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A man in his sixties with acute dyspnoea, chest pain and syncope

EDUCATIONAL CASE REPORT

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A man in his sixties was admitted as an emergency due to dyspnoea, chest pain and syncope. The medical team quickly established that he was in a state of shock. Multidisciplinary management resulted in successful diagnosis and treatment of a condition with high mortality.

A man in his sixties with inflammatory bowel disease and myopathy treated with prednisolone called for an ambulance during the night due to acute worsening of dyspnoea. He had been short of breath 3–4 weeks beforehand, but experienced a considerable worsening during the night in question. He fainted twice before he called for help. When the ambulance arrived, he reported pain throughout his body, including severe central chest pain. The patient had central cyanosis, with SpO_2 of 70 % without oxygen, increasing to 93 % with 12 litres of oxygen through a mask with a reservoir. His blood pressure was 115/70 mmHg, and his pulse fluctuated between 130

bpm and 145 bpm.
While transporting the patient to hospital, the ambulance sent an ECG to the cardiology department for prehospital assessment (Figure 1). The ECG revealed sinus tachycardia at a rate of 143 bpm, increased P-wave amplitude in leads II and III consistent with P pulmonale, slight ST elevation in leads V1 and aVR, and ST depression in V4–V6, II, III and aVF.

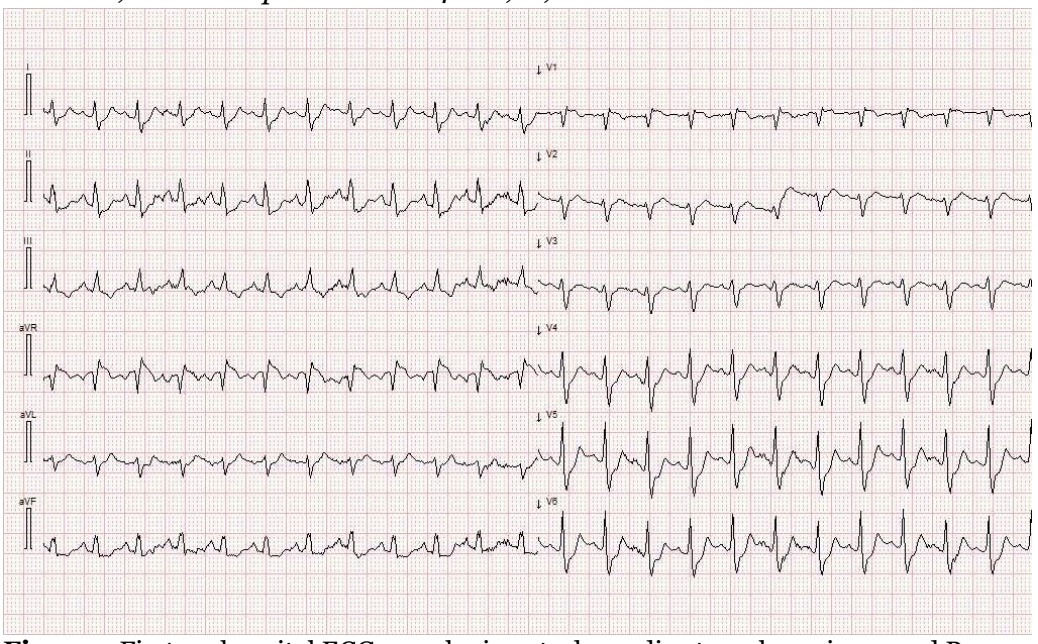

Figure 1 First prehospital ECG reveals sinus tachycardia at 143 bpm, increased P-wave amplitude in leads II and III, slight ST elevation in V1 and aVR, ST depression in V4–V6, II, III and aVF.

Acute coronary syndrome with widespread ischaemia was considered in the differential diagnosis due to findings of ST depression in multiple leads and ST elevation in V1 and aVR. Infarction with subsequent development of acute heart failure and pulmonary oedema was also considered, as well as pulmonary embolism due to findings of sinus tachycardia and P pulmonale, which indicates right atrial dilation.

Ambulance staff sent another ECG 25 minutes later (Figure 2), which revealed atrial fibrillation with a heart rate of 120 bpm, widened QRS complex with right bundle branch block pattern and T-wave inversion in III and V1–V3.

