

High-risk pulmonary embolism in a patient with acute dissecting aortic aneurysm

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Abstract

In the last decades, an increased incidence of pulmonary embolism (PE) and acute dissection (AD) of aortic aneurysms has been registered mostly due to increased availability of advanced imaging techniques. They seldom occur concomitantly in the same patient. In this paper, we present the clinical challenges and controversies of diagnosis and therapy in a 70-year-old male patient with an atypical presentation of high-risk PE occurring concomitantly with a silent AD of a thoracic aortic aneurysm.

Key words: Acute dissection of an aneurysm, anticoagulant therapy, pulmonary embolism, thoracic aortic aneurysm

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Introduction

Pulmonary embolism (PE) and acute dissection (AD) of thoracic aortic aneurysm (TAA) are serious and life-threatening conditions which seldom occur concomitantly.

PE results in a high percentage of deaths annually being underdiagnosed because its positive diagnosis is elusive and the differential one very wide.^[1]

In the last decades, an increased incidence of AD has been registered. Despite remarkable advances in the diagnosis,^[1,2] the most important factor is a high index of suspicion by the examining physician.

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Case Report

A 70-year-old man, ex-smoker, with systemic hypertension, stable angina, metabolic syndrome, hypercholesterolemia, and hyperuricemia was diagnosed since 2011 with an abdominal aortic aneurysm (AAA). On February 23, 2014, he accused dyspnea and chest discomfort after heavy exertion (shoveling snow) and was admitted to a district hospital with suspicion of angina. Because the dyspnea progressed and the patient's condition got worse, he was transferred after 3 days to the County Emergency Hospital of Timisoara on February 26th. On admission, the physical examination revealed cyanosis, wheezing, blood pressure of 110/70 mmHg, pulse 88 b/min, and a diastolic murmur in the aortic area. Oxygen saturation was 78%. On the electrocardiogram, there was evidence of sinus rhythm, right ventricular hypertrophy (S>R in V5-V6), and deep, negative T-waves in V1-V4. PE was suspected and the contrast enhanced thoracic computed tomography revealed a large, bilateral thromboembolism of the principal and lobar branches of the pulmonary arteries and TAA [Figure 1].

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Because of the newly detected TAA and of the previous AAA, fibrinolysis was postponed. Therapy with oxygen and heparin 5000 UI in intravenous (IV) bolus followed by a continuous IV infusion of 1000 UI/h (activated partial thromboplastin time [APTT] adjusted) was started. APTT values were maintained between 47 and 69 s. The general state of the patient stabilized. Thus, after 4 days, acenocoumarol was added to therapy with 5 days superposition until the repeated international normalized ratio (INR) was between 2 and 3. Heparin was stopped on the 9th day; the mean daily dose was 25200 UI/day. The other administered medication was bisoprolol 5 mg/day and later, after the blood pressure rose, perindopril 5 mg/day.

At admission, the echocardiography revealed enlarged aortic ring (29 mm), dilated root (46 mm), and ascending aorta (60 mm), with a dissection flap, right ventricle (RV) of 38 mm, enlarged left atria (40 mm), left ventricular hypertrophy, and marked septal hypokinesia. Spectral Doppler detected third degree, central, aortic regurgitation,

third-degree tricuspid regurgitation, and an estimated systolic pulmonary artery pressure (PAPs) of 55 mmHg [Figure 2].

Re-evaluation on March 11, 2014, revealed the reduction of RV to 33 mm and of the PAPs to 38 mmHg.

The leg veins ultrasonography demonstrated deep thrombosis of the right internal saphena and popliteal vein as well as the presence of bilateral spontaneous and Valsalva-induced reflux at the level of the saphenofemoral junction.

Spirometry documented a reduction of FEV1 (71%) and forced vital capacity (69%).

Laboratory tests were normal excepting the following revealed pathological data: D dimmer - 18.6 µg/mL, blood glucose - 124 mg/dL, high-density lipoprotein-cholesterol - 33 mg/dL, Low-density lipoprotein-cholesterol - 128 mg/dL, triglycerides - 195 mg/dL, and uric acid - 9.2 mg/dL.

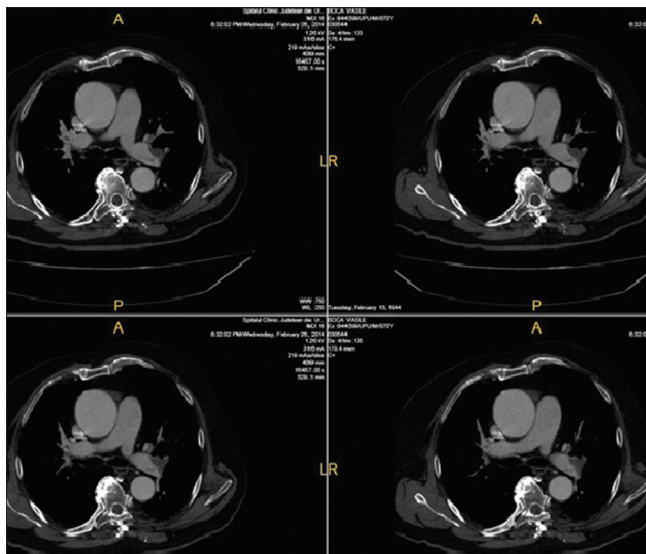


Figure 1: Contrast enhanced transmission computed tomography evidencing subtotal thrombosis of the left and right pulmonary arteries and the lobar branches and presence of a thoracic aortic aneurysm

