CASE REPORT

An unusual presentation of pulmonary embolism leading to infarction, cavitation, abscess formation and bronchopleural fistulation

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SUMMARY

We report an unusual presentation of pulmonary embolism (PE) where a 58-year-old man first developed symptoms of community-acquired pneumonia. Despite antibiotic therapy, he remained unwell with rising inflammatory markers, general malaise and persistent cough. He developed stony dull percussion and absent breath sounds to his left mid to lower zones. Serial chest x-rays showed progression from lobar consolidation to a large loculated left-sided pleural collection. CT chest showed left-sided lung abscess, empyema and bronchopleural fistulation. Incidentally, the scan revealed acute left-sided PE and its distribution corresponded with the location of the left lung abscess and empyema. The sequence of events likely started with PE leading to infarction, cavitation, abscess formation and bronchopleural fistulation. This patient was managed with a 6-month course of rivaroxaban. After completing 2 weeks of intravenous meropenem, he was converted to 4-week course of oral co-amoxiclav and metronidazole and attained full recovery.

BACKGROUND

The classic presentation of pulmonary embolism (PE) is a sudden onset of or worsened breathlessness and pleuritic chest pain with or without haemoptysis. It is not uncommon for PE to present with only some or occasionally none of these symptoms. We present here an unusual case of PE whereby the initial presentation mimicked that of a pneumonia, which transpired to have been a PE that caused pulmonary infarction, cavitation, abscess formation and subsequent bronchopleural fistula (BPF). This case highlights the importance of maintaining high clinical suspicion for alternative diagnosis when pneumonia does not resolve with appropriate choice and escalation of antibiotic therapy.

CASE PRESENTATION

A 58-year-old man presented to ED with 3-day history of fevers, sore throat, hoarse voice, cough productive of green sputum and loss of appetite while on a cruise ship. Medical history included obesity (body mass index (BMI) of 36.4), type 2 diabetes mellitus, hypertension, spinal disc prolapse, sciatica and irritable bowel syndrome. He was a current smoker with a 40-pack-year history. He had longstanding ulcers on his feet that limited his mobility. His medications at the time of

presentation were pregabalin, liraglutide, humulin M3, atenolol, bendroflumethiazide, tramadol, amitriptyline, mebeverine, atorvastatin, lansoprazole and naproxen.

Admission observations showed a temperature of 36.3°C, blood pressure of 136/84 mm Hg, heart rate of 75/min, respiratory rate 15/min and oxygen saturation 93% on air. On examination, there were crackles bibasally and polyphonic wheeze, and his calves were soft and non-tender. He was started on treatment for community-acquired pneumonia (CAP).

Despite continued antibiotic treatment, by day 6 of admission he appeared clinically unwell with symptoms of clamminess and sweatiness. He developed haemoptysis with blood-streaked sputum, and on auscultation, he had reduced air entry to the left lower zone and left basal crackles. In the next few days, he developed stony dull percussion notes to the left mid to lower zones as well as absent breath sounds in the same territory. The examination finding correlated well with progressive lung changes found on serial chest x-rays (CXRs). The clinical impression was of loculated empyema in the context of left lower lobe pneumonia, and he was scheduled for a video-assisted thoracic surgery (VATS) initially to wash out the empyema. However, CT chest with contrast visualised a moderate-sized PE and the VATS was cancelled. He received antibiotic treatment for the next 6 weeks. He improved clinically by the end of the hospital admission—he remained afebrile, with no further haemoptysis and reduced production of purulent sputum.

INVESTIGATIONS

Laboratory investigations on admission (table 1) showed elevated white cell count (WCC) of $14.8 \times 10^9/L$, C reactive protein (CRP) of 252 mg/L and low platelet count of $144 \times 10^9/L$. Urea and creatinine were normal, potassium was 3.0 mmol/L and liver function test showed raised bilirubin of 37 $\mu\text{mol/L}$, alkaline phosphatase of 185 U/L but normal alanine aminotransferase. Viral swabs tested negative to parainfluenza type 1, 2 and 3, RSV, metapneumovirus, adenovirus, influenza A and B, mycoplasma pneumoniae antibody and legionella urinary antigen.

CXR on day of admission showed peribronchial thickening and a 2 cm area of round and defined airspace shadowing in the left mid zone (figure 1).

