Hemorrhagic infiltration of the pulmonary artery connective sheath: a complication of acute aortic dissection

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Learning objectives

Hemorrhagic infiltration of the pulmonary artery connective sheath is a rare complication that may result in a case of acute aortic dissection, most frequently, Stanford type A (1, 2). The radiological interpretation of this finding may be of a diagnostic challenge.

Multidetector computed tomography (MDCT) is the modality of choice for the diagnosis of this entity. MDCT can also be used to monitor the evolution of this life threatening disease. It is of great importance that radiologists can recognize and properly diagnose this complication, since the hemorrhagic infiltration of the pulmonary artery sheath usually has a good prognosis and generally requires no specific treatment. Thus, our goal is to:

1. Define the process of hemorrhagic infiltration of the pulmonary artery connective sheath secondary to acute aortic dissection, supported by a well-established pathological correlation.

2. Explain the pathophysiology of this entity, including a review of the literature on this subject.

3. Describe the radiological findings of this rare complication through a case series.

Background

Anatomically, pulmonary arteries and bronchi along with the lymphatic channels are closely surrounded by a sheath of connective tissue. This sheath is connected to the mediastinum (3).

In aortic dissection, especially Stanford type A, this sheath loses its firm tightness, resulting in the formation of a hemomediastinum (3). At this point, this condition can lead to many complications. Frequently, hemorrhagic extravasations into an extrapleural route can create a 'apical cap'. Also, the pressure exerted by the hemomediastinum can fissure the pleura and form a hemothorax (1, 4).

Exceptionally, an increased in mediastinal pressure may rupture the adventitia of the aorta to result in a hemorrhagic infiltration along the sheath shared between the aorta and the pulmonary artery. Rarely, extravasation of blood spreads along the pulmonary arteries to the lung interstitium.

Previously, the connective 'sheath' was described as an 'adventitia' shared by the aorta and the pulmonary trunk (2, 3). However, we believe that it is actually a sheath composed of connective tissue, which is common to the aorta and the pulmonary trunk (**Fig. 1**).

Images for this section:

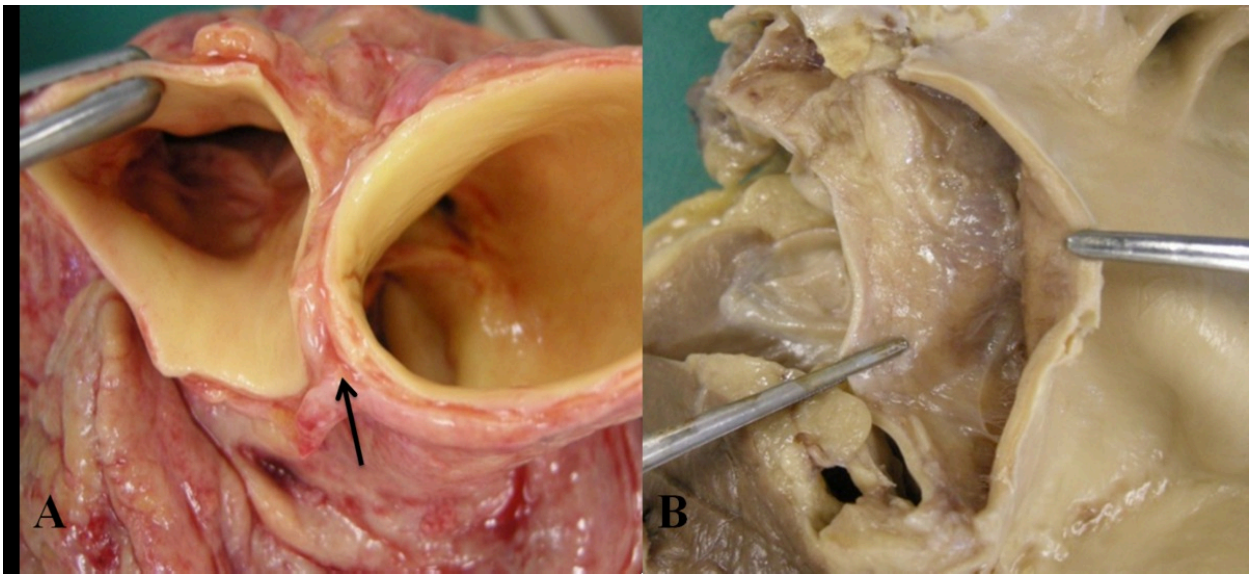

Fig. 1: A) Normal heart pre-fixation. Intact connective aortopulmonary sheath (arrow) between the pulmonary trunk (left) and the aorta (right). B) Heart post-fixation. Connective aortopulmonary sheath separated from the ascending aorta, forming a virtual space in a normal patient.

Imaging findings OR Procedure details

Materials and Methods

Literature review:
A literature review using PUBMED ('aortic dissection' infiltration of the lung parenchyma ',' interstitial pulmonary hemorrhage ') has led to the identification of seven reported cases, illustrated by computed tomography (1, 2, 5, 6, 7, 8, and 9).

Our radiological cases:
Case series of twelve aortic dissections with hemorrhagic infiltration of the aortopulmonary sheath, from five academic centers was reported from 2008 to 2010. The medical records were reviewed. Computed tomography (MDCT) was performed for each patient.

