Pulmonary Embolism through the CT Angiography looking glass

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Authors: M. Tapp\textsuperscript{1}, A. Mannava\textsuperscript{2}, F. C. Lyall\textsuperscript{3}; \textsuperscript{1}Torquay/UK, \textsuperscript{2}Torbay Hospital, Torquay, Devon/UK, \textsuperscript{3}Torbay Hospital, Torquay/UK
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Learning objectives

The value of a CTPA study can be improved by:

a) Using optimal techniques in conducting and viewing a scan.

b) Systematically reviewing images to derive maximum useful information i.e. diagnosis, chronicity, severity, background lung/cardiac pathology, other pathology.

We have attempted to address these two issues in this poster, referencing current literature as well as our local protocols and experience at Torbay Hospital, South Devon NHS Foundation Trust, UK.

Background

Pulmonary thromboembolism (PE) is a potentially life threatening, relatively common cardiovascular emergency.

Early diagnosis can improve clinical outcome in acute embolism and often aids management in chronic embolism. Thin slice Multidetector-row spiral CT pulmonary angiography (MD-CTPA) is steadily becoming the modality of choice for radiological detection of pulmonary thromboembolism. The advent and continued protocol improvement of CT for detecting pulmonary embolism has led to a radical improvement in the standards of care worldwide in managing this condition.

A few features of PE to remember:

- PE is a master of masquerade, both clinically and in imaging. Common clinical presentations of PE are dyspnoea, pleuritic chest pain, cough and haemoptysis. In severe cases, right heart failure causes dizziness or syncope.

- Emboli do not necessarily result in infarction.

- Less than 30% are associated with symptomatic deep vein thrombosis (DVT).

- PE occurs mostly in the lower lobes and is often multiple (62%) and bilateral (50%)[1].
• Chest radiographs are most often normal. However, signs such as an enlarged pulmonary artery, peripheral ‘wedge’ opacity (implying lung infarction), regional oligoaemia, pleural effusion and raised hemidiaphragm may be evident.

• Acute right-sided heart failure is known to be responsible for circulatory failure and death in patients with severe PE.

Risk factor history such as immobility, recent fracture, trauma, surgery, previous embolic disease, and malignancy maybe sometimes deduced from previous imaging, if available. Provision of these details, clinical signs and scores (such as the Wells Score recommended by NICE[2]) when requesting a scan is very useful.

Findings and procedure details

**CTPA Technique**

Common obstacles to a diagnostic study are:

· Motion artefacts.

· Flow and streak artefacts.

· Poor opacification of arteries.

· Background lung pathology.

Patient habitus, pregnancy and kidney disease pose certain constraints.

CTPA is contraindicated in severe kidney failure and if the patient is allergic to contrast.

At Torbay Hospital we use 64 slice MDCT, GE Healthcare scanners.

· Scanning requires the patient to lie supine and still, holding their breath for 3-5 seconds, ideally with their arms above their head. Dyspnoeic patients are aided by continuing oxygenation throughout the scan and sometimes raising the head end with supports. Motion artefacts can be caused by a patient hyperventilating or cardiac motion (Fig 1).
An abrupt shift in path or position of vessels on contiguous imaging indicates a motion artefact.

**Fig. 1**: The double wall appearance of the main pulmonary trunk is due to a motion artefact from cardiac motion. The arrow points to the true wall.

**References**: NHS, Torbay Hospital - Torbay Hospital, Torquay/UK

- We recommend prior intravenous access with an 18G catheter. 80-100ml uniphase contrast bolus is injected at 4.5 ml/second. This is followed by a saline flush. The flush decreases streak artefact caused by high density contrast material in the superior vena cava obscuring adjacent pulmonary vasculature[3](Fig 2). Streak artefacts from metallic implants cannot be overcome and alternate modality of imaging maybe needed.
Fig. 2: The streak artefact from SVC makes it difficult to visualize right pulmonary artery. Here the blue arrows point to streak artefacts while red arrow points to true emboli.

References: NHS, Torbay Hospital - Torbay Hospital, Torquay/UK

- Timing scan acquisition is vital to optimal imaging. Contrast undergoes first pass metabolism and recirculates, accumulating in pulmonary vasculature with time. Empirical scanning delay uses this principle to obtain better opacification in vessels. However, there is a risk of non-diagnostic early or late scanning, as in either case suboptimal opacification of the pulmonary tree can occur. In addition, a rapid inspiratory effort just before the scan can draw in unopacified blood from the inferior vena cava reducing the diagnostic quality of the scan, so the patient should be advised to temporarily stop breathing rather than taking in a large breath for the scan. We use Smart prep software[4] to prompt the radiographer to start the scan. This method of bolus tracking is widely used, with the intention of having opacification of at least 250 HU in the pulmonary tree for optimal contrast enhancement.