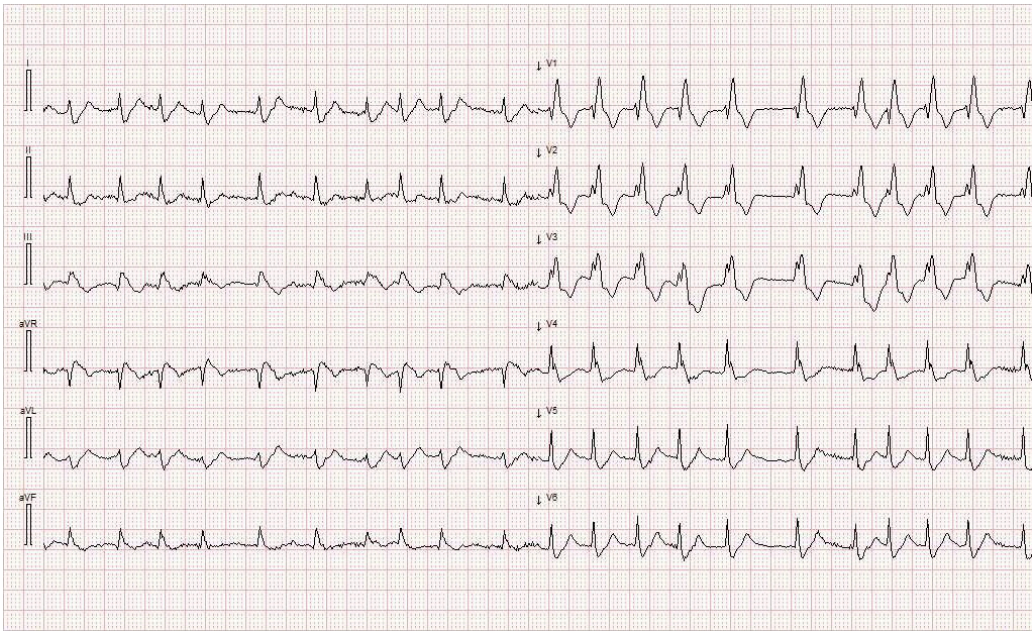

Figure 2 Second prehospital ECG 25 minutes later reveals atrial fibrillation at 120 bpm, development of right bundle branch block, and T-wave inversion in leads III and V1–V3.

Both new onset atrial fibrillation with slower rate than in sinus rhythm and right bundle branch block are signs of failure of the conduction system. Additionally, inverted T-waves in the leads described are a typical finding of right ventricular dilation.

The patient was received by a medical team shortly after the last ECG arrived. He was awake and responded appropriately to questioning. In the initial examination, he had patent airways. His oxygen saturation was 100 % with 12 litres of O_2 and his respiratory rate was 17 breaths per minute. Clear lung sounds were heard on auscultation. His skin was cold and clammy with no peripheral pulses, but with palpable pulses in the groin bilaterally. His blood pressure measured using an arterial line was 50/30 mmHg. The patient monitor showed sinus tachycardia with heart rate of 120 bpm and a QRS configuration associated with right bundle branch block. The patient was afebrile with a temperature of 37.0 °C. Arterial blood gases taken with 12 litres of supplemental oxygen found pH 7.36 (reference range 7.35–7.45), pCO_2 4.45 kPa (4.7–5.9), pO_2 13.8 kPa (11.1–14.4) and lactate 4.6 mmol/L (0–2.5).

Screening echocardiography revealed considerable right ventricular dilation; the diameter of the right ventricle (RV) was larger than that of the left ventricle (LV), with an RV/LV ratio of 1.3. A normal ratio is 0.66, and anything over 1.0 is clearly abnormal (1). The right ventricle appeared to have reduced long axis function with TAPSE (tricuspid annular plane excursion) measured at 10 mm (normal > 17). A large flapping mass was observed in the right atrium, which raised suspicion of a thrombus in transit (Figure 3, video). The left ventricle had decreased septal contractility and was compressed by the dilated right heart chambers. Doppler assessments were consistent with underfilling of the left ventricle. The diameter of the ascending aorta was normal, but aortic regurgitation was found, which was hard to grade in the emergency situation.

