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Case Presentation

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Early Video-Assisted Thoracic Surgery for Primary Spontaneous Hemopneumothorax causing Life - Threatening Hemorrhage: Two Cases Report

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Abstract

Spontaneous hemopneumothorax is a rare thoracic disease that can be life-threatening if not diagnosed and treated promptly. We report two cases of primary spontaneous hemopneumothorax occurred in two young males, who underwent early video-assisted thoracoscopic surgery for hemorrhage and recovered uneventfully. In both cases, video-assisted thoracoscopic surgery allowed the site of bleeding to be identified and blocked. Conservative treatment with drainage is only useful for diagnosis and to stabilize patients before surgery. Surgical treatment with video-assisted thoracoscopic surgery should be performed as soon as possible after the diagnosis of spontaneous hemopneumothorax.

Introduction

Spontaneous Hemopneumothorax (SHP) is defined as the spontaneous accumulation of air and blood within the pleural space, occurring without any history of trauma or specific cause or iatrogenic intervention [1,2]. SHP is a rare thoracic disease, occurring in only 2-5% of patients with spontaneous pneumothorax [3]. It is a life-threatening condition, if not diagnosed and treated promptly, causing potentially fatal hemorrhage shock [4-6]. In this paper, we report two cases of primary SHP occurred

in two young males, who underwent early Video-Assisted Thoracoscopic Surgery (VATS) for hemorrhage and recovered uneventfully.

Case Presentation

Case 1

A 21-year-old male presented in the Emergency Room (ER) with a 24-hour history of severe right-sided chest pain suddenly arose after coughing and with shortness of breath. There was no recent history of trauma, surgery, or medication use, nor a previous history of similar symptoms. The patient denies previous diseases and smoking habits. Clinical examination revealed a reduction in right-sided breath sounds. A chest X-ray was performed, which showed a right-sided spontaneous pneumothorax **(Figure 1)**. A right chest tube was placed. After the positioning of the endopleural drainage, there was a progressive leakage of 500 ml of fresh blood, for which the patient was urgently subjected to a chest CT scan **(Figure 2)**, which showed a large right pleural effusion and confirmed an ispilateral pneumothorax. The level of his serum hemoglobin was 11.0 g/dl. The patient's hemodynamic conditions remained stable. The patient was adequately hydrated and drainage carefully monitored. Chest computed tomography scans taken 7 hour post admission showed a massive right pleural effusion and confirmed an ispilateral pneumothorax **(Figure 3)**. An initial deterioration of the patient's clinical condition and a further leakage of 500 ml of blood from the drainage were observed; therefore, an emergent VATS was indicated. The operative findings showed a large collection of blood and clots in the pleural cavity **(Figure 4)**. After evacuation of the blood and clots, continuous bleeding from a small aberrant vessel at the apex of the pleural cavity was discovered **(Figure 5A)**. The aberrant vessel was clipped **(Figure 5B)**. For the detection of an area of bullous dystrophy a segment resection of the right upper lobe apex was performed. The postoperative course was uneventful. A chest X-ray was performed postoperatively to confirm lung expansion.

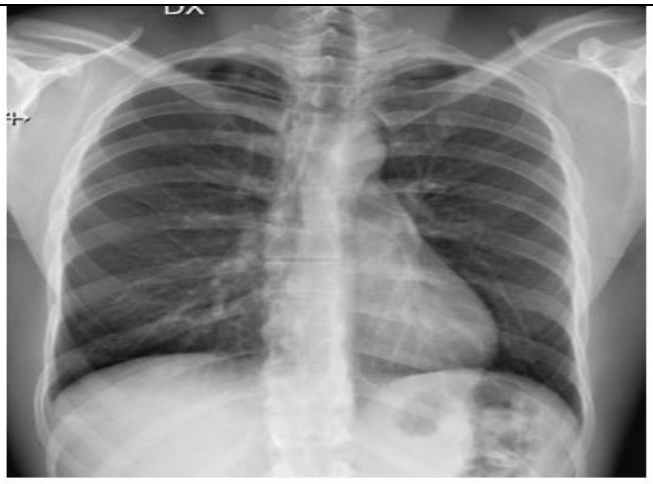


Figure 1: Chest X-ray of the patient on arrival at ER revealed a right-sided spontaneous pneumothorax.

Figure 2: Chest computed tomography scans taken post-chest tube insertion showed large right pleural effusion; an ispilateral pneumothorax is confirmed.

