CASE REPORT

Acute COPD exacerbation presenting with pronounced intrabullous haemorrhage and haemoptysis

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SUMMARY

A 54-year-old man with history of chronic obstructive pulmonary disease (COPD) presented with subacute onset of chest pain, shortness of breath, productive cough with haemoptysis and night sweats. There were no fever or recent weight loss reported. The chest radiograph showed right upper lobe bullae with adjacent opacification and an emphysematous lung. Due to worsening haemoptysis and persistent chest pain, CT of the chest with contrast was performed, which revealed moderate to severe emphysema and numerous blood-filled bullae. Cardiac work-up for chest pain was negative for myocardial ischaemia and for aortic dissection. Further infectious work-ups for mycobacterial and invasive fungal infection were negative. The patient was treated for acute COPD exacerbation and responded well to the antibiotics with the resolution of haemoptysis. Follow-up CT of the chest revealed the gradual resolution of the haemorrhage, while the patient remained asymptomatic.

intermittent wheezing over 3 weeks. He also had a pleuritic chest pain with a cough. The patient has known a diagnosis of COPD, Global Initiative for Chronic Obstructive Lung Disease (GOLD) stage II airflow limitation with forced expiratory volume in 1 s (FEV₁) of 2.56 L, 70% of the predicted value and moderate diffusion impairment. There was no history of recent COPD exacerbation or dyspnoea on exertion. The patient did develop progressive shortness of breath with haemoptysis and noticed to have night sweats. However, there were no other infectious symptoms including fever, rigours, chills or recent weight loss. He denied illicit drug usage, tuberculosis exposure, and recent travel or incarceration. He reported negative HIV and purified protein derivative (PPD) skin testing. His medical history also includes hypertension, peripheral vascular disease and chronic kidney disease.

On examination in the emergency department, the patient was afebrile though tachycardiac. Heart rate was 107 beats per minute, blood pressure 119/68 mm Hg and the respiratory rate was increased to 20 breaths per minute with the oxygen saturation of 96% while breathing ambient air. Auscultation of the chest revealed diminished breath sounds at the right lung with mild expiratory wheezing. The remainder of the physical

BACKGROUND

Chronic obstructive pulmonary disease (COPD) is a disease with a significant morbidity and mortality worldwide.¹ It is characterised by progressive airflow limitation that is not fully reversible and is caused by pathophysiological processes including the destruction of the alveolar walls which leads to the development of bullous emphysema.² Acute exacerbations are common among patients with COPD and characterised by significant worsening of the symptoms and complications, which may result in death.^{3–5} Intrapulmonary or intrabullous haemorrhage are rarely reported in patients without an anticoagulation therapy. There is one recent report describing a bullous haematoma during acute COPD exacerbation with dual antiplatelet medication.⁶ Here, we present a unique case of acute exacerbation of COPD with bullous emphysema, complicated with intrabullous haemorrhage and haemoptysis without anticoagulation therapy.

CASE PRESENTATION

A 54-year-old man with 40-pack-year smoking history presented in the emergency department with chest pain. The pain was sudden in onset, persistent with mid sternum location and radiating to his neck, upper arms and back. He had a productive cough with increasing amount of blood tinged sputum and

Figure 1 Hyperinflated lungs consistent with COPD. Confluent airspace opacity at the right upper lung with no pleural effusion or pneumothorax.

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Figure 2 CT images of the patient were obtained in February (A1–A3), March (B1–B3) and July 2016 (C1–C3). (A1–A3) Axial sections showing moderate to severe emphysema, primarily paraseptal, with a lesser centrilobular component. There are numerous non-enhancing soft tissue opacities in upper lobe paraseptal bullae. (A2) Confluent ground glass opacities in the adjacent relatively normal lung adjacent to the bullae affected by the described soft tissue opacities. (B1–B3) Axial sections showing a marked improvement of the soft tissue opacities within paraseptal bullae in size and number. The confluent ground glass opacities noted in A2 have near completely resolved. No new or progressive finding. (C1–C3) The soft tissue opacities within paraseptal bullae in A1 and B1 continue to improve. No ground glass opacities, No new or progressive finding.

examination was unremarkable. Chest radiograph showed right upper lobe bullae with adjacent opacification and emphysematous lung (figure 1). Additional laboratory workup revealed mild leucocytosis with white blood cell count of $14\,400/\mu\text{L}$, mild anaemia with haemoglobin of 11.9 g/dL but normal platelet count $268 \times 10^3/\mu\text{L}$. Coagulation profile including prothrombin time (PT) and partial thromboplastin time (PTT) was within normal limits. Serial troponins were in normal range and the

