Cardiac arrest immediately after CT Pulmonary Angiogram in the setting of multiple bilateral pulmonary emboli - report of three cases

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Learning Objectives

• Appreciate that patients with massive bilateral pulmonary emboli (PE) may develop cardiac arrest while undergoing Computed Tomography Pulmonary Angiography (CTPA),
• Recognize the clinical features of acute right strain secondary to massive pulmonary emboli, and,
• Counsel referring doctors about the risk of cardiac arrest during CTPA and recommend cardiac monitoring of such patients by staff appropriately trained in advanced life support and thrombolysis administration.
Background

Although formal pulmonary angiography has historically been regarded as the gold standard for the diagnosis of pulmonary embolism, computed tomography pulmonary angiogram (CTPA) is now considered to be the safer and, with the widespread implementation of multidetector CT (MDCT), the more accessible alternative with high sensitivity and specificity [1,2].

There have been reported cases in the literature, however, of cardiopulmonary arrest occurring in patients during CT scanning, especially in the context of trauma, earning the CT scanner the moniker of "the donut of death" [3]. There is one reported case occurring in the context of bilateral massive PE immediately after CTPA [4]. We report three additional cases.
Imaging Findings OR Procedure Details

Patient 1

A 40 year old female presented to the Emergency Department of a district hospital with a 1 day history of sudden onset dyspnoea and pleuritic chest pain, one week after an elective right knee replacement. She reported being immobile since the operation. Her relevant past medical history included morbid obesity and 1 pack per day of smoking. Clinical examination revealed she was hypotensive (BP 100/80 mmHg) and tachycardic (HR 130 bpm). Pulse oximetry was 94% on room air. ECG showed sinus tachycardia.

A presumptive diagnosis of PE was made, and the patient was commenced on therapeutic heparin infusion and transferred to the nearest regional hospital for CTPA, which showed multiple pulmonary emboli almost occluding both interlobar arteries (Fig. 1 on page 6 and Fig. 2 on page 6) as well as enlarged right ventricle relative to the left (Fig. 3 on page 7).

Immediately after the CT was performed, the patient suddenly became agitated. During the transfer of the patient from the CT machine to the Emergency bed, she developed cardiac arrest, and despite 30 minutes of cardiopulmonary resuscitation, IV tPA and anticoagulation, she passed away.

Patient 2

A 68 year old male presents to the Emergency Department with right calf swelling and pleuritic chest pain on the background of previous deep vein thrombosis (DVT) and PE in 2009 and acute myeloid leukaemia. His other relevant past medical history included pulmonary sarcoidosis and interstitial nephritis. On examination, he was tachycardic (HR 90). ECG showed sinus tachycardia (Fig. 4 on page 8).

A presumptive diagnosis of pulmonary embolism was made, and the patient was commenced on therapeutic clexane prior to CTPA, which showed multiple bilateral segmental PEs (Fig. 5 on page 9 Fig. 6 on page 10 Fig. 7 on page 11). It also demonstrated bowing of the interventricular septum into the left ventricle (Fig. 8 on page 12).

Immediately after the CTPA the patient suffered a cardiac arrest. Intravenous tPA, adrenaline and 30 minutes of cardiopulmonary resuscitation was performed before
spontaneous cardiac activity returned. The patient was intubated and was discharged after a lengthy stay in the intensive care unit (ICU).

Patient 3

A 58 year old female presents with 1 week history of recurrent syncope and dyspnoea on the background of known left DVT and non-small cell lung cancer treated recently with chemotherapy. She denied any chest pain. She had been commenced on clexane by her family doctor one week earlier. On examination, she was tachycardic (HR 130), tachypnoeic (RR 20) and had mild hypotension (BP 105/76). Her pulse oximetry read 94% on room air. Her left calf was mildly erythematous, tender and swollen. ECG showed S1Q3T3 changes, with inverted T waves in the right chest leads (Fig. 9 on page 13).

A presumptive diagnosis of pulmonary embolus was made and the decision was made to confirm the diagnosis by CTPA, which showed a saddle pulmonary embolus extending into both main pulmonary and left interlobar artery (Fig. 10 on page 14), as well as bowing of the interventricular septum into the left ventricle (Fig. 11 on page 15) and reflux of contrast agent from the right atrium into the inferior vena cava and liver (Fig. 12 on page 16).

