

Pulmonary oedema in hypertensive crisis – from failed femoral cannulation to diagnosis

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INTRODUCTION

Pulmonary oedema is a very common clinical presentation in the hospital setting with the management steps memorised by most medical students from an early stage. This management works on the basis that the patient is fluid overloaded from left ventricular systolic dysfunction (LVSD). In reality however, this is not always the case, with diastolic dysfunction also causing pulmonary oedema. In the case of diastolic dysfunction there is little data to guide management.¹ We present a case of a patient who developed flash pulmonary oedema secondary to a hypertensive crisis. Complications during cannulation of femoral vessels prompted a diagnosis of mid aortic syndrome.

CASE PRESENTATION

This 44-year-old man (Heavy Smoker) presented to the emergency department in 2015 with acute shortness of breath (SOB), chest pain and a cough. He explained that he had experienced a productive cough for 2 weeks prior to admission. On the day of admission he developed acute severe SOB, heart palpitations and sharp central chest pain and tightness. The patient had taken Gaviscon suspecting reflux as the cause, but found no relief from his symptoms. He reported feeling sweaty and clammy.

- He had a past medical history of cerebrovascular accident (CVA) in 2012, which left him with some residual left-sided weakness. He also had chronic obstructive pulmonary disease (COPD) and hypertension with poor compliance to medications resulting in multiple admissions with a systolic blood pressure (BP) of more than 200. There was no relevant family history of note.

On admission, the patient's BP was 198/116, HR 93 (regular), RR 18, SpO₂ 96% (room air) GCS 15/15. Cardiovascular and gastrointestinal examinations were unremarkable and there was no evidence of pedal oedema.

Investigations revealed – creatinine 112, urea 7.5, electrolytes normal. Chest x-ray was unremarkable and infective markers were moderately raised. ECG showed T-wave inversion in the lateral leads however troponin I was <0.04 with the second at 12 hours 0.06.

At this point, the working diagnoses were lateral non-ST elevation myocardial infarction, lower respiratory tract infection/exacerbation of COPD and hypertension. He was started on antibiotics, treated for acute coronary syndrome (ACS) with dual antiplatelet and fondaparinux and a referral was sent to the cardiologists.

Some 11 hours later he was reviewed in the early hour of the morning by the medical registrar on call. The patient had deteriorated significantly with worsening SOB, wheeze and productive cough of white frothy sputum. Arterial blood gas showed type 1 respiratory failure without acidosis with a pO₂ of 5.72 (35% FiO₂).

Respiratory examination revealed bilateral crepitations and expiratory wheeze. He was treated for an acute exacerbation of COPD with IV steroids and nebulisers added to his medications. The following day he was reviewed by the cardiology team whose differential diagnoses were uncontrollable hypertension and acute exacerbation of COPD. ACS treatment was stopped and amlodipine was added to his medications.

That night in the early hours of the morning the patient deteriorated once more with acute SOB and severe hypertension. He was treated for flash pulmonary oedema with furosemide and a glyceryl trinitrate infusion but did not respond so was transferred to the ICU. The initial plan was to start loading fluid with the use of a heamofilter so cannulation of the femoral vessels was attempted. Cannulation of the right femoral vein failed (very small vessels visualised with US aid with same problem on the contralateral side) and this was equally difficult but seemed successful on the left. Once in a vessel, the wire stopped advancing at some 20-30 cm but this was deemed enough to dilate and insert a catheter. In view of the difficulty in advancing the wire, a pressure transducer was connected to measure the direct blood pressure within that vessel. This pressure came back as 60/20 mmHg, with peripheral blood pressure reading: 210/115mmHg.

At this stage an ECHO was performed by the on-call consultant and this revealed a poorly performing left heart but no signs of fluid overload – although it was not possible at this stage to visualise inferior vena cava both internal jugular veins were not distended but were collapsible during inspiration. Only then was it realised that the cause of pulmonary oedema was a hypertensive crisis with possible aortic stenosis and infusion of labetalol was started and continued for 24 hours. The patient's clinical condition improved with the infusion though unfortunately he subsequently developed an acute kidney injury 2 days after. Hemofiltration was attempted but failed due patient agitation and line position issues. Fortunately his renal function started to improve spontaneously. Several weeks later, once stable and with a renal function that was amenable to contrast, the patient underwent a CT angiogram of the aorta. This showed significant atherosclerosis within the upper aorta leading to occlusion just inferior to the renal arteries. There was 2-vessel supply to the right kidney and extremely poor supply to the left kidney, which was shrunken and demonstrated perinephric stranding.