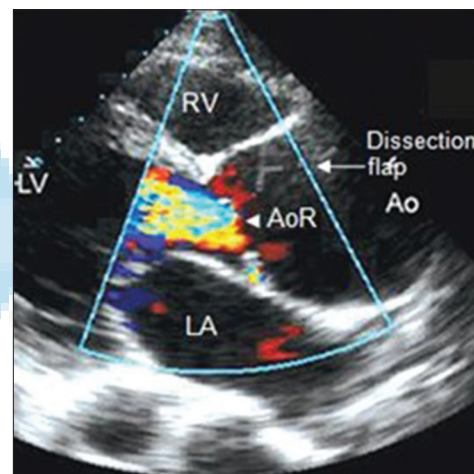


Figure 2: 2M mode and spectral Doppler echocardiography performed at admission evidencing enlarged RV and ascending Ao with a dissection flap: Ao=Aorta; LA=Left atrium; LV=Left ventricle; RV=Right ventricle; AoR=Aortic regurgitation

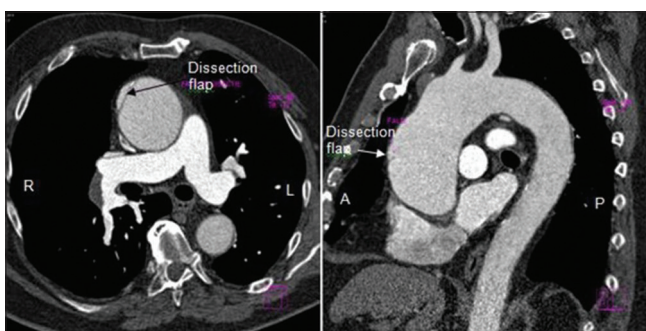


Figure 3: Multislice transmission computed tomography demonstrating the presence of acute dissection of thoracic aortic aneurysm and residual thrombi in the pulmonary arteries



Figure 4: Multislice transmission computed tomography with tridimensional reconstruction demonstrating the presence of thoracic aortic aneurysm and the dissection

A control multislice transmission computed tomography (TCT) with tridimensional reconstruction, performed on March 14, 2014, revealed an aneurysm of the ascending aorta (Stanford A): 4.38/4.28 cm in the supra-avalvular and 6.58/5.93 cm in the tubar region, with a dissection flap and an AAA. Some unobstructive thrombi were still present in both principal branches of the pulmonary artery remained some unobstructive thrombi, extended to the proximal segments of the lobar arteries [Figures 3 and 4].

After 1 month, the patient underwent angiocoronarography, which revealed two 75% stenosis of the proximal left anterior descending. He was scheduled for the replacement of the ascending aorta with aortic annuloplasty and aorto-coronarian bypass. The patient was stable while undergoing therapy with perindopril 10 mg/day, bisoprolol 5 mg/day, atorvastatin 40 mg/day, and acenocoumarol 3 mg/day, INR adjusted, until September, when surgery was performed. Unfortunately, the patient died shortly after surgery because of acute respiratory distress syndrome.

Discussion

There are a few cases of PE associated with AD of aortic aneurysms reported in medical literature. Most existing papers postulate about compression or even occlusion^[1] of the right pulmonary artery by a dissecting TAA, resulting, eventually, in pulmonary thrombosis^[2] or pulmonary hypertension.^[3,4] Even those cases are very rare, so that Semiz-Oysu *et al.*,^[2] by analyzing, using multislice TCT, the pathological aspects, detected using multislice TCT, in the lungs of 134 patients with AD, could not find any evidence of PE. Other authors describe the association of PE with various types of TAA dissection: Involving the descending aorta^[5] or other Stanford B forms.^[5,6] The concomitant presence of a massive PE with AD of an ascending aortic TAA is even rarer. We found very few cases describing similar associations^[7,8] and none with the dilatation of the aortic ring, and aortic regurgitation, and concomitant AAA.

Our case presented many peculiar aspects. Although the patient had few risk factors for thrombosis, he developed an atypical deep vein thrombosis of the right limb. The patient went to the hospital after accusing progressive dyspnea. It was very difficult to suppose that the PE occurred concomitantly with the dissection of a former undiagnosed TAA since the last one was completely silent. It is probable that our patient developed an asymptomatic TAA because of uncontrolled systemic hypertension associated with multiple cardiovascular risk factors, but we cannot exclude some inherited conditions.

Another challenging problem was the treatment of this patient. Although he presented a high-risk PE, the

administration of fibrinolytic therapy was avoided due to the presence of TAA and the suspicion of AD. Anticoagulant therapy represents a huge problem in such situations that the aspect discussed in other papers, as well.^[8,9] Taking into account the recommendations of the European Society of Cardiology,^[10] anticoagulant therapy is mandatory in patients with PE. Our patient stabilized under therapy with heparin, allowing the introduction of oral anticoagulant treatment, but the possibility of a rupture in the dissected region existed at all times. Anticoagulant therapy, associated with a good control of blood pressure values, gave us the possibility of further investigation and the preparation of the patient for surgery.

Conclusions

Although very rare, the concomitance between an acute dissecting TAA and a PE exists. Both can have an atypical presentation or can even be silent. The decision about anticoagulant therapy, in the presence of aortic dissection, is difficult and must be taken individually. Failure to use such therapy despite contraindications can result in an unfavorable clinical outcome.

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Conflicts of interest

There are no conflicts of interest.

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