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Aortic Dissection and Venous Thromboembolisms (Comprising Deep Vein Thrombosis and Pulmonary Embolisms): A Fatal Association Whose Treatment of the Second may complicate the First

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ABSTRACT
Pulmonary embolism and aortic dissection are serious and life-threatening conditions which seldom occur concomitantly. We present a rare case of uncomplicated type B aortic dissection associated at the same time to deep vein thrombosis and pulmonary embolism, in our case, the question arises on the therapeutic decision, since the anticoagulant treatment of venous thromboembolic disease presents a risk of complication of the aortic dissection.

Introduction

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

Venous thromboembolisms (VTEs), comprising deep vein thrombosis (DVT) and PE, are a major cause of morbidity and mortality among hospitalized patients [1]. The overall age- and gender-adjusted annual incidence is 117 per 100,000 population [2, 3]. Widely accepted predisposing factors for VTEs can be grouped into 3 categories, termed the [a]Virchow's triad[o]: endothelial injury, stasis of blood flow and, hypercoagulability [4].

Our patient suffered from chronic, uncomplicated type B AD, and at the same time from DVT and PE, in that case, the question arises on the therapeutic decision, since the anticoagulant treatment of venous thromboembolic disease presents a risk of complication of the aortic dissection.

Case presentation:

A 34 years women, without cardiovascular risk factors, and without any notable pathological antecedents, was diagnosed since one year and 4 months for Stanford type B aortic dissection, type III of DE BAKEY, extending from the aortic arch to the renal abdominal aorta, with dilatation of the ascending aorta, sinus aneurysm coronary and significant aortic insufficiency, put on medical treatment with Bisoprolol 5 mg and converting enzyme inhibitor 5 mg with good evolution. (figure 1,2)

She was admitted via the emergency department of the Mohammed VI University Hospital Center Oujda (Morocco) with dyspnea and orthopnea. She accused a progressive increase of volume of the two lower limbs and of the right upper limb 3 days before its admission.

Physical examination on admission, revealed regular general condition, without chest pain, heart rate was 82/ minute, right arm blood pressure (BP) was 90/50 mmHg, left arm BP - 100/40 mmHg, respiratory rate 28 breaths/min., pulse oxygen saturation of 90 % on room air. Her height was 160 cm and her weight was 65 kg.

The cardiovascular examination found a diastolic murmur in the aortic area, signs of right heart failure with edema of the two soft white bilateral lower limbs taking the scoop, reaching the root of the thighs, respiratory sounds were clear, and no murmurs were detected, the abdominal examination found a pulsatile peri-umbilical mass, and the rest of the somatic examination found soft white edema not taking the bucket occupying the arm and right forearm.

A laboratory examination were normal, and demonstrated a white blood cell count of 10660 u/L,a hemo globin concentration of 14.4 mg/dl, and a platelet count of 321000 /ul, a blood creatinine level of 7.57 mg/dl.

Electrocardiography revealed a normal sinus rhythm, Left ventricular hypertrophy with secondary repolarization disorder.

A chest X-ray film revealed widening of the media stinum with a cardiothoracic ratio of 71 % and normal lung fields, but no pleural effusion was observed.

A transthoracic echocardiographic examination did detect tricuspid aorta, important aortic regurgitation due to lack of coaptation of aortic sigmoid, aorta dilated at the level of the ring at 29 mm with a coronary sinus aneurysm measuring 67 mm. enlarged left atria (31 cm^2). Normal left ventricular contraction (ejection fraction,50%) with left ventricular dilatation at 60 mm, and an estimated systolic pulmonary artery pressure(PAPs) of 55 mmHg.Right ventricle not dilated with good systolic function, without pericardial effusion. (figure 3)

In front of the edema of the right upper limb, a Doppler ultrasound of the right upper limb was performed showing an ultrasonographic appearance in favor of a deep vein hrombosis of the right internal jugular vein, axillary vein and subclavian right extended to the venous trunk brachiocephalic

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Our patient had a strong suspicion of PE and the contrast enhanced thoracic computed tomography (CT) revealed a right lobar pulmonary embolism with focal infarction and thrombus of the left pulmonary vein and a stationary appearance of the dissection. (figure 4,5)

Our decision was to anticoagulate the patient, so the patient was put on heparin therapy with relay by VKA until the repeated international normalized ratio (INR) was between 2 and 3. Heparin was stopped on the 9th day, with good evolution.

The case we present raises the question of curative anticoagulation in patients suffered from AD and at the same time from DVT and PE.