Four months later the patient was admitted once more with community-acquired pneumonia and as an inpatient he went on to develop a hypertensive crisis and pulmonary oedema requiring forced diuresis. An MRI angiogram revealed a complete occlusion of the aorta at the level of the renal arteries with collateral formation (suggestive of chronicity). There was a 1cm length of severe stenosis in the proximal part of the right renal artery and the left renal artery was not visualised making high-grade stenosis

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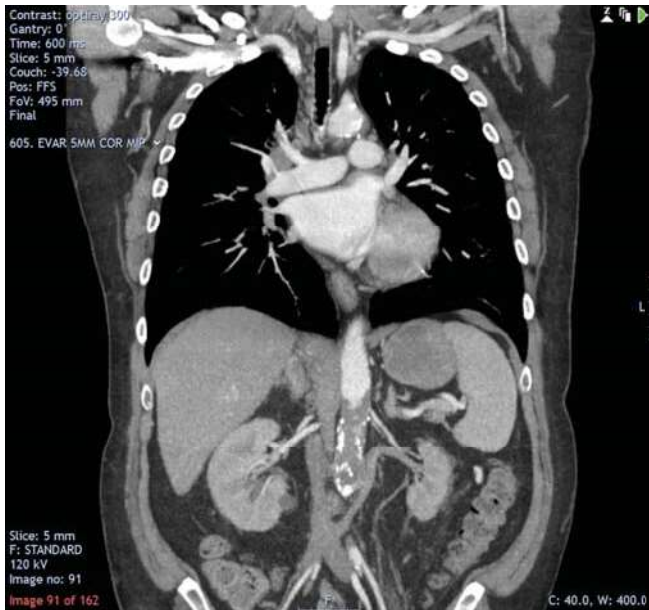


Figure 1: Coronal view of CT aortic angiogram showing severe occlusion of the aorta.



Figure 2: 3D reconstruction of CT aortic angiogram showing severe occlusion at level of the renal arteries.

probable. The left kidney was contracted. Urgent right renal artery stenting and left renal artery angioplasty were performed with good results. Post-operatively the patient's hypertension was better controlled and his renal function improved.

On review of the patient's history it was noted that he had a long history of chronic pain in his lower back, and both legs (right more than left) going to his ankles. Particular points of pain were his hip and the posterior aspect of both knees, which worsened when walking or sitting down on a hard seat for long periods. He had Perthes disease to his right femur requiring a total hip replacement at the age of 21 due to unmanageable pain. His symptoms after this operation were much the same.

The pain physicians managed him for some time with little change to his symptoms, which were having a big impact of his quality of life. A CT scan of his spine revealed a disc prolapse at the level of L5/S1 so he underwent a laminectomy. Again post-op his symptoms were not much improved. He used 2 walking sticks for mobility even after the recovery periods were complete.

On review of his blood pressure it is clear that it was markedly elevated from an early age – 120/70 at the age of 13 and 150/90 at the age of 23.



Figure 3: MR aortic angiogram showing severe occlusion of the aorta at the level of the renal arteries.

DISCUSSION

Middle aortic syndrome (MAS) describes cases of narrowing of the descending and abdominal aorta causing clinical signs and symptoms.² It is likely congenital and is most common in children and young adults.^{2,3} MAS most commonly presents as uncontrolled hypertension and has a proclivity to affect both visceral and renal arteries.⁴ It has been shown to be highly treatable with the hypertension often curable with endovascular or surgical intervention.^{5,6}

Left ventricular systolic dysfunction (LVSD) is not always the cause for pulmonary oedema. In fact, one study designed to support the theory that LVSD is usually the cause of acute pulmonary oedema, even in patients that had a preserved ejection fraction (EF) after the acute episode, suggested that diastolic dysfunction appears to be a cause of pulmonary oedema in a large proportion of patients.⁷ A further study found that as much as 40% of elderly patients who developed pulmonary oedema had an EF greater than 50%.⁸ Vasoconstriction can exacerbate pulmonary oedema by increasing preload. The mechanism for this vasoconstriction appears to be due to angiotensin II action.⁹ Interestingly, more than 85% of patient with acute pulmonary oedema have systolic hypertension.¹⁰ The mechanisms by which pulmonary oedema arises are explored below.

There are three main physiological mechanisms that contribute to flash pulmonary oedema (FPO).¹¹ These are:

1. Defective natriuresis
2. Increased haemodynamic burden and exacerbation of diastolic dysfunction
3. Failure of the pulmonary capillary blood flow.

Defective natriuresis

Studies show differences in pathophysiology between unilateral and bilateral renal artery stenosis (RAS) causing flash pulmonary oedema. The main difference is that in unilateral RAS is that the functioning kidney can compensate for the incorrectly activated renin-angiotensin system that causes increased BP with sodium retention, by so-called pressure natriuresis. In this circumstance, the unaffected kidney can suppress renin and increase sodium excretion. In bilateral RAS this is unable to occur, thus increased intravascular volume and sodium retention persists.¹²

