Incidence of chronic pulmonary thromboembolism in patients with suspected acute pulmonary thromboembolism

Poster No.: C-1279
Congress: ECR 2014
Type: Scientific Exhibit
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Keywords: Embolism / Thrombosis, Diagnostic procedure, CT-Angiography, Thorax
DOI: 10.1594/ecr2014/C-1279

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Aims and objectives

Most pulmonary thromboemboli resolve without sequelae. For reasons that are still unclear, in a small percentage of patients, the thromboemboli do not resolve but rather form endothelialized fibrotic obstructions of the pulmonary vascular bed. The result is vascular stenosis, which may lead to severe pulmonary hypertension and cor pulmonale (1).

The difficulties in estimating the true prevalence of chronic pulmonary thromboembolism are largely due to the insidious nature of the presentation, clinically silent acute events, and the absence of long-term follow-up of all acute pulmonary emboli although studies suggest an incidence of about one case per 1,000 people per year (2).

Chronic thromboembolic pulmonary hypertension (CTPH) is a late-occurring complication of pulmonary embolism that is associated with considerable morbidity and mortality. Previous studies have reported an incidence of CTPH between 0.1% and 3.8% in patients with previous venous thromboembolism (3).

In the presence of chronic thromboembolic disease, the bronchial and nonbronchial systemic circulation is markedly increased as a result of the development of systemic-to-pulmonary anastomoses, which help to maintain pulmonary blood flow in the presence of vessel obstruction (1).

Chronic pulmonary thromboembolism (cPTE) is often identified during the diagnostic work-up in patients with unexplained pulmonary hypertension. At our institution, most cases of chronic pulmonary thromboembolism are discovered at CT pulmonary angiography performed to rule out acute pulmonary thromboembolism (aPTE).

The purpose of this study was:

- to determine the incidence of cPTE in patients with suspected aPTE.
- to determine the incidence of hypertrophy of the bronchial arteries in patients with cPTE.
- to determine whether patients with cPTE had a history of thromboembolic disease (aPTE and/or deep-vein thrombosis (DVT).
Methods and materials

We consecutively included all patients with suspected aPTE who underwent pulmonary CT angiography between January 2006 and September 2013. The majority of patients came from the emergency department.

We performed CT pulmonary angiography (CTPA) with a 16 multidetector CT scanner (120 kV, 70-120 mAs, 0.5 second scanning time, 0.75 mm detector width, pitch of 1.5) from January 2006- November 2012. Images were reconstructed with a 1-mm section thickness at a 0.7-mm interval. Patients received 100 mL of contrast material (iopromide, Ultravist 300; Schering, Berlin, Germany) at an injection rate of 4 mL/sec followed by the injection of 40 ml of saline media. We used the automatic bolus track system with the circular region of interest on the main pulmonary artery (threshold of 120 HU, 6 seconds scanning delay). Between December 2012-September 2013 because of updating of our equipment we performed CTPA with a 128 MDCT scanner (120 kV, 150 mAs, (SAFIRE adjustment); gantry time rotation 0.33seg, collimation 0.6mm and pitch of 1.2; 1 mm section thickness reconstruction at 0.7 mm interval. Patients receive 60 ml of the same contrast material at the same rate, followed by the injection of 40 ml of saline media.

Because some signs of chronic thromboembolism (eg, bands) may be overlooked with the high contrast mediastinal window settings, we view the images by using three different grey scales for interpretation: a lung window (window width, 1500 HU; window level, -600 HU), a mediastinal window (window width, 350 HU; window level, 40 HU), and a pulmonary thromboembolism-specific window (window width, 700 HU; window level, 100 HU).

Diagnostic criteria for cPTE were mainly direct vascular signs:

- **complete** obstruction with decreased vessel size: abrupt decrease in vessel diameter and absence of contrast material in the vessel segment distal to the total obstruction.

- **partial** obstruction:
  - crescent-shaped intraluminal defect that forms obtuse angles with the vessel wall,
  - bands or webs, thin lines surrounded by contrast material most frequently found in lobar or segmental arteries and rarely are seen in the main pulmonary artery. Many branching bands form a web.
We recorded also **indirect signs of chronic pulmonary emboli:**

- hypertrophy of bronchial arteries: abnormal dilatation of the proximal portion of the bronchial arteries (diameter of more than 2 mm) and arterial tortuosity.

- mosaic pattern: sharply demarcated regions of decreased and increased attenuation because of irregular perfusion, with larger vessels in regions of increased attenuation

- bronchial dilations: cylindrical bronchial airway dilatation at the level of segmental and subsegmental bronchi adjacent to severely stenosed or completely obstructed and retracted pulmonary arteries.