Our pathological cases:
A search of autopsy reports from 2006 to 2012, at the Montreal General Hospital was performed. Two reports describing acute aortic dissection with infiltration of the aortopulmonary sheath were identified. They also contain photographs of this disease.

MDCT technique:

Images from the MDCT were performed with computed tomography (CT) 16-MDCT (LightSpeed 16, GE Healthcare), 64-MDCT (LightSpeed VCT, GE Healthcare), or 256-MDCT (iCT, Philips Healthcare). The study initial CT acquisition is usually obtained from a non-contrast CT, followed by a study with IV injection of iodinated contrast. The nonionic contrast medium (100 mL iopamidol 370, Isovue 370, Bracco, or 100 mL iodinanol 320 Visipaque 320, GE Healthcare) is injected intravenously at a rate of 5 mL/s in the right antecubital vein through a 18-gauge needle, followed by a bolus of saline (bolus-tracking method).

Radiological Findings

Our case series includes a total of twelve patients (5 M, 7 F) (see Table 1), with an average age of 71.1 years (range 49-90 years). Six patients presented with chest pain, and seven had a history of poorly controlled hypertension. In one patient, the aortic dissection was likely secondary to an iatrogenic dissection of the right coronary artery, which occurred during conventional coronary angiography (aortocoronary dissection).

MDCT without contrast findings:

When available, the MDCT without contrast showed hyperdense foci at the periphery of the proximal pulmonary arteries (**Fig. 2**, images on the left). In the interstitium, hemorrhagic extravasations along the subsegmental pulmonary arteries would be seen on MDCT as ground glass opacities.

In the particular case of the patient with iatrogenic aortocoronary dissection (Patient 5), MDCT without contrast (performed immediately after coronary angiography) showed very hyperdense areas at the periphery of the ascending aorta and proximal pulmonary arteries. This hyperdensity is caused by remaining contrast agent from the recent angiography (**Fig. 3**).

MDCT findings using iodized contrast:

The opacification of the pulmonary arteries' lumen shows the infiltrated appearance surrounding the periphery of these arteries in continuity with the infiltrated appearance of the circumference of the ascending aorta. That is to say, the presumed site of the common aortopulmonary sheath. We may also note the compression, to varying degrees, of the pulmonary trunk and of the right and left main pulmonary arteries, which in one case, to a near-total luminal obstruction (**Fig. 4**).

This peri-vascular infiltration involves right and left main pulmonary arteries in 6 cases, the right main pulmonary artery in 2 cases (**Fig. 5**), and left alone in one case. In 2 cases, the infiltration is limited only to the pulmonary trunk without reaching the bifurcation.

In 4 cases, the presence of ground-glass opacities is displayed at the periphery of the pulmonary arteries (**Fig. 6**). One hypothesis explaining this finding is that these opacities are the result of localized alveolar hemorrhage secondary to ischemia. This would nevertheless have to be confirmed pathologically. It is important to note that the presence of ground-glass opacity is mainly observed in cases where the compression of the pulmonary trunk is greater than or equal to 40%.

Pathology findings

The first of two autopsy cases, with the most extensive disease, is the one of a 90 year-old woman, who died suddenly in an emergency, following a presentation of severe chest pain. The autopsy showed an aneurysm of the ascending thoracic aorta measuring 7cm in diameter, with a transverse parietal fissure of 9 cm in length (**Fig. 7a**). This fissure transected the intima, media and adventitia to reach the sheath located between the aorta and the pulmonary trunk. The hemorrhage infiltrated along the sheath and entered the lung parenchyma through the hilum (**Fig. 7b**), spreading around the bronchi, arteries, and pulmonary veins (**Fig. 7c**).

The second case is a 45 year-old woman who presented with severe epigastric pain. She died suddenly of cardiac arrest soon after her arrival in the emergency department. The autopsy showed an aneurysm of the ascending aorta measuring 4.1 cm in diameter with a transparietal fissure of 1cm in length, located in the left lateral wall of the ascending aorta, likely due to a rupture with dissection. Extensive hemorrhagic infiltration was noted reaching the right hilum and infiltrating the circumference of the right main bronchus and pulmonary arteries of middle lobe (**Fig. 8**).

Images for this section:

Patients	Age (years)	Sex	Risk Factors	Presenting Symptoms	MDCT	Site of aortic dissection	RAA, site of flap	Pulmonary artery		Other findings
								Involved	Luminal compression (%)	
1.	72	M	HTN	Thoracic pain	C+, CXR	A	P	LPA, RPA	90% **	RLL pulmonary infarct, hemomediastinum ground glass,
2.	49	M	HTN	Thoracic pain	C+/C-	A	AN, LL, P	LPA, RPA	20%	Hemopericardium
3.	90	F	HTN	Epigastric pain	C+/C-	A	P	PT	10%	Hemopericardium
4.	62	M	HTN	Retrosternal pain	C+/C-	A,T	P, RL	RPA, LPA	60%	Hemopericardium, ground glass
5.	70	F	HTA, DLPD, iatrogenic diss.*	Thoracic pain	C+/C-	A	Right coronary sinus***	PT	25%	
6.	89	F	DM2, HTN, AAA	Inconscious	C+/C-	A	P, LL	LPA, RPA	50%	Hemopericardium, ground glass
7.	68	F	Severe cyphoscoliosis, dwarfism	Hemoptysis, thoracic pain	C+	A, T, D	AN, RL, LL	RPA	40%	Ground glass
8.	66	M	HTN, DLPD	Back pain	C+	A	AN, RL, P	RPA	25%	
9.	N/A	N/A	N/A	N/A	C+	A, T, D	P, LL	PT	70%	
10.	74	M	aortic atheromatous ulcer	N/A	C+	A	P	LPA	10%	
11.	N/A	N/A	N/A	N/A	C+	A, T, D	LL	LPA, RPA	50%	Hemopericardium
12.	N/A	N/A	N/A	N/A	C+	A	P, LL	LPA, RPA	30%	Hemopericardium

