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

Robbie Carrington, BMBS, Peninsula College of Medicine and Dentistry
Andrzej Szymczakowski, MA, Institution Akademia Medyczna, Warsaw, Poland

ABSTRACT

Pulmonary oedema is a very common clinical presentation in the hospital setting with the management steps memorised by most medical student 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 (FPO) secondary to a hypertensive crisis.

KEYWORDS

Critical care, hypertensive crisis, diastolic dysfunction, central venous catheterisation, pulmonary oedema.

INTRODUCTION

Pulmonary oedema is a very common clinical presentation in the hospital setting with the management steps memorised by most medical student 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.[2] We present a case of a patient who developed flash pulmonary oedema secondary to a hypertensive crisis. Complication during cannulation of femoral vessels prompted a diagnosis of mid aortic syndrome.

CASE PRESENTATION

This 44-year-old man 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 two 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 of his symptoms. He reported feeling sweaty and clammy. He was a heavy smoker with an 80-pack year history.

He had a past medical history of cerebrovascular accident (CVA) in 2012, which had left him with some residual left-sided weakness. He 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 examination on admission, the patient's BP was 198/116, HR 93 (regular), RR 18, SpO2 96% (RA) GCS 15/15. Cardiovascular and gastrointestinal examinations were unremarkable; 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 NSTEMI, LRTI/exacerbation of COPD and hypertension. He was started on antibiotics, treated for acute coronary syndrome (ACS) with dual antiplatelet and fondiparinux and a referral was sent to the cardiology doctors.

Some 11 hours later he was reviewed in the early hours of the morning by the medical registrar on call. The patient had deteriorated significantly with worsening SOB, wheeze and productive cough for white frothy sputum. ABG 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 GTN infusion, but was not responding and so was transferred to the intensive care unit. The initial plan was to start off loading fluid with use of heamofilter therefore cannulation of femoral vessels was attempted. Cannulation of the right femoral vein failed (very small vessels visualised with ultrasound 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 to advance at some 20-30 cm but this was deemed enough to dilate and insert a catheter. In view of the described difficulty with 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 at that time reading: 210/115mmHg.

At this stage an ECHO was performed by the on call consultant and this revealed 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 it was 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; unfortunately he subsequently developed an acute kidney infection 2 days after. Hemofiltration was attempted but failed due to 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 aortosclerosis 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 a community acquired pneumonia. Whilst as an inpatient he developed 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 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 his history it was noted that the patient had a long history of chronic pain in his lower back, and both legs (R>L) 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 of time. 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.

DISCUSSION

Middle aortic syndrome (MAS) describes cases of narrowing of the descending and abdominal aorta causing clinical signs and symptoms.[3] It is likely congenital and is most common in children and young adults.[2, 4] MAS most commonly presents as uncontrolled hypertension and has a proclivity to affect both visceral and renal arteries.[5] It has been shown to be highly treatable with the hypertension often curable with endovascular or surgical intervention.[6, 7]

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.[8] A further study found that as much as 40% of elderly patients who developed pulmonary oedema had an EF greater than 50%.[9] Vasoconstriction can exacerbate pulmonary oedema by increasing preload. The mechanism for this vasoconstriction appears to be due to angiotensin II action.[10] Interestingly, more than 85% of patient with acute pulmonary oedema have systolic hypertension.[11] The mechanisms by which pulmonary oedema arises are explored below.

There are three main physiological mechanisms that contribute to flash pulmonary oedema.[12] 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 supress renin and increase sodium excretion. In bilateral RAS this is unable to occur, thus increased intravascular volume and sodium retention persists.[13]

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[14, 15] thereby further augmenting systolic ventricular pressure load.[16] Stiffening of the left ventricular (LV) wall causes increase end-diastolic pressures as well as higher left atrial and pulmonary venous pressures.[17] Tachycardia can exaggerate this elevation in pressure leading to FPO. If the LV wall is stiff there is less time available to refill during diastole further increasing retrograde vascular pressures.[18]

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 capilliaries is unable to enter the alveolar space due to the impermeable alveolar epithelial barrier. Instead it is reabsorbed throught the lymphatic system.[11] In acute heart failure the stiff LV 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.[19]

Sympathetic crashing acute pulmonary oedema (SCAPE) is likely to be what 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.[20] 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.[21] Similar to that described above the high alveolar capillary pressures cause stress failure and increase permeability.[22] An increase in sympathetic tone causes release of catecholamines, which increase heart rate therefore reducing diastolic time exacerbating left sided cardiac pressures.[23] 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 furosemide has little evidence and 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 acute pulmonary oedema, it is surprising that the current method of management remains based on the theory of fluid overload and LVSD. Getting the hypertensive crisis under control is essential for a good clinical outcome[24] 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 may be diagnosed accidentally by a non-advancing wire, as in our patient.