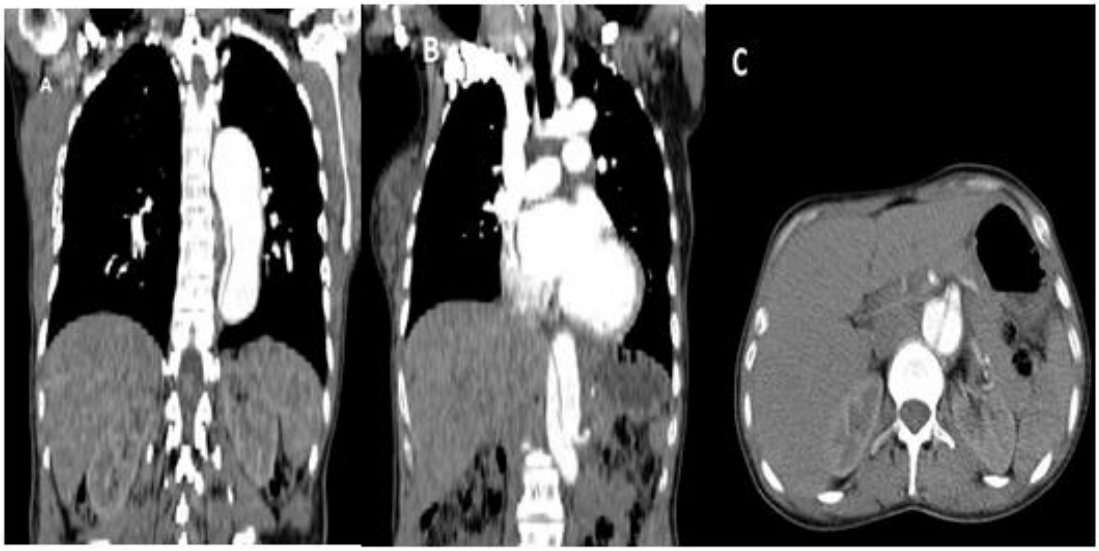

Figure 1. Contrast enhanced transmission computed tomography evidencing aortic dissection involving the thoracic aorta with an unrolled and ectasic aspect (a), abdominal aorta (b) with individualization of a circulating false channel. This dissection extended to the emergence of the superior mesenteric artery (c), without repercussion on visceral organs.

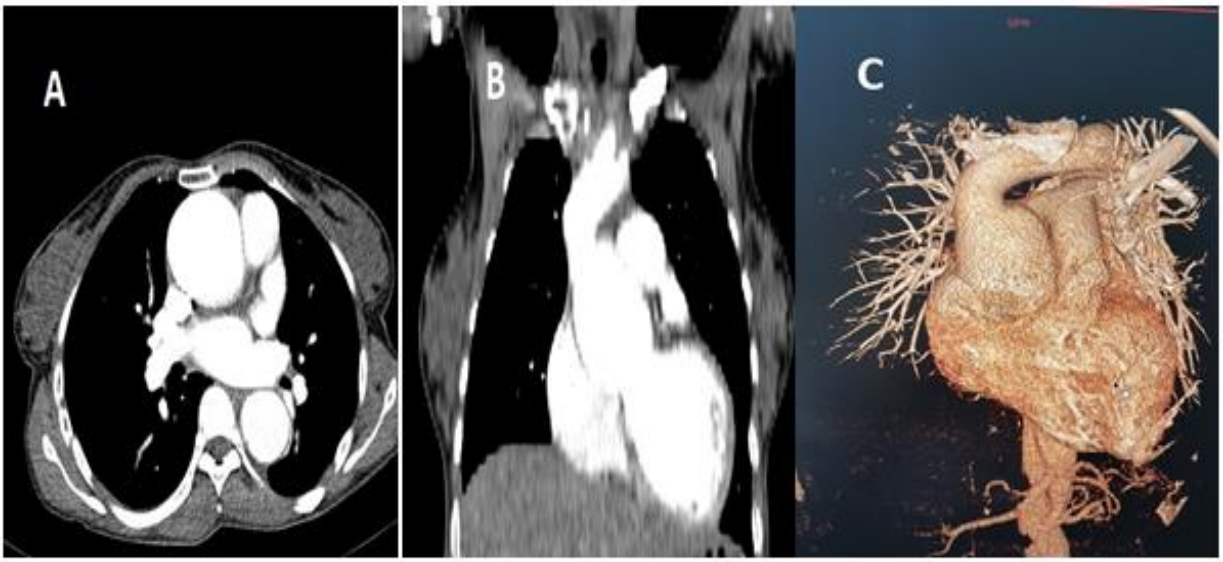

Figure 2. Aneurysm of the ascending aorta respecting the supra-aortic trunks, measuring 57 mm in diameter (a): axial section, (b): coronal section (c): With tridimensional reconstruction.

