CASE REPORT

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Post-Traumatic Loculated Haemothorax

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Abstract

Haemothorax is a collection of blood in the pleural cavity usually from traumatic injury. A chest X-ray has historically been the imaging modality of choice upon arrival to the hospital. If the size or severity of a haemothorax warrants intervention, tube thoracostomy has been and still remains the treatment of choice. Most cases of haemothorax will resolve with tube thoracostomy. If residual blood remains within the pleural cavity after tube thoracostomy, it is then considered to be a retained haemothorax (RH), with significant risks for developing late complications such as empyaema and fibrothorax. Once late complications occur, the only definitive treatment is surgery. In order to avoid surgery, research has been focused on removing an RH before it progresses pathologically. The most promising therapy consists of fibrinolytics, which are infused into the pleural space, disrupting the haemothorax, allowing for further drainage. If medical therapy and early procedures fail to resolve the RH, surgery is usually indicated. Surgery historically consisted solely of thoracotomy but has been largely replaced in non-emergent situations by video-assisted thoracoscopy.

Key words: Retained Haemothorax, VATS, intrapleural fibrinolytics, intra-fissure loculation.

Introduction

Blood in the pleural space is referred to as a haemothorax. The differential diagnosis for a patient who has not experienced substantial thoracic trauma is becoming more challenging due to an increase in medical problems and iatrogenic consequences[1].

Blood can enter the pleural space from a variety of vascular structures with consequences dependent upon arterial or venous source, the size of vascular injury, and localisation within lung, chest wall, mediastinum, diaphragm, or retroperitoneum. Fortunately, an invasive diagnosis to define the bleeding vessel is not required in all patients with haemothorax[2]. Yet that fact complicates efforts to define any cohort of specific diagnoses. A further complicating factor to any study of the anatomical causes of haemothorax is the tissue planes through which blood may pass before rupture into the pleural space.

Although the mesothelial cell has a fibrinolytic potential to convert blood clot to a liquid haemothorax rich in fibrin degradation products, transient clotting or lack of fibrinolysis following mesothelial cell injury may occur[1]. These blood clots are difficult to drain through even large-bore chest tubes.

Small amounts of blood in otherwise serous pleural effusions can cause red discoloration and prompt an incorrect diagnosis. For that reason, most clinicians define haemothorax as a pleural fluid haematocrit greater than or equal to 50% of the serum haematocrit[2].

There are 2 different physiological stages of haemothorax resolution: early and late[3]. Some have speculated that an early defibrinating of the haemothorax may occur with an increased pleural fluid protein concentration and a corresponding increase in intrapleural hyperosmotic pressure. This promotes the development of a pleural effusion[4-5]. As a haemothorax remains within the pleural cavity, it will typically complete spontaneous reabsorb within several weeks, especially if the volume is under 300 mL[6].

If it does not reabsorb, it will become a retained haemothorax (RH). RH has been defined as blood occupying at least one-third of the pleural space that cannot be drained by thoracostomy after 72 hours or as clots of at least 500 mL volume. RH can begin to form as early as 24 hours after chest tube placement[6].

Case history

A 44-year-old male patient with a history of chronic smoking presented to our department with complaints of gradually increasing right-sided chest pain which increased on inspiration, and occasional non-productive cough for the past 2 months. Through a comprehensive history it was

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established that patient the patient had suffered a road traffic accident 2 months ago for which an ICD (inter costal drainage) was placed because of haemopneumothorax. Patient's ICD was subsequently removed after resolution of the underlying condition and was discharged.

