CASE REPORT

Muddy clinical waters: a missed pulmonary embolus

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SUMMARY

The diagnosis of pulmonary embolism (PE) can be difficult to make. The consequences of missing a PE can be fatal and clinicians should always be vigilant. This case presents a patient admitted with fluctuations in consciousness and in a septic state. A preliminary diagnosis of aspiration pneumonia was made, and the patient went on to have an exploratory surgical procedure with no remarkable findings. Only after further investigation of the patient's fluctuations in consciousness was a retrospective diagnosis of a large PE made. The patient was anticoagulated and made a full recovery, with no outstanding pulmonary issues at follow-up 6 weeks after discharge. A prolonged immobile state, likely in part due to the patient's septic state and psychiatric history, along with recent surgery and hospitalisation were the most obvious risk factors.

BACKGROUND

The diagnosis of pulmonary embolism (PE) can be difficult to make—particularly as the symptoms and signs may vary and the presentation can be atypical. The impact of missing the diagnosis can be fatal.^{1 2} With the widespread use of CT the number of incidental PEs discovered has also increased, and on most occasions, in retrospect, the patient had non-specific symptoms such as fatigue or breathlessness.³ Clinicians must always be vigilant, both in the clinical assessment and review of investigations, in order to prevent PEs from going undiagnosed.

CASE PRESENTATION

A 55-year-old woman was admitted to the acute medical unit with a 3-day history of drowsiness, vomiting, confusion and clinical anorexia. A clear history was unable to be taken from the patient, who asked to have some coffee and to go home after denying feeling any pain. A collateral history from family members was unavailable at the time. She had been referred by her general practitioner, who was called to her home in sheltered accommodation after reports from the coordinator of her inability to cope. She was previously seen in A&E and in the acute medical unit for short admissions with multiple collapses, tachycardia, subjective fevers and dizziness. During her last admission a diagnosis of urinary sepsis was made, and she was discharged on a course of antibiotics when her clinical condition and inflammatory markers improved. Her only medical history of note is schizophrenia managed with citalopram, lorazepam PRN and clozapine.

During the day 1 admission date, the patient was found to be drowsy but rousable. She had fever at 38.5°C with an increased respiratory rate. She was

normotensive, but found to have a sinus tachycardia on an ECG tracing. On examination, she was clearly using her accessory muscles of inspiration but she did not have any notable additional breath sounds. There was no other remarkable examination finding. Her Glasgow Coma Scale (GCS) was 12/15 (6 motor; 3 verbal; 3 eyes) with no focal neurological deficits. A urine dipstick test was unremarkable for infection. An arterial blood gas showed that she had a hypoxic respiratory failure on 15 L non-rebreather mask; with a pH of 7.49, partial pressure oxygen 7.23 kPa, partial pressure of carbon dioxide 4.15 kPa, calculated lactate 1.9 mmol/L, calculated HCO₃⁻ 25 mmol/L and a calculated base excess 0.7 mmol/L. On admission she was found to have markedly raised C reactive protein (269 mg/L; normal range 0–10 mg/L), but a normal white blood cell count (5.5×10⁹/L; normal range 4–10×10⁹/L) and neutrophil count (4.1×10⁹/L; normal range 2–7×10⁹/L). In addition, she was found to have an acute kidney injury with an estimated glomerular filtration rate of 32 mL/min, creatinine level 155 µmol/L and urea 15.8 mmol/L. A chest radiograph was unremarkable (figure 1).

A diagnosis was made on the admission date of a sepsis due to a lower respiratory tract infection which was possibly secondary to an episode of aspiration. The alternative diagnosis was a non-resolved urinary sepsis. Although a PE was considered, a D-dimer was not performed as this was expected to be raised in a hyperinflammatory state and was excluded based on the presence of several more likely diagnoses. She was started on tazocin 4.5 g intravenously three times a day. The cause of the vomiting was unclear, and a surgical review was requested to exclude an intestinal obstruction.

Figure 1 Admission chest radiograph.

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INVESTIGATIONS

On review by the surgical team on day 2, further imaging was requested to exclude an intestinal obstruction. A CT scan of her abdomen without contrast was performed on the same day. The major findings were those of a moderate-to-severe distension of the jejunal and ileal loops. No obvious obstructive mass lesions, inflammation or evidence of perforation present. A diagnosis of small bowel obstruction was made.

TREATMENT

This patient was moved to the high dependency unit for continuous monitoring and a nasogastric tube was inserted. A diagnostic laparotomy performed on day 3 showed a dilated small intestine but no obvious cause of obstruction. Her surgery was uneventful. Following her surgical procedure, she was discharged from the high dependency unit back to the care of the medics on day 6. Later on in the day, she suddenly became unresponsive with a calculated GCS grade of 3. On urgent assessment, she was found to be haemodynamically stable, profusely sweating but feverish, with normal capillary blood glucose and unremarkable arterial blood gas results. ECG did not show any remarkable features. A CT scan of her head was ordered to look for any signs of an acute cerebrovascular event. This was unremarkable, but further review of previous scans by the consultant radiologist revealed a large, saddle emboli located in the pulmonary trunk (figure 2). The patient's unresponsive state spontaneously resolved without any acute intervention or persisting deficit.

