Case Report

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Giant left atrium with left lung damage: a case report

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Abstract

Giant left atrium is most commonly associated with rheumatic mitral valve disease, causing a series of cardiac and extracardiac complications. Cardiac complications are often reported, such as atrial fibrillation, decreased cardiac output, and atrial thrombus formation. Extracardiac complications are rarely described in the literature. We report an unusual case of a 55-year-old woman who was diagnosed with rheumatic heart disease 20 years earlier. Her chief complaints were episodes of chest tightness and difficulty breathing, which she had for more than 30 years. Echocardiography showed severe mitral stenosis with severe mitral insufficiency. Contrast-enhanced chest CT showed that the left thoracic cavity was occupied by a giant left atrium. The left main bronchus was compressed, and the left lung showed complete consolidation without pulmonary function.

Keywords

Giant left atrium, damaged lung, rheumatic heart disease, mitral stenosis, mitral insufficiency, extracardiac complications

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Introduction

Giant left atrium (GLA) is a rare condition closely associated with rheumatic mitral valve disease, seen in only 3%–4% of patients with rheumatic valvular heart disease.[1] The atrial cut-off size for defining GLA differs among investigators;[1,2] however, an anterior-posterior dimension of 65 mm is commonly used in the literature.[2–4] Because of the location of the left atrium, as it increases in size, adjacent organs may be compressed, resulting in complications. Compression of the main bronchus, for example, can result in pulmonary

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complications. This report describes the case of a patient with a history of GLA with damaged left lung.

Case presentation

A 55-year-old woman presented to the Department of Cardiac Surgery, Dongyang People's Hospital, in Zhejiang Province, China, with the chief complaint of repeated episodes of chest tightness, difficulty breathing, and hoarseness for more than 30 years. She was diagnosed with rheumatic heart disease 20 years prior to presentation. Her symptoms had worsened over the preceding month after catching a cold. She denied fever, chest pain, dysphagia, headache, nausea, vomiting, or other symptoms.

Physical examination revealed that the patient had mitral facies (bluish tinge except for flushed cheeks), orthopnea, and jugular venous distension. Her left lung breath sounds were absent, and right lung breath sounds were rough, with scattered moist rales. The apex beat was located in the seventh intercostal space at the anterior axillary line. Both systolic and diastolic murmurs were present at the apex area.

Echocardiographic findings were consistent with rheumatic valvular heart disease. There was severe mitral stenosis with severe mitral insufficiency. The area of the mitral valve orifice was $0.62\,cm^2$, measured during the diastolic phase. There was mild aortic valve stenosis and mild to moderate tricuspid regurgitation. The left atrium was markedly enlarged at $14 \times 8 \times 11$ cm (Figure 1).

Contrast-enhanced chest CT showed that the left thoracic cavity was occupied by a GLA with an anteroposterior diameter of 15 cm. The mitral valve and left atrial wall showed irregular calcification shadows. The left main bronchus was compressed, and the entire left lung was retracted without significant parenchymal air or bronchial shadow. The upper trachea was pulled to the left side by the left atelectatic lung.

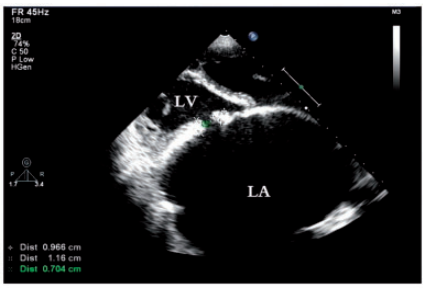

Figure 1. Echocardiography revealed massive enlargement of the left atrium; the mitral valve leaflets were thickened with narrowing of the valve orifice.
LA, left atrium; LV, left ventricle.