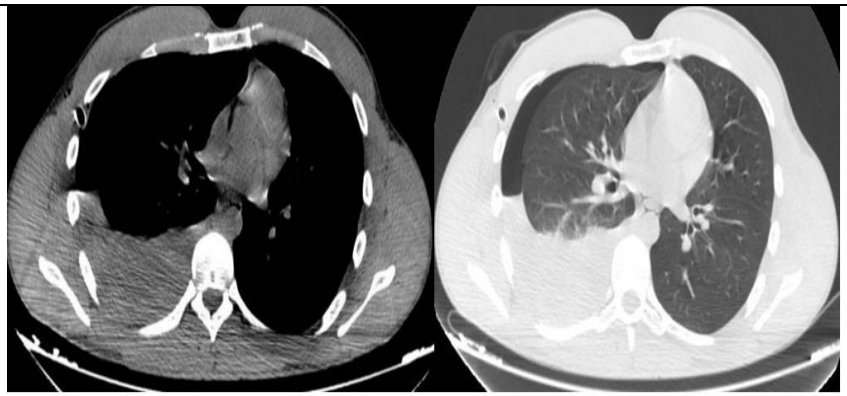


Figure 3: Chest computed tomography scans taken 7 hour post admission showed massive right pleural effusion; an ispilateral pneumothorax is confirmed.

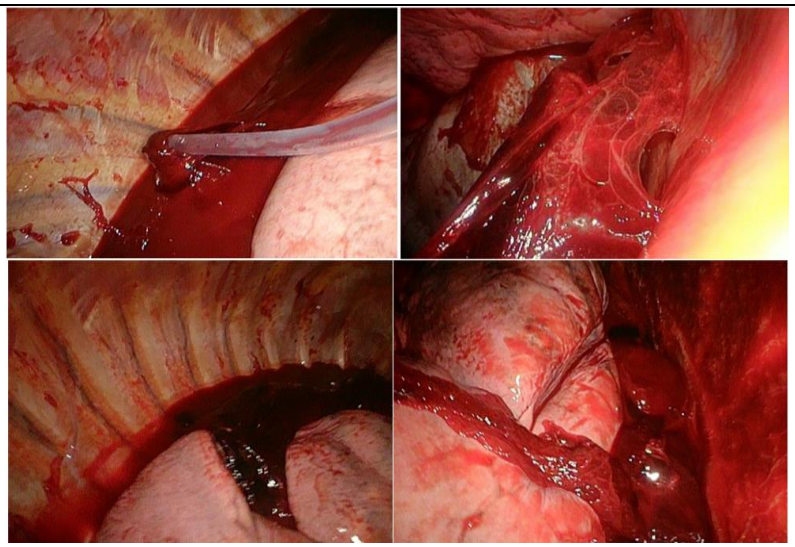
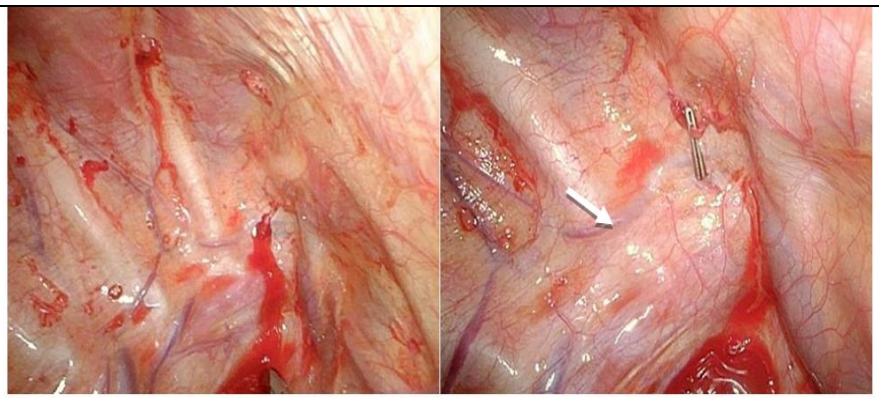


Figure 4: The operative findings showed a large collection of blood and clots in the pleural cavity.



A B

Figure 5: After evacuation of the blood and clots, continuous bleeding from a small aberrant vessel at the apex of the pleural cavity was discovered (white arrow) (A). The aberrant vessel was clipped (B).