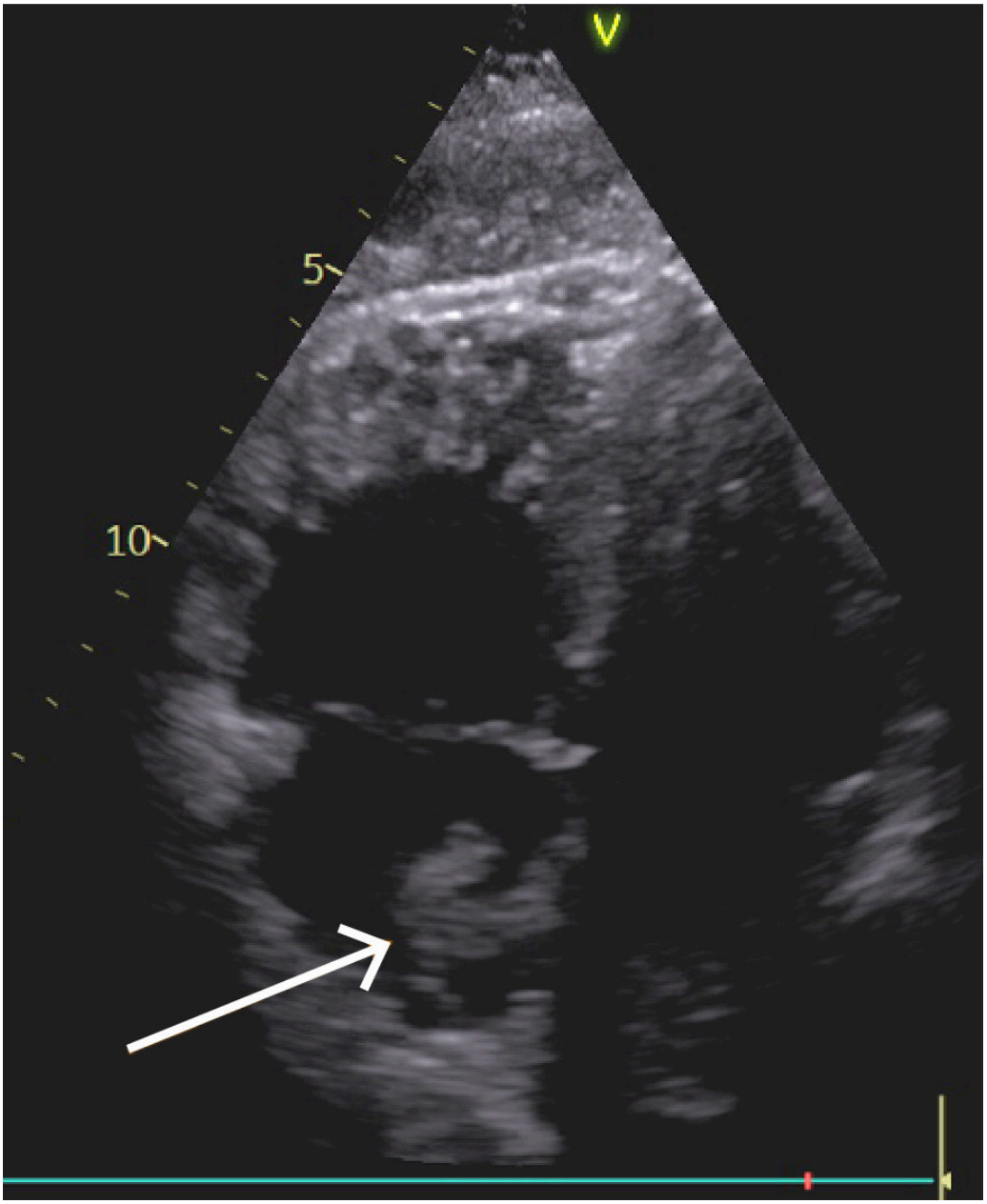

Figure 3 Echocardiography. Right ventricle, upper left; right atrium, lower left. Flapping mass in right atrium (arrow), raising suspicion of thrombosis – so-called 'clot in transit'.

Acute central pulmonary embolism with subsequent right heart failure, reduced cardiac output and circulatory failure seemed to be the most likely cause of the patient's condition. At the same time, findings of aortic regurgitation meant that aortic dissection could not be ruled out with certainty following the echocardiography review. Therefore, a CT scan was needed before administering thrombolytic treatment with alteplase.

After a team discussion, 5000 IU heparin was administered intravenously ten minutes after the patient arrived in the emergency department, in accordance with local and international guidelines (2). To increase the patient's blood pressure, 0.01 mg adrenaline was administered intravenously, and a noradrenaline infusion was started at an infusion rate of 0.3 µg/kg/min. An interventional radiologist and radiographer were also called.

The patient was awake and lucid, his respiratory status was stable, but haemodynamic instability persisted following administration of heparin and initiation of a vasopressor. The patient was transported to the radiology department connected to a defibrillator and with alteplase prepared for intravenous administration.

A chest CT scan with pulmonary embolism protocol revealed a saddle embolism, with thrombi in most of the major pulmonary arteries and involvement of all lobes of the lungs at a lobar and segmental level (Figures 4 and 5). The largest impact was in the lower lobes. Aortic dissection was not detected.

A bolus dose of 10 mg intravenous alteplase was administered immediately once the diagnosis of pulmonary embolism had been verified and aortic dissection ruled out, approximately 40 minutes after arrival of the patient in hospital. A maintenance dose of 90 mg alteplase was then started.

