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CT pulmonary angiographic imaging manifestations of chronic pulmonary thromboembolic disease

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Background

Chronic pulmonary thromboembolism (PE) is usually a consequence of incomplete thrombus resolution (1). The recognition of chronic pulmonary thromboembolism is important because it remains a potentially curable condition with a pulmonary endarterectomy (2).

In the majority of patients acute PEs resolve spontaneously, however in a minority endothelialised fibrotic obstructions of the pulmonary vascular bed form resulting in a longstanding vascular stenosis (2). These stenoses can go on to cause pulmonary hypertension and cor pulmonale.

The CT features of chronic PEs can be classified into three categories; either direct pulmonary arterial signs due to thrombus itself, signs of pulmonary hypertension or those of a systemic collateral supply.

As a result of the growing number of chest CT examinations being undertaken worldwide, incompletely resolved emboli are an increasingly common finding. Radiologists must be aware of the imaging findings associated with chronic thromboembolic disease as it remains a potentially treatable cause of pulmonary hypertension.

CT Technique

In our institution CT pulmonary angiograms are performed using a General Electric (GE) Lightspeed VCT 64 section system (120 KV, 80-750 mAs, 0.5s rotation time). The images are acquired with the patient in the supine position from the arch of the aorta to the top of the diaphragm. 60-100 mls of ioversol contrast is administered (300 mg I/ml; Optiray 300, Covidien Imaging Solutions, Hazelwood, MO, USA) delivered via an Optivantage pump injector (Covidien Imaging Solutions) at 4 ml/s.

The use of multi-planar reformatted and maximum intensity projection images have been suggested as helpful in interpretation as they provide longitudinal views of vessels which may help clarify questionable findings that could represent obstructions (1).

CT features of chronic pulmonary thromboembolic disease

Direct pulmonary arterial signs: Filling defects

An organized thrombus within the arterial wall can cause vessel narrowing, irregularities within the intima, bands or webs (3). Organised thrombus is often seen running parallel to the lumen and appears as arterial wall thickening

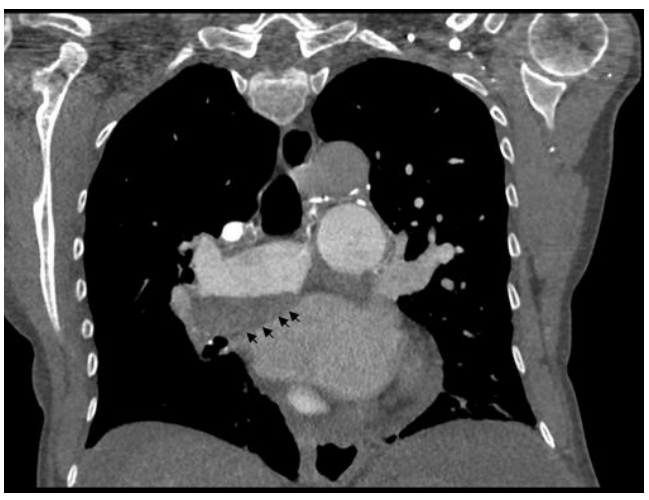

Figure 1a: Coronal CT reconstruction (W800 L100) showing peripherally layered thrombus (black arrows) in the right main pulmonary artery.

Figure 1 b: Axial contrast enhanced CT scan (W800 L100) showing chronic thrombus (white arrows) causing narrowing of the left main pulmonary artery.

(Figures 1a and 1b).
Axial slices through chronic arterial thrombus can have the appearance of cresenteric shaped intra-luminal filling defects with adherence to the vessel wall (Figure 2).

Figure 2:

Magnified axial contrast enhanced CT image (W800 L100) showing chronic thrombus (white arrows) with a broad based adherence to the vessel wall.

Resultant post-stenotic dilatation can also often be seen (Figure 3).

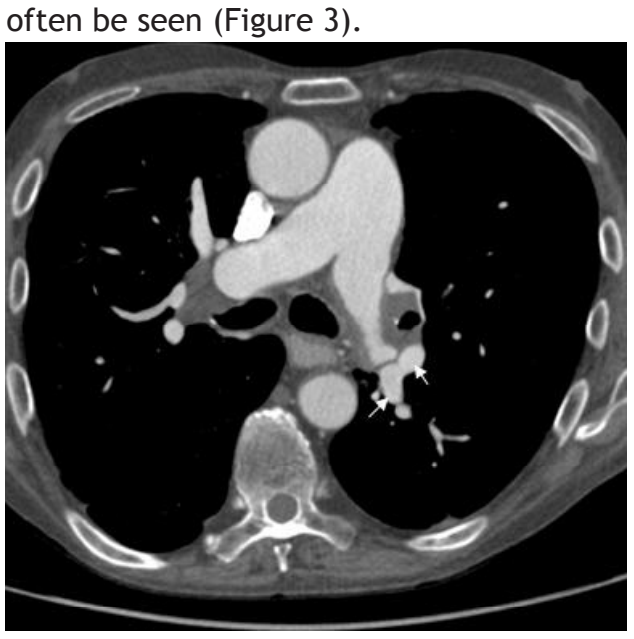

Figure 3: Axial contrast enhanced CT scan (W800 L100) showing post-stenotic dilatation (white arrows) of segmental vessels secondary to extensive chronic thrombus in the left main pulmonary artery.