Table 1: Patient presenting with acute aortic dissection and infiltration of the common connective aortopulmonary sheath. Legend: HTN: hypertension. DLPD: dyslipidemia. DM2: type 2 diabetes. + C: with contrast agent. C-: without contrast agent. A: ascendant. T: transverse. D: descendant. RAA: rupture of the ascending aorta. AN: anterior. LL: left lateral. RL: right lateral. P: posterior. LPA: left pulmonary artery. RPA: right pulmonary artery. PT: pulmonary trunk. RLL: right lower lobe. N/A: not available. *Conventional coronary angiography. ** Subtotal compression of the pulmonary trunk by hematoma. ***Dissection limited to the anterior portion of the right coronary sinus of the ascending aorta.

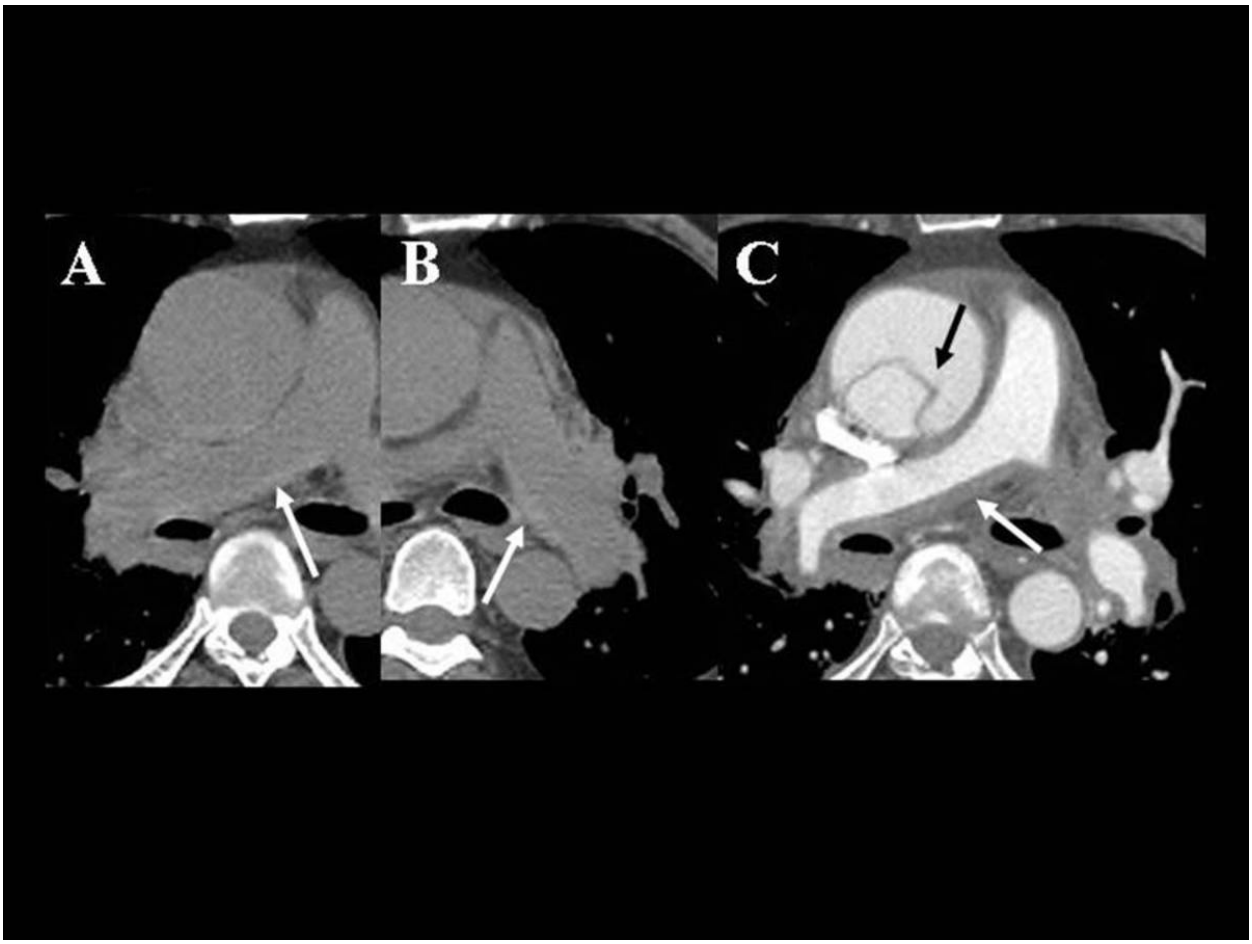

Fig. 2: 49-year-old man with chest pain. Normal coronary angiography, a chest MDCT is performed (Patient 2). A) Slightly visible hyperdense edging in the wall of the right (A, white arrow) and left proximal pulmonary arteries(B, white arrow). C) MDCT with iodinated contrast shows aortic dissection (black arrow), Stanford type A, and the posterior parietal thickening of the right pulmonary artery (white arrow).