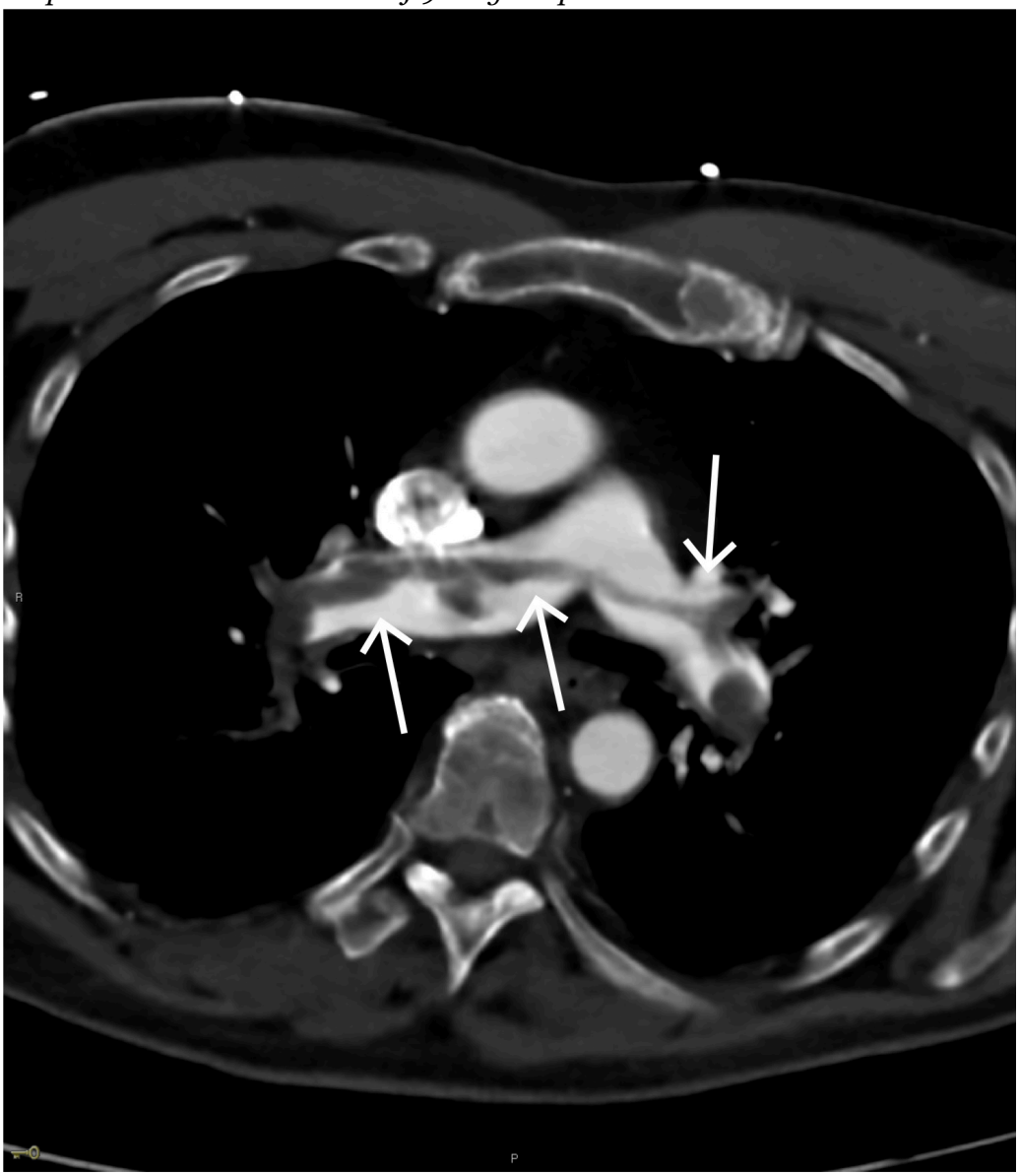

Figure 4 CT angiography. Saddle embolism is seen as a dark filling defect in the pulmonary artery (arrows).