A band is a linear defect anchored at both ends, with an unattached segment in its mid-portion, which is often orientated in the direction of blood flow (Figure 4). Multiple bands that form a network are webs and are seen as thin lines surrounded by contrast material.

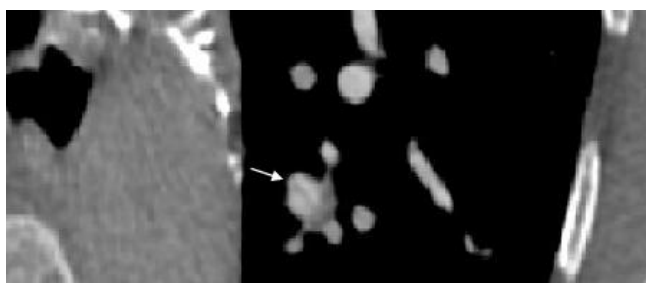

Figure 4: Magnified contrast enhanced axial image (W800 L100) showing a fibrotic band within a segmental pulmonary artery (white arrow).

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CT pulmonary angiographic imaging manifestations of chronic pulmonary thromboembolic disease (continued)

Signs of pulmonary hypertension

The signs related to pulmonary hypertension include enlargement of the main pulmonary arteries, at herosclerotic calcification of the pulmonary arteries (4), and signs of right heart disease (5).

Pulmonary hypertension of any cause can result in enlargement of the main pulmonary artery. Frazier et al suggest that a diameter of greater than 29mm, when the main pulmonary artery is measured in the scanning plane at right angles to its long axis just lateral to the ascending aorta, is indicative of pulmonary hypertension (4) (Figure 5).

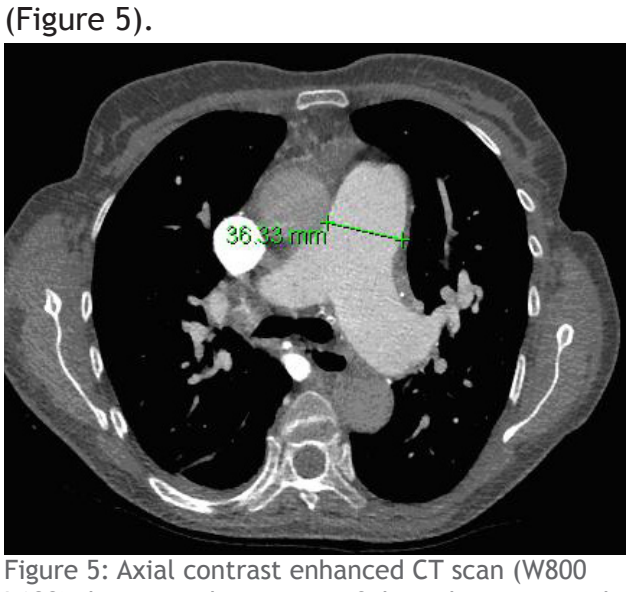

Figure 5: Axial contrast enhanced CT scan (W800 L100) showing enlargement of the pulmonary trunk indicating the presence of pulmonary hypertension.

Moreover if the ratio of the diameter of the pulmonary artery to that of the aorta is greater than 1:1 then this is highly suggestive of elevated pulmonary arterial pressures (6).

Right heart failure is a common finding in association with pulmonary hypertension. The increased strain put on the right sided chambers as a result of increased vascular resistance in the pulmonary circulation leads to right ventricular hypertrophy and enlargement. The presence of right ventricular dilatation is confirmed when the diameter of the right ventricle exceeds that of the left ventricle (7). Measurements are made in the axial plane and are taken at the widest point of the right and left ventricular chambers in diastole (Figure 6).

