Atypical Right Pulmonary Artery Dissection Complicating Severe Blunt Chest Trauma

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Pulmonary artery dissection is a rare albeit life-threatening event and it mostly occurs as the spontaneous rupture of pulmonary artery aneurysm complicating chronic pulmonary hypertension. Here, we describe a case of blunt traumatic pulmonary artery dissection.

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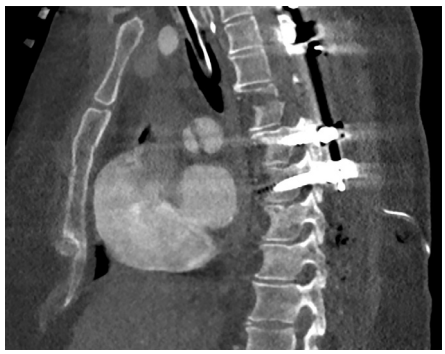

Computed tomography scan. Sagittal view showing right pulmonary artery dissection.

Central Message

Facing traumatic PAD, we opted for nonoperative management considering: (1) its anatomic presentation, (2) the conceivable need for extracorporeal circulation, and (3) the potential for spontaneous healing.

INTRODUCTION

Pulmonary artery dissection (PAD) is a rare and life-threatening event, which usually complicates chronic pulmonary artery hypertension, ultimately leading to pulmonary artery aneurysm rupture.[1] Recent improvements in imaging techniques have allowed earlier diagnosis and timely treatment of PAD including both medical and surgical approaches.

To the best of our knowledge, isolated traumatic PAD (without contemporary aortic isthmus rupture) has been mentioned as a cause of PAD but never thoroughly described in a surviving patient without surgical cure.[2] In this report, we describe a case of isolated, traumatic PAD complicating severe, blunt thoracic trauma whose originality lies both in its atypical anatomical presentation and its favorable outcome after nonoperative management.

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CASE REPORT

Patient Profile

A 56-year-old female was involved in a high-velocity car accident. She had no past medical history except obesity (body mass index: 39 kg m^{-2}) without known chronic sleep apnea, and her last cardiology consultation, 8 years beforehand, was unnoticeable.

At first medical intervention on scene, the patient was hemodynamically stable, though hypoxemic with polypnea. Chest auscultation revealed left lung hypoventilation. Her Glasgow Coma Scale was 14/15, and she suffered from an obvious open fracture of the left femur shaft. She was given analgesia, sedation and was subsequently intubated, while fluid expansion and transfusion were provided.

Full body computed tomography scan revealed no cerebral abnormalities, multiple fractures (ribs, sternum, cervical, and

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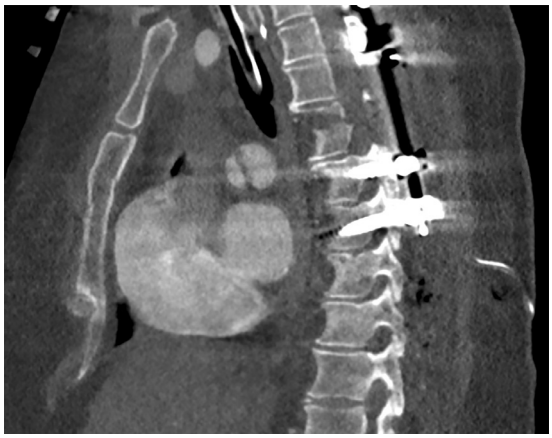


Figure 1. Computed tomography scan. Sagittal view showing right pulmonary artery dissection on the path between the sternal body fracture, anteriorly and Chance-Type fracture of the fifth thoracic vertebral body, posteriorly.

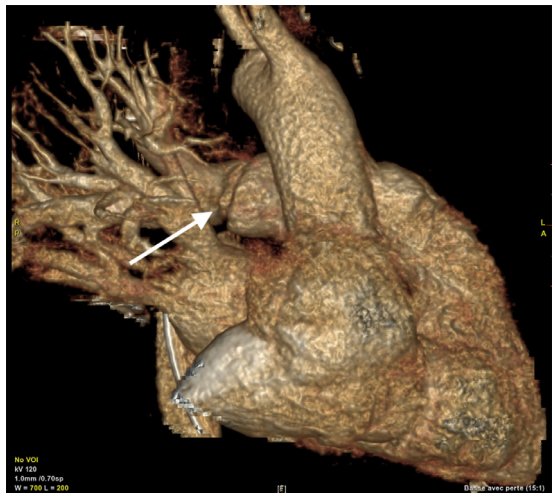


Figure 3. Tridimensional reconstruction of the pulmonary angiogram showing right pulmonary artery dissection (arrow).

thoracic vertebras (Fig. 1), left femur shaft and left ulna), right PAD, bilateral lung contusions with contemporary pleural effusions and left diaphragmatic crus disinsertion with stomach herniation.