We reviewed the patients’ medical records; in addition to sex and age, charts were reviewed for comorbid conditions and history of previous thromboembolic disease (aPTE and/or DVT).
Results

A total of 2715 patients were included; 104 patients (4%) had inconclusive examinations that were due to suboptimal contrast enhancement and/or movement artefacts, so we had 2611 evaluable patients. From these 2611 patients, 723 (27%) had aPTE.

80 patients (60% men, mean age 74 years ± 15) out of 2611 had direct vascular signs of cPTE:

- signs of both cPTE and aPTE were present in 46 (6.4% of the patients with aPTE) and only signs of cPTE were present in 34 (1.3% of the total of patients).

- 40 patients (50%) presented signs of total occlusion of one or more pulmonary arteries (Fig. 1).

![Fig. 1a](image1) ![Fig. 1b](image2)

Fig. 1: 43-year-old man with cPTE. (a) Axial contrast-enhanced CT scan shows complete occlusion and marked reduction in size of the right middle lobe pulmonary artery (arrow) in comparison with the lingular lobe pulmonary arteries. (b) Axial CT scan (lung window) shows almost absence of segmental and subsegmental vessels in the right middle lobe (arrow).

References: UDIAT, Parc Taulí Sabadell - Barcelona/ES

- 66 patients (82.5%) presented signs of partial occlusion of one or more pulmonary arteries (Fig. 2).
Fig. 2: Fig. 2 a ,b. Chronic pulmonary thromboembolism in an 85-year-old woman. a) Axial and b) coronal MIP images showing eccentric chronic thrombi producing irregular contours of the intimal surface of right main pulmonary (arrows) and also surrounding the right interlobar pulmonary artery (arrowheads).

References: UDIAT, Parc Tauli Sabadell - Barcelona/ES
32 out of 40 patients with some total occlusion presented some partial pulmonary artery occlusion.

-46 patients (57.5%) presented bands/webs (Fig. 3), in 8 out of 80 the exclusive vascular sign of CPTE were bands.
Fig. 3: Fig. 3 a, b. Residual bands from a pulmonary thrombus in an 73-year-old man. (a) Axial contrast-enhanced CT image shows a linear structure anchored to the vessel wall in the left lower lobe pulmonary artery (arrow). (b) Coronal maximum intensity projection CT image shows the bilateral bands anchored to the vessel wall in more detail (arrows).

References: UDIAT, Parc Tauli Sabadell - Barcelona/ES

About indirect signs of cPTE:

- hypotrophy of the bronchial arteries (Fig. 4) was present in 74 % of the patients (59 out of 80) with cPTE; in 9 patients it was not possible to assess the presence of bronchial arteries dilation due to the lack of contrast opacification in the aorta.

Fig. 4: Fig. 4 a, b. Chronic pulmonary thromboembolism in an 50-year-old woman with a history of previous acute pulmonary thromboembolism. a) Axial contrast-enhanced CT scan shows enlargement of the right bronchial arteries (arrows) (b) Oblique coronal maximum intensity projection CT image better depicts the course of the hypertrophied and tortuous right bronchial arteries (arrows).

References: UDIAT, Parc Tauli Sabadell - Barcelona/ES

- mosaic pattern of perfusion (Fig. 5) was seen in 50% of patients (40 out of 80).
Fig. 5: Fig. 5. Chronic pulmonary thromboembolism in a 65-year-old man. CT scan (lung window) shows a mosaic perfusion pattern with marked regional variations in attenuation of the lung parenchyma and disparity in the size of the segmental vessels, with larger-diameter vessels in regions of increased attenuation (arrowheads). A peripheral parenchymal band or scar (arrow) from infarction also is depicted.

References: UDIAT, Parc Tauli Sabadell - Barcelona/ES

Bronchial dilations (Fig. 6) were present in 31% (25 out of 80) always matching with complete obstruction of the pulmonary arteries.
Fig. 6: Chronic pulmonary thromboembolism in an 80-year-old woman. (a) Axial CT scan (lung window) shows increased bronchial diameters and an absence of normal distal tapering of the segmental and subsegmental bronchi of the left lower lobe (arrows). Note the small arterial segments at the lateral border of each dilated bronchus.

References: UDIAT, Parc Tauli Sabadell - Barcelona/ES

Most patients, 60% (48 out of 80) with cPTE had a history of thromboembolic disease: 52% (42 out of 80) aPTE, 32% (26 out of 80) DVT, and 22% (18 out of 80) both aPTE and DVT.