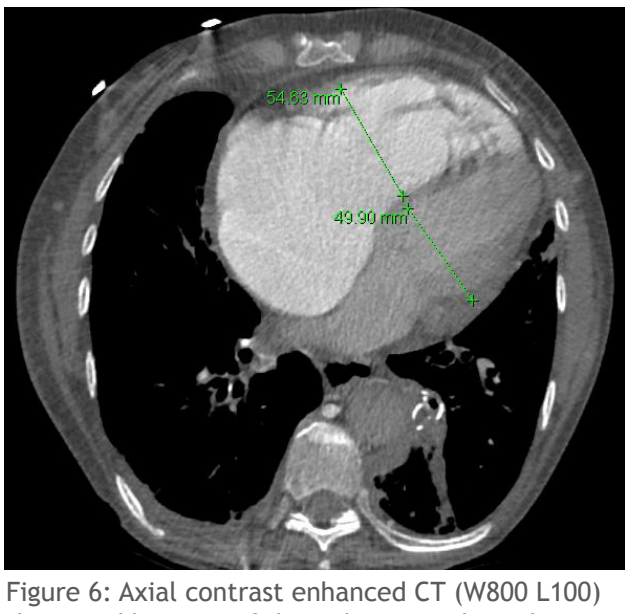

Figure 6: Axial contrast enhanced CT (W800 L100) showing dilatation of the right ventricle with a ratio of greater than 1:1 in comparison with the left ventricle.

Right heart failure may also be accompanied by dilatation of the tricuspid valve annulus and resultant tricuspid regurgitation (1) (Figure 7).

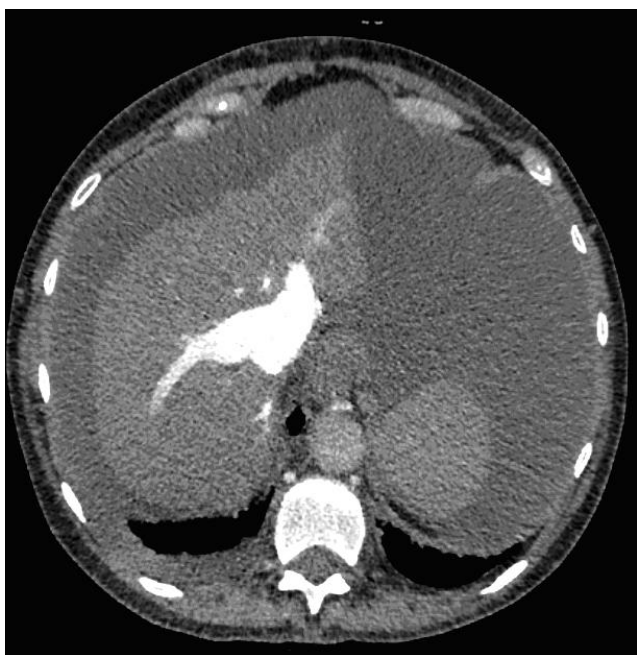

Figure 7: Axial contrast enhanced CT (abdominal window settings) showing opacification of the IVC and retrograde filling of the hepatic veins secondary to tricuspid regurgitation. Note is made of the ascites which may be a feature of right heart failure.

Collateral systemic supply

Collateral systemic arterial supply manifests as the enlargement of bronchial and non-bronchial systemic arteries. Bronchial arterial flow increases in response to a chronic obstruction of the pulmonary vasculature (1) (Figure 8).

Figure 8: Magnified contrast enhanced axial image (W800 L100) showing dilated bronchial arteries as a result of chronic obstruction of the pulmonary vasculature.

Whilst in physiological normality the bronchial arteries are solely responsible for nutritive supply to bronchi, states of diminished pulmonary arterial circulation results in an increase in flow through the bronchial vessels as they are now required to participate in blood oxygenation (8). In addition, trans-pleural systemic collateral vessels, such as intercostal arteries, have been described (9).

The authors have also observed pulmonary/systemic venous collateralization via the bronchial venous plexus in a case of chronic PEs with pulmonary hypertension. Dilated tortuous vessels were noted within the mediastinum that, because of their differential density from the systemic arterial system and anatomical continuation with the venous system could not be dilated bronchial arteries as described above, but represent bronchial venous collaterals (Figure 9).

Figure 9: Coronal maximum intensity projection CT image showing dilated tortuous mediastinal vessels of venous origin representing pulmonary/systemic venous collateralization. Note is made of the density of contrast within the mediastinal collaterals and in the venous system and that is different from the density in the aorta and systemic arterial system.

Conclusion

The presence of any of the above radiological signs should prompt the radiologist to suspect a diagnosis of chronic pulmonary thromboembolic disease. This is of particular importance when the signs of pulmonary artery hypertension or a systemic collateral supply are recognised, as the chest CT may not have been performed using a pulmonary angiogram protocol and one may therefore miss the opportunity to make a diagnosis of chronic PEs.

Its recognition is vital as the condition is potentially treatable, significantly improving the prognosis for these patients.

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