CASE REPORT

Spontaneous pulmonary haemorrhage into an existing emphysematous bulla

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SUMMARY

A patient with chronic obstructive pulmonary disease (COPD) and pulmonary fibrosis presented dyspnoeic with productive cough and large-volume haemoptysis, 1 month after coronary stenting and commencement of clopidogrel. A chest radiograph showed a well-circumscribed opacity in the left lower zone with surrounding consolidation, where previously an emphysematous bulla had been. A CT scan of the thorax confirmed a 14 cm bulla in the left lower lobe, which was 70% filled with blood with associated surrounding consolidation. A diagnosis of pulmonary haemorrhage into an existing emphysematous bulla with co-existing infective exacerbation of COPD was performed. Owing to his significant comorbidities, the patient was managed conservatively, returning to baseline over several days. He had two further admissions due to pulmonary infection over 2 months, where the haematoma was nominally smaller, and died during the second of these.

BACKGROUND

Chronic obstructive pulmonary disease (COPD) is very common and can be complicated by emphysematous bullae; these can be of little consequence but often compound dyspnoea and occasionally precipitate pneumothorax or intrabullous infection.[1] We describe a unique case of massive haematoma formation within a giant emphysematous bulla, preceded by infection and antiplatelet medication. This case is of interest because pulmonary haematoma is rarely spontaneous, and in those cases without preceding trauma or underlying vascular malformation, there is often contributory haemophilia or oral anticoagulation therapy.[2 3]

CASE PRESENTATION

A 65-year-old man with COPD reported of being generally unwell for 3 days, with cough productive of green sputum. On the day of presentation to his local emergency department, he reported of large-volume haemoptysis with visible clots and pleuritic chest pain, and was markedly more dyspnoeic.

His observations demonstrated tachypnoea (respiratory rate 34 bpm), tachycardia (heart rate 110 bpm), hypotension (blood pressure 85/53 mm Hg), oxygen saturation 77% on room air and temperature 37.1°C. These improved with intravenous fluids and high-flow oxygen. On examination, he was cool peripherally with conjunctival pallor. He had equal chest expansion and, on auscultation, no cardiac murmurs but widespread crackles in both lungs and decreased air entry at the left lung base were noted. His abdomen was soft and non-tender. There was clinically no deep vein thrombosis.

He had a background of interstitial lung disease and COPD. Owing to non-attendance, his most recent pulmonary function tests were 3 years prior to this episode, showing forced expiratory volume in 1 s (FEV1) 2.49 L (78% of predicted), forced vital capacity (FVC) 5.22 (128% of predicted), with FEV1/FVC ratio 47% (55% of predicted). Transfer factor was 0.36 (26% of predicted). The patient's medications included long-acting β agonist, long-acting muscarinic and corticosteroid inhalers and oral mucolytics. He was no longer smoking, but had a 45-pack-year history. Over the preceding 12 months, the patient had several exacerbations of COPD, two requiring hospital admissions. One month prior to this episode, during an admission for COPD exacerbation, he underwent coronary angiography for stable angina, having a stent placed in the circumflex artery, and was started on clopidogrel, in addition to aspirin, which he had been taking long-term. At this point, he was started on long-term home oxygen therapy, for at least 16 h per day. He lived alone in his own home, with no external help, and was able to walk 10–15 m when well.

INVESTIGATIONS

Blood tests

Arterial blood gas showed type 1 respiratory failure (pH 7.43; PO_2 5.4; PCO_2 3.9; lactate 3.0) on 2 L oxygen. Haemoglobin was 78 g/dL (40 g/dL lower than the most recent sample); leucocyte count 23.1×10^9/L with marked neutrophilia; and C reactive protein 35.1 mg/L. Platelet count was 330×10^9/L and clotting was normal.

Chest radiograph

AP chest radiograph showed 8×8 cm opacity in the left lower and mid zones; it was difficult to define inferiorly and laterally, but appeared to have an air-fluid level (figure 1). There were fibrotic and emphysematous changes in both lungs, more prominent in the lower lobes, and consolidation surrounding the previously mentioned left-sided opacity.

Pleural ultrasound & aspiration

Bedside pleural ultrasound demonstrated a partially-loculated collection in the left lower zone with some associated pleural effusion. Pleural aspiration was performed with 10 mL frank blood aspirated.