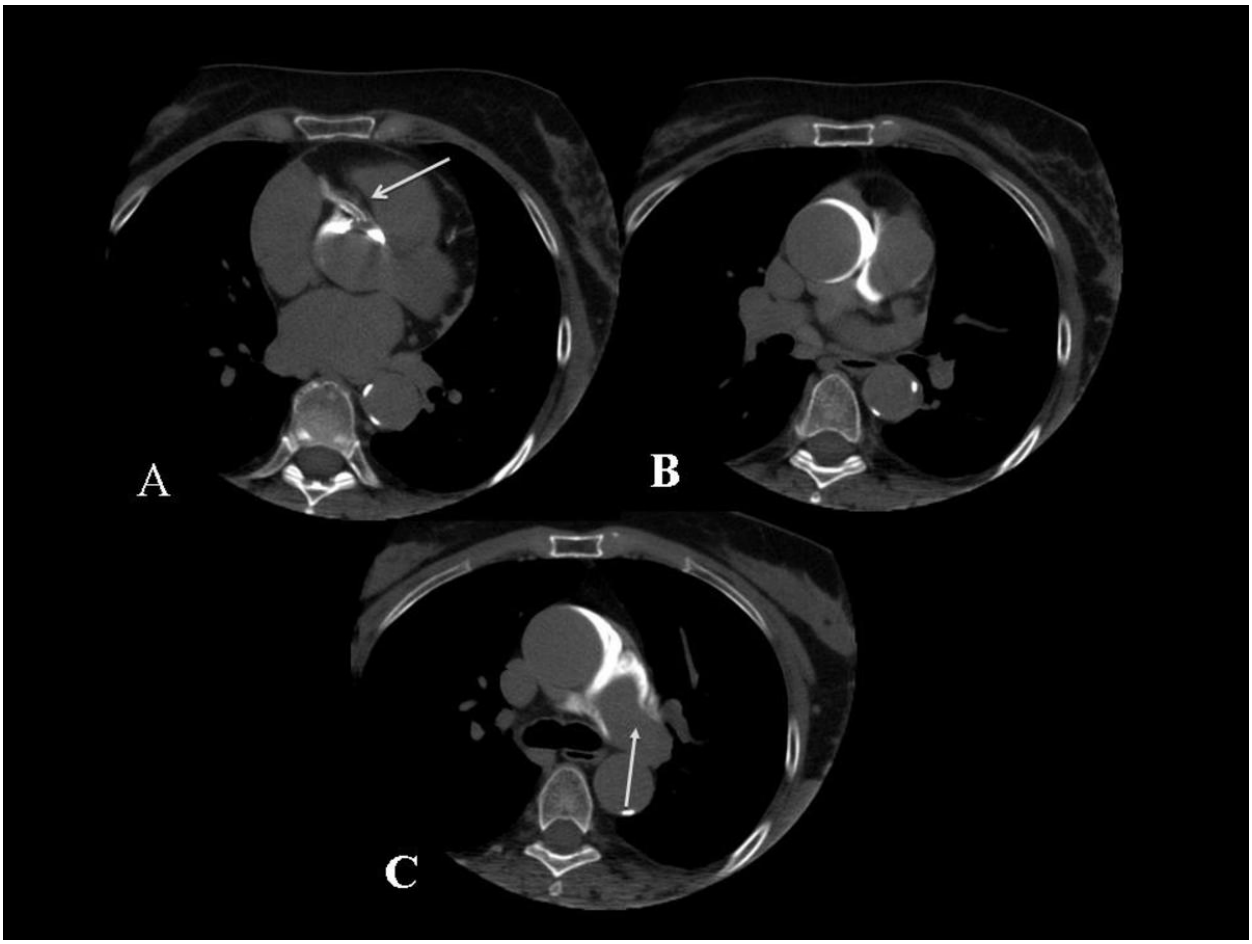

Fig. 3: 70-year-old woman presenting with symptoms of angina (Patient 5). A) hyperdensity located in the wall of the right coronary sinus, adjacent coronary artery (white arrow). A coronary stent is also visible. B)and C) hyperintensity along the pulmonary trunk and ascending aorta whose appearance and location suggests that this hyperintensity is located in the aortopulmonary sheath (white arrow).