PAD Description

More precisely, contrast-enhanced pulmonary angiogram revealed a linear endovascular defect developed on the middle portion of the right pulmonary artery (Fig. 1), consistent with an intimal flap, without parietal hematoma (Fig. 2). The pulmonary artery trunk was nonaneurysmal and 28 mm width (Fig. 3).

Transthoracic echocardiography revealed a globally hypokinetic, dilated right ventricle with severe tricuspid valve regurgitation. We also noticed pulmonary artery hypertension with estimated systolic pulmonary artery pressure of 40 mm Hg and a noncircumferential pericardial effusion of less than 5 mm. Even if abnormal regional right ventricular wall motility was not evidenced on the first echocardiography, initial troponin Ic was 2.50 μg/L and raised up to 4.34 μg/L on the first day, which was highly suggestive of a traumatic contusion of the right ventricle. Transesophageal echocardiography revealed right PAD with intimal flap of 13.2 mm; right pulmonary artery diameter was 22.7 mm (Video 1).

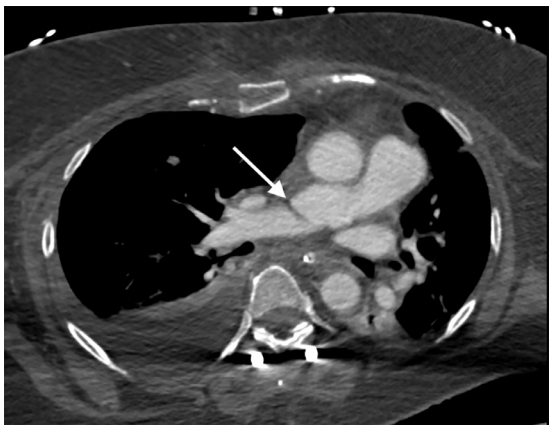


Figure 2. Computed tomography scan. Axial view showing right pulmonary artery dissection with intimal flap (arrow), endovascular defect on the path between the sternal body fracture, anteriorly and Chance-Type fracture of the fifth thoracic vertebral body, posteriorly.

Treatment and Outcome

Initial support consisted in several peripheral surgical interventions including spine osteosynthesis, femoral shaft osteosynthesis, and exploratory laparotomy allowing diaphragmatic suture. Right ventricular dysfunction was managed by reducing right ventricular afterload (protective, low-volume, low-pressure mechanical ventilation, inhaled nitric oxide), by reducing right ventricular dilation (loop diuretics) and optimizing right coronary perfusion pressure (norepinephrine to maintain diastolic arterial pressure above 45 mm Hg) and then antihypertensive drugs to avoid excessive strain on the pulmonary artery wall. During her stay in the ICU, the patient was sequentially assessed with echocardiography and tricuspid regurgitation had almost totally vanished on the third day, together with a normalization of her pulmonary artery pressure. Preventive anticoagulation (deep vein thrombosis prophylaxis) was also introduced early.

Postoperative course was favorable with hemodynamic and respiratory improvements. The patient left intensive care unit 3 weeks after the accident. At 6 weeks, she was asymptomatic, described neither chest pain nor dyspnea and presented no clinical signs of cardiac failure. On her consultation with the thoracic surgeon (3 months after trauma), she did not report any functional limitation on exercise but did not present to the radiology suite for subsequent computed tomography follow-up.

COMMENT

Mechanisms and Typical Presentations of PAD

In most cases, spontaneous PAD occurs as a complication of chronic pulmonary artery hypertension (either essential or complicating postrhumatismal mitral valve stenosis) leading to pulmonary aneurysm rupture. Traumatic PAD cases described in the literature are mainly iatrogenic traumas following pulmonary artery catheterization. In severe blunt chest trauma, aortic isthmus is the preferred site of large vessels injury,[3] traumatic PAD remaining very scarce. Infectious etiologies of PAD such as endocarditis or tuberculosis are also described. More rarely, conjonctive tissue abnormalities, atherosclerosis, or amyloses are mentioned.