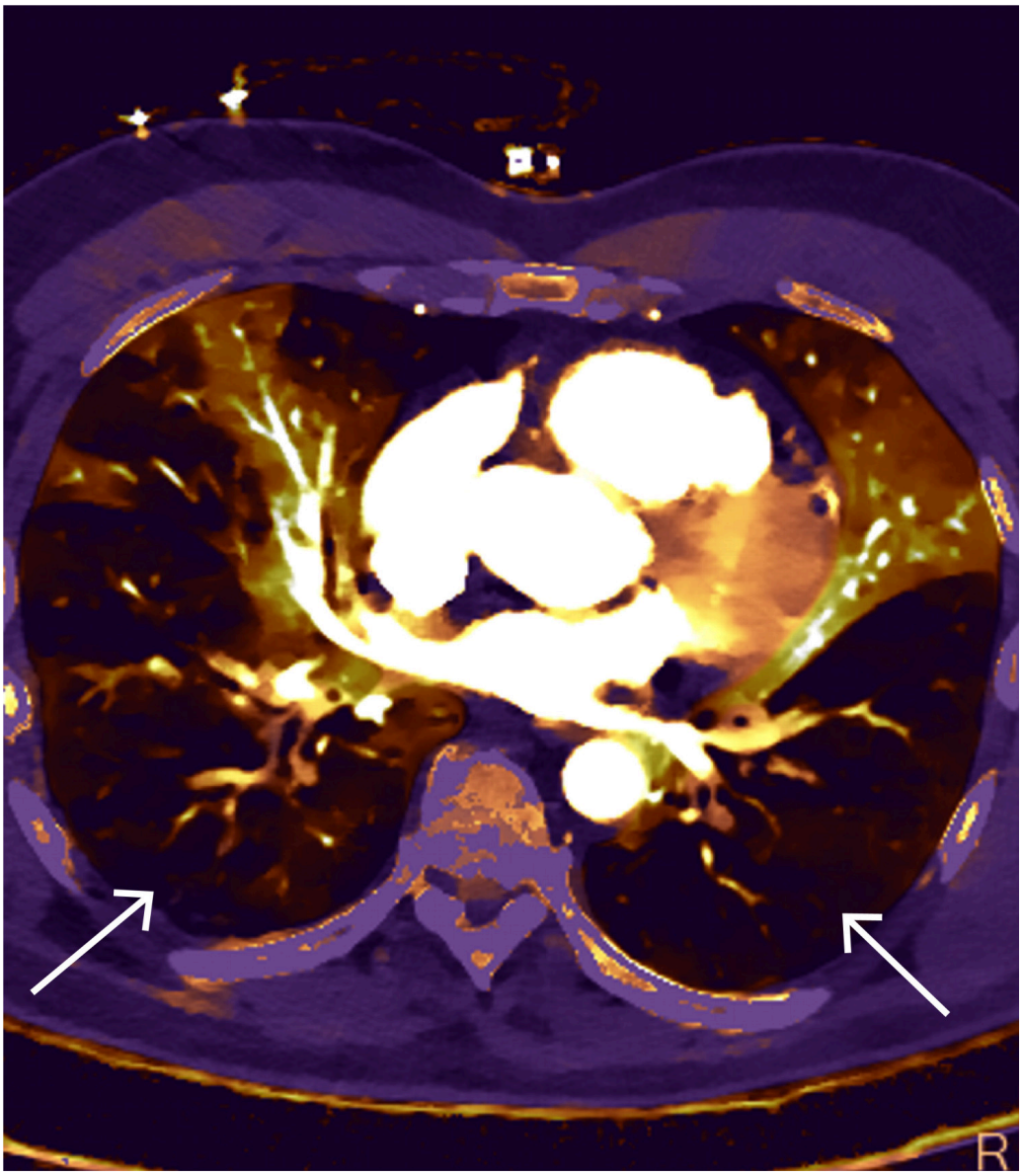

Figure 5 Spectral CT enables reconstructions showing iodine density. This gives a map showing both loss of and preserved perfusion. Here there were large areas with perfusion impact (arrows) in both lower lobes, the middle lobe and the right upper lobe. Only the left upper lobe was spared.

An interventional radiologist and interventional radiographer are available 24 hours per day with a 30-minute response time. When the patient arrived in the CT department, the interventional team were already in place, so the images were reviewed immediately, whilst the patient was transported to the cardiac intensive care unit for further stabilisation and assessment of treatment effect.

In the cardiac intensive care unit, the patient had persistent dyspnoea, severe chest pain and mean arterial pressure (MAP) of approximately 45 mmHg despite the alteplase infusion, titration of noradrenaline up to 0.40 µg/kg/min and initiation of vasopressin at an infusion rate of 0.03 IU/min. Due to haemodynamic instability, repeated doses of dilute adrenaline were administered to prevent further decompensation.

Due to the lack of effect from systemic alteplase and considerable right ventricular failure, the team agreed to take the patient directly to the operating theatre for urgent catheter-based thrombectomy. The patient was not considered haemodynamically stable enough for transport to a hospital with

thoracic surgery availability and access to extracorporeal membrane oxygenation (ECMO). It was 1 hour and 30 minutes in total since the patient's arrival in the emergency department before he was taken to the operating theatre.

Under local anaesthetic, ultrasound-guided puncture of the common femoral artery was performed, and an angled pigtail catheter was advanced via the inferior vena cava through the right heart chambers to the pulmonary artery (Figure 6). Mean pulmonary artery pressure at the start of the procedure was 37–39 mmHg (<20 mmHg). The thrombectomy device used was the FlowTriever (Inari Medical). Thrombi were aspirated from the vasculature of both lungs using 24 Fr and 16 Fr catheters (Figure 7). In total, ten aspirations were performed.

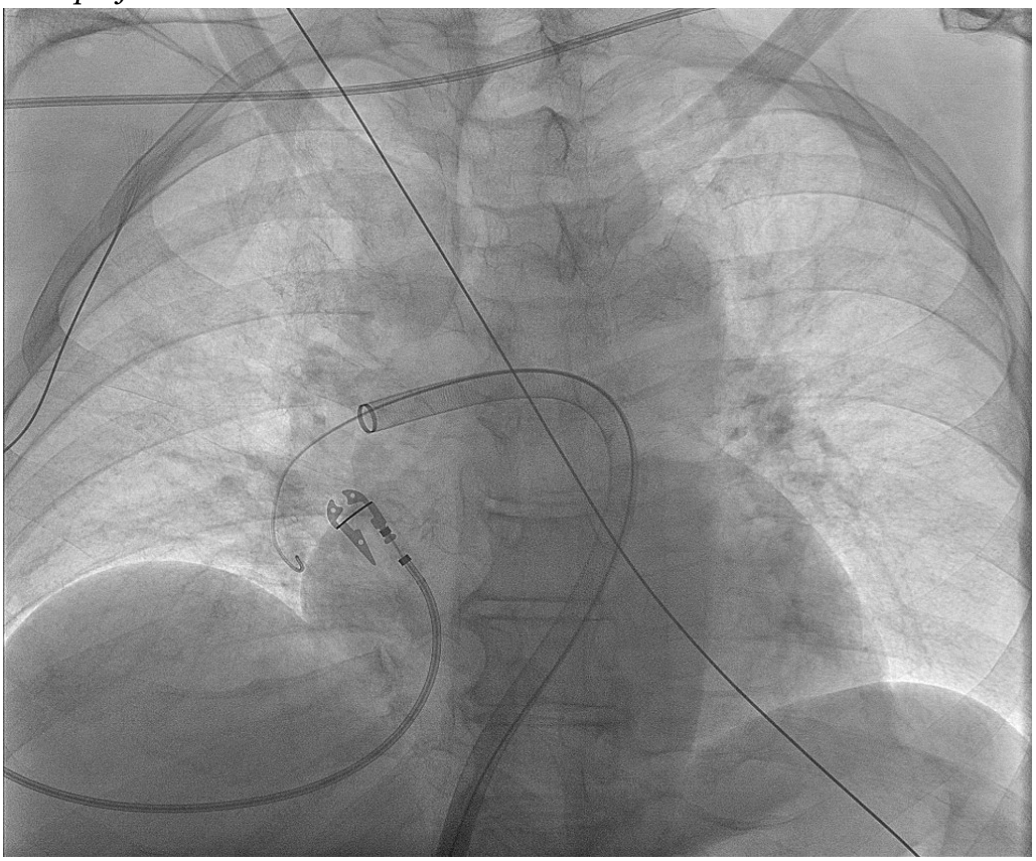

Figure 6 Fluoroscopy showing catheter placement in the pulmonary artery.

