

Idiopathic Hemothorax in a Patient with Status Asthmaticus

ABSTRACT

INTRODUCTION Idiopathic spontaneous hemothorax has been rarely described in the literature.

CASE STUDY A case of status asthmaticus and spontaneous hemothorax is described in a 29-year-old female of African descent who presented to the emergency room after two days of severe cough productive of yellow sputum, otalgia, sore throat, subjective fevers, chills, headache, progressive wheezing, chest tightness and dyspnea. She had a history of seven years of asthma and was non-adherent with her controller asthma medications. Prophylactic subcutaneous administration of enoxaparin 40 milligrams was initiated upon hospitalization. The patient initially had a normal chest radiograph but subsequently developed a large, left hemothorax that required tube thoracostomy placement followed by video-assisted thoracoscopic surgery (VATS).

RESULTS The patient was transferred to the Intensive Care Unit (ICU) and tube thoracostomy resulted in evacuation of 1,400 milliliters of blood-like fluid, which had a pleural fluid hematocrit greater than 50% of the serum hematocrit. A contrast-enhanced computed tomography (CT) scan of the chest did not reveal any source for the bleeding and a technetium bone scan of the chest was normal. The patient required transfusion of five units of packed red blood cells. She was then taken to the operating room for VATS because of continued chest tube drainage (3,200 milliliters of fluid over a 48 hour period).

CONCLUSION The etiology of the hemothorax was unknown despite surgical exploration but was felt to be secondary to cough and bronchospasm associated with status asthmaticus.

INTRODUCTION

A severe asthma exacerbation is defined by some, but not necessarily all of the following signs and symptoms: dyspnea at rest, upright sitting position, inability to speak in phrases or sentences, respiratory rate greater than 30 breaths per minute, use of accessory muscles of respiration, pulse greater than 120 beats per minute, pulsus paradoxus greater than 25 mmHg, peak expiratory flow rate less than 50% of predicted or personal best, hypoxemia, and eucapnia or hypercapnea. An altered mental status, paradoxical respirations, bradycardia, a quiet chest on auscultation and absence of pulsus paradoxus can indicate imminent respiratory arrest. (1)

Status asthmaticus has been associated with several complications. These include barotrauma resulting in pneumothorax and pneumomediastinum; mucus plugging, which may cause lobar atelectasis or pneumonia; rib fracture; respiratory failure; and aspiration pneumonia. Intubation for respiratory failure may result in aspiration pneumonia, barotrauma, severe hypotension secondary to a reduction in right heart preload and possibly cardiac arrest.

Hemothorax is the presence of pleural fluid with a hematocrit greater than 50% of the serum hematocrit. (2) As pleural fluid may impart a red color, even with a hematocrit of 5% or greater, it may be indistinguishable from blood. Therefore, the hematocrit of a hemorrhagic pleural effusion should always be measured. The hematocrit may be less than the required 50% of the blood hematocrit after several days, making it appear as a hemorrhagic exudate. The definition of hemothorax therefore includes a pleural fluid hematocrit ranging from 25–50% of a serum hematocrit. (3)

Idiopathic spontaneous hemothorax (4, 5, 6, 7) has been rarely described in the literature, and in one case, the patient did not have confirmatory surgical exploration of the chest cavity. In another patient, spontaneous

hemothorax occurred seven years apart in opposite thoracic cavities. (8) Only one other report of a spontaneous hemothorax has been described associated with status asthmaticus. The patient was an 11-year-old child diagnosed with Ewing's sarcoma as a cause of the hemothorax. (9) This paper describes a second patient with idiopathic spontaneous hemothorax who had severe bronchospasm and cough secondary to status asthmaticus.

CASE STUDY

A 29-year-old female of African descent presented to the emergency department with a two-day history of a severe cough productive of yellow sputum, otalgia, sore throat, subjective fevers, chills, headache, progressive wheezing, chest tightness and dyspnea. The patient has a seven-year history of asthma and was non-adherent with her controller medications. She had several emergency department visits, urgent office visits and hospitalizations for asthma. Likewise, she had a history of migraine headache, allergic rhinitis, chronic sinusitis and flexural atopic dermatitis. She denied any history of pneumonia, pleurisy, tuberculosis, chest trauma, connective tissue diseases and thromboembolic diseases. She denied smoking, excessive alcohol intake or illicit drug use.

A physical examination revealed a well-developed, well-nourished, cooperative female oriented in time, place, and person whose BMI was 22. Her respiratory rate was 26/min, heart rate was 134/min and blood pressure was 133/77 mm Hg in the right arm sitting. Her oxygen saturation was 97% on two liters of oxygen via nasal cannula and oral temperature was 97.5°F. She was using accessory muscles of breathing and was only able to answer questions in a staccato fashion, 1 or 2 words at a time. Coughing episodes would result in even greater dyspnea. Examination of the chest demonstrated diffuse inspiratory and expiratory wheezing and rhonchi throughout both lung fields. The remainder of her physical examination was otherwise normal.

She was treated with methylprednisolone 125 milligrams intravenously every six hours, levofloxacin 750 milligrams intravenously daily, nebulized albuterol every three hours, and subcutaneous enoxaparin 40 milligrams for venous thromboembolism prophylaxis. The initial chest radiograph was within normal limits and the complete blood count (CBC) revealed a hematocrit of 40.1% with a white blood cell count (WBC) of 10,700 cells per milliliter. The WBC differential revealed 89% segmented neutrophils and no immature forms. Previous office spirometry was consistent with moderate obstruction with a FEV1 of 72% predicted.