On examination, vitals were in range, bilateral chest movements were equal on inspection with dull note on percussion in the right infrascapular and axillary regions. Auscultation revealed decreased breath sounds on the right infrascapular and axillary regions. CXR (PA) done initially suggested a homogenous opacity in the right lower zone. Subsequent CECT chest was done and a loculated collection of size 8.7 x 8.6 x .8.0 cm with an approximate volume of 275 mL was seen with an air focus within the right posterior pleural cavity with adjacent atelectasis of right lower lobe

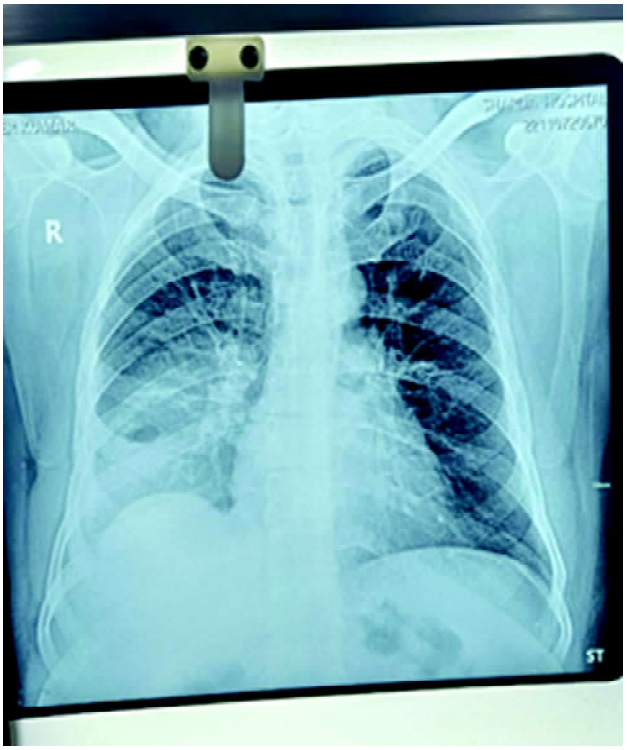

Fig. 1: *Pre-procedural X-ray.*

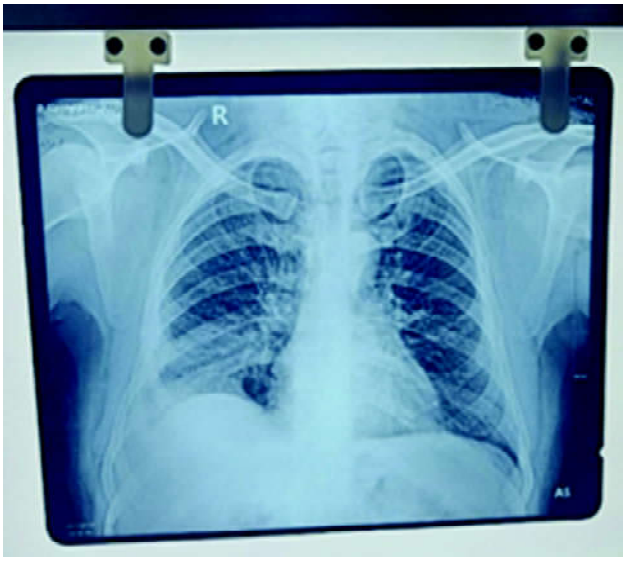

Fig. 2: *Post-procedural X-ray after ICD removal.*

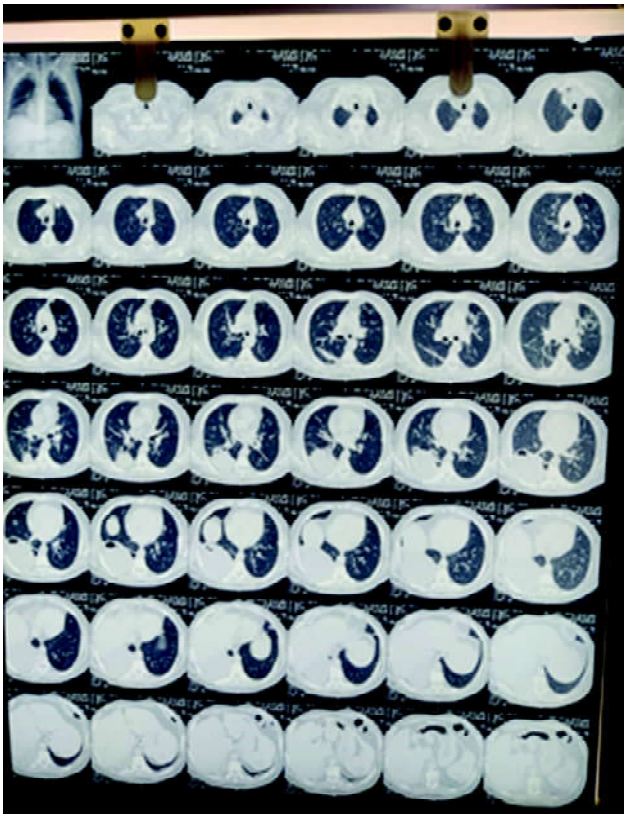

Fig. 3: *Pre-procedural CT scan.*

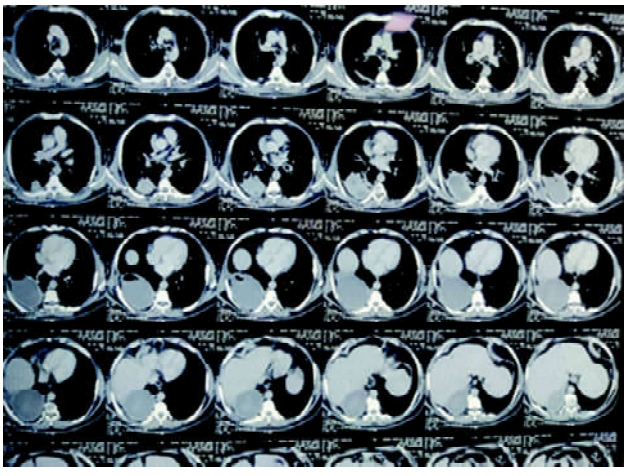

Fig. 4: *Pre-procedural CT scan localising the haemothorax.*

suggestive of right-sided hydropneumothorax.