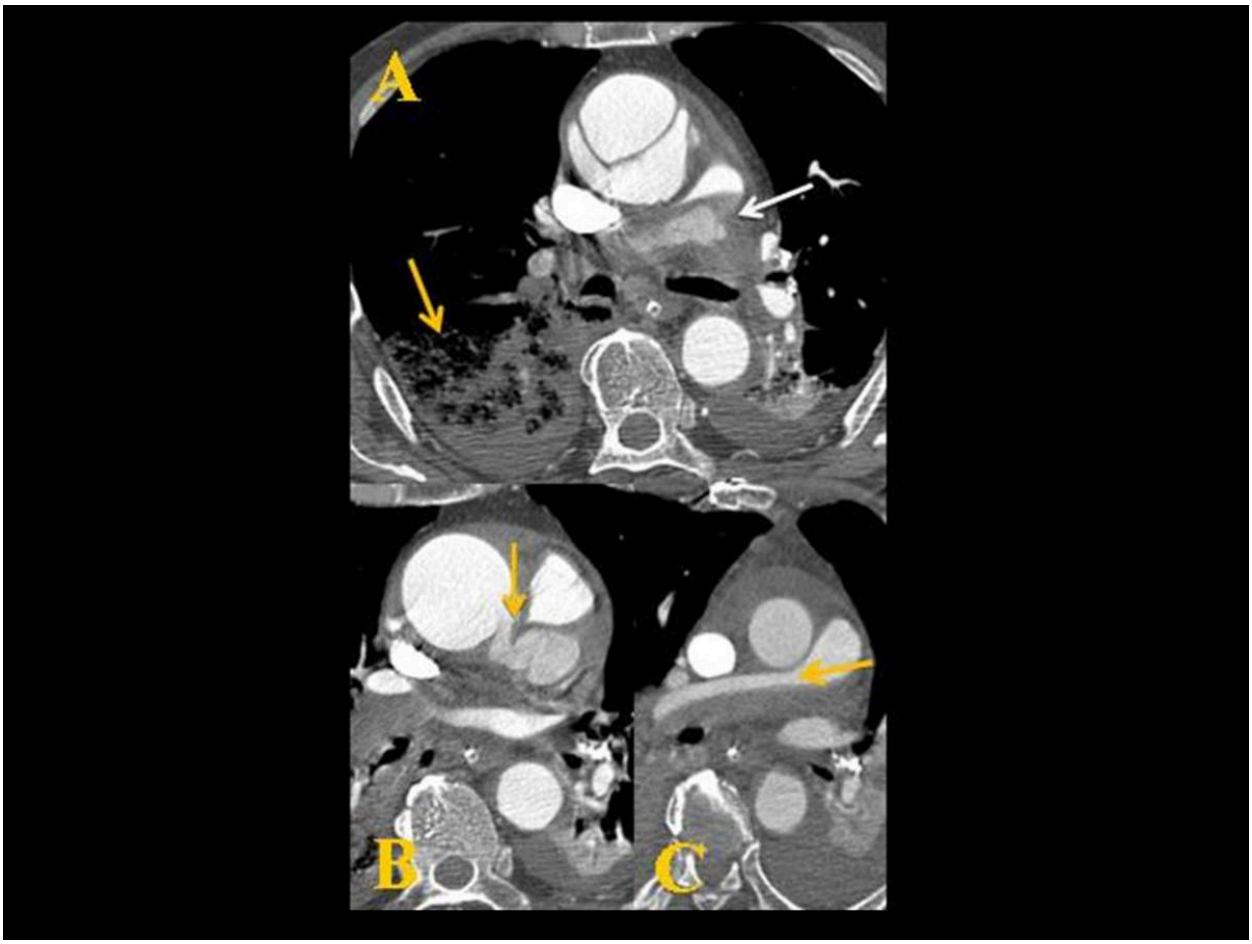

Fig. 4: 72-year-old man, presenting with chest pain (Patient 1). A) hematoma with subtotal compression of the lumen of the pulmonary trunk (white arrow) and of the right pulmonary artery. Edematous-hemorrhagic complication of the right lower lobe lung parenchyma(yellow arrow). B) Dissection of the ascending aorta and contrast extravasation adjacent to the left coronary sinus with extension in the aortopulmonary sheath (yellow arrow). C) Six days post-replacement of the ascending aorta and hemi-arch. Note the partial recanalization of the right pulmonary artery (yellow arrow).