The patient developed cardiac arrest immediately after the CTPA. Despite 50 minutes of cardiopulmonary resuscitation, intravenous adrenaline and tPA, the patient passed away.
**Fig. 1:** Patient 1 - CTPA axial image - bilateral pulmonary emboli almost occluding both interlobar arteries

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Fig. 2: Patient 2 - CTPA Coronal Maximum Intensity Projection (MIP) - multiple bilateral pulmonary emboli almost occluding both interlobar arteries and occluding the segmental artery to the lingula.

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Fig. 3: CTPA Axial Image - bowing of the interventricular septum into the left ventricle

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Fig. 4: Patient 2 ECG - sinus tachycardia

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Fig. 5: Patient 2 - Coronal CTPA - Segmental PEs in the upper lobes

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Fig. 6: Patient 2 - coronal CTPA - multiple PEs in both interlobar arteries

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Fig. 7: Patient 2 - coronal CTPA - segmental PEs in right upper and lower lobes

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Fig. 8: Patient 2 - axial CTPA - bowing of interventricular septum into left ventricle

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**Fig. 9:** Patient 3 - ECG - S wave in lead I, prominent Q wave in lead II and inverted T waves in lead III and anteroinferior chest leads (V1 to 3), suggestive of right ventricular strain.

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**Fig. 10:** Patient 3 - axial CTPA - filling defects straddling the pulmonary trunk bifurcation (saddle PE)

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**Fig. 11:** Patient 3 - axial CTPA - bowing of interventricular septum into left ventricle

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**Fig. 12:** Patient 3 - axial CTPA - reflux of intravenous contrast into IVC and right hepatic vein

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Conclusion

All three patients suffered cardiopulmonary arrest during or immediately after undergoing CTPA. Although pulmonary embolus is a well-recognised cause of cardiac arrest [5] and is likely the cause in these three patients, one cannot exclude the possibility that the process of performing the CTPA may have hastened the progression of the disease.

Both formal and CT pulmonary angiography involve the injection of intravenous radiographic contrast under pressure - is this injection to blame? Mills et al described one patient with acute and chronic pulmonary PE who was hypotensive before the formal angiogram and developed cardiopulmonary arrest after contrast injection [6], but the type of contrast medium was not stated, and the patient had documented pre-existing pulmonary hypertension.

The haemodynamic and pulmonary effects of radiographic contrast media in the context of formal cardiac and pulmonary angiography has been documented in both in vitro and vivo studies [7-10], ranging from systemic hypotension, severe pulmonary hypertension, non-histamine induced bronchospasm, reduced lung function and increased right atrial pressures. The use of older non-ionic and ionic contrast media in these studies is a potential cofounder, however. Ultravist (iopromide) 300 and 370mgI/ml, a non-ionic contrast agent, is used at John Hunter, and has been associated with non-histamine induced bronchospasm [10] as well as rises in pulmonary arterial pressures in rat studies [11]. Iopromide remains hypertonic to saline, and hypertonic solutions themselves in the pulmonary circulation are associated with reduced systemic blood pressure and raised pulmonary pressures [12,13].

Other possible mechanisms include the ventilation perfusion changes that occur in the supine position required to perform the CTPA, in contrast to the erect position that dyspnoeic patients are usually nursed. No current literature exists to support this hypothesis, however.

Our last patient had a high risk for PE, electrocardiographic evidence of right heart strain and clinically was hypotensive and tachycardic. One could argue that the delay in obtaining the CTPA and the possible contrast or postural changes prevented her from obtaining timely and potentially life-saving thrombolytic therapy. Her history of malignancy, however, was a contra-indication to such a course and the clinician gave thrombolytic therapy during the arrest as a last measure to revive her.

The referring clinician of a patient with high risk for PE should be informed that there is a possible risk of cardiopulmonary arrest during CTPA, and that should the patient
show clinical or electrocardiographic evidence of right heart strain and therefore be considered for possible thrombolytics\cite{14}, consideration be given to using transthoracic echocardiography (TOE) to confirm the diagnosis and avoid contrast injection. If TOE is not readily available, then CTPA may be performed with cardiac monitoring performed by staff trained in advanced life support and thrombolysis administration.
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References