- Images are acquired caudo-cranially. This ensures that lower lobes are imaged early where most PE tend to occur. If the patient breathes during the scan, we prefer imaging at least the lower lobes without motion artefact.
Acquired imaging is viewed on multiple window settings.

1) Lung window (WW 1400HU, WL 600HU).

2) Mediastinal window (WW 440HU, WL 40HU).

3) Pulmonary embolism specific window (WW 500HU, WL 150HU).

4) Bone window (WW 2500HU, WL 200HU).

5) Liver window (WW 155HU, WL 115HU).

Viewing images on different grey scales helps identify emboli and assess severity with more clarity. It aids distinguishing pathology mimicking as PE and artefacts, from true thrombo-emboli better. The variation in windows may add useful information about existence of co-morbidities, haemodynamic and ventilatory status.

Routine review of upper abdomen, lymph nodes, bone and soft tissue is important.

These may give clues for detection of PE or may add information alternate/additional to suspected diagnosis.

It is important to take background lung pathology into account for understanding flow artefacts. Local atelectasis or consolidation can lead to increased local resistance, causing a slow flow (Fig 3), thereby reducing opacification in the vessel mimicking PE.
Fig. 3: The left lower lobe arteries are poorly opacified making it difficult to differentiate from subsegmental PE.

References: NHS, Torbay Hospital - Torbay Hospital, Torquay/UK

- Patients with a high BMI or greater girth width may prove difficult to image. Our CT tables carry up to 227kg; table width being 43cm and table to gantry height being 50cm. In larger patients we require more contrast and/or increased radiation for optimal imaging. This tends to increase display image noise. We have a protocol at Torbay, to change radiation dose, scan timing and reconstruction of image based on patient weight to optimize the image, reducing noise.

- In pregnant ladies with symptoms of PE, our institution performs bilateral lower limb Doppler ultrasound examinations. If these are positive we treat the patient empirically as having a PE to avoid the radiation dose. If the ultrasound scans are negative, we perform perfusion only nuclear medicine imaging instead of CTPA. However if CTPA is still required, we use 80ml contrast, varying the timing of the scan to minimise radiation.
· In patients with severe acute kidney injury we try to hydrate the patient sufficiently and look for improvement in kidney function before conducting the scan.

· We may have to repeat CTPA scan or perform doppler ultrasound lower limbs if the original scan is indeterminate, depending on the level at which it is indeterminate. CTPA showing a thrombus up to the segmental level can be taken as adequate evidence of PE in most instances, whereas the necessity to treat isolated sub-segmental thrombi in an asymptomatic patient without a DVT is unclear[6]. In patients with a non-high clinical probability, CTPA may be used as a stand-alone test. Further testing in patients who have a negative CTPA despite a high clinical probability is based on individual physician’s judgment.

**Reviewing Imaging**

The value of developing a systematic method in reviewing any imaging cannot be overstressed. In the case of CTPA we have four areas that must be evaluated.

We like calling them the ABCD of CTPA-

<table>
<thead>
<tr>
<th>A</th>
<th>B</th>
<th>C</th>
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<tbody>
<tr>
<td><strong>Arterial signs</strong></td>
<td><strong>Breathing</strong></td>
<td><strong>Cardiac signs</strong></td>
<td><strong>Don’t Forget</strong></td>
</tr>
<tr>
<td>Are there filling defects in the pulmonary arteries?</td>
<td>How well is the lung perfused?</td>
<td>Is there right heart strain?</td>
<td>What else is the CTPA telling us?</td>
</tr>
<tr>
<td>At what level is there an obstruction?</td>
<td>-Is there any wedge infarction?</td>
<td>Is there pulmonary hypertension?</td>
<td>Is there a change from previous?</td>
</tr>
<tr>
<td>acute PE</td>
<td>-Any ground glass or mosaic opacity?</td>
<td>-Any interventricular septum change?</td>
<td>-review in different windows</td>
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<tr>
<td>-Dilatation of artery</td>
<td>-Any associated venous congestion?</td>
<td>-Any reflux into the inferior vena cava reflux?</td>
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Arterial signs

Emboli appear as filling defects in an otherwise opacified pulmonary vessel.

Acute emboli may

- occlude a vessel completely.
- be located at the centre of a pulmonary vessel.
- be abutting a wall.

