SPONTANEOUS HAEMOPNEUMOTHORAX DUE TO CAVITATING PULMONARY INFARCTION - A RARE CONDITION REVISITED

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ABSTRACT

Background: Secondary spontaneous haemopneumothorax (SHP) is a potentially fatal and rare complication which occurs in 1 – 12% of patients with spontaneous pneumothorax and has a male preponderance (male-to-female ratio of incidence rates of 25.4:1). It involves accumulation of air and at least 500mls of blood within the pleural space in the absence of trauma. SHP is usually a complication of spontaneous pneumothorax associated with lung disease but can also be secondary to active lung infection or trauma. Consequently, patients with SHP often have limited cardiorespiratory reserves that increase the risk of mortality associated with this disease entity.

Materials and Methods: We report a case of spontaneous haemopneumothorax secondary to lung cavitation complicating pulmonary infarction in a middle-aged lady with imaging proven pulmonary thromboembolism.

Result: Patient presented with a short history of increasing tachypnea and tachycardia with reduced oxygen saturation of 58% on room air. Cardiac enzymes were negative but D-Dimer level was elevated. Chest radiograph showed a cavitating lesion in the right lower lobe with pleural effusion and right pneumothorax. Computed tomography pulmonary angiogram (CTPA) revealed filling defects in the branches of the pulmonary arteries, in keeping with thromboembolism. A cavitating lesion was noted in the right lung as well as dense right hydropneumothorax. Ultrasound-guided percutaneous catheter drainage of the hydrothorax tapped frank blood.

Conclusion: Spontaneous haemopneumothorax complicating pulmonary infarction is an important clinical entity to be recognized. Previously, the diagnosis was made at autopsy or based on plain chest radiograph appearance correlated with clinical findings. The utility of timely and appropriate radiological diagnostic tools such as CT thorax or CT pulmonary angiography scan can accurately make the diagnosis. In certain instances the aetiological factor(s) can also be identified to improve the management of this disease entity.

Keywords: spontaneous haemopneumothorax, pulmonary thromboembolism, computed tomography
1.0 Introduction

Secondary spontaneous haemopneumothorax (SHP) is a potentially fatal and rare complication which occurs in approximately 1 – 12% of patients with spontaneous pneumothorax (Tatabe, 1996). It has a male preponderance with male-to-female incidence rates ratio of 25.4:1 (Kiser, 2000). It involves accumulation of air and at least 500mls of blood within the pleural space in the absence of trauma. The concentration of the haemothorax should be at least 40% of the hematocrit of the peripheral blood. This disease entity was discovered first in 1828 by Laennec during an autopsy of a patient who died of chest infection; and was managed successfully with repeated pleural aspirations by Whitaker in 1876 (Wu et al, 2002).

In the absence of lung infection and trauma, secondary spontaneous haemopneumothorax is usually a complication of spontaneous pneumothorax. Spontaneous pneumothorax is commonly associated with lung disease. Consequently, the patients have limited cardiorespiratory reserves that increase the risk of morbidity and mortality associated with this disease entity. Previously, diagnosis was made by high index of clinical suspicion; guided by plain chest radiograph films; and empirical treatment was instituted. However, with the utility of computed tomography (CT) scan of the thorax the diagnosis is made with accuracy. Therefore, facilitating faster management with the treatment of choice which is video-assisted thoracotomy (VATS) for evacuation of the pleural space haematoma (Chiang et al, 2003 and Chong et al, 2011).

We report a case of spontaneous haemopneumothorax secondary to lung cavitation complicating pulmonary infarction in a middle-aged lady with medical diagnostic imaging proven pulmonary thromboembolism.

