





CASE REPORT

Left atrium and pulmonary artery compression due to aortic aneurysm causing heart failure symptoms



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KEYWORDS

Thoracic aortic aneurysm; Heart failure; Pulmonary edema; Left atrium **Abstract** Patients with thoracic aortic aneurysm (TAA) are mostly asymptomatic and TAA is rarely related to heart failure (HF). We report the case of an 80-year-old female patient, with type A TAA without dissection, with right pulmonary artery and left atrium compression, who presented with HF, preserved ejection fraction and acute pulmonary edema. © 2018 Sociedade Portuguesa de Cardiologia. Published by Elsevier España, S.L.U. All rights

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PALAVRAS-CHAVE

Aneurisma da aorta torácica; Insuficiência cardíaca; Edema pulmonar; Átrio esquerdo

Compressão do átrio esquerdo e artéria pulmonar por um aneurisma da aorta causando sintomas de insuficiência cardíaca

Resumo Pacientes com aneurisma da aorta torácica (AAT) são geralmente assintomáticos e o AAT é raramente associado à insuficiência cardíaca (IC). Nós relatamos o caso de uma paciente feminina, 80 anos, com um AAT tipo A sem sinais de dissecção, que apresentou compressão do átrio esquerdo e da artéria pulmonar direita e desenvolvimento de IC com fração de ejeção preservada e edema agudo de pulmão.

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Introduction

* Corresponding author. *E-mail address:* lagoeiro@globo.com (A.J.L. Jorge). Patients with thoracic aortic aneurysm (TAA) are mostly asymptomatic and TAA is rarely related to heart failure (HF). TAA may present acutely with rupture or dissection,

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Figure 1 Chest radiography: (A) mediastinal enlargement and left pleural and pericardial effusion; (B) After medical treatment: thoracic aortic aneurysm.



Figure 2 Echocardiogram with Doppler IVS: interventricular septum; LA: left atrium; LV: left ventricle; RV: right ventricle.

or chronically, with symptoms related to surrounding structures.¹ Extrinsic compression of the left atrium (LA) and the pulmonary vessels is an uncommon cause of hemodynamic compromise and may be secondary to the involvement of mediastinal structures, including TAA. This compression can lead to increased atrial and pulmonary artery pressures and may consequently cause pulmonary hypertension or pulmonary edema.^{2,3} We report the case of a patient with type A TAA with right pulmonary artery and LA compression, who had HF with preserved ejection fraction (HFpEF) and acute pulmonary edema (APE).

Case report

An 80-year-old female patient, with a previous diagnosis of hypertension and HFpEF, was hospitalized with dry cough, progressive dyspnea on exertion, New York Heart Association (NYHA) functional class III/IV, orthopnea and lower limb edema. She also reported moderate atypical chest pain and dysphagia for solids.

On physical examination, blood pressure was 162/82 mmHg; heart rate was 88 bpm and respiratory rate was 27 ipm. Pathological jugular venous distention was present. The patient had a regular heart rhythm, with no murmurs;

fine crackles in both lung bases and bilateral lower limb edema (++/4+).

Complete right bundle branch block was observed on the electrocardiogram. Chest radiography showed cardiomegaly, mediastinal widening, and left pleural effusion that resolved with medical treatment. (Figure 1). Echocardiogram revealed left ventricular ejection fraction of 72%, increased LA, TAA diameter of 7.2 cm, moderate pericardial effusion with no signs of restriction, and left pleural effusion.

Twelve hours after admission she progressed with APE, which was controlled with medication. Seventy-two hours later she had atrial fibrillation without hemodynamic impairment. Clinical improvement occurred following drug treatment and she was discharged in NYHA class II.

Echocardiogram one week later showed giant TAA with left atrial compression, diastolic dysfunction and preserved left ventricular ejection fraction, without pericardial or pleural effusion. (Figure 2)

Chest angiography was performed and revealed a giant 8.3×7.7 -cm TAA, which compressed the right pulmonary artery, the left and right atriums, the right ventricle and the esophagus, without any evidence of dissection (Figures 3 and 4).

Her clinical status significantly improved after administration of carvedilol, enalapril and furosemide. Correcting