a normal left ventricular segmental kinesis and EF, but a surprising abnormal LA dilatation and the LV compressed by the huge LA (Figures 1A and 1B). The estimated LA volume was between 822.6 and 1035.1 ml with an anteroposterior diameter between 141.08 and 149.59 mm (Figures 1C and 1D). There was also right atrium (RA) dilated (Figure 2) with a systolic pulmonary artery pressure was 40 mmHg.

Also in this patient, the instrumental examinations performed showed not only a giant LA, but that the LV, the right ventricle (RV) and the RA were compressed, mainly causing dyspnea and congestive heart failure. The same patient underwent a mitral valve replacement, an LA reduction, and tricuspid valve repair, with a post-operative chest X-ray 6 months following surgery showed a marked reduction in the cardiothoracic ratio.

Probably a rapid dilatation of LA could be the result of congenital aneurysms [8] (or predisposition to develop them), or even a chronic atrial fibrillation with the presence of endocardiac thrombi [9].

In our case the normal wall motion of the LV and preserved EF suggest that the cause of dyspnea is the compression of the LV by the GLA, because the altered diastolic function of LV due to an incomplete and inadequate ventricular filling. Therefore, blood accumulates upstream of the left heart in the pulmonary veins, and gradually leads to airway compromise and thus worsening dyspnea.

In fact the size of the left atrium (for indexed volumes greater than 32 ml/m²) quantifies the risk of progression to symptomatic heart failure (1.97 times higher the normal) independently of the other risk factors, as demonstrated by Takemoto et al. on the usefulness of LA volume in the prediction of the first congestive heart failure in patients aged ≥ 65 years with well-preserved left ventricular systolic function [10].

Although surgical therapy may give a seemingly better reduction of the problems related to the size of LA, more evidence of changes in the size of the left atrium implemented with medical therapy alone would be needed, to hope for a less invasive and challenging approach both for the operator and the patient.

References


Xie et al. recently reported in literature an interesting case of giant left atrium in a patient with longstanding rheumatic heart disease [7].