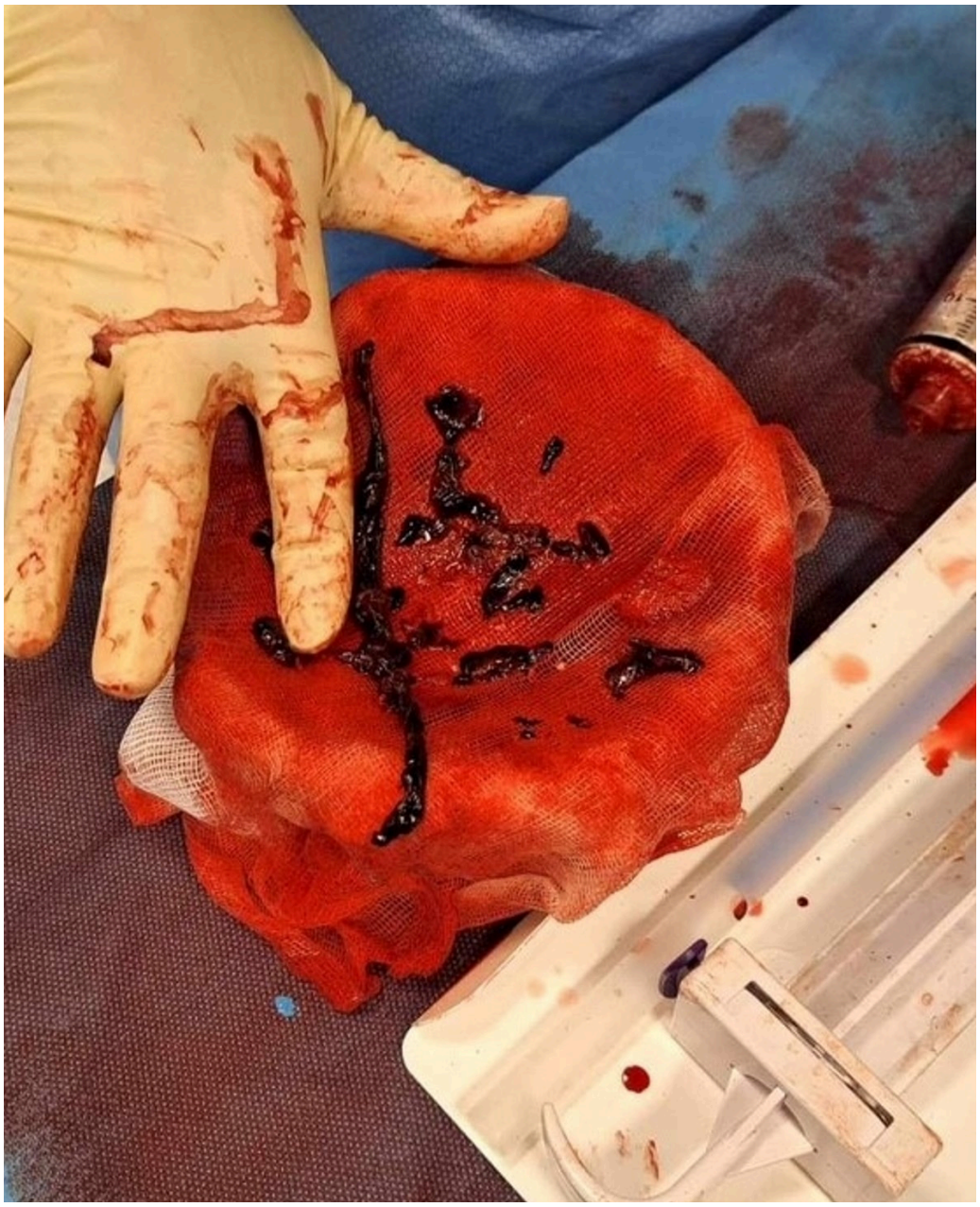

Figure 7 Aspirated thrombi.

Blood loss was estimated to be 50–80 ml in total. The patient's systemic pressure rose immediately following aspiration of the thrombi, and the vasopressor treatment could be tapered. Vasopressin was discontinued and the noradrenaline infusion reduced to 0.02 µg/kg/min. There was a rapid objective and subjective improvement in observed dyspnoea. At the end of the procedure, mean pulmonary artery pressure was 19–20 mmHg. Angiography demonstrated acceptable and rapid filling in the major segments of the lungs bilaterally.

Following the procedure, the patient was transferred back to the cardiac intensive care unit. A heparin infusion was started at 1320 IU/hour as further anticoagulant therapy. The infusion rate was guided by the activated partial thromboplastin time (APTT) (reference range 1.5 × baseline value). Clinically, the patient was not in pain at this time, and there was no objective or subjective dyspnoea. SpO_2 was 97 % on 1 litre of oxygen through nasal canula. His skin was dry and warm. His blood pressure rose to 111/62 mmHg as the vasopressor was tapered.

