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Pulmonary hypertension as a result of an aortic aneurysm compressing to pulmonary arteries

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ABSTRACT

Thoracic aortic aneurysm is a major health problem with multiple etiologies and potentially devastating consequences. Acute aortic dissection should be included in the differential diagnosis of patients with sudden onset of chest or back pain, syncope, stroke, or acute heart failure. However, these symptoms are not typical of chronic aneurysm without dissection. Furthermore as a rare complication large aneurysms may cause symptoms via mass effect. Herein we report a case of a giant thoracic aortic aneurysm causing severe pulmonary hypertension due to compression of the pulmonary arteries. The it pulmonary hypertension via the mass affect of an aneurysm is considered to be rare, but should also be kept in mind, especially in the absence of an observable reason for pulmonary hypertension and when the size of the aneurysm is extremely large.

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1. Introduction

Thoracic aortic aneurysm is a major health problem with multiple etiologies and potentially devastating consequences. Currently, no large randomized trial has shown that medical therapy can significantly slow or halt the progressive dilatation that eventually leads to dissection and rupture (Danyi et al., 2012). Most aneurysms are clinically silent. There are no specific symptoms of a chronic aneurysm of the ascending aorta. Acute aortic dissection should be included in the differential diagnosis of patients with sudden onset of chest or back pain, syncope, stroke, or acute heart failure. However, these symptoms are not typical of chronic aneurysm without dissection (Lavall et al., 2012) . Furthermore as a rare complication large aneurysms may cause symptoms via mass effect. When aneurysms compress the pulmonary artery, pulmonary arterial hypertension and right heart failure may develop. Herein we report a case of a giant thoracic aortic aneurysm causing severe pulmonary hypertension due to compression of the pulmonary arteries.

2. Case

A 82-year-old woman was examined in the cardiology outpatient clinic with the complaint of dyspnea. She had been suffering from shortness of breath for a few years but her complaints were increased in the last six months. She had no history of cardiovascular disease. She had diabetes mellitus for twenty years which was managed by oral antidiabetics. Her electrocardiogram showed atrial fibrillation with a rapid ventricular rate of 135 per minute. In physical examination a loud S2 was heard with an early diastolic murmur. In contrast to her complaints about dyspnea, it was surprising to find no decompensation finding, such as pretibial edema, hepatomegaly, jugular venous distention and pulmonary overloading. Her lungs were clear and she had no rale or rhoncus dur-

ing auscultation. At first, dyspnea was considered to be due to rapid ventricular rate atrial fibrillation. We administered intravenous digoxin for tachycardia and provided a heart rate within normal limits. Although we achieved a normal heart rate, she was still complaining from shortness of breath. After the management of tachycardia, echocardiography was performed, which showed a normal left ventricular ejection fraction, an ascending aorta of 11 cm width, first degree aortic regurgitation, fourth degree tricuspid regurgitation with a dilated right ventricle and systolic pulmonary arterial pressure of 105 mmHg. Severe pulmonary hypertension with dyspnea primarily suggested acute pulmonary embolism, despite any clinical signs for venous thrombosis. It was planned to refere her to a cardiovascular surgeon but aortic aneurysm was not solely enough to explain dyspnea and increased pulmonary arterial pressure.

Computed tomography also confirmed a giant aortic aneurysm. Radiologists reported that no thrombus was present in pulmonary vasculature. D-dimer levels were also lower than the the cut-off value for pulmonary embolism. In the further evaluation of the computed tomographic images, in the sequences where the ascending aorta was the largest, it was noticed that aortic aneurysm pushed right and left pulmonary arteries both sides and pressed them (Fig. 1). The reason of increased pulmonary arterial pressure was compression of a giant aortic aneurysm to the right and left pulmonary arteries.

In conclusion, although pulmonary embolism is the first diagnosis coming to mind, mass affect of a giant aneurysm has also to be considered. Herein there was no thrombus formation in the pulmonary tract so acute pulmonary hypertension had to be explained by a different mechanism. In the light of the literature, it can be appreciated that secondary pulmonary hypertension due to the mass affect of an aneurysm is not very often. Previously, Tomey et al., (2011) reported a case with a 11 cm width aneurysm causing pulmonary hypertension and right heart failure. A case of an aortic dissection presenting with secondary pulmonary hypertension caused by compression of the pulmonary artery by dissecting hematoma was also presented by Kim et al., (2004), Neri et al., (2001), Desai et al., (1991) and Iskandrian et al., (1977) also reported similar cases. Therefore it can be seen that pulmonary hypertension via the mass affect of an aneurysm is considered to be rare, but should also be kept in mind, especially in the absence of an observable reason for pulmonary hypertension and when the size of the aneurysm is extremely large.

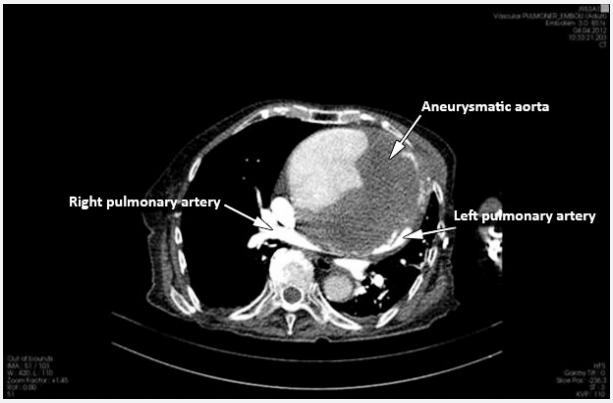

Fig. 1. Giant aortic aneurysm compressing to both right and left pulmonary arteries

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