On axial section this may appear as non-opacification, central filling defect or peripheral filling defect forming an acute angle, respectively.
Fig. 4: Peripheral filling defect of acute PE at bifurcation of left pulmonary artery.

References: NHS, Torbay Hospital - Torbay Hospital, Torquay/UK
Fig. 5: Filling defects of multiple acute PE. The central filling defect of partial occlusion the the arrow points to, is like a ‘polo’ mint (Polo mint sign/donut sign/railway track sign). The peripheral defects making acute angle to wall are also visible. 

References: NHS, Torbay Hospital - Torbay Hospital,Torquay/UK

Fig. 6: In acute PE the vessel becomes dilated compared to those surrounding of the same order as seen in the left lower lobe (arrow pointing to embolus).

References: NHS, Torbay Hospital - Torbay Hospital,Torquay/UK

Saddle filling defect - when a large acute PE bridges over two bifurcating vessels it looks like a horse saddle, hence described as a saddle PE.
Fig. 7: Acute saddle PE. This person had multiple bilateral pulmonary emboli, including a central saddle PE straddling the bifurcation of the pulmonary trunk.

References: NHS, Torbay Hospital - Torbay Hospital, Torquay/UK

In chronic PE the vessels retract and appear of smaller calibre compared to surrounding vessels of the same order. Also, the peripheral embolus makes an obtuse angle, crescentic defect within artery wall [7].

Another feature common to chronic PE is pulmonary hypertension. When a clot matures and retracts it causes vessel stenosis and leads to pulmonary hypertension. This may be seen as dilated, sometimes tortuous main pulmonary arteries. When suspicious it is worth measuring:

- Pulmonary artery diameter at the level of bifurcation, lateral to ascending aorta at right angle to long axis. Measurement of >30mm favours diagnosis of pulmonary hypertension.

- Diameter of pulmonary artery: Diameter of aorta is usually 1:1. Any dilatation will increase this ratio. This has a stronger correlation in patients under 50 years of age.

Ancillary features of pulmonary hypertension such as development of systemic collateral blood supply, atherosclerotic calcification and presence of aneurysms maybe visible.
Parenchymal changes are varied in the population. The key feature to assess from lung parenchyma is perfusion status. Under perfused tissue has low attenuation. We need to take into account where the anatomical obstruction is visualised, collateral and variable bronchial arterial supply and presence of pre-existent cardiopulmonary disease.

Fig. 9: The main pulmonary artery when compared to adjacent ascending aorta is grossly enlarged suggesting pulmonary hypertension.

References: NHS, Torbay Hospital - Torbay Hospital, Torquay/UK

The lung parenchyma has dual blood supply from pulmonary and bronchial arteries. Pulmonary arteries fan out from a central hilum to a wider periphery; hence infarctions tend to be wedge shaped (Fig.10) with a wide peripheral base. The periphery of the lung is also more susceptible to infarction as the bronchial arteries supply more mediastinal lung tissue.
**Fig. 10:** Wedge shaped area of consolidation in left lower lobe with areas of internal air lucencies, representing infarcted lung tissue with some aerated lung areas.

**References:** NHS, Torbay Hospital - Torbay Hospital, Torquay/UK

Thromboembolic infarction presents on CTPA usually as

- Mosaic perfusion abnormalities. Decreased perfusion leads to low attenuation in affected areas often accompanied by an asymmetry in the number and calibre of vessels. This is more commonly seen in chronic PE.
- Focal ground glass pattern (Fig 11).
Fig. 11: Right upper lobe ground glass opacification.

References: NHS, Torbay Hospital - Torbay Hospital, Torquay/UK

There may be non-infarcted aerated parenchymal tissue adjacent to infarcted tissue. Associated haemorrhage may appear as a peripheral concave lesion.

Following treatment of acute PE the lung changes mature and involute, often leading to areas of cavitation and atelectasis.

Whilst non thromboembolic PE are uncommon, they may have characteristic imaging. These may be discrete nodules with cavitation (septic emboli), widespread mosaic homogenous or heterogeneous increased opacification 12-24 hours following trauma (fat emboli) or fine miliary nodules which coalesce at a later stage (talcosis).

Parenchymal scarring can result from previous infarctions.

Rarely in chronic PE webs/bands/linear opacities can be visualised within the vessel. Bronchial dilatation with bronchiectasis is also common in chronic PE. Thrombi of long standing can calcify mimicking diffuse calcified pulmonary metastasis on CT chest.
Fig. 12: The thickened-wall appearance of the main pulmonary trunk is suggestive of chronic PE.

References: NHS, Torbay Hospital - Torbay Hospital, Torquay/UK

Cardiac signs

Newer CT associated imaging modalities and studies are often directed to yield more prognostic scoring data.