RESULTS

She had some improvement in her dyspnea but the severe coughing and wheezing persisted over the next 60 hours. The patient suddenly developed severe, left-sided chest pain which prompted an evaluation with a bolus contrast CT study of the chest. (FIGURE 1) It revealed a small left upper lobe pulmonary infiltrate and a new, small left pleural effusion. The patient became more dyspneic over the next 24 hours. A repeat chest radiograph revealed an increased left pleural effusion. (FIGURE 2) The serum hematocrit now was 24.5%.

Figure 1: Bolus contrast computer tomography (CT) of the chest revealing a left upper lobe pulmonary infiltrate (thick arrow) with a new, small left pleural effusion (thin arrow).

Figure 2: Chest radiograph revealing an increased left pleural effusion.

The patient was transferred to the ICU and tube thoracostomy resulted in evacuation of 1,400 milliliters of blood that had a hematocrit greater than 50% of the serum hematocrit. A new contrast-enhanced CT examination of the chest did not reveal a source for the bleeding and a technetium bone scan did not reveal any abnormal chest uptake. The patient was transfused with five units of packed red blood cells and because of continued chest tube drainage (3,200 milliliters over a 48-hour period), was taken to the operating room for VATS. At surgery, a thrombus within the pleural space was noted and removed, resulting in complete expansion of the left lung. The left upper lobe apical region had an adherent clot that was

removed but no etiology for the hemothorax could be identified. A urinary *Legionella* antigen test was positive but the blood cultures, sputum cultures, left upper lobe biopsy specimen and pleural fluid did not yield growth of *Legionella*. Likewise, the pleural fluid pH, cell count, protein and LDH did not shed any additional light on the etiology of the pleural effusion.

The patient gradually recovered after the VATS procedure and the chest tube was removed and left lung remained expanded. A vasculitis and autoimmune work-up were negative. The patient was discharged on levofloxacin for 21 days, a two-week oral prednisone taper and her controller asthma medications. She now has been observed for 30 months with no recurrence of a hemothorax, and the most recent chest radiograph was normal other than minor pleural thickening at the left lung base.

DISCUSSION

Blood entering the pleural space is exposed to the motion of the diaphragm, lungs, and other intrathoracic structures that can result in defibrination of the blood and incomplete clotting. Within several hours of cessation of bleeding, existing clots undergo lysis secondary to pleural enzymes. However, when lysis is incomplete or bleeding is significant, clot formation usually occurs. Once the clot organizes, it adheres to the lung and pleura, and becomes difficult to remove. The agitation of cardiac and respiratory movement rapidly defibrinates the blood and results in a fibrin clot which becomes deposited on the visceral and parietal pleura, establishing ideal circumstances for a “trapped” lung. In the clot's early development, the thin membrane on the surface has little substance and is attached loosely to the underlying pleural surface. However, angioblastic and fibroblastic proliferation occurs by the seventh day. The membrane continues to thicken by progressive deposition and organization of the coagulum within the cavity making removal and decortication difficult.

There are three major clinical effects of a hemothorax—two acute and a third more chronic. The acute, early effects of a hemothorax are associated with hypovolemia and altered lung mechanics. The changes in pulmonary function are the result of lung compression secondary to the hemothorax. The more chronic effects of a hemothorax result from significant amounts of retained clot and subsequent empyema formation or development of a fibrothorax. This mostly results from inadequate, early management of a hemothorax.

Tube thoracostomy is the treatment of choice, particularly for a hemothorax that impairs breathing. Indications for surgical exploration and tube thoracostomy are a blood loss of 1,500 milliliters in 24 hours or 200 milliliters per hour during several successive hours necessitating repeated blood transfusions. [\(10, 11\)](#) Patients with active blood loss but with stable hemodynamics can be treated with VATS, not only to stop the bleeding but also to evacuate blood clots and break down adhesions.

A series of 50 VATS procedures performed in patients with traumatic hemothorax demonstrated active blood loss in 11 subjects. [\(3\)](#) In patients with incomplete resolution of clots and blood loss after chest tube drainage, VATS resulted in shorter hospital stay and duration of tube drainage when compared to a group just treated with tube drainage in patients with incomplete resolution of clots and blood loss after chest tube drainage. [\(12\)](#) Removal of loculated collections of blood or clotted blood is necessary since retained blood in the pleural space is a risk factor for empyema and fibrothorax. [\(13,14\)](#)

Thoracotomy is the procedure of choice for patients with hemodynamic instability due to active bleeding. [\(15\)](#) The source of bleeding can be controlled and the hemothorax evacuated at the time of surgical exploration. Thoracotomy is usually required for adequate empyema drainage or if decortication is required. In addition, a longer time span between the appearance of a hemothorax and a VATS procedure increases the need for intraoperative conversion to a thoracotomy. [\(16\)](#) A thoracotomy is necessary to control and treat a hemothorax in 10% of patients. [\(11\)](#)

CONCLUSION

A number of atraumatic etiologies of spontaneous hemothorax are described in the literature. These include spontaneous hemopneumothorax, coagulopathy, vascular disorders, neoplasia and miscellaneous causes such as pulmonary sequestration and endometriosis. (17,18) There is severe dynamic hyperinflation and pleural pressure changes present in patients with status asthmaticus secondary to breathing against obstructed airways. This theoretically could lead to large changes in intrathoracic pressure as a consequence of severe airflow obstruction producing transient shearing and pulling of the vasculature in the chest cavity resulting in a large hemothorax. The patient in this report had status asthmaticus. Initially, she had a normal chest radiograph, but subsequently developed a large, spontaneous hemothorax that required tube thoracostomy placement followed by VATS. Although the etiology of the hemothorax is unclear despite surgical exploration, it was concluded that severe bronchospasm and cough secondary to status asthmaticus led to the patient's spontaneous hemothorax.

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