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Case Report

Sudden death due to dissection of the thoracic aorta associated with dissection and rupture of the pulmonary artery: Report of two cases

Sorin Hostiuc [a,*], Dan Dermengiu [a], Mihai Ceauşu [b], Corneliu Octavian Capatina [a], Lacramioara Luca [b], Mihaela Hostiuc [c]

[a] Department of Forensic Pathology, National Institute of Legal Medicine, Bucharest, Romania
[b] Department of Pathology, National Institute of Legal Medicine, Bucharest, Romania
[c] Department of Internal Medicine, Floreasca Clinical Emergency Hospital, Bucharest, Romania

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ABSTRACT

We present two cases of dissection of the thoracic aorta associated with dissection and rupture of the pulmonary artery. In both cases the initial dissection was hypothesized to occur in the thoracic aorta, with secondary dissection and rupture of the pulmonary artery.

1. Introduction

Arterial dissection is caused by in-vessel hemorrhages, either secondary to intimal ruptures, to lacerations or other pathology of the vasa vasorum. The dissection leads to the formation of a false dissecting lumen; physio-pathologically it can lead to vessel occlusion with distal ischemia, artery-to-artery thromboembolism or hemorrhage [1–5]. If dissection affecting systemic vessels is frequent in clinical practice, those involving pulmonary vessels are much rare, less than 100 cases being cited in articles from Web of Knowledge [6–93]. Concomitant, aortic dissection associated with compression, dissection or rupture of the pulmonary artery is even less common [6,11,15,17,20,21,29,37,40,73,75,77,78,80,86]. Due to its rarity and unspecific but severe clinical findings the pulmonary artery dissection is difficult to diagnose in the emergency department, being often only proven at the autopsy.

We present here two cases of dissection of the thoracic aorta associated with dissection and rupture of the pulmonary artery. In both cases the initial dissection was hypothesized to occur in the thoracic aorta, with secondary dissection and rupture of the pulmonary artery.

2. Case reports

2.1. Case 1

A 53 years old man, with morbid obesity (140 kg), was brought in the ICU in respiratory arrest. At admission the patient was in a deep coma (GCS = 3), with upper body cyanosis, $PaO_2/FiO_2 > 244$, bradycardia (around 50 bpm), hypotension (90/50 mmHg), hemodynamic instability in need for vasopressor and inotropic support. Past history (obtained from the family) identified an episode of pulmonary thromboembolism seven years ago, and varicose veins in the lower limbs. ECG showed sinus bradycardia (30/min), a narrow QRS interval, biphasic T waves in DII, DIII, aVF, V2, V3, V4 and negative T wave in V1, a slow R wave progression in V1–V3, QT = 560 ms. Lab works showed increased leukocyte count, metabolic acidosis, hyperglycemia, negative cardiac enzymes, increased D-dimers (>5000 ng/ml). Cardiology examination revealed a difficult cardiac auscultation, apparently without murmurs and suggested hemorrhagic stroke as a possible diagnosis. Cerebral CT identified incipient cortical atrophy and low-density areas in the frontal–parietal subcortical white matter. Abdominal ultrasound was normal except for a moderate fatty infiltration of the liver and pancreas. The chest X-ray showed an aortic heart, enlarged thoracic aorta, without visible pulmonary pathology. In about 12 h from the initial admission the patient entered in cardiac arrest (asystole), non-responsive to CPR and was pronounced dead. The initial cause of death in hospital was coma of an unknown origin. The autopsy was performed the next day. At

* Corresponding author at: Sos.Vitan Barzesti 9, 042122 Sector 4 Bucuresti, Romania. Tel.: +40 723 791072.
E-mail addresses: soraer@gmail.com, sorin.hostiuc@legmed.ro (S. Hostiuc).

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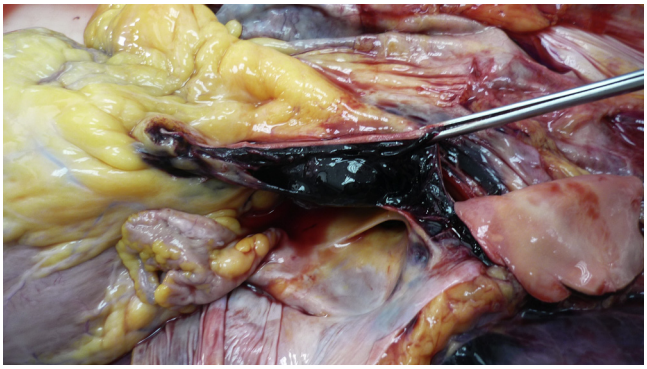

Fig. 1. Dissection of the pulmonary artery, case 1.

