Chronic pulmonary thromboembolism: Pictorial review of CT pulmonary angiographic findings

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Learning objectives

To present CT angiographic findings in chronic pulmonary thromboembolic disease

To present CT findings which differentiate acute from chronic thromboembolic disease

To identify patients suitable for pulmonary thromboendarterectomy

Background

The pathophysiology of chronic clot formation

In greater than 90% of cases of acute pulmonary embolus, treatment with anticoagulation results in complete resolution to normal pulmonary haemodynamics within 30 days. This occurs due to mechanical fragmentation and endogenous fibrinolysis of the clot. Why some people go on to have chronic clot is uncertain. There does not appear to be a problem with fibrinolysis, rather it is felt that the problem has to do with ongoing in situ thrombosis leading to incomplete thrombus resolution. This tends to occur in patient's with large or recurrent thrombi.

As thrombus resolves, it shrinks allowing partial recanalisation (figures 1 and 2). The remaining thrombus is incorporated into the vessel wall and covered with a layer of endothelial cells. As the thrombus resolves, its overall effect on the thrombosed vessel can present in a variety of ways. There can be complete vascular obstruction, retraction of the clot with partial recanalisation, or residual fibrous cords (webs and bands).

Pulmonary hypertension resulting from chronic thromboembolic disease is thought to occur as a complex interplay of factors including development of secondary arteriopathy in the pulmonary vascular bed, effects of circulating vasoconstrictors, and even genetic factors and is not simply due to mechanical stenosis or obstruction by the clot.

Imaging findings OR Procedure details

Vascular signs
Chronic thrombus causing complete obstruction of an artery is seen as a filling defect causing an abrupt convex cutoff to the contrast within the vessel (figure 3). This is called a pouch defect on catheter angiography. It is difficult to identify on CT pulmonary angiogram (CTPA) as the clot and affected vessel usually shrinks (figure 3, 4 and 15). It is easier to identify indirect sign such as the sudden decrease in diameter of the occluded vessel and those distal to it as compared with adjacent patent vessels. On lung windows this calibre change will be seen as a vessel markedly smaller than the accompanying bronchus (figure 4 and 13).

When a thrombus becomes organised it can appear as partial filling defects such as luminal narrowing, intimal irregularities, bands and webs. Narrowing is due to recanalisation through a large thrombus seen as a narrow lumen of contrast flowing through a thick-walled artery (Figure 1, 2, 5, 6). The wall thickening is due to thrombus contracting, organising and becoming endothelialised into the vessel wall. The thrombus lining the walls can sometimes be have a nodular contour, seen as intimal irregularities outlined by contrast in the vessel (figure 1, 5). An indirect sign of this luminal narrowing is post stenotic vessel dilatation or even aneurysm. When the vessel is seen perpendicular to the scan plane, thrombus lining the wall appears as a crescent-shaped filling defect forming obtuse angles with the vessel wall (figure 2 and 6).

**Bands** (figure 7, 8 and 9) are delicate linear structures attached to the vessel wall at two ends with an unattached mid portion. They are usually aligned in the direction of blood flow, i.e. with the long axis of the blood vessel. They vary from 3 to 20 mm in length and 1 to 3 mm in width. **Webs** (figure 9) are narrow bands with branches which form a network. On CT angiogram webs and bands are seen as thin curvilinear structures within an opacified vessel, occurring most frequently in the lobar or segmental arteries.

**Signs of pulmonary hypertension**

Most of the imaging features of pulmonary hypertension are common to all forms regardless of cause. One of the main signs of pulmonary hypertension is enlargement of the main pulmonary artery (measured proximal to the bifurcation and adjacent to the ascending aorta) to a diameter of 30 mm or more (figure 10). A ratio of the main pulmonary artery to the adjacent ascending aorta of greater than 1.1 strongly correlates with pulmonary hypertension (figure 10). Other signs of pulmonary hypertension include atherosclerotic calcification of pulmonary arteries (figure 10) and tortuous pulmonary vessels. Slightly more specific to chronic thromboembolism is asymmetry in the size of the right and left pulmonary arteries.

The right heart bears the brunt of the work in pulmonary hypertension resulting in right ventricular dilatation and hypertrophy. Right ventricular dilatation is seen when the ratio of right to left ventricular diameter is greater than 1.1 (figure 10). On CT, the ventricular diameter can be measured in the axial planes at its widest points. It is
measured as the contrast filled ventricle between the inner surfaces of the free wall and the interventricular septum. In severe cases the right ventricular dilatation can cause **leftward bowing** of the interventricular septum (figure 10). **Pericardial effusion** may also be seen in severe pulmonary hypertension.