- The ECG gated CT uses gating techniques to improve temporal resolution and minimize imaging artefacts caused by cardiac motion. It provides details of cardiac function and therefore is useful in evaluating prognosis[8].
- Dual energy CT uses iodine uptake to visualise perfusion impairment. These are co-related to areas of anatomical obstruction in order to derive the true degree of perfusion impairment and estimate collateral blood supply[9].

The magnitude of PE can be calculated on CT pulmonary angiography by applying dedicated CT scores or angiographic scores. Right heart failure, pulmonary hypertension and large central PE all indicate poor prognosis. On CTPA imaging we look out for signs
of pulmonary hypertension and right ventricular failure i.e. right ventricular dilatation and dysfunction as well as its effect of peripheral venous congestion. These can be seen as

- Straightening of the interventricular septum and sometimes even bowing of septum into left ventricle (Fig 13).
- 3D cardiac volumetric reconstruction may better be correlated to right ventricular hypertrophy/dilatation and related morbidity than ratio of diameters of the ventricles RV: LV being >1:1. However the ratio of ventricular diameters measured as the greatest short-axis of each, still gives useful information[10].
- Reflux of contrast into the inferior vena cava and retrograde congestion of veins indicate poor right ventricular function.

The presence of large central arterial PE is always going to have high value in the scoring systems, indicating poor prognosis regardless of whether or not signs of pulmonary hypertension or right heart failure are present.

![Fig. 13: Bowing of interventricular septum signifying right heart strain.](image)

*References*: NHS, Torbay Hospital - Torbay Hospital, Torquay/UK
**Fig. 14**: Despite motion artefacts, an enlarged right ventricle is obvious with straightening of the interventricular septum.

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Fig. 15: There is reflux of contrast material into the inferior vena cava.

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Fig. 16: Axial CTPA section are both from patient with severe right heart failure and retrograde venous congestion in liver.

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Fig. 17: Coronal CTPA section from the same patient as of previous image with severe right heart failure and retrograde venous congestion in liver.

References: NHS, Torbay Hospital - Torbay Hospital, Torquay/UK

Do not forget!

The CTPA report should ideally comment on -

· Contrast agent and volume.
· Clinical information available.
· Comparison made to previous films.
· Diagnostic quality of film.
· Pulmonary embolus.
· Right heart strain.
· Pulmonary arteries.
· Lung parenchyma.
· Pleural effusion.
· Central airways.
· Lymphadenopathy.
· Heart and great vessels.
· Upper abdomen.
· Bone and soft tissues.

Impression is based on reviewing all of these.

When a scan is indeterminate it is important to establish the level to which we have clarity (e.g. - mention sub-segmental or segmental).

Routine assessment on different windowing settings is required to ensure full assessment of the examination, to aid in the assessment of other pathology such as bone or liver lesions as well as pulmonary emboli.
Fig. 18: While PE was not seen this patient had a left breast mass detected on CTPA.
References: NHS, Torbay Hospital - Torbay Hospital, Torquay/UK

Fig. 19: This patient had a left lower lobe lung collapse and associated pleural effusion which were the cause of hypoxia and dyspnea.
References: NHS, Torbay Hospital - Torbay Hospital, Torquay/UK
Fig. 20: This patient with pancreatic cancer metastasising to bone and liver was seen to have progression of disease on CTPA. We see a lytic lesion in the distal right clavicle which is at risk of a pathological fracture.

References: NHS, Torbay Hospital - Torbay Hospital,Torquay/UK
**Fig. 21**: These sclerotic spinal metastases were noticed prominently on bone window settings.

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**Images for this section:**
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![Image of CT scan showing filling defects in the lung with an arrow pointing to the polo mint sign]

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Fig. 8: Chronic long strand of thrombus in the right lower lobe pulmonary artery. The vessel has retracted and is of smaller caliber.
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Fig. 21: These sclerotic spinal metastases were noticed prominently on bone window settings.
Conclusion

Pulmonary thromboembolism is a common cardiovascular cause of death. It is potentially reversible in the acute setting with early detection and potentially curable in the chronic setting. Systematically conducting scanning, viewing and reading images improves accuracy and can prevent pulmonary thromboembolism from being missed, adding information regarding chronicity, severity, ventilatory and haemodynamic status of patient. A non-diagnostic CTPA scan can still yield information about likely alternate diagnosis.

While CTPA is unlikely to be challenged in the near future as the first choice of imaging to detect pulmonary thromboembolism, we may see newer modalities address prognostic information.

Personal information

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