Case 2

A 24-year-old male presented in the Emergency Room (ER) with a 12-hour history of severe left-sided chest pain, dyspnea and cough; the symptoms worsened in the last 2 hours. The patient has no significant pathologies, has not suffered trauma and denies similar previous symptoms. He was chronic smoker with 6-years history of smoking with 10 cigarettes per day. The patient was conscious, tachycardiac, tachypneic, and presents moderate respiratory distress. Chest computed tomography performed urgently showed a large left-sided compressive hydropneumothorax with lung collapse and pleural effusion with tracheal deviation to the contralateral side **(Figure 6)**. A left chest tube was urgently placed with the leakage of a large quantity of air and 600 ml of fresh blood. The level of his serum hemoglobin was 9.6 g/dl. A chest X-ray taken 6 hour post-chest tube insertion showed blood residuals and pneumothorax on the left side **(Figure 7)**. The patient was subsequently taken for an emergent VATS. The operative findings showed a large collection of clots in the pleural cavity **(Figure 8).** After evacuation of the clots the source of the bleeding was found to be a ruptured adhesion in the pulmonary apex **(Figure 9)**; hemostasis was achieved via cauterization and hemostatic devices. For the detection of a large area of bullous dystrophy at the lung apex **(Figure 9)** wedge resection of the left upper lobe apex was performed. The patient remained stable post-operatively. Postoperative chest X-ray showed resolution of hemopneumothorax with fully lung expansion **(Figure 10).**

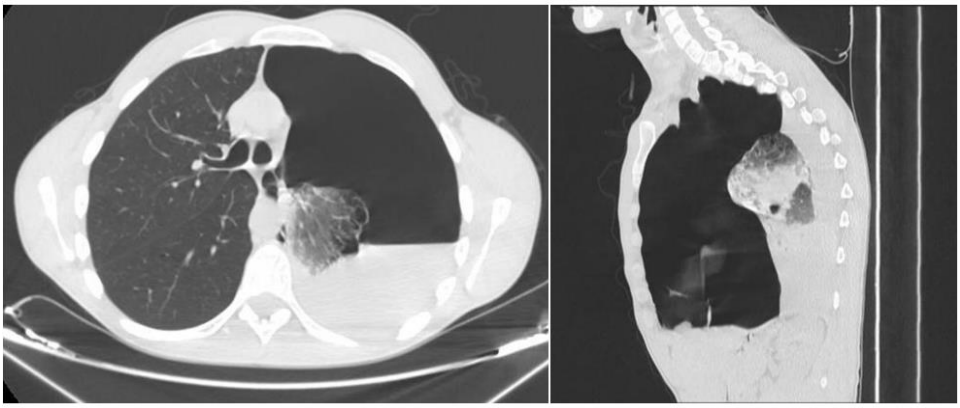


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Figure 6: Axial (A) and sagittal (B) view of chest computed tomography on arrival at ER showed a large left-sided compressive hydropneumothorax with lung collapse and pleural effusion with tracheal deviation to the contralateral side.

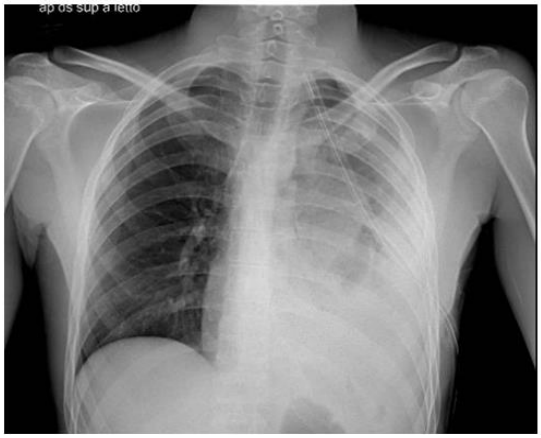

Figure 7: A chest X-ray taken 6 hour post-chest tube insertion showed blood residuals and pneumothorax on the left side.

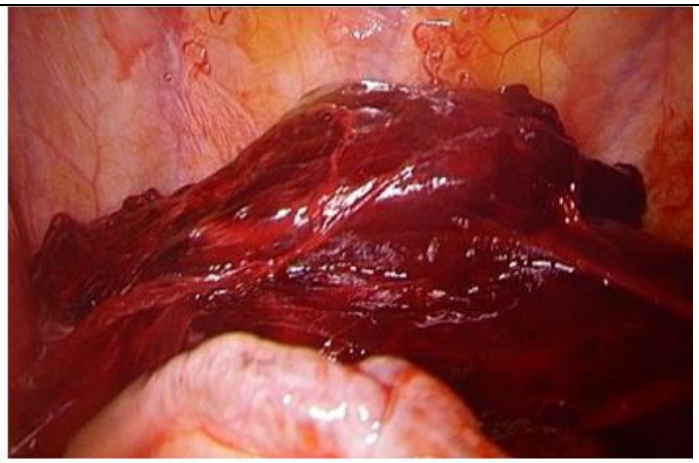

Figure 8: The operative findings showed a large collection of clots in the pleural cavity.