The results of laboratory tests taken on arrival were ready after the intervention, with findings of elevated troponin T at 284 ng/l (0–15) and NT-proBNP at 1571 ng/L (<210).
The following day, the patient had improved, and vasopressor treatment could be discontinued. The patient's anticoagulant therapy was switched to subcutaneous dalteparin (100 U/kg twice daily) since it was considered unlikely that thrombolysis with alteplase would be required again. Echocardiography follow-up the day after admission found normalised cardiac function and normal dimensions of both ventricles. Pulmonary artery systolic pressure was calculated to be 31 mmHg, which is considered normal. The patient was transferred to the haematology ward and discharged after a total of five days in hospital. He should continue oral anticoagulation with rivaroxaban 20 mg once daily for the first six months, followed by 10 mg once daily indefinitely.

Discussion

Right ventricular failure is a concerning complication of massive pulmonary embolism and is seen in 45 % of acute pulmonary embolism patients in varying degrees of severity (3). The mechanisms involved in the development of right ventricular failure in this situation are complex. Central pulmonary emboli that occlude large parts of the pulmonary vasculature mean that the right ventricle must pump against high pulmonary vascular resistance (increased right ventricular afterload). The right ventricle is thin walled and not constructed to withstand acute increases in afterload. When this happens, it leads to reduced right ventricular function and compensatory dilation (Figure 8). Secondary to this, the interventricular septum is pushed toward the left ventricle, causing reduced diastolic filling. This results in reduced cardiac output and coronary hypoperfusion, with secondary ischaemia and further biventricular failure. The ischaemia is exacerbated by hypoxia and the increased oxygen demand of the right ventricle. At the same time, right ventricular dilation causes stretching of the annulus of the tricuspid valve, with secondary tricuspid regurgitation, which also contributes to reduced left ventricular filling. All of this can lead to circulatory decompensation in the form of obstructive shock, and in the worst case to cardiac arrest (2, 3).

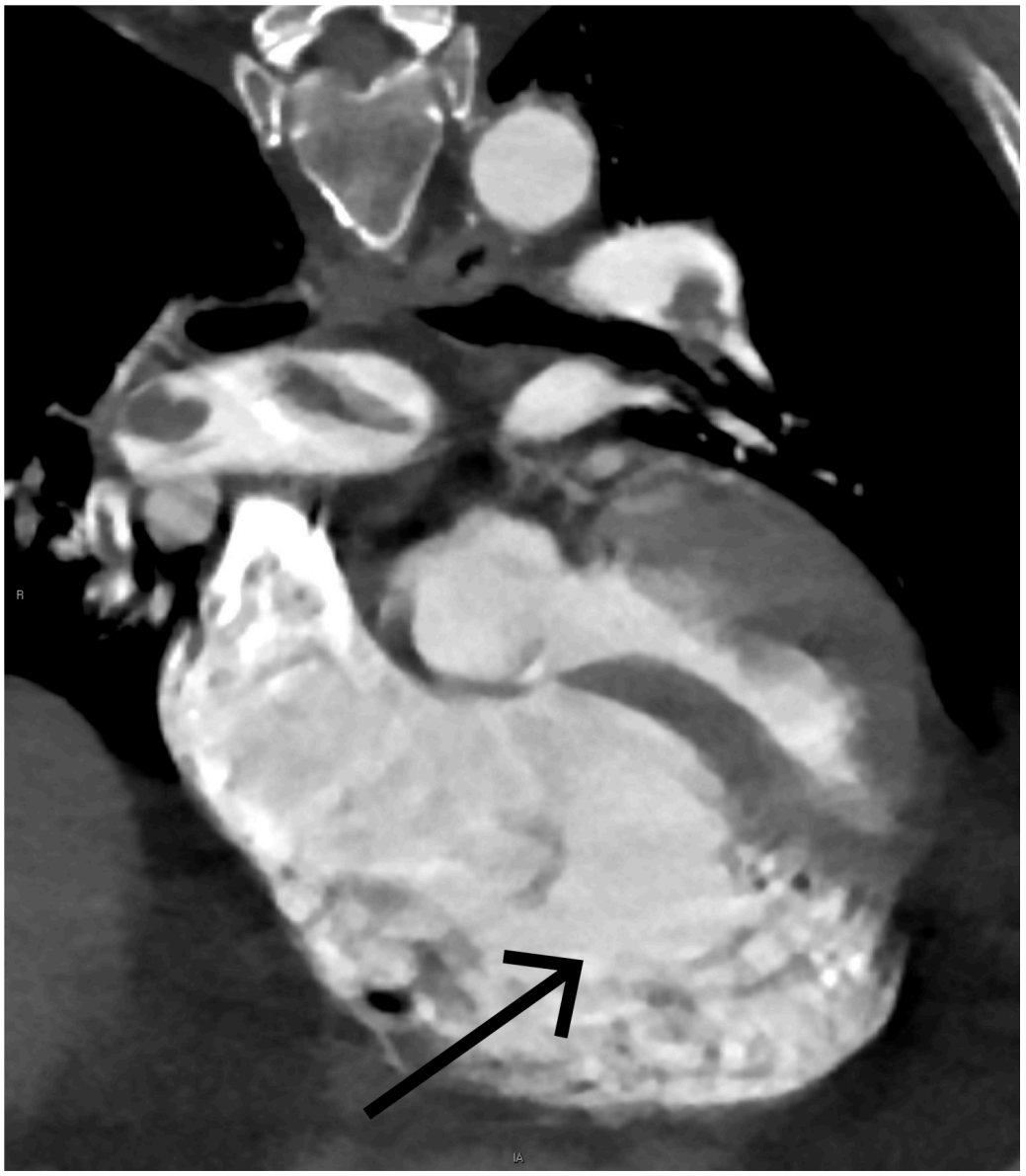

Figure 8 Dilated right heart chambers shown on CT angiography.