the opening of the chest cavity was identified a hemopericardium of about 900 ml. In order to seek the origin we started dissecting the thoracic aorta where we found a rupture in the beginning of the descending part associated with severe mediastinal hemorrhage, and a separation of the intimal layer – with numerous atherosclerotic plaques, from the media and adventitia. The dissection was retrograde, involving the initial part of the descending aorta, aortic arch and ascending aorta. During the examination of the pulmonary artery we also found clotted blood dissecting between the intimal and adventitial layer (Fig. 1), starting at the origin of the artery, and continued toward the left pulmonary artery. At the emergence of the left pulmonary artery we found a first intimal rupture, with irregular margins, of about 0.8 cm. The dissection continued on both right lobar arteries, with a second intimal rupture at the emergence of inferior right segmental artery. Other gross pathology findings were: cerebral stasis and a moderate degree of atrophy, pulmonary edema, hepatic steatosis, and multiple liver cavernous hemangiomas. Histological examination was performed using the following stains: Hematoxylin-Eosin, Weigert, Alcian Blue, Masson, and PAS. On histological slides of the aorta we identified a dissection between the medial and adventitial layers with hemorrhagic infiltrate, disseminated to the periadventitial tissues. Intima contained frequent atherosclerotic, sometimes calcified plaques and mucoid deposits. Weigert stain identified fragmentation and disorganization of the elastic fibers from the media, and external and internal limiting elastic membranes. Alcian Blue stain (pH = 2.5) identified frequent ortochromatic deposits at the level of the media (mucopolysaccharides). PAS stain was negative. On histological slides of the pulmonary artery we identified two breaches: one at the level of the intimal and medial layer, and one at the level of the medial and adventitial layers, with massive hemorrhagic infiltrate, relatively recent, continued in the sub-adventitial tissue. On histological slides of the segmentary pulmonary artery we identified a focal breach in the intimal layer, with abundant perivascular hemorrhage (Fig. 2A, C, D, E and F).

2.2. Case 2

A 60 years old men, with second degree obesity was found dead at home, by the family. The crime scene investigation (including declarations from witnesses) did not reveal any known chronic diseases. The autopsy was performed the next day. At the opening of the chest cavity we noted cardiac tamponade (more than 400 ml of blood and clots). In order to seek the origin of the bleed we started examining the aorta, where we found a dissection without signs of intimal tear, at the level of the ascending aorta, aortic arch and partially descending thoracic aorta (about 12 cm). The hemorrhagic infiltrate affected the proximal portion of the wall of the right ventricle and was continued in the pulmonary artery, where it dissected in the media layer. The anterior pulmonary wall was ruptured at about 4.5 cm from the emergence of the artery (Fig. 3). The dissecting sac stopped at about 1 cm after the rupture.

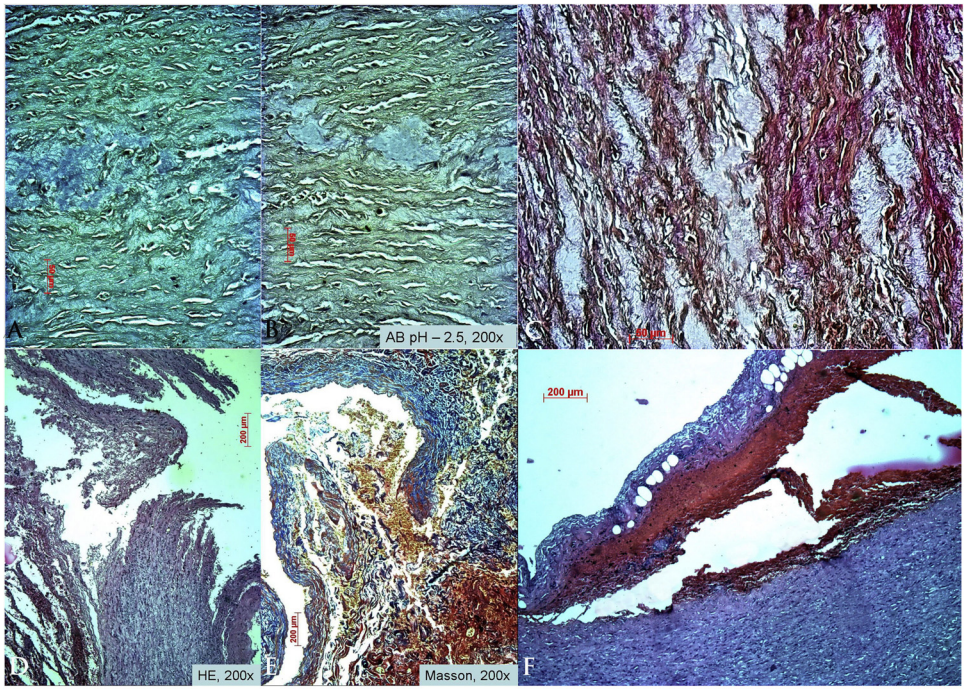

Fig. 2. Histological slides. Amorphous mucoid ground substance (mucopolysaccharides) staining ortochromatic (blue) with Alcian Blue pH 2.5 in the media of the ascending aorta, 200× (A) case 1 and (B) case 2; (C) disruption and irregular arrangements of the elastic fibers due to focal deposition of mucopolysaccharides in the media of the ascending aorta, Weigert's stain, 200×, case 1; (D) tear of the aortic wall between media and adventice with hemorrhagic infiltrate, HE, 50×, case 1; (E) severe tearing of the media of the pulmonary artery tracking blood into the surrounding connective tissues, HE, 200×, case 1; (F) severe tearing of the media of the pulmonary artery tracking blood into the surrounding connective tissues, Masson's trichrome, 200×, case 1.