The extreme majority of PAD cases are symptomatic, patients reporting dyspnea, chest pain with hemodynamic instability, or cardiogenic shock. If death occurs, it is most often due to dissection progression toward pericardium with major cardiac tamponade. When PAD is diagnosed early enough, treatment is mostly operative because of immediate vital prognosis engagement. However, there is no definitive consensus all the more as surgical treatment remains associated with high perioperative mortality.

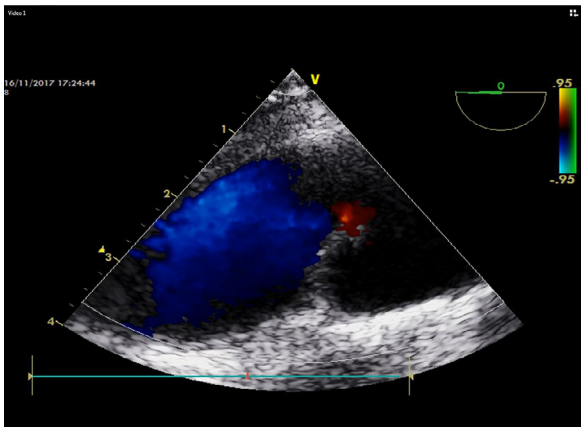
Originality of the Case

In this case, we faced a traumatic PAD associated with right ventricular failure. If preexisting pulmonary hypertension cannot be ruled out in this obese patient with irregular medical follow-up, analysis of the different imaging techniques corroborate an exclusive traumatic mechanism. The association between right PAD on the one hand, and tricuspid regurgitation and right ventricular dysfunction on the other hand cannot be ruled out. Indeed, right PAD may have increased right ventricular afterload but another likely (and potentially associated) reason was right ventricular contusion. It appears that our patient probably had preexisting cardiomegaly with leftward lateral deviation of the heart, right pulmonary artery becoming median and ascending aorta being deviated to the left. Right pulmonary artery was therefore crushed between the sternal body fracture, anteriorly, and Chance-Type fracture of the fifth thoracic vertebral body, posteriorly, all of them being fractured by the shock wave. As the endovascular defect was precisely located, between these bony fractures, it was diagnosed as traumatic PAD. This anatomic variation may also explain aortic isthmus integrity, whose rupture is often associated with high-velocity shocks. Clinically speaking, the main originality lied in the asymptomatic nature of the PAD and the absence of previous chronic pulmonary hypertension, prompting a nonoperative treatment, which deserves further discussion. We did not opt for pulmonary artery stenting as it would have imposed an overly harsh radial strain on the dissected pulmonary artery wall. Concerning a potential surgical approach, we had to take into account (1) the anatomic presentation of the PAD, (2) the conceivable need for extracorporeal circulation, and (3) the potential for spontaneous tissue healing. In our patient, the dissected right pulmonary artery was situated between the descending thoracic aorta (anteriorly) and the trachea (posteriorly). Both aorta and trachea are histologically resilient and allow for a firm contention of the dissected pulmonary artery. In such a situation, resection of the dissected part and anastomosis of the healthy pulmonary artery borders would have been technically challenging. Since the patient suffered from left diaphragmatic rupture and associated left lung contusion, surgical repair would have required extracorporeal circulation during right pulmonary artery clamping. As the patient had stable hemodynamic status, the chest drain did not evidence any residual bleeding, and serial echocardiographic exams revealed nonexpanding and noncompressive hemopericardium (0.5 cm width), we opted for a medical, nonoperative treatment. We managed pulmonary artery hypertension, limited anticoagulation to deep vein thrombosis prophylaxis and expected that the progressive sclerosis of the healing process would strengthen the pulmonary artery wall and prevent both rupture and pseudoaneurysm.

The patient gave written informed consent for unrestricted use of her medical data.

SUPPLEMENTARY MATERIAL

The following is the supplementary data to this article:



Video 1. Transesophageal, upper mediastinal view of the right pulmonary artery (long axis, color Doppler) showing a 13.2 mm long intimal flap of the right pulmonary artery whose diameter was 22.7 mm wide.

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