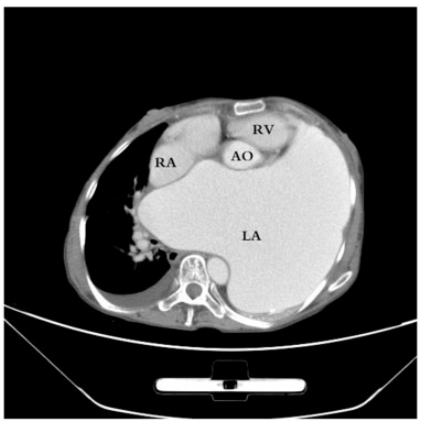

Figure 2. Contrast-enhanced chest CT showed that the giant left atrium occupied the left thoracic cavity.
AO, aorta; LA, left atrium; RA, right atrium; RV, right ventricle.

A filling defect was seen within the left pulmonary artery on CT (Figures 2 and 3).

The patient was treated with cardiac stimulants, diuretics, and vasodilators without improvement in cardiac function, and she ultimately succumbed to heart failure 3 days after admission. The patient's representative refused an autopsy.

This report was approved by the Investigational Ethical Review Board of Dongyang People's Hospital, and written

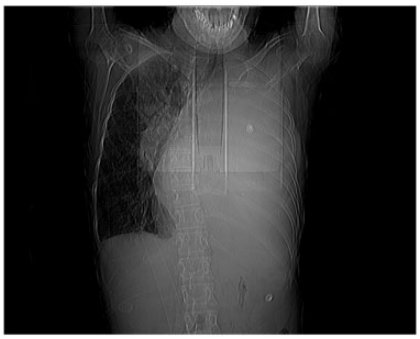

Figure 3. Contrast-enhanced chest CT revealed damage to the left lung.

informed consent was obtained from the patient's representative.

Discussion

GLA is the end-stage result of long-standing hemodynamic or electrical overload. It is most commonly associated with rheumatic mitral valve disease, such as mitral stenosis or mitral insufficiency. Hurst pointed out that mitral regurgitation played a more important role than mitral stenosis in left atrial enlargement in these patients.[5] In this model, rheumatic pancarditis damages the entire heart, and the left atrium dilates more easily when mitral regurgitation fills the chamber with a large volume of blood.[5] A study by Wang et al. showed that genetic mutations can contribute to the remodeling, resulting in left atrial enlargement.[6]

With increased left atrial volume, a series of complications can occur, collectively known as GLA syndrome. This condition can include dysrhythmia (atrial fibrillation) and decreased cardiac output, as well as atrial thrombus formation and other thromboembolic events.[2,5] Our patient was confirmed to have a filling defect within the left pulmonary artery on CT (artery embolization), which might have been related to local blood stasis or deep vein thrombosis. The severely enlarged left atrium may also compress other mediastinal structures and cause symptoms. Esophageal compression can cause dysphagia. Compression of the left main bronchus and/or right middle lobe can cause pulmonary ventilation and air exchange dysfunction.[7] The enlarged left atrium and/or dilated pulmonary artery can compress the left recurrent laryngeal nerve between the aortic arch and the pulmonary artery, resulting in left vocal cord paralysis. It was thought that our patient had a cardiovocal syndrome (Ortner's syndrome). She had hoarseness that may have developed from compression of the left recurrent laryngeal nerve by the huge left atrium.[8]

CT images showed that the osseous structures of chest were intact and demonstrated no significant abnormality. The trachea and tracheal bifurcation were midline, no right pulmonary compensatory changes were seen, and there was no obvious hypoplasia of the left pulmonary artery and its branches. These findings can be helpful in confirming that the left lung pathology was not a result of a developmental defect or rapidly progressing disease. Tuberculosis was not a consideration in this patient, and no satellite lesions were seen on CT images. Consequently, the left lung damage was considered to be related to compression by the GLA.

In summary, our patient had a long history of rheumatic heart disease, and severe mitral stenosis and mitral insufficiency were the notable pathophysiological causes of GLA. As the left atrium continued to expand, it compressed the left main bronchus, causing severe damage to the left lung, seen on CT imaging. To our knowledge, GLA with pathology of this type involving the entire left lung has never been reported.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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