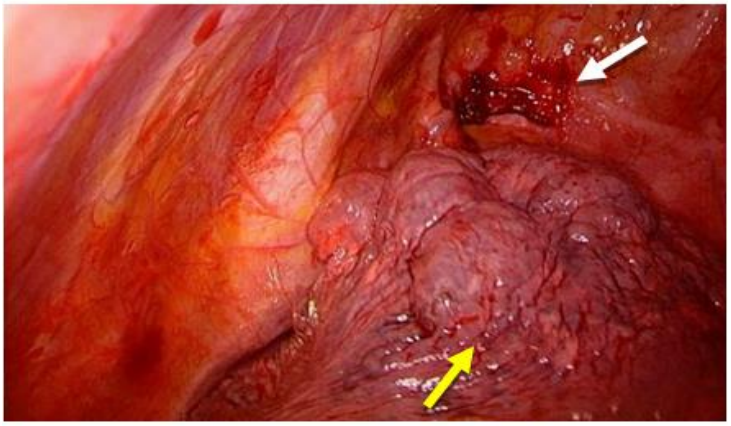

Figure 9: After evacuation of the clots the source of the bleeding was found to be a ruptured adhesion in the pulmonary apex (white arrow). Large area of bullous dystrophy is visible at the lung apex (yellow arrow).

Figure 10: Postoperative chest X-ray showed resolution of hemopneumothorax with fully lung expansion.

Discussion

SHP is a rare but serious thoracic disease, defined as the concurrent and spontaneous presence of hemothorax and pneumothorax without prior trauma or iatrogenic intervention [1]. It is 30 times more frequent in males, especially at a young age [7]. SHP begins acutely with chest pain, dyspnea, tachycardia, and hypotension. SHP is distinguished from Primary Spontaneous Pneumothorax (PSP) by the possibility of developing hemodynamic instability due to hemorrhagic shock with consequent clinical worsening [2,6]. Bleeding in SHP cases may result from a tearing of adhesions between the parietal and visceral pleura (as in our second case), or of aberrant vessels (as in our first case), due to lung collapse for pneumothorax; another mechanism is the rupture of both the vascularized bulla and the underlying lung parenchyma [2,8]. In about a third of cases, the origin of bleeding cannot be identified [5]. The chest X-ray performed upright shows an air-fluid level; however, in approximately 10% of cases the X-ray performed upon admission may only show pneumothorax (as in our first case), whereas hemothorax appears later on. CT is performed in doubtful cases or to exclude other pathologies [2].

Initial treatment of SHP consists of resuscitation and placement of a chest drain. The chest drain is placed to achieve hemostasis by re-expansion of the lung; however, its success rate is not high [5] and most patients treated with chest drainage require subsequent surgical treatment [6]. Furthermore, the reduction in the amount of blood drained may be secondary not to hemostasis but to obstruction of drainage by a blood clot [5]. SHP can be treated conservatively if the bleeding subsides within 24 hours and in case of hemodynamic stability [8]. In cases of rapid clinical deterioration, lack of or reduced lung expansion due to collection of blood clots in the pleural cavity or rebleeding [2], early surgical intervention is indicated. Surgical intervention also prevents late complications of conservative treatment, such as residual hemothorax, empyema, fibrothorax [2,5]. Compared to conventional thoracotomy, VATS allows, due to better view of the pleural cavity, to identify and directly stop bleeding, to evacuate blood clots, to resect areas of dystrophic lung parenchyma and to

perform pleurodesis **[10].** Furthermore, VATS causes less surgical trauma and therefore leads to less postoperative pain, shorter hospital stay and quicker recovery of lung function. In our two cases, VATS surgery was performed early as recommended by recent medical literature **[2,5,9]**.

Conclusions

SHP is a rare pathology potentially life-threatening; it should be suspected in young male patients presenting with acute onset of chest pain and dyspnea, with or without signs of shock, and with signs of hemopneumothorax on chest X-ray. Prompt diagnosis and early surgical treatment are essential. Conservative treatment with drainage is only useful for the diagnosis and stabilization of the patient before surgery. VATS should be performed as soon as possible after the diagnosis of SHP.

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