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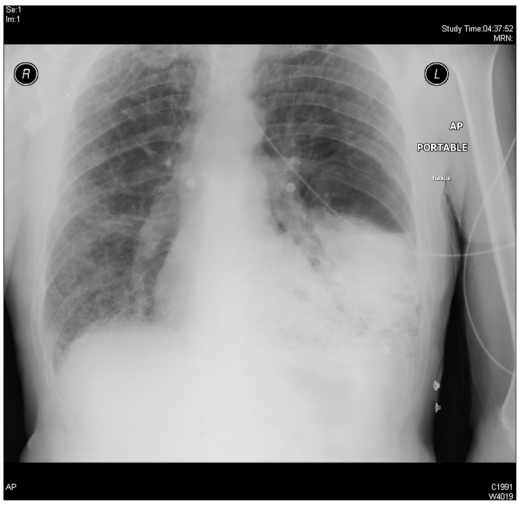

Figure 1 Anteroposterior (AP) chest radiograph showing chronic emphysematous and fibrotic changes in both lungs and an 8×8 cm soft tissue opacity in the left lower zone with surrounding consolidation.

CT scan of the thorax

Arterial phase contrast CT scan of the chest showed gross generalised centrilobular and panacinar emphysema and subpleural honeycombing consistent with known COPD and interstitial lung disease, respectively (figures 2 and 3). There was a large 14×8×8 cm bulla in the lateral left lower lobe, substantially filled with loculated, relatively high-density material, likely representing haematoma. No source of bleeding or vascular malformation was identified. Extensive consolidation was seen elsewhere in the left lower lobe, with prominent hilar lymph nodes.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for a single, discrete soft tissue density seen on chest X-ray typically includes primary lung neoplasm or solitary pulmonary metastasis, tuberculoma, aspergilloma, hamartoma, organising pneumonia, Wegener's granulomatosis, intrapulmonary lymph node, arteriovenous malformation and rheumatoid nodule.[4] In patients with known bullous emphysema, this should also include infection or fluid within a bulla.

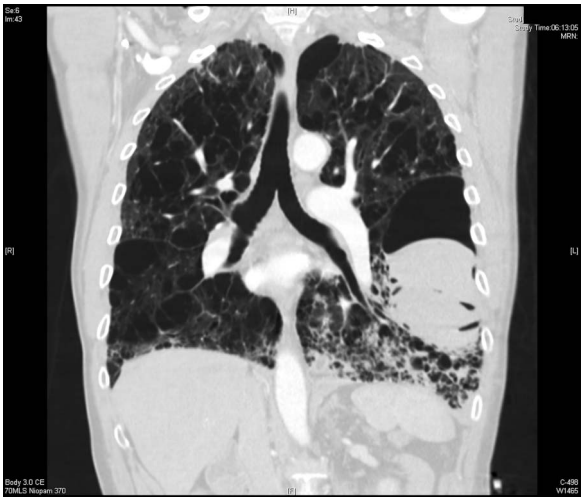

Figure 2 CT scan of the thorax (coronal view; lung window) showing gross panacinar emphysema with a 14×8 cm bulla in the lateral left lower lobe containing partially loculated, high-density material representing haematoma.

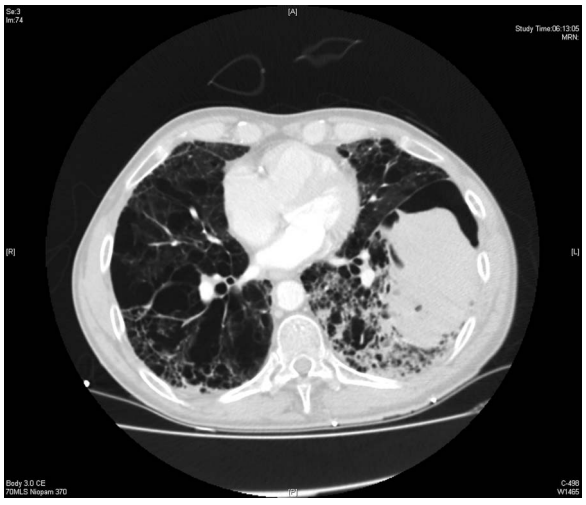

Figure 3 CT scan of the thorax (axial view; lung window) showing an 8×8 cm emphysematous bulla in the left lower lobe, containing partially loculated 35–45 Hounsfield Unit-density material, likely representing blood; and surrounding consolidation, multiple emphysematous bullae in right lung and no cardiomegaly.

In this patient, the combination of haemoptysis, significant drop in haemoglobin, opacity on radiograph where an emphysematous bulla was previously known, relatively high-density bulla contents on CT and frank blood obtained during pleural aspiration, led to the diagnosis of pulmonary haematoma into an existing emphysematous bulla.