The most frequent comorbidities were: neoplasm 20% (16 out of 80), cardiomyopathy 19% (15 out of 80), COPD 10% (8 patients), diabetes mellitus 9% (7 out of 80), presence of a thrombotic risk factor 5% (4 out of 80), usual interstitial pneumonia 4% (3 out of 80).
Conclusion

cPTE is a challenging diagnosis. Early recognition of cPTE is important because if chronic thromboembolic pulmonary hypertension ensues, pulmonary endarterectomy is a very effective treatment. The difficulties in estimating the true prevalence of cPTE are largely due to the insidious nature of the presentation, clinically silent acute events, and the absence of long-term follow-up of all acute pulmonary emboli, although studies suggest an incidence of about one case per 1,000 people per year (2).

cPTE is often identified during the diagnostic work-up in patients with unexplained pulmonary hypertension. At our institution, most cases of chronic pulmonary thromboembolism are discovered at CT pulmonary angiography performed to rule out aPTE. In this context is not unusual to confuse the signs of chronic thromboembolism with signs of acute thromboembolism. It is important to mention that it is not infrequent to encounter concurrent signs of both acute and chronic emboli in patients with recurrent thromboembolic disease; in our study 46 out of 80 patients (representing a 6.4% of the patients diagnosed with aPTE) presented both acute and chronic signs of pulmonary embolism.

In cases of acute complete obstruction, the diameter of the pulmonary artery may be increased because of impaction of the thrombus by pulsatile flow (4). Conversely, in chronic thromboembolic disease, the diameter of the vessel distal to a complete obstruction is markedly decreased. An acute partial filling defect may be central or eccentric in location. In acute thromboembolism, a nonobstructive eccentric filling defect forms acute angles with the vessel wall. Conversely, partially obstructive chronic thromboembolism appears as a peripheral crescent-shaped defect that forms obtuse angles with the vessel wall. An acute partial central defect appears surrounded by contrast-enhanced blood (1).

The features of chronic pulmonary emboli on CT scans can be categorized into vascular or direct signs (total occlusion, partial occlusion, bands/webs) or indirect signs (parenchymal findings, hypertrophy of bronchial arteries) (1). The most frequent direct sign of cPTE in our study was the partial pulmonary occlusion (82 % of patients). It is also remarkable that a10 % of patients had only residual band images, as the unique sign of cPTE.

Hypertrophy of the bronchial arteries was present in the majority of patients, 74%, and was underestimated due to the lack of opacification in the aorta in some studies (9 out of 80) using our routine protocol to rule out aPTE. Acute pulmonary embolism does not appear to cause dilatation of the bronchial arteries; in patients in whom the distinction between acute and chronic or recurrent pulmonary embolism at CTPA is unclear, the
presence of dilated bronchial arteries should favour the diagnosis of chronic or recurrent pulmonary embolism (5).

Other indirect signs such as parenchymal findings (e.g., mosaic attenuation with asymmetric artery size, bronchial dilation) are nonspecific, in the appropriate clinical setting they may be regarded as supportive of the diagnosis of chronic thromboemboli (1). Mosaic pattern of perfusion was seen in 50% of our patients; oligemia distal to occluded vessels causes a redistribution of blood away from the affected areas (1, 2). These irregularities in perfusion are demonstrated on CT scans as a sharply demarcated, mosaic pattern of attenuation. Hypoperfused areas are of low attenuation, with increased attenuation where the vessels have become larger and more prominent. Mosaic attenuation is nonspecific and can be caused by a variety of other pulmonary conditions, including small airways disease and primary disorders of the parenchyma (6).

Another indirect sign that has been described is cylindrical bronchial dilatation adjacent to occluded or severely stenosed pulmonary vessels in the segmental and subsegmental level (1, 6). In our patients bronchial dilations was seen in 31% of patient who presented signs of total occlusion of some pulmonary arteries, in the literature (7) this sign is found with more frequency (64%), this fact is probably related to the increased number of partial obstructions instead of complete occlusion in our study. The concomitant presence of other signs of cPTE can help distinguish between bronchial airway dilatation caused by cPTE and those due to obstructive airways disease (1).

A previously documented venous thromboembolic event (aPTE and/or DVT) was found in a high percentage of patients (60%), percentage similar to the found in patients with thromboembolic pulmonary hypertension (8). About other comorbidities we highlight a 20% of patients with a history of neoplasm. This precedent should contribute to the high percentage of patients with cPTE in our study.

In conclusion in our study the incidence of cPTE in patients with suspected aPTE was high. Most patients with cPTE have hypertrophied bronchial arteries. Most patients with cPTE have a history of thromboembolic disease (previous aPTE and/or DVT).
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References