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Table 1 Trend of laboratory results

	On admission	Day 5	Day 9	Day 15	Day 30
Hb (g/L)	158	146	137	128 (L)	130
WCC ($10^9/L$)	14.8 (H)	18.2 (H)	12.3 (H)	9.0	8.0
CRP (mg/L)	252 (H)	302 (H)	187 (H)	167 (H)	22 (H)
Albumin (g/L)	34 (L)	18 (L)	18 (L)	17 (L)	22 (L)
Bilirubin ($\mu\text{mol/L}$)	37 (H)	41 (H)	22 (H)	14	8
ALP (U/L)	185 (H)	175 (H)	235 (H)	251 (H)	155 (H)
ALT (U/L)	23	22	52 (H)	50 (H)	20
Sodium (mmol/L)	134	128 (L)	129 (L)	131 (L)	133
Potassium (mmol/L)	3.0 (L)	4.2	4.3	4.5	4.3
Urea ($\mu\text{mol/L}$)	5.1	9.7 (H)	5.2	3.4	3.4
Creatinine (mmol/L)	65 (L)	68 (L)	55 (L)	54 (L)	45 (L)
eGFR (mL/min/1.73 m ²)	>90	>90	>90	>90	>90

ALP, alkaline phosphatase; ALT, alanine aminotransferase; CRP, C reactive protein; eGFR, estimated glomerular filtration rate; Hb, haemoglobin; WCC, white cell count.

On day 3, there was evidence of consolidation with associated hilar lymphadenopathy. By day 8, there was clear sign of loculated pleural collection, consolidation and signs of empyema (figure 2). On day 11, a clear fluid level was superimposed on the signs of consolidation (figure 3). This reflected the presence of a bronchial fistula causing rapid build-up of air-fluid level, faster than would be expected for a pure bacterial infection. An ultrasound scan of the collection showed a rim of fluid around the lung at the left lower zone that was 1.5 cm in maximum depth. Aspiration of the fluid was attempted but unsuccessful.

A CT chest with contrast was done to further assess the empyema and possible lung abscess. The scan showed a large complex collection arising from the left lower lobe, which consisted of an empyema and a coexisting lung abscess (figure 4). There were also signs of BPF as hinted by previous CXR (figure 3). To our surprise, the scan revealed a large filling defect at the bifurcation of the left main pulmonary artery extending to the upper and lower lobe pulmonary arteries, consistent with acute PE (red arrow, figure 5). The distribution of the clot corresponded well with the location of the left lung abscess and empyema.

TREATMENT

Our patient was initially diagnosed with acute bronchitis with an element of bronchospasm. He was started on empirical antibiotics (doxycycline), prednisolone and salbutamol nebulisers on admission. On the second day of admission, he lacked wheeze on examination and the clinical impression was of a CAP without airway disease. Doxycycline was switched to clarithromycin and prednisolone was stopped in view of this modified diagnosis. His inflammatory markers increased further with WCC rising from $14.8 \times 10^9/L$ on day 1 to $17.0 \times 10^9/L$ on day 4, and CRP from 252 to 336 mg/L (table 1). He developed haemoptysis and became increasingly unwell and clammy. Clarithromycin was changed to moxifloxacin on day 4 as this patient had a history of penicillin allergy. The serial CXRs suggested development of empyema refractory to moxifloxacin. He was tested for meropenem allergy and found to be non-allergic, and he was started on meropenem.

When it was apparent that the patient was not clinically and radiologically improving with the escalated antibiotic treatment, we initially planned a VATS to wash out the empyema.

Figure 1 Day 1: CXR. CXR, chest x-ray; PA, posteroanterior.

Figure 2 Day 8: CXR illustrates a deterioration in the disease progression. AP, anteroposterior; CXR, chest x-ray.

Figure 3 Day 11: CXR shows an air fluid level in the left mid zone that measures 10 cm. The pleural collection in the left lateral chest wall remains static. CXR, chest x-ray.

However, as anticoagulation was started immediately on diagnosis of PE, this was a relative contraindication and VATS was cancelled.¹ Transthoracic echo showed the absence of right heart strain with normal left ventricular systolic function and preserved right ventricular function. Our patient was started on treatment dose enoxaparin and metronidazole was added to his meropenem therapy to cover anaerobic bacteria. Meanwhile he received allergy testing which revealed that he was not allergic to penicillin. After he completed a 2-week course of intravenous antibiotics, his inflammatory markers were on a clear downward trend and he was converted to a 4-week course of oral co-amoxiclav and metronidazole. A 6-month course of rivaroxaban was started for the single episode of PE.

Figure 4 Lung window shows left lower lobe complex collection that includes empyema and lung abscess.

Figure 5 CT chest with contrast shows left-sided pulmonary filling defect consistent with acute PE. PE, pulmonary embolism.

OUTCOME AND FOLLOW-UP

A month after admission, our patient was no longer coughing up purulent nor blood-stained sputum, his appetite returned and he felt well in himself. He was mobilising on the ward independently with little shortness of breath. On day 30, his WCC decreased to 8.0 and CRP was 22. Chest radiograph on day 30 (figure 6) showed resolving pulmonary consolidation and the air fluid level had disappeared. Given the clinical, biochemical and radiological improvement, he was discharged back into the community with oral antibiotics and rivaroxaban.