REFERENCES

1. Eagle KA, Quertermous T, Singer DE, Mulley AG, Reder VA, Boucher CA et al. Left ventricular ejection fraction: physician estimates compared with gated blood pool scan measurements. Arch Int Med 1988;148: 882–5.
2. Eagle KA, Quertermous T, Singer DE, Mulley AG, Reder VA, Boucher CA et al. Left ventricular ejection fraction: physician estimates compared with gated blood pool scan measurements. Arch Int Med 1988;148: 882–5.
3. Delis KT, Gloviczki P. Midlde aortic syndrome: from presentation to contemporary open surgical and endovascular treatment. Perspect Vasc Surg Endovasc Ther 2005;17:187–203.
4. Connolly JE, Wilson SE, Lawrence PL, Fujitani RM. Middle aortic syndrome: distal thoracic and abdominal coarctation, a disorder with multiple etiologies. J Am Coll Surg 2002;194: 774–81.
5. Chitrakar A, Shrestha KR, Shrestha UK. Middle aortic syndrome with renal artery stenosis. J Surg Case Rep. 2017 Sep 29;2017(9):rjx177. doi: 10.1093/jscr/rjx177.
6. Messina LM, Reilly LM, Goldstone J, Ehrenfeld WK, Ferrell LD, Stoney RJ. Middle aortic syndrome. Effectiveness and durability of complex arterial revascularization techniques. Ann Surg 1986;204:331–9.
7. Taketani T, Miyata T, Morota T, Takamoto S. Surgical treatment of atypical aortic coarctation complicating Takayasu's arteritis: experience with 33 cases over 44 years. J Vasc Surg 2005;41:597–601.
8. Little WC. Hypertensive pulmonary oedema is due to diastolic dysfunction. European Heart Journal 2001;22:1961-1964.
9. Gandhi SK, Powers JC, Nomeir AM, Fowle K, Kitzman DW, Rankin KM et al. The pathogenesis of acute pulmonary edema associated with hypertension. N Engl J Med 2001; 344:17-22.
10. Heitzer T, Just H, Brockhoff C, Meinertz T, Olschewski M, Münzel T. Long-term nitroglycerin treatment is associated with supersensitivity to vasoconstrictors in men with stable coronary artery disease: prevention by concomitant treatment with captopril. J Am Coll Cardiol. 1998;31:83e88.
11. Kramer K, Kirkman P, Kitzman D, Little WC. Flash pulmonary edema: association with hypertension and reoccurrence despite coronary revascularization. Am Heart J 2000;140: 451–5.
12. Syammakh MH, Anthonius FR Hypertensive crisis induced pulmonary oedema in emergency care unit Sumbawa hospital: a case report, International Journal of Research in Medical Sciences 2018;6(12): 4122-4127.
13. Gavras H, Brunner HB, Vaughan ED, Laragh JH. Angiotensin-sodium interaction in blood pressure maintenance of renal hypertensive and normotensive rats. Science 1973;180:1369-1371.
14. Avolio AP, Deng FQ, Li WQ, Luo YF, Huang ZD, Xing LF et al. Effects of aging on arterial distensibility in populations with high and low prevalence of hypertension: comparison between urban and rural communities in China. Circulation 1985;71:202-210.
15. Laurent S. Arterial wall hypertrophy and stiffness in essential hypertensive patients. Hypertension 1995;26:355-362.
16. Roman MJ, Pickering TG, Schwartz JE, Pini R, Devereux RB. Relation of arterial structure and function to left ventricular geometric patterns in hypertensive adults. J Am Coll Cardiol 1996;28:51-756.
17. Grisk O, Rettig R. Interactions between the sympathetic nervous system and the kidneys in arterial hypertension. Cardiovasc Res 2004;61:238-246.
18. Messerli FH, Bangalore S, Makani H, Rimoldi SF, Allemann Y, White CJ et al. Flash pulmonary oedema and bilateral renal artery stenosis: the Pickering Syndrome. European Heart Journal 2001;32(18):2231–2235. Available from: https://doi.org/10.1093/eurheartj/ehr056 (accessed 30.6.19).
19. Rimoldi SF, Yuzefpolskaya M, Allemann Y, Messerli F. Flash pulmonary edema. Prog Cardiovasc Dis 2009;52:249-259.
20. Mebazaa A, Gheorghiade M, Piña IL, Harjola VP, Hollenberg SM, Follath F, et al. Practical recommendations for prehospital and early in-hospital management of patients presenting with acute heart failure syndromes. Crit Care Med 2008;36(1 Suppl):S129–39.
21. Agrawal N, Kumar A, Aggarwal, P Jamshed N. Sympathetic crashing acute pulmonary oedema. Indian J Crit Care Med 2016;20(12):719-723.
22. West JB, Mathieu-Costello O. Stress failure of pulmonary capillaries in the intensive care setting. Schweiz Med Wochenschr 1992;122:751–7.
23. Rimoldi SF, Yuzefpolskaya M, Allemann Y, Messerli F. Flash pulmonary edema. Prog Cardiovasc Dis 2009;52:249–59.
24. Hsieh Y et al. Treating acute hypertensive cardiogenic pulmonary edema with high- dose nitroglycerin. Turkish Journal of Emergency Medicine 2018;18(1):34-36. Available from: https://doi.org/10.1016/j.tjem.2018.01.004 (accessed 30.6.19).

Correspondence to:
Robbie.carrington@doctors.org.uk