2.0 Materials and Methods/ Case report

We report a case of a 57-year-old woman who presented with history of right arm swelling and pain as well as difficulty in breathing for a period of one week. The right arm swelling was progressive with no history of trauma. Concurrently, the patient gave history of cough with productive yellowish sputum and low-grade fever. However, she did not have chills and rigor. Due to her condition, she had not been mobilizing well. She had past medical history of hypertension and cerebrovascular accident. She also had ischaemic heart disease for which she underwent coronary artery bypass graft (CABG) ten years ago.

On examination, the right arm was swollen, warm, red and tender. There was serous discharge from the pores at the anterior aspect of her right arm, just above the elbow; but there were no ulcers seen. She had a low-grade fever and was tachycardic, with a pulse rate of 102 beats/minute. Upon auscultation, air entry was reduced in both lower lung zones with crepitations heard on the left side more than the right.

Laboratory findings showed increasing trend of white cells counts which increased from 7.0 x 10^9/L to 17.1 x 10^9 /L, with neutrophil predominance, in a span of 2 days. Her hemoglobin level also dropped progressively from 10g/dL to as low as 6.7g/dL. Plain radiograph of the chest revealed consolidations in both lung bases and minimal bilateral pleural effusions (Figure 1a). There were also some basal atelectatic changes in the right lower lobe. Swab from the right arm grew Acinetobacter baumanii. However, all other initial cultures eg. blood
and urine were negative for infectious organism. Therefore, the patient was treated for necrotizing fasciitis of her right arm. She was also treated for pneumonia with empirical administration of intravenous antibiotics.

Approximately one week later in the ward, patient’s condition deteriorated with increasing tachypnea and tachycardia with reduced oxygen saturation of 58% in room air and only picked up to 96% with 5L/min oxygen supplementation via high flow mask. ECG revealed sinus tachycardia and right axis deviation but did not show ischaemic changes. Cardiac enzymes were negative but she was noted to have an elevated D-Dimer level.

### 3.0 Results

#### 3.1 Further investigations

Repeat chest radiograph showed a cavitating lesion in the right lower lung lobe with right-sided pleural effusion and right pneumothorax (Figure 1b).

Computed tomography pulmonary angiogram (CTPA) revealed filling defects in the right descending pulmonary artery (Figure 2). Coronal multiplanar reformatted (MPR) CT scan images showed filling defects in the right ascending, right descending and left ascending pulmonary arteries as well as their segmental branches in keeping with massive pulmonary embolism (Figure 3). Consolidative changes were seen in both upper lobes and in the right lower lobe. There was a cavitating lesion noted in the consolidated lung in the right lower lobe (Figure 4a). Bilateral pleural effusions with collapse of both adjacent lungs were also noted. Dense right pleural space collection, in keeping with haemothorax was also noted.

Consequently, ultrasound-guided percutaneous catheter drainage was performed. A 6 French Bioteq pigtail catheter was inserted into the right pleural space. The procedure was uneventful. The drainage tube was connected to an underwater seal bottle, which demonstrated bubbling. An initial amount of 500mL of frank hemorrhagic fluid was drained.

#### 3.2 Management

Patient was treated conservatively with medical supportive treatment. Although culture results from the aspirate did not show any significant colony of organism, the patient was empirically treated with intravenous antibiotics ie. IV Imipenem 500mg 8 hourly and IV Clindamycin 600mg 8 hourly. She was also given a stat dose of IV Tazocin (Vancomycin). She was started on anticoagulation therapy by being given subcutaneous injection of Clexane 0.6mg daily. IV Transexanemic acid 500mg 8 hourly was also administered.