DISCUSSION

PE leads to pulmonary infarction in 10%–31% of cases. The likelihood of infarction occurring has been postulated to

Figure 6 Day 30: a dense linear band is seen across the left mid zone which likely represents the margins of the previous cavity. The air fluid level is no longer seen. The area of dense pulmonary consolidation is now smaller and resolving compared with the previous radiograph.

increase with increasing body height, active smoking, congestive heart failure and presence of collateral blood supply.^{2,3} Of those who develop infarction, 4%–7% go on to develop cavitation as evidenced by autopsy findings.² Cavitation may occur due to aseptic necrosis (also known as bland infarctions) or as a result of superinfection of the dead tissue. The finding of our CT scan suggested that the process in our patient began as an infarction of a segment of the lung secondary to the PE. In retrospect, the admission CXR (figure 1) provided the first hint to pulmonary infarction. The left lung shadowing was round and well demarcated unlike typical pneumonia, and the left hilar artery could not be traced distally in contrast to the right hilar artery. The infarction led to cavitation, abscess formation and later, bronchopleural fistulation. While VATS could theoretically have been employed to drain the abscess, anticoagulation was a contraindication and this was held off. The patient subsequently recovered well on medical management alone, suggesting surgical intervention was not required.

While infected cavitating pulmonary infarction has been documented in numerous case reports, BPF secondary to infection complicating infarction in the context of PE is a rare complication.^{4,5} BPF is a sinus tract that connects the bronchus and the pleura. It may be found in large infarctions extending to one or more lobes or infected cavitating pulmonary infarcts. It has been reported to occur 2–30 days (average 17 days) after infarction.^{2,5} Radiological changes on chest radiographs that suggest BPF include continuous increase in intrapleural airspace, new appearance of or changes in air-fluid level, new onset tension pneumothorax or a drop in the air fluid level of >2 cm in the absence of a chest tube.⁶ It is associated with 18%–67% mortality rate and can be seen with complications including acute respiratory distress syndrome and tension pneumothorax.⁶ Fortunately, follow-up CXR in our patient 2 weeks after his CT scan showed no sign of pneumothorax or lung collapse. The air fluid level previously seen in figure 3 had disappeared and the pulmonary consolidation was resolving.

BPF may be managed conservatively by postural drainage and antibiotics to treat any underlying infection, bronchoscopically or surgical closure of the fistula.⁶ Ricci *et al* reported that of the patients who had BPF visible on CT scan, 73% (8/11) required surgical intervention compared with 30% (3/10) when BPF was not visible. CT was found to be useful in guiding surgery overall as it helped to identify and localise BPFs.⁷ Tsubakimoto *et al* examined the clinical outcome of 11 patients who had BPF. None of them had closure by surgery or bronchoscopy, and 10 of the 11 patients were managed successfully by conservative management.⁸ One of the 11 patients had BPF missed by the radiologist and clinician, and the outcome was death from multi-organ failure. The authors noted that this patient should have received surgical closure of the BPF, and they recommended urgent interventional closure in those with ‘a large pulmonary defect communicating to the pleural space ... depicted on CT’. However, conservative management was successful in 80% where BPF was visible on CT scan (4/5). The likelihood of BPF requiring surgery may be increased by, but not necessarily dictated by, CT finding of BPF.

The likelihood of PE can be stratified using the relative risk of a patient’s risk factors. According to the British Thoracic Society guidelines, our patient had the major risk factor of immobilisation secondary to long-standing ulcers on his feet.⁹ His other minor risk factors included hypertension and obesity.

Learning points

- It is not uncommon for pulmonary embolism (PE) to present without breathlessness, pleuritic chest pain or haemoptysis. One must maintain high clinical suspicion to diagnose this condition.
- When community-acquired pneumonia does not resolve with appropriate antibiotics, it may represent an alternative condition with similar symptoms mimicking this diagnosis (such as PE).
- Complicated PE can lead to infarction, cavitation, abscess formation and in rare cases, bronchopleural fistulation. This may be treated conservatively, bronchoscopically or by surgical closure of the fistula.

Most patients (>85%) with PE present with sudden onset or worsened shortness of breath, and it can also present with a constellation of other symptoms including pleuritic chest pain, cough, haemoptysis and syncope.¹⁰ Despite the many clues to this condition, PE may mimic clear clinical features of an alternative diagnosis, for example, pneumonia as in our case. Where there is doubt with regards to the diagnosis, a list of differentials needs to be generated and followed up with serial focused clinical examinations, laboratory investigations and radiological scans where appropriate to identify an alternative explanation.

Contributors ET: contributed to collecting the relevant data/result (eg, blood test result and radiograph findings), as well as the writing of the case report; conducted literature review. LB: contributed to the planning of this case report, as well as the writing of the case report. TM: contributed to the writing of the case report and proofread the final draft. AB: contributed to the idea and planning of the case report, edited multiple drafts and proofread the final version; interpreted the radiological images.

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