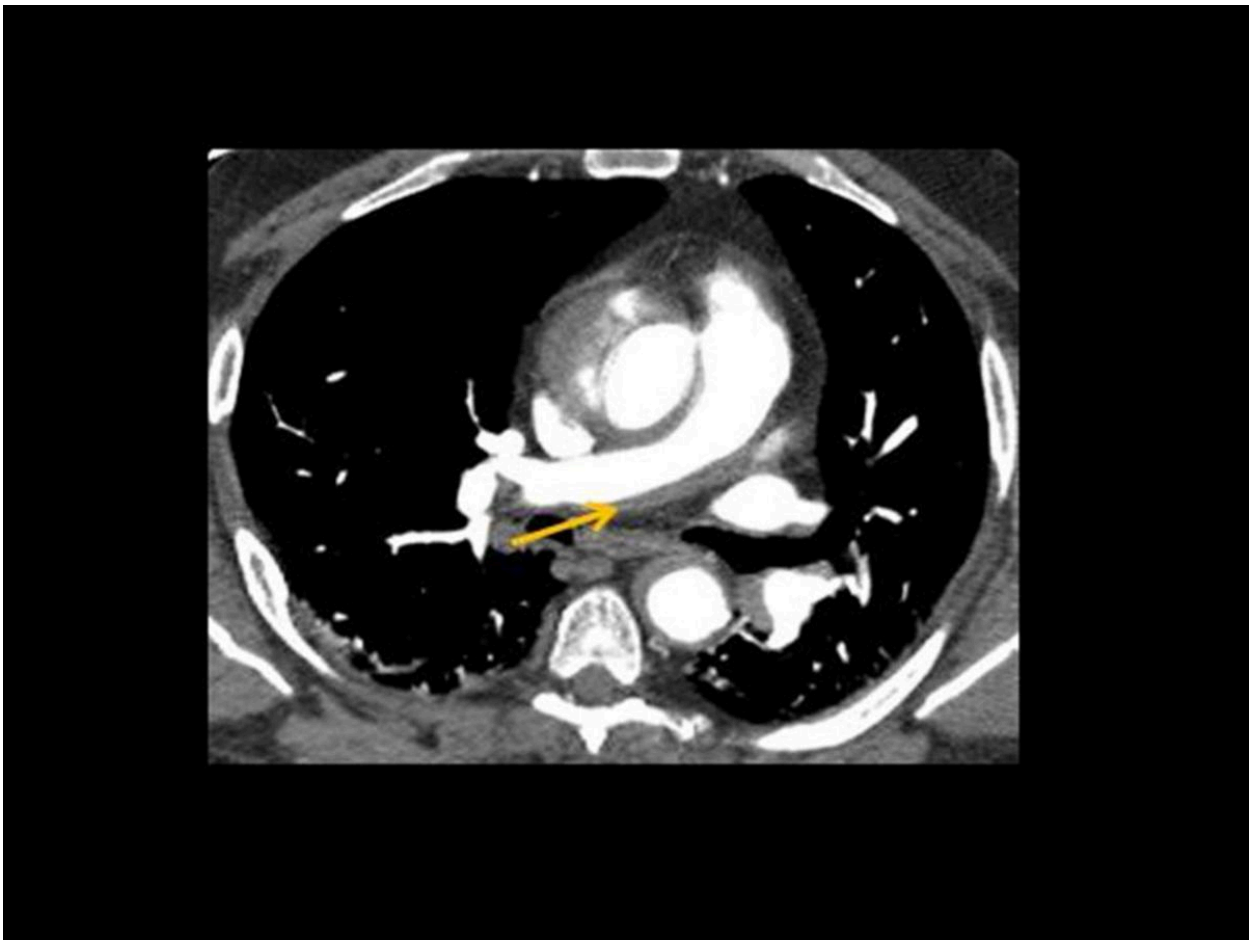

Fig. 5: 66-year-old man with thoracic pain. Posterior parietal thickening limited to the pulmonary trunk (arrow).

