Case report

Spontaneous rupture of the left pulmonary artery—caused by long-term steroid use?

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Abstract

We describe a 71-year-old woman with spontaneous rupture of the left pulmonary artery. She was admitted with pulselessness of her left arm and lack of sensibility in her left arm and leg. Echocardiography and thoracic CT did not confirm aortic dissection. CT only showed hematoma around the descending aorta. She underwent left-sided thoracotomy. Intraoperatively, a rupture of the left pulmonary artery without any evidence of an aneurysm was found. Potentially predisposing factors for this rupture were long-term use of steroids due to COPD and her age.

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1. Introduction

Spontaneous rupture of the pulmonary artery (PA) is a rare but often fatal event. It has been linked to trauma [1], infection after lung transplantation [2], cystic media necrosis [3], preexisting PA aneurysms [4], pulmonary hypertension [5] and Swan–Ganz catheters [6].

We describe the case of a 71-year-old lady with steroid therapy for 6 years who suffered a spontaneous PA rupture initially thought to be an aortic dissection. The unusual clinical presentation and the pathology findings are discussed.

2. Case report

A 71-year-old woman with a history of chronic obstructive pulmonary disease (COPD) was admitted to another hospital due to an acute exacerbation of her COPD. She had been treated with 10 mg prednisone daily for 6 years. Additionally, she had received 200 mg theophylline b.i.d. and ipratropiumbromide q.i.d. Concomitant diseases included bronchiectasis, arterial hypertension, hyperlipoproteinemia and diabetes mellitus.

The first chest X-ray showed two infiltrates in the right middle and the left lower lobe, respectively. She was quickly stabilized by reproterole infusion and terbutalin s.c. One day later after admission she suddenly complained of severe chest pain, dyspnea and lack of sensibility in her left arm and leg. The preliminary diagnosis of an aortic dissection type B was made by Duplex ultrasound. Consecutively, the patient developed hypovolemic shock and was intubated. She was transferred to our hospital requiring inotropic support with high-dose epinephrine and norepinephrine but arrived with systolic pressures of 60 mmHg only.

A transesophageal echo showed a moving structure in the descending aorta but not an intimal flap typical for aortic dissection. Therefore, the patient underwent a CT scan, which showed hematoma around the descending thoracic aorta. There seemed to be a connection with the pulmonary vessels. Again, no intimal flap could be visualized (Fig. 1). It was decided to perform a left-sided diagnostic thoracotomy given the dismal prognosis of the patient without surgical intervention.
After thoracotomy little blood was found in the left cavity. There was major bleeding from the left upper lobe, which could be controlled by application of vascular clamps in the area of the aortic isthmus. The entire ascending aorta was absolutely normal. A rupture in the back wall of the left pulmonary artery was identified as the bleeding source (Fig. 2). A circumscript aneurysm of the left PA could not be identified. Despite rapidly closing the ruptured PA with a 5-0 Prolene running suture and maximum inotropic support the patient died from cardiac failure after prolonged hypotension.

A specimen from the rupture site was sent into pathology for further examination. An autopsy was refused by the patient’s relatives.

3. Discussion

The experience with the management of spontaneous PA rupture is limited. Risk factors for such catastrophic events include: age above 60 years, pulmonary hypertension, and cardiopulmonary bypass.

Pulmonary hypertension is a common complication of COPD thought to be caused by hypoxic pulmonary vasoconstriction, which in turn induces permanent medial hypertrophy [7]. The patient described herein had a long history of COPD but the pathologic specimen did not show signs of PA hypertension. Hardy et al. [8] showed on human cadaveric lungs that PA hypertension poses no additional risk of PA rupture. They demonstrated that PAs in patients older than 60 years were more likely to rupture at a given pressure than similar arteries in younger patients. Our patient was a 71-year-old lady meeting this particular risk factor.

Chronic steroid use may increase the fragility of the vascular system by its negative effect on collagen formation and connective tissue strength, thereby making vessel rupture more likely. The patient described herein had been on 10 mg of prednisone for 6 years. A possible relation to the PA rupture has to be discussed. On the other hand, many patients require steroids for various clinical conditions and do not suffer a PA rupture. Therefore, the patient’s long-term steroid medication can only be considered a contributing factor to the rupture.

Hemoptysis is the leading symptom after PA rupture. In contrast, our patient presented with very misleading symptoms of pulselessness and loss of sensibility on her left side. Taken together with her history of hypertension type B aortic dissection was suspected. Since this diagnosis was not established by echocardiography in the referring hospital the patient underwent time-consuming studies, i.e. another TEE and—due to its failure to deliver an appropriate diagnosis—a CT scan. One could argue that the patient should have been taken to surgery immediately but we were not sure about the best operative approach. If the patient had a type A dissection a left-sided thoracotomy would have been a rather unfortunate approach. Therefore, we decided to perform these two diagnostic studies. Nevertheless, we were not able to rule out the differential diagnosis of type B aortic dissection by both studies. The clinical symptoms of pulselessness in her left arm and lack of sensibility in her left arm and leg may have been caused by prolonged hypotension due to hypovolemia and perhaps concomitant carotid artery stenosis. After all, the patient also suffered from arterial hypertension, diabetes mellitus and hyperlipoproteinemia—all confounding risk factors for the development of carotid artery stenosis.

In conclusion, clinical symptoms generally attributed to acute type B dissection may be caused by spontaneous PA rupture. It can occur in older patients with long-term steroid use. If older patients with such drug history and long lasting
COPD suffer circulatory collapse rupture of a PA needs to be excluded as a differential diagnosis.

References