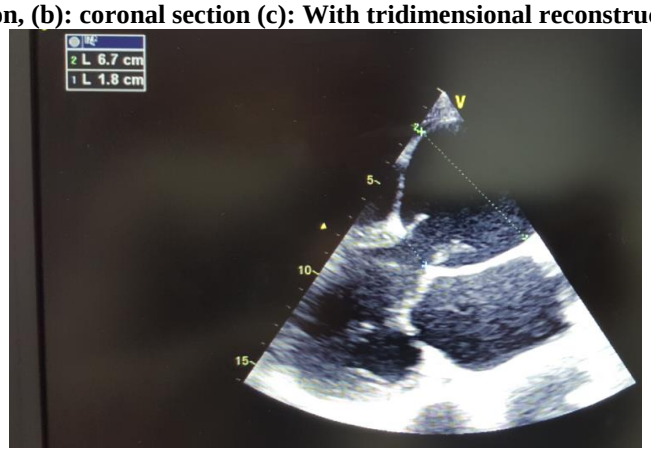

Figure 3. 2M mode echocardiography evidencing a coronary sinus aneurysm measuring 67 mm.

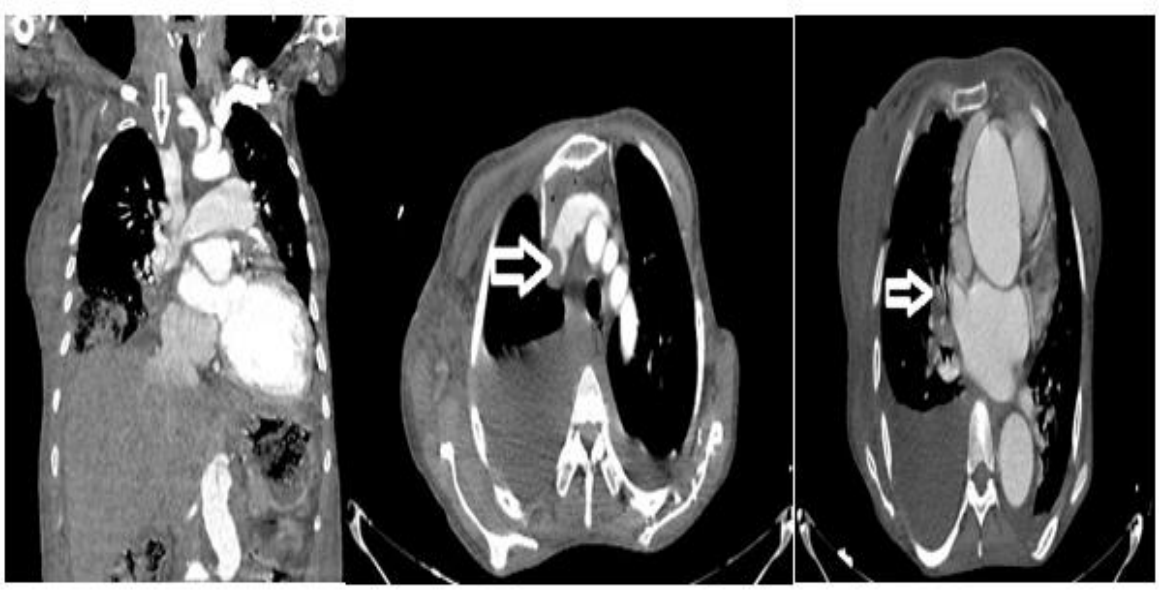

Figure 4, 5. Thrombus of the right venous trunk brachiocephalic extended to the right jugular vein, pulmonary embolism of the right branch of the pulmonary artery extended to its lobar and segmental branches with bilateral pleural effusion.

Discussion:

Aortic dissection is a disease of the tunica media of the vessel allowing blood to flow between the media and intima layers [5]. Incidence is three cases for every 100,000 inhabitants per year, with a bimodal age distribution [6]. There are many factors that can lead to degeneration of the tunica media and to occurrence of dissection, including atherosclerosis, hypertension, smoking, male sex, and inflammatory arteriopathies [7]. One of many peculiar aspects in our patience is the absence of these factors.

Dissections of the aorta are evaluated according to the DeBakey or Stanford classifications. The Stanford classification is according to involvement of the ascending aorta (type A) or the descending aorta (type B).[8] The DeBakey classification refers to involvement of the ascending aorta, divided into type I (both descending and ascending involved), type II (restricted to the ascending), or type III (involving the descending aorta) [9].