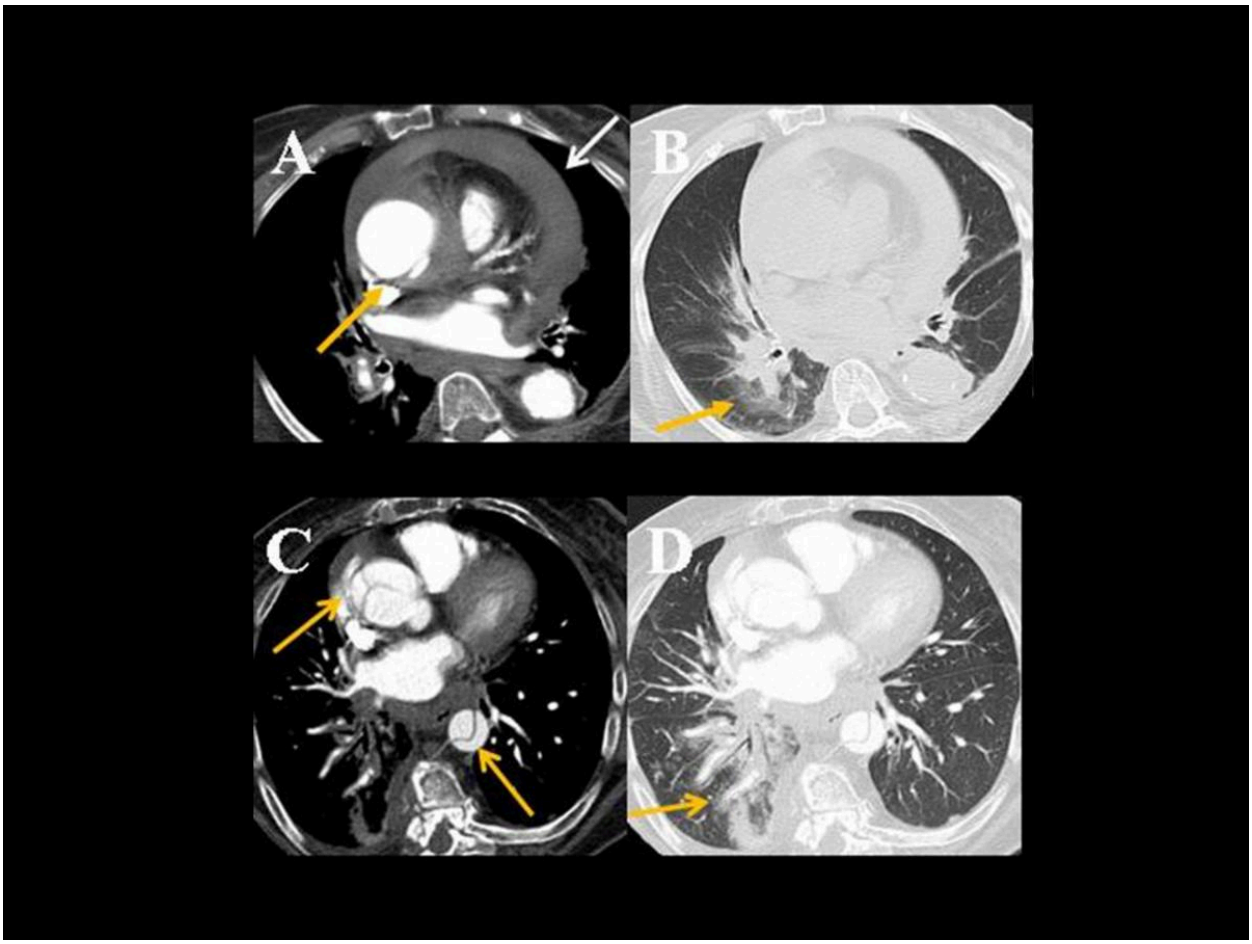

Fig. 6: (A, B) 89-year-old woman found unconscious at home (Patient 6). Aortic dissection, Stanford type A (yellow arrow), with hemorrhagic pericardial effusion (white arrow). B) peribronchovascular ground glass opacity in the right lower lobe (yellow arrow). (C, D) 68-year-old woman with chest pain (patient 7). C) acute aortic dissection, Stanford type A (arrows). D) Ground glass opacity around the pulmonary artery in the right lower lobe (yellow arrow).

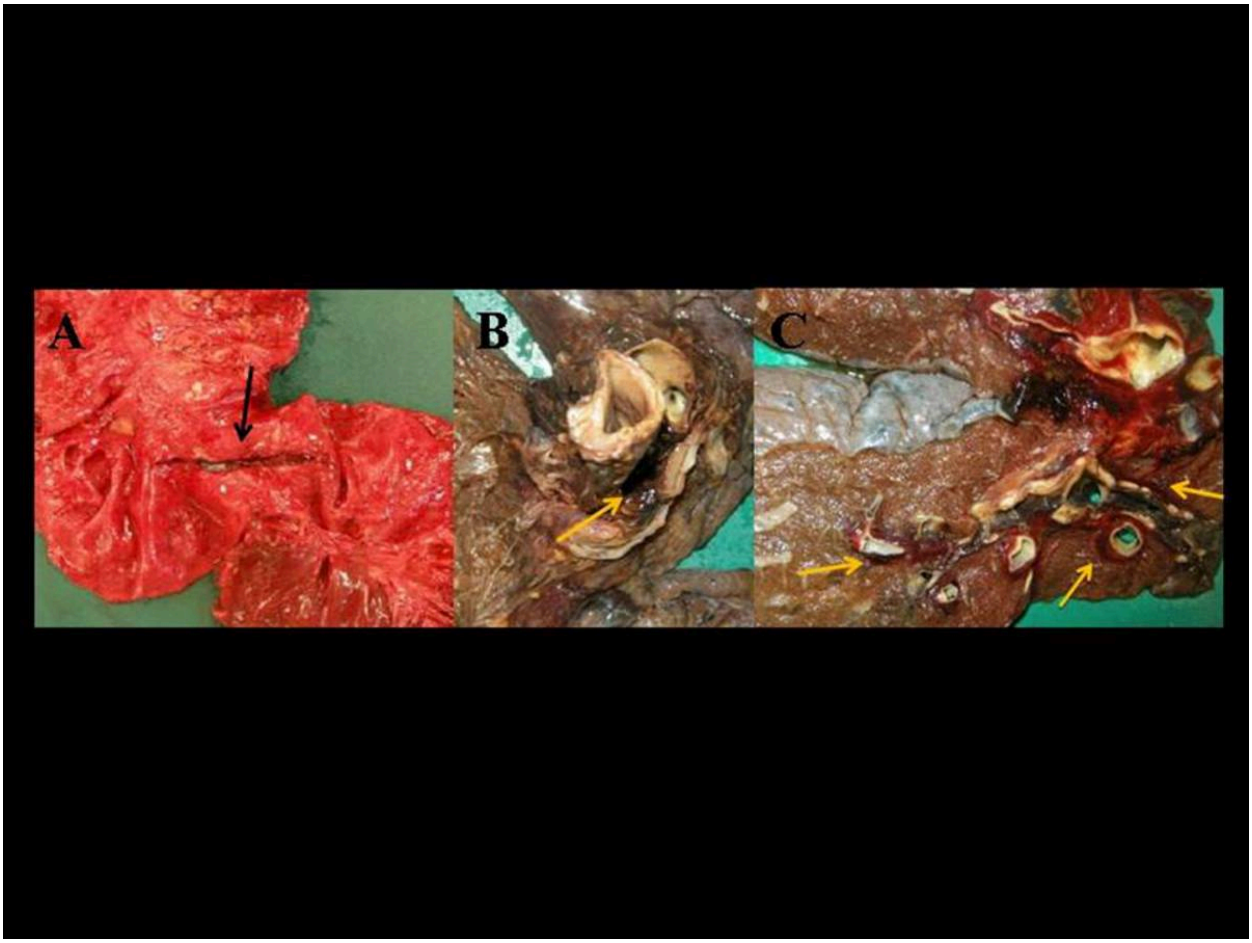

Fig. 7: 90-year-old woman with severe thoracic pain. A) Site of the aortic rupture (arrow). B) Left lung. Hemorrhagic infiltration of the pulmonary hilum (arrow). C) Coronal cut of the left lung. Hemorrhagic infiltration along the pulmonary arteries, veins, and bronchi (arrows).