TREATMENT

The patient was managed holistically using a multidisciplinary approach. For the large intrapulmonary haematoma, aspirin and clopidogrel were held briefly and the patient was given oral tranexamic for 3 days to encourage haemostasis. He was transfused 2 units of packed red cells to correct his anaemia, which was contributing to his symptoms. The patient was considered for surgical intervention but due to poor baseline lung function, the large size of the bulla, and patient preference, he was managed medically. For the co-existing infectious exacerbation of COPD, the patient was managed with regular salbutamol and ipratropium bromide nebulisers, intravenous co-amoxiclav and oral prednisolone.

Over the following 3 days, the patient's oxygen demand decreased to baseline, haemoptysis improved, haemoglobin level remained stable and inflammatory markers improved. He was switched to oral antibiotics and scheduled to complete a 4-week course due to the high likelihood of infection developing within the haematoma. As there was a high risk of coronary stent thrombosis, dual antiplatelet therapy was recommenced.

There is a paucity of evidence regarding the management of pulmonary haematoma into an existing emphysematous bulla. Conservative or medical management may be appropriate in small, sterile collections, but, often, these patients require antibiotics and reversal of preceding coagulopathies.[5 6] In young, fit patients with small haematomas, bullectomy or lobectomy has been successful in several cases.[7 8] In future, we suggest that there could be a role for percutaneous fluoroscopy-guided embolisation of the offending vessel.

OUTCOME AND FOLLOW-UP

The patient improved and was discharged to his own home. Follow-up chest radiograph (figure 4) demonstrated the bullous

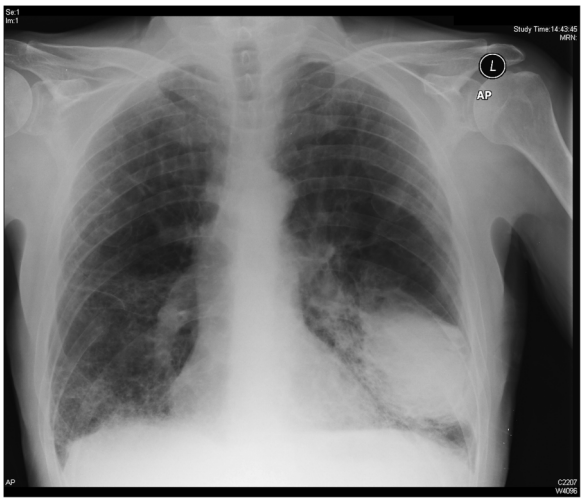

Figure 4 Anteroposterior (AP) chest radiograph 3 weeks after admission shows minimal decrease in size of pulmonary haematoma compared to previous image; and partial resolution of consolidation.

haematoma decreasing in size, although only modestly, and the surrounding consolidation had improved. The patient had two further admissions in the 2 months after discharge, and died during the second from further pulmonary infection despite multiple courses of antibiotics and no blood or sputum culture growth.

DISCUSSION

Pulmonary bullae are common in patients with COPD, and can be complicated by pneumothorax or more rarely by sterile or infected intrabullous fluid collection.[1] Blood-containing pulmonary cavities or lesions are rare.[6]

Almost all cases of pulmonary haematoma are due to blunt or penetrating chest trauma. Other causes include bleeding into existing bulla during invasive mechanical ventilation, ruptured arteriovenous malformation and extrapulmonary causes such as aortic aneurysm rupture.[6] Seemingly spontaneous haematoma has occurred de novo, or in pre-existing emphysematous bullae or tuberculous cavities in patients with predisposing coagulopathies, such as haemophilia or therapeutic and supratherapeutic anticoagulation.[3 5] There are two published cases describing spontaneous pulmonary haematoma without coagulopathy, both much smaller than this case, and managed surgically.[7 8]

The predominant theory for haematoma formation within an emphysematous bulla, supported by this case, is that an initial intrabullous infection causes angionecrosis and consequent bleeding into the pre-existing cavity.[8]

This case is the first to describe non-traumatic pulmonary haematoma occurring in an existing emphysematous bulla, likely precipitated by infection and compounded by dual antiplatelet therapy. It highlights the importance thorough history-taking, review of previous imaging, early investigation and instigation of management, and a multidisciplinary, holistic approach to patient care.

Learning points

- Pulmonary haematoma can occur inside an existing emphysematous bulla.
- Pulmonary haematoma is an important but rare differential diagnosis for a single, discrete opacity on chest radiograph.
- Dual antiplatelet therapy is a risk factor for pulmonary haemorrhage.
- Arterial phase CT scan is the investigation of choice for pulmonary haemorrhage.
- The definitive management of pulmonary haematoma into a bulla is lobectomy, but this should be dependent on patient circumstances.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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