Left ventricular hypertrophy and diastolic dysfunction

Sustained BP elevation in patients with RAS can cause left ventricular hypertrophy and diastolic dysfunction. Associated arterial stiffening increases pulse wave velocity so that the reflected pulse wave returns during systole instead of during diastole^{13, 14} thereby further augmenting systolic ventricular pressure load.¹⁵ Stiffening of the left ventricle wall causes increased end-diastolic pressures as well as higher left atrial and pulmonary venous pressures.¹⁶ Tachycardia can exaggerate this elevation in pressure leading to flash pulmonary oedema (FPO). If the left ventricle wall is stiff there is less time available to refill during diastole further increasing retrograde vascular pressures.¹⁷

Failure of pulmonary capillary blood flow

The third mechanism by which FPO is caused is due to leaky capillary endothelium from high pressure and stress. In normal conditions, fluid that escapes the alveolar capillaries is unable to enter the alveolar space due to the impermeable alveolar epithelial barrier. Instead, it reabsorbs throughout the lymphatic system.¹¹ In acute heart failure, the stiff left ventricle causes high pressure to back up through the cardiovascular system to the alveolar capillaries. Once the intracapillary pressure exceeds 20–25 mmHg, fluid leaks through the endothelium into the alveolar space causing the ventilation perfusion mismatch seen in FPO.¹⁸ Other neurohumoral mediators may also affect the permeability of alveolar capillaries.¹⁸

Sympathetic crashing acute pulmonary oedema (SCAPE) is likely to have occurred in the above case. It differs slightly to acute heart failure in that it is a more rapid onset of pulmonary oedema and describes a more severe clinical picture requiring fast and effective management for optimal patient outcomes.¹⁹ It develops in three distinct steps: distension of pulmonary capillaries due to high left atrial pressures, interstitial oedema and fluid escaping into the alveolar airway,¹⁹ thereby hindering

gaseous exchange.²⁰ Similar to that described above, the high alveolar capillary pressures cause stress failure and increase permeability.²¹ An increase in sympathetic tone causes the release of catecholamines, which increase heart rate, reducing diastolic time and exacerbating left sided cardiac pressures.²² Activation of the renin-angiotensin system may aggravate diastolic pressures increasing alveolar fluid overload.²⁰ Recommendations for management rely on non-invasive ventilation and nitrate infusions.²⁰ The authors suggest there is little evidence for the use of furosemide and what exists suggests it may even cause harm.²⁰

CONCLUSION

Mid aortic syndrome (with or without involvement of renal arteries) may present as a crash pulmonary oedema with hypertensive crisis. Given the quantity of evidence suggesting that diastolic dysfunction plays a large role in acute pulmonary oedema, it is surprising that the current method of management is still based on the theory of fluid overload and LVSD. Getting the hypertensive crisis under control is essential for a good clinical outcome²³ and is often achieved with nitrate infusions. However, in patients with renal artery stenosis this can severely affect renal perfusion with the potential to cause severe harm. Current guidelines for CT diagnosis of acute aortic syndrome do not include middle aortic syndrome – which sometimes, as in our patient, may be diagnosed accidentally by a non-advancing wire.

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This article has been re-published with the medical photographs.

Editor

A “Catonite” with nine lives

Terry Ainsworth, Football Historian & Hospital Volunteer

As I am a new patient representative, I think I should introduce myself with a little background information. I was born in Caton and the Royal Lancaster Infirmary (RLI) has always been MY hospital and link to the NHS. A cat supposedly has nine lives and this “Catonite” certainly has three of his, thanks to the RLI. When I was about six years old, I was with my parents on the platform at Morecambe Promenade railway station (now the Platform venue) when I fainted, fell on to the track, broke an arm and fractured my skull. Treatment at the RLI followed and after a worrying time, they saved my life. Fast-forward to my early thirties when I snapped an Achilles tendon playing squash and had an operation. Whilst, I was nearing full fitness again, I started to struggle to get my breath. My doctor rushed me into the RLI where they diagnosed a pulmonary embolism, two blood clots having gone to my lungs. The RLI once more saved my life. Many years later, 2017-18 to be precise, I fainted twice over a period of 15 months and ended up in Accident and Emergency (A & E). For some time, I had been experiencing severe stomach pain but after many tests during my first A & E visit, they found no cause. The second visit in September 2018 solved the problem. I remember the doctor coming into my cubicle in A & E with a big smile on his face saying:

“We think we have solved the problem and a surgeon is on her way to see you.” The surgeon came to see me and said that I had a Spigelian Hernia. She said she would put me on her list for an operation as soon as she had carried out more tests because she was worried that when I fainted, I was unconscious for 7 or 8 minutes. Many tests were carried out, all positive, and the surgeon asked her senior colleague to assist in the operation, which took place on November 16th, 2018. Those two surgeons were my guardian angels, as well as all the staff who assisted them. This experience has stayed with me and I resolved to put something back into the NHS by becoming a volunteer in the Charity Office (more on that in future editions).

For twenty years, I have written articles for the Lancaster Guardian primarily about local football and its history. In those years, I have also written 13 books on the same subject although my last two books have been about the first 100 years of football in England from 1872-1972. In football parlance, I suppose I am making my debut in the journal and I hope that I can produce the occasional article for Shadaba to publish.

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