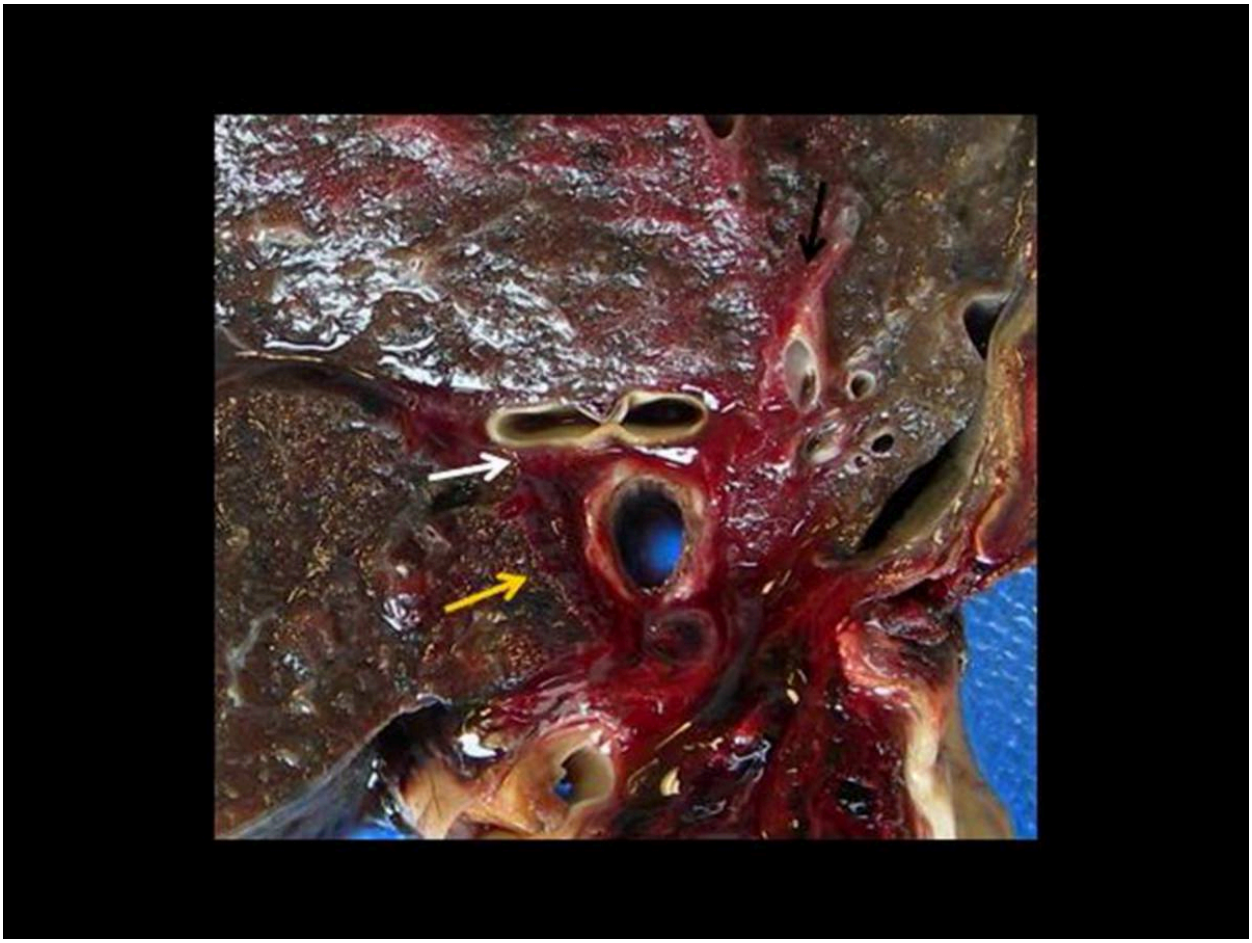

Fig. 8: 45-year-old woman with severe epigastric pain. Right lung. Hemorrhagic infiltration surrounding the right main bronchus (yellow arrow), pulmonary artery (white arrow) and pulmonary vein (black arrow) of the middle lobe.

Conclusion

Proposed mechanism

The suggested mechanism to explain this entity involves dissection with contained rupture of the ascending aorta and the formation of a hemorrhagic infiltration limited to the extravasation of aortic blood along the aortopulmonary connective sheath (usually a virtual space in a normal patient), with consequent compression of the pulmonary arteries. This vascular compression would result in distal edema with hemorrhage of the lung parenchyma.

Conclusion

Hemorrhagic infiltration of the common aortopulmonary connective in acute aortic dissection, primarily Stanford type A, is a rare complication and not well known by the radiological community.

So far, few attempts were made to provide a detailed description of the pathophysiology of this complication. We consider our research as the first study describing the radio-patho-physiology of hemorrhagic infiltration of the aortopulmonary sheath in acute aortic dissection with supporting pathological and radiological evidence.

Early detection and recognition would allow to distinguish this entity from other acute diseases of the mediastinum, and could lead to appropriate management.

MDCT is a quickly available diagnostic tool, which is relatively specific in cases of acute aortic dissection. MDCT may also be used for monitoring if necessary.

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