ECG did not reveal acute ST-T changes. Infectious workup including sputum culture and respiratory viral panel was requested. Due to worsening haemoptysis and persistent chest pain, CT of the chest with contrast was performed. The CT chest revealed moderate to severe paraseptal emphysema predominant in the upper lobe. There were non-enhancing soft tissue opacities in paraseptal bullae without air-fluid levels, along with adjacent ground-glass opacities in right upper lobe. Hounsfield

unit (HU) of the soft tissue densities was ranging from 50 to 70 consistent with haemorrhage or blood clots. There was no filling defect in the pulmonary arteries and no radiological evidence of endobronchial lesions was observed (figure 2). The patient received empirical antibiotics with azithromycin and ceftriaxone with oral prednisone with the presumed diagnosis of pneumonia and acute COPD exacerbation. Due to worsening haemoptysis on presentation, bronchoscopy was planned. However no recurrent haemoptysis was noticed after the treatment, so airway surveillance was deferred. Further diagnostic workups for invasive fungal infections, mycobacterium disease and vasculitis were performed including acid fast bacilli, serum galactomannan, anti-neutrophil antibodies (ANA) and antineutrophilic cytoplasmic antibodies (ANCA), which were all negative. The urine sample for 23 valent pneumococcal antigen was positive, but this was attributed to the 23 valent pneumococcal polysaccharide vaccine administered at hospital admission. The patient improved clinically with no recurrent haemoptysis or chest pain.

OUTCOME AND FOLLOW-UP

Subsequent CT scan of the chest 5 weeks after discharge revealed no new pulmonary opacities and prior soft tissue opacities within paraseptal bullae had markedly improved in numbers and sizes (figure 2). Clinically, the patient was asymptomatic with improved dyspnoea, without weight loss or night sweats. He denied a cough or recurrent haemoptysis. Follow-up CT scan 3 months later demonstrated further improvement of the multiple soft tissue opacities within the paraseptal bullae (figure 2). The patient quit smoking and remained asymptomatic at 6-month follow-up visit.

DISCUSSION

Emphysema is defined as 'an abnormal permanent enlargement of the airspace distal to the terminal bronchioles accompanied by destruction of the alveolar walls and without obvious fibrosis'.⁷ Bullous emphysema is a condition in which bullae occur in conjunction with the emphysematous lungs.⁸ The pathogenesis of the bullae is complex and poorly understood. Chronic inflammation of the airways, especially terminal bronchioles causes airway obstruction leading to progressive airway trapping, distension of the airspaces and eventually destruction.⁸ In our case, the history of extensive smoking along with chest imaging was suggestive of emphysema. The patient had ongoing symptoms for 3 weeks with haemoptysis, night sweats and further developed acute chest pain. The initial chest radiograph was consistent with large bullae with adjacent airspace disease supporting the diagnosis of underlying pulmonary infection and exacerbation of COPD. However, with acute chest pain and worsening haemoptysis, vascular involvement including aortic dissection, pulmonary vascular occlusion or possible vasculitis needed to be evaluated. Pneumothorax remained in differentials as well. By CT scan of the chest major vascular involvement and pneumothorax could be excluded. It was further helpful in evaluating the pathogenesis of the numerous soft tissue densities by measuring Hounsfield units, which suggested intrabullous haemorrhage. However, it was initially difficult to exclude the presence of concurrent invasive fungal disease, mycobacterial infection or inflammatory processes including vasculitis. The unilateral location of cystic lesions filled with the pretentious material as observed in our patient is not characteristic of a vasculitis such as granulomatosis with polyangiitis (GPA: formerly called Wegener's granulomatosis) which usually occurs bilaterally. However, ground-glass opacity and consolidation in GPA may be

variable, from bilateral to perihilar and peribroncho-vascular in distribution.⁹ In our case, the vasculitis workup did not yield positive ANA or ANCA. The treatment response to anti-bacterial agents with resolution of haemoptysis supported the diagnosis of bacterial infection causing COPD exacerbation. Pneumococcal urinary antigen was detected while the patient received the pneumococcal vaccine. This raised, however, the possibility of false positive testing.¹⁰

Follow-up CT of the chest at 5 weeks and 3 months demonstrated significant regression of soft tissue densities consistent with a benign haemorrhagic process.

A non-traumatic intrabullous haemorrhage in COPD exacerbation is rarely reported. Two publications describe surgically managed spontaneous pulmonary haemorrhage without anticoagulation or coagulopathy.^{11 12} Furthermore, there is one case published in which a pulmonary haematoma occurred during pulmonary infection and which was likely facilitated by dual antiplatelet therapy.⁶ However, in our case, a presumed bacterial infection triggered an acute COPD exacerbation with pronounced intrabullous haemorrhage in the absence of antiplatelet or anticoagulation therapy. We further demonstrate that a conservative management remains a feasible treatment option in large intrabullous pulmonary haemorrhage with haemoptysis.

Learning points

- ▶ Intrabullous haemorrhage with haemoptysis is a rare complication in acute chronic obstructive pulmonary disease exacerbation.
- ▶ Intrabullous haemorrhage might occur even in the absence of antiplatelet or anticoagulation therapy.
- ▶ The Hounsfield unit is helpful in assuming the pathophysiology of the abnormal CT findings.
- ▶ Conservative management remains a feasible option in large intrabullous haemorrhage.

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