**Systemic collaterals** can develop in pulmonary hypertension. Most often collateralised vessels are the bronchial arteries. They arise from the aorta at the level of the carina. Normal bronchial arteries take around 1 - 2% of cardiac output but when they collateralise they can take up to 30%. Collateralised bronchial arteries are identified by their tortuosity and by having a diameter greater than 2mm (figure 12). Other systemic arteries that collateralise are the inferior phrenic, intercostals (figure 12) and internal mammary arteries.

**Parenchymal signs**

These are generally non-specify signs but can be used as supportive evidence of chronic thromboembolism. **Mosaic perfusion** (figure 4 and 13) can be seen in patients with chronic PE. This is seen on lung windows as well defined areas of low and high attenuation. This is due to under-perfusion in affected vessels (giving low attenuation) and hyper-perfusion in patent vessels (giving high attenuation). In the under-perfused regions the vessels are often noted to be smaller and less numerous than in the perfused areas.

**Wedge shaped pleural based opacity** is most suggestive of infarction. With time these can constrict leaving a linear **parenchymal band** (figure 14). These scars are often linear, multiple, have adjacent pleural thickening and found in the lower lobes. They are also often found in regions that are underperfused.

**Differentiating acute from chronic thromboembolism**

(figures 1, 2 and 16)

In acute embolism when there is complete obstruction, non-enhancing thrombus obscures the whole lumen and causes the vessel to **expand** (figure 16). Chronic thrombus causes the vessel to **contract**.

A partial filling defect is seen in the centre of the artery, surrounded by contrast. This is seen as "**polo-mint sign**" (figure 16) when imaged perpendicular to the vessel, and "**railroad track sign**" (figure 16) when imaged parallel to the vessel. When the thrombus is seen eccentrically, it forms **acute angles** with the adjacent arterial wall (figure 16).
In chronic embolism, thrombus is predominantly seen peripherally as **thickening of vessel wall** which when seen perpendicular to the vessel forms an **obtuse angle** with the adjacent wall. When thrombus is seen centrally, they are seen as thin linear bands or webs.

**Patients suitable for pulmonary endarterectomy**

Pulmonary thromboendarterectomy is indicated for patients who despite adequate anticoagulation continue to have pulmonary haemodynamic impairment at rest, or those have normal haemodynamics at rest but have marked pulmonary hypertension on exercise. This can be a curative procedure leading to normalisation of pulmonary haemodynamics but carries a high mortality ranging from 4 to 14%. The main cause of operative failure is presence of surgically inaccessible distal thrombus or development of secondary hypertensive arteriopathy in the pulmonary vascular bed. Therefore to be suitable for endarterectomy, the occluding thrombus must be in the main, lobar or proximal segmental arteries. Signs on CTPA predictive of good response include the presence of significant central vessel but limited distal vessel disease and the presence of dilated bronchial arteries. It is thought that patients with dilated bronchial arteries have less severe distal disease, correlating with better prognosis.

**Images for this section:**
**Fig. 1:** Figure 1: 89 year old with serial imaging of thrombus (arrowheads) in the left lower lobe pulmonary artery. (a) Axial CTPA image of acute thrombus forming acute angle with the vessel wall. (b) Axial CTPA image at 4 months. There has been retraction of the thrombus with irregular intimal surface. (c) Axial CTPA image at 9 months. The thrombus has shrunk further with more irregularity to the intimal surface. Suggestion of thin bands radiating toward the anterior surface was thought to be artifactual rather than true web.

**Fig. 2:** Figure 2: Same patient as figure 1. Coronal images taken from the same vessel. (a) coronal CTPA image of acute thrombus forming acute angle with the vessel wall. (b) coronal CTPA image at 4 months. There has been retraction of the thrombus now forming obtuse angles with the vessel wall. (c) Coronal CTPA image at 9 months. The thrombus has shrunk further with now near complete recanalisation of the vessel lumen.
**Fig. 3:** Figure 3: 83 year old with long hospital stay and worsening shortness of breath. (a) coronal MIP image shows chronic thrombus with complete cut-off of the right lower lobe artery with a convex margin to the contrast called "pouch defect" when described on catheter angiography (arrow). Note also the obtuse angle the thrombus forms with the vessel wall. (b) shows right lower lobe artery in sagittal section. Again note the convex margin and obtuse angles formed with the vessel wall (arrow). Note also the change in calibre (narrowing) of the distal vessel (arrowhead).
**Fig. 4:** Figure 4: 42 year old with a history of multiple chronic thromboemboli. (a) Axial MIP image orientated in the line of the lingula artery shows chronic thrombus causing complete obstruction and shrinkage of vessel. (b) Lung windows show low attenuation (mosaic) perfusion in the lingual with fewer and smaller vessels as compared with the rest of the lung.