The following day, the patient’s condition further deteriorated. Arterial blood gases revealed respiratory acidosis: pH was 7.28, pCO₂ was 36.2mmHg and pO₂ was 42.2mmHg with bicarbonate level of 17.1mmol/L. Oxygen saturation has dropped further to 73.3%. The patient was electively intubated for Type 1 respiratory failure. A larger chest tube (32 French) was also inserted to drain the thick hemorrhagic pleural fluid. Subsequently, pus was also drained from this site. Therefore, a provisional diagnosis of empyema thoracis complicating right pleural hematoma was made.
Inevitably, patient developed nosocomial infections. Blood cultures grew methicillin resistant Staphylococcus aureus (MRSA), which was sensitive to Tazocin. Swabs from her wound sites i.e. right hand and sacral sores grew Acinetobacter baumanii and Pseudomonas aeruginosa. Cultures from the tracheal aspirate showed colonization of Acinetobacter baumanii and cultures taken a week later isolated Pseudomonas aeruginosa. Intensive intravenous antibiotic regime, meticulous arterial blood gases monitoring, and ventilator settings adjustments were conducted. She was also started on Dopamine and Norepinephrine inotropes as her blood pressure kept dropping.

Over the next ten days following the diagnosis of pulmonary thromboembolism complicated by cavitating lung infarction and haemopneumothorax; the patient was transfused with 3 pints of packed cells. Her hemoglobin level picked up from 6.7g/dL to 10.5g/dL but again dropped to 8.9g/dL. Persistent haemopneumothorax was noted on her chest radiograph (Figure 4b) and haemoserous fluid continued to drain out from the chest tube. The total amount of fluid drained via her chest tube was 1830mls of bloody and haemoserous fluid. Although the patient has reaccumulation of right haemothorax in keeping with active bleed, surgical intervention was not carried out as she was deemed not physically fit for the procedure. The patient succumbed to her illness after approximately 6 weeks in the ward. The cause of death was declared as sepsis from multiple sources including right hand necrotizing fasciitis, sacral sore, and nosocomial pneumonia.

**Figure 1**: (a) Plain chest radiograph showed evidence of bilateral basal lung consolidations, more marked in the right lower lobe (yellow arrow) with atelectatic changes in the right lung base (white arrow). Cardiomegaly (red arrow) and median sternotomy sutures were also noted. (b) Subsequent chest radiograph showed cavitating lesion in the right lower lobe (blue arrow) and right pneumothorax (curved white arrow).
Figure 2: Computed tomography pulmonary artery angiogram in axial plane in soft tissue window (Window Width: 350, Window Level: 50) showed evidence of pulmonary artery embolism. Filling defects (white arrow) were seen in the right descending pulmonary artery and its segmental branches in keeping with pulmonary thromboembolism. Right-sided dense pleural collection in keeping with haemothorax was also noted.

Figure 3: Coronal view of multiplanar reformatted image of computed tomography pulmonary artery angiogram in soft tissue window (Window Width: 350, Window Level: 50) showed evidence of pulmonary artery embolism. Filling defects (red arrows) were seen in the right ascending and descending pulmonary arteries and their segmental branches as well as in the left ascending pulmonary artery in keeping with massive pulmonary thromboembolism.
Figure 4: (a) Coronal view multiplanar reformatted image of CT pulmonary angiogram in lung window (Window Width: 1500, Window Level:-500) showed a cavitating lesion within the right lower lobe consolidation (straight white arrow). A large right haemopneumothorax was also noted (curved white arrow). (b) Despite aggressive intervention, chest radiograph several days later showed persistent pneumothorax (curved white arrow).

4.0 Discussion

Spontaneous haemopneumothorax (SHP) is a potentially life-threatening condition. The incidence of SHP complicating spontaneous pneumothorax ranges from 0.5-12% (Kakaris, 2004). The aetiological factors include malignancies, anticoagulant medications, vascular ruptures (aortic dissection, arteriovenous malformations [AVMs]), endometriosis, pulmonary infarctions, adhesions with pneumothorax, and hematologic abnormalities such as haemophilia (Kakaris, 2004). Pneumothorax has also been reported as a complication of cavitating pulmonary infarction (Hall et al, 1977).