High prevalence of asymptomatic PE suggests the usefulness of an early detection of DVT in high risk patients, in order to establish an intense antithrombotic therapy, PEs are found in about 12% of autopsies [10]. Pineda et al. [11] reported that 45% of patients did not complain of the classic symptoms of PE and 19% were asymptomatic before death. Our patient had a strong suspicion of pulmonary embolism due to clinical presentation, the presence of deep vein thrombosis of the right internal jugular vein, axillary vein and subclavian right extended to the venous trunk brachiocephalic, prolonged bed rest, absence of prophylactic anticoagulation, aortic dissection and presence of aneurysm, PE and Stanford type B AD in the same patient is rare with limited cases reported [12,13].

There are a few cases of PE associated with AD reported in medical literature. Most existing papers postulate about compression or even occlusion [14] of the right pulmonary artery by a dissecting thoracic aortic aneurysm (TAA), resulting, eventually, in pulmonary thrombosis [15] or pulmonary hypertension.[16,17] Even those cases are very rare, so that Semiz-Oysu et al.,[15] by analyzing, using multislice transmission computed tomography (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 thoracic aortic dissection: Involving the descending aorta [18] or other Stanford B forms.[18,19].

In our case, the patient had a very strong risk factor for DVT and PE : bedridden with very low physical activity, associated the decision to don't use a prophylactic anticoagulant therapy. However, hypercoagulability can develop during medical treatment of aortic dissection associated with aortic aneurysm, resulting in a high risk of venous thromboembolism. On the other hand, anticoagulation therapy can cause AD to worsen, resulting in the recanalization of false lumens and re-dissection. Unfortunately we did not use any mechanical methods of prophylaxis such as compression stockings or intermittent pneumatic compression devices to reduce venous stasis. The decision to introduce anticoagulant therapy in the patient suffering from aortic dissection and pulmonary embolism, at the same time, was difficult. Nevertheless, failure to use such therapy could have resulted in an unfavorable clinical outcome.

There are no standard treatments for PE and DVT that occur during medical treatment for AD. The challenging problem was the treatment of this patient. Anticoagulant therapy represents a huge problem in such situations that the aspect discussed in other papers, as well [20, 21]. Taking into account the recommendations of the European Society of Cardiology [22], anticoagulant therapy is mandatory in patients with PE, but in our case, the possibility of a rupture existed at all times. Howerever, since thromboembolic risk was very high and pulmonary embolism was extensive and duration of contraindication to adequate anticoagulant might be temporal, we selected anticoagulation therapy as first choice, with daily biological and CT control at the slightest clinical sign, we didn't installed an IVC filter because of the clinical presentation and data from CT and venous echodoppler. After the initiation of anticoagulation therapy, the patient suffers from lower back pain, CT scan shows stationary appearance of the dissection with normal renal ultrasound. (Figure 6) In cases in which the risk of re-dissection increases or a false lumen enlarges during medical therapy, stent grafting might be indicated depending on the anatomical location. [23]

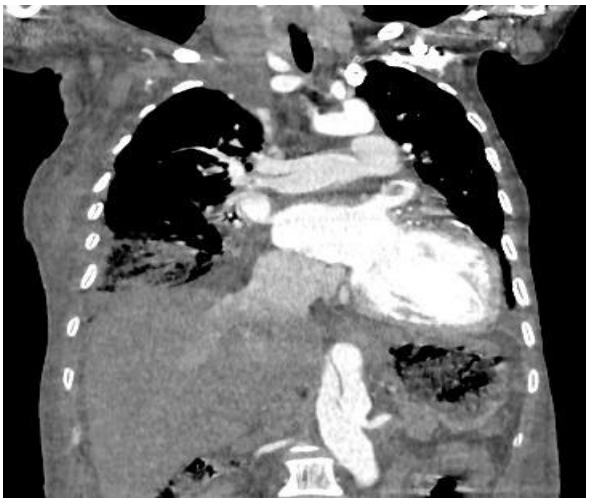

Figure 6. Stationary appearance of the dissection with normal renal ultrasound.

Here, we reported a case of PE and DVT during medical treatment of AD in which the decision of anticoagulation was effective. This does not indicate that anticoagulation therapy is safe for all AD patients. The dose and timing of anticoagulant therapy are thought to be important.

Conclusion:

It is important to prevent venous thromboembolisms during medical treatment for type B AD. The decision about anticoagulation prophylaxis in the presence of aortic dissection in bedridden patients is difficult and must be always made individually. Failure to use such therapy despite contraindications can result in an unfavorable clinical outcome.

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