OUTCOME AND FOLLOW-UP

The patient was started on low-molecular-weight heparin anticoagulation therapy after excluding neurological pathology, which was subsequently converted to coumarin therapy. She had also developed diarrhoea which was culture positive for *Campylobacter*, for which she continued her course of tazocin with oral clarithromycin 500 mg twice a day. On completion of antibiotics she was discharged back to her sheltered accommodation.

The patient was followed up in the respiratory clinic in the following 6 weeks, and the chest radiograph appearance showed no remarkable features (figure 3). The patient was then discharged from further follow-up.

Figure 2 Non-contrast CT scan at the level of carina. Large saddle embolus noted.

Figure 3 Six weeks postdischarge follow-up chest radiograph.

DISCUSSION

On first presentation the differential diagnosis on this patient appeared complex and this may have accounted for the delay in arriving to the final diagnosis. The septic state, difficulty in communication and question of bowel obstruction on admission complicated the differential.

Patients with a primary presentation of fluctuations in consciousness and collapse can have a wide range of diagnoses and the National Institute of Care Excellence (NICE) has issued extensive guidance on the assessment of fluctuation and temporary loss of consciousness.⁴ Narrowing these diagnoses down will be based mainly on the quality of the collateral history if the patient is unresponsive. Neurological causes are an important consideration, particularly cerebrovascular accidents and epileptic events. Cardiac causes such as arrhythmias or myocardial infarctions are also another important consideration. Other notable differentials can include hypoglycaemic states and drug overdoses. Fluctuations in consciousness and tachycardia due to septic states are a possibility, and in this case report, are supported by the raised inflammatory markers and suggestive urinalysis findings. The patient's fluctuations in consciousness were likely secondary to a septic state, supported by the absence of hypoperfusion or hypoxic pathological features.

Of course, a PE should always be considered in cases of dyspnoea, tachycardia and unremarkable chest radiograph findings. Factors particular to this case which, in retrospect, lend authority to the diagnosis of a PE are as follows: (1) extensive PE may present with syncope due to lowering of cardiac output and may present as symptomatic hypotension, evidence of poor perfusion (ie, confusion), and an elevated jugular venous pressure.¹ (2) The septic presentation in this patient may have been secondary to the pulmonary embolic event rather than a pure septic origin; this has been reported in some instances previously in the literature⁵ and is supported by the clinical findings of a hypoxic respiratory failure, tachycardic state and normal chest radiograph. This case report describes a clinical scenario of a PE diagnosed alongside a sepsis, with management for each started as appropriate as both conditions may have contributed to a deteriorated clinical state.

Pulmonary emboli represent a clinical condition that can be notoriously difficult to diagnose.^{3 6} NICE guidelines suggest the use of clinical probability scores and D-dimer testing as a first-line approach, followed by more definitive investigations such as ventilation perfusion scans and CT pulmonary angiograms.⁵ The treatment will depend largely on haemodynamic stability on presentation, symptoms at presentation and ultimately long-term anticoagulation. Unfortunately, there are still reported cases of

missed PE's, silent PE (largely asymptomatic) and PEs reported incidentally on imaging (ie, when not investigating for PE).^{2,7} This case report describes a clinical incident of PE that can be classified as 'missed' or 'incidental'. Incidental pulmonary emboli appear to carry a similar risk in recurrence and poor long-term prognosis as symptomatic PEs and should therefore be treated with anticoagulation such as was the case with this patient.¹

Interestingly, there have been reported cases in the literature linking psychiatric conditions and antipsychotics use with the incidence of PE.^{8–11} The aetiology of this link remains unknown; possible mechanisms include antipsychotic drugs enhancing platelet aggregation, cardiolipin bodies associated with increased risk of thrombosis, venous stasis exacerbated by sedation and antipsychotics promoting the secretion of epinephrine leading to a procoagulant state.^{8,10}

This case may represent another manifestation of this pattern, and the characteristic factors found in our case are immobility, a septic state, surgical intervention and hospitalisation.⁸

Contributors NL holds responsibility for authorship and planning of the case report. SRB provided critical revisions and specialist input, and managed the case clinically.

Competing interests None.

Patient consent Obtained.

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Learning points

- ▶ Maintain a high level of suspicion for pulmonary emboli in hypoxic patients with normal chest radiographs.
- ▶ It is not uncommon for sepsis and pulmonary emboli to present together, and should be managed accordingly.
- ▶ Consider pulmonary emboli in states of fluctuating consciousness and collapse.
- ▶ Systematic examination of imaging by the clinical team should accompany clinical reports.
- ▶ Clinicians should be aware of the risk of developing venous thromboembolic disease in patients with a psychiatric history or currently active psychiatric problems.

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