During a span of approximately one millenium, between the years 915 to 1977, there have been 24 cases of pneumothorax complicating pulmonary infarction which have been reported in the world literature (McFadden, 1969). Many of the early cases of this complication were based entirely upon autopsy material, without clinical or radiologic documentation.
The mechanisms of bleeding described in SHP are bleeding either of a torn apical vascular adhesion between the parietal and visceral pleura or of torn congenital aberrant vessels between the parietal pleura and the bullae or due to rupture of vascularized bullae (Ali, 2008). SHP may result from tears in the vascularized adhesions between the visceral and parietal pleura that occur as the lung collapses. The rigid chest wall prevents the bleeding vessel from contracting. Vessels with poorly developed muscular walls have been identified within fibrous pleural adhesions that when, do not retract and are not hemostatic (McFadden et al, 1969). In our patient, the mechanism of secondary SHP is likely due to a ruptured cavitation of the infarcted lung. The subpleural location of our patient’s cavitating infarction is likely the cause of pneumothorax.

There is also a recognizable syndrome for this condition which is characterized by 3 phases whereby initially there is massive infarction followed by blood tinged and purulent sputum indicating excavation of infarcted area (Hall et al, 1977). This is followed by bronchopleural fistula formation leading to haemopneumothorax. We suspect that due to her prolonged immobilization, our patient had already developed pulmonary embolism at presentation which would account for her breathlessness. She had deteriorated in the ward when the infarcted area cavitated leading to a bronchopleural fistula and ultimately a SHP.

SHP is life-threatening because of acute huge blood loss leading to hypovolemic shock. Complications include empyema thoracis, and superimposed infection (McFadden et al, 1969) as was the case in our patient. Early thoracotomy for evacuation of any residual pleural blood after tube thoracostomy has been recommended, because in unoperated patients a restrictive pleural cortex with pulmonary dysfunction may develop (McFadden et al, 1969).

Generally, the aim of treatment is to achieve resuscitation, hemostasis, and re-expansion of the lung. Initial treatment consists of fluid resuscitation, drainage of the pleural fluid and blood transfusions (Kakaris et al, 2004). Definitive therapeutic policies for SHP are decided upon individually based on the patient’s condition and clinical appearance of the disease. The clinical features depend primarily on the amount of blood loss and air leakage.

Management options include tube thoracostomy, aspiration, thoracotomy, and decortication after the initial period of rest. Management by video-assisted thoracic surgery (VATS) has also been used successfully with added advantages of less tissue trauma and less postoperative pain when compared with traditional thoracotomy approach. More recently, VATS is increasingly being used for the management of SHP with favorable results (Hwong et al, 2004). We have provided a recommended algorithm for management of non-traumatic acute chest pain and shortness of breath (Figure 5).

Indications for thoracotomy are hypovolemic shock, continuous bleeding (100 ml/h), persistent air leak, impaired lung expansion, pachypleuritis, or recurrent pneumothorax (Wu et al, 2002). In the absence of these features, conservative management by tube thoracostomy is recommended (Wu et al, 2002). Considering that our patient was septic and haemodynamically decompensated, she was deemed not stable for surgery and was treated conservatively by chest drains.
Figure 5: Algorithm for management of sudden onset of chest pain and shortness of breath.
[Modified from Chiang et al, 2003]
5.0 Conclusion and recommendation

Spontaneous haemopneumothorax complicating pulmonary infarction is an important clinical entity to be recognized. With the aid of radiological investigations such as CT thorax or CT pulmonary angiography, the diagnosis can be made faster and accurately. In certain instances the aetiological factor(s) can be identified for the appropriate treatment to be initiated. The triad of pulmonary embolism, cavitating pulmonary infarction and dense hydropneumothorax need to be identified and emergency treatment by percutaneous catheter drainage is of utmost importance. Computed tomography pulmonary angiography scan, when performed timely, is a useful diagnostic tool that can help improve the management of this complication thus reduce patient mortality.

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Declaration

The authors declare that there is no conflict of interest in the preparation of this article and for publication in this journal. This article has not been previously published in any other journal or conference proceeding.

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References