**Fig. 5:** Figure 5: 79 year old being investigated for acute on chronic dyspnoea. Coronal oblique MIP image orientated along the left main pulmonary artery. Organised thrombus is seen lining the vessel wall. Thrombus produces an irregular contour to the intimal surface (arrows).
Fig. 6: Figure 6: 80 year old admitted with increasing shortness of breath. Scans done 6 weeks apart. (a, b, c) Axial, coronal and sagittal CTPA showing acute clot. Note the bulging of the clot (a) into the right pulmonary artery (RPA) forming concave margin with the contrast and acute angles with the vessel wall (arrowhead on (c)). (d, e, f) Axial, coronal, sagittal CTPA done 6 weeks later shows partial resolution. The remaining thrombus forms a convex border with the contrast on axial and coronal scans (d and e). Thrombus lining the vessel wall now forms an obtuse angles with it (arrow on (e))
**Fig. 7:** Figure 7: 80 yr old admitted with increasing dyspnoea over 6 months. (a) axial and (b) sagittal scan shows well defined linear structure anchored to the vessel on the both ends. (b) sagittal MIP in the line of the artery shows free edge inferiorly (arrow).

**Fig. 8:** Figure 8: 55 year old investigated for continuing dyspnoea having had a PE 3 months ago. (a) axial scan shows well defined band (arrow) with (b) coronal scan showing superior attachment (long arrow) and inferior free edge (short arrow).
Fig. 9: Figure 9: 85 year old with worsening dyspnoea and previous history of DVT. (a) Multiple thin bands (arrows) forming a web right lower lobe artery (long arrow). Thin bands (short arrows) in left lower lobe (a) and lingual arteries (b).
Fig. 10: Figure 10: 53 year old with a history of chronic thromboembolism. Axial CTPA shows enlargement of the pulmonary trunk (3.4cm) measured just proximal its bifurcation (black line). The adjacent ascending aorta measured 2.8cm. The resultant ratio of pulmonary to aortic dimensions is 1.2, correlating strongly with pulmonary hypertension. Note the atherosclerotic calcification in the right and left pulmonary arteries (arrows).
**Fig. 11:** Figure 11: (a) Axial CTPA image of 80 year old showing normal heart. The right ventricular (RV, black line) to left ventricular ratio (LV, red line) was

**Fig. 12:** Figure 12: (a) Coronal MIP image of 55 year old with history of multiple PEs. Note the enlarged and tortuous intercostal arteries. (b) 42 year old with pulmonary embolus 8 months previously. Note the bilateral tortuous bronchial arteries (arrows). Right is larger than the left. Note also chronic thrombus in the left main pulmonary artery (arrowhead).
**Fig. 13:** Figure 13: 89 year old with thromboembolism 4 months previously. (a) Axial CTPA shows mosaic perfusion pattern in both lungs peripherally. Note the generally diminished vessel size in regions of low attenuation. (b) 42 year old with a history of multiple chronic thromboemboli (same patient as figure 4 and 15). Note the lower attenuation and generally smaller vessels in the left apex as compared to the right.

**Fig. 14:** Figure 14: (a) 84 year old with predominantly left sided pulmonary thromboembolism 2 months previously showing linear parenchymal scarring (arrows) in
apical segment of left lower lobe. (b) 83 year old with history of chronic thromboembolism showing right lower lobe parenchymal scar (arrow).

**Fig. 15:** Figure 15: 42 year old with a history of multiple chronic thromboemboli (same patient as figure 4). Coronal MIP image showing thrombus in right upper lobe, left lingula and left lower lobe arteries. Note the diminished size of the left thrombus filled left lower lobe vessels (arrow) as compared with the opacified right lower lobe vessels. Note also the prominent right bronchial artery.
Fig. 16: Figure 16: 84 year old with history of pancreatic cancer admitted with sudden dyspnoea. (a) Axial CTPA shows filling defect (arrow) surrounded by contrast in a right upper lobe segmental vessel ("polo mint sign"). (b) Same vessel as in (a) imaged in sagittal section shows contrast on either side of a linear filling defect ("railroad track sign", arrow). (c) Axial image of same patient with an eccentric filling defect in a different vessel (right middle lobe segmental) forming acute angles with the vessel wall (arrow). (d) Same patient with axial image showing complete obstruction (arrow) in a left lower lobe segmental vessel. Note the expansion of the vessel compared with other patent vessels (arrowhead).
Conclusion

With more patients undergoing CT scans for suspected thromboembolism, the number of chronic pulmonary emboli being diagnosed is increasing. With pulmonary thromboendarterectomy for treatment of chronic thromboembolism becoming more available, radiologists will need to be comfortable making the diagnosis of chronic thromboembolic disease and in identifying patients suitable for endarterectomy.

Personal Information

References