Patients with acute pulmonary embolism are stratified by risk into one of four groups based on their risk of short-term mortality. These groups are high risk, intermediate-high risk, intermediate-low risk and low risk. Classification takes place based on the development of haemodynamic instability, cardiac right ventricular function, troponin levels and the Pulmonary Embolism Severity Index (PESI) score (3). Short-term survival associated with pulmonary embolism varies widely, with a 30-day mortality ranging from 0 % in the low-risk group up to 24.5 % in the high-risk group (2).

Acute treatment of pulmonary embolism is selected based on risk stratification and ranges from treatment with direct oral anticoagulants (DOACs) to systemic thrombolysis, endovascular or surgical thrombectomy, support with extracorporeal membrane oxygenation (ECMO) and cardiopulmonary resuscitation (2). The endovascular treatment options are relatively new as the device has only been on the market for a short time. Most guidelines, including the 2019 European Society of Cardiology (ESC) guidelines on pulmonary embolism (2), still recommend primarily systemic thrombolysis in high-risk patients and predominantly low-molecular-weight heparin in patients with

intermediate-high risk. At our hospital, there is scope to consider systemic thrombolysis for the latter patient group in cases with substantial right ventricular dysfunction and compensatory tachycardia.

These ESC guidelines state that percutaneous catheter-based treatment, including catheter-based thrombolysis or thrombectomy, can be considered in high-risk patients in whom thrombolysis has failed or is contraindicated (2). At our hospital, ten catheter-based thrombolysis procedures and ten endovascular thrombectomy procedures have been performed respectively since being started in 2023. The recommendations for endovascular approaches are limited by several factors. The main reason is a lack of safety data and comparative studies with drug treatment, as well as the fact that treatment provision is highly centre- and operator-dependent. Eventually, as the device becomes more widely available and tried and tested, it is likely that more data in this area will be produced, and recommendations may potentially be changed (4). For example, a multicentre, prospective registry study (FLASH) looked at 1000 patients with high and intermediate risk of pulmonary embolism treated with endovascular thrombectomy. The results are promising and demonstrate considerable improvement in right ventricular function and reduced need for supplemental oxygen in the first 48 hours following the procedure. There were no procedure-related deaths (5). Furthermore, a small prospective study from Germany found no serious complications following treatment with thrombectomy with the FlowTriever system in patients with acute pulmonary embolism and diagnosed right ventricular failure (6).

At our hospital, a Pulmonary Embolism Response Team (PERT) has been established for the rapid multidisciplinary discussion of treatment options for pulmonary embolism patients in intermediate-high and high risk groups. This is in line with the recommendations in the 2019 ESC guidelines on pulmonary embolism (2). Our team consists of a haematologist, cardiologist in the echocardiography division (cardiology specialty registrar at night) and an interventional radiologist.

Chronic thromboembolic pulmonary hypertension is a potentially serious long-term complication of pulmonary embolism. The diagnosis can only be made after three months of appropriate anticoagulant therapy (7). Symptoms are persistent dyspnoea, which is particularly noticeable on physical activity, and fatigue. Chronic thromboembolic pulmonary hypertension occurs secondary to right ventricular failure due to chronic changes in the pulmonary vasculature with secondary right ventricular hypertrophy and dilation. One of several risk factors for the development of pulmonary hypertension following acute pulmonary embolism is the thrombus or thrombi in the acute phase being large or located centrally (7).

The risk stratification of the patient in this case report was without doubt as a high-risk patient. There was clear consensus on initial treatment with systemic alteplase in accordance with applicable national and international guidelines since there were no contraindications (2). The interventional team responded to a suspected central pulmonary embolism and possible need for catheter-based intervention because experience has found that alteplase may be less effective in long and obstructive thrombi.

Patient's perspective

In the emergency department, I was overwhelmed by how everyone stood ready, organised and drilled to the smallest detail. It was well-organised chaos. I felt I was in the safest hands in the world. After the procedures, I was visited by empathetic and friendly doctors who gave explanations in words I understood and listened to. Tears were shed, it was overwhelming. I know that I could have died on the operating table several times.

I don't know how I can thank you enough.

The decision to perform either surgical or endovascular thrombectomy in a high-risk patient, such as our patient, is more straightforward than in other pulmonary embolism patients since recommendations are given in the guidelines. There is great interest in identifying which patients in the intermediate-high risk group would also benefit more from these treatment options than from conventional drug treatment, both in the short term (survival), but also in the long term in terms of cardiac function and risk of developing chronic thromboembolic pulmonary hypertension. There are currently several large studies ongoing to identify these patients, including the PE-TRACT and HI-PEITHO studies (8, 9).

The patient has consented to the publication of this article.

The article has been peer-reviewed.

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