USG guided aspiration was done which was able to retrieve around 5 mL of pleural fluid, which was sent for work up, but no NAAT based test could be done as the fluid received was haemorrhagic in nature. Patient was further planned for thoracoscopy with the intention of retrieving tissue for biopsy.

During the thoracoscopy, it was noted that there was a loculated haemorthorax within the the right oblique fissure. This loculation and surrounding fluid was evacuated and ICD was placed. At this point the patient's initial fluid reports were made available and had ruled-out tuberculosis. An HPE sample was taken and send for testing which too was in conclusive.

Patient had ICD in situ for subsequent three days, with a total fluid collection of 850 mL after which the ICD was taken out and he was discharged and asked to follow-up.

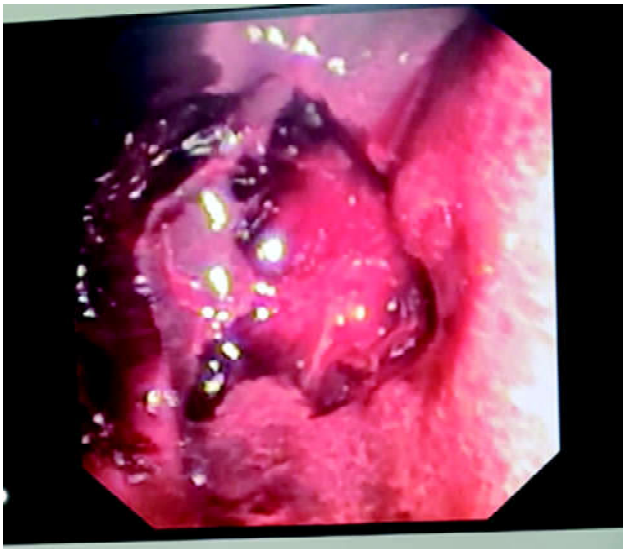

Fig. 5: *Loculated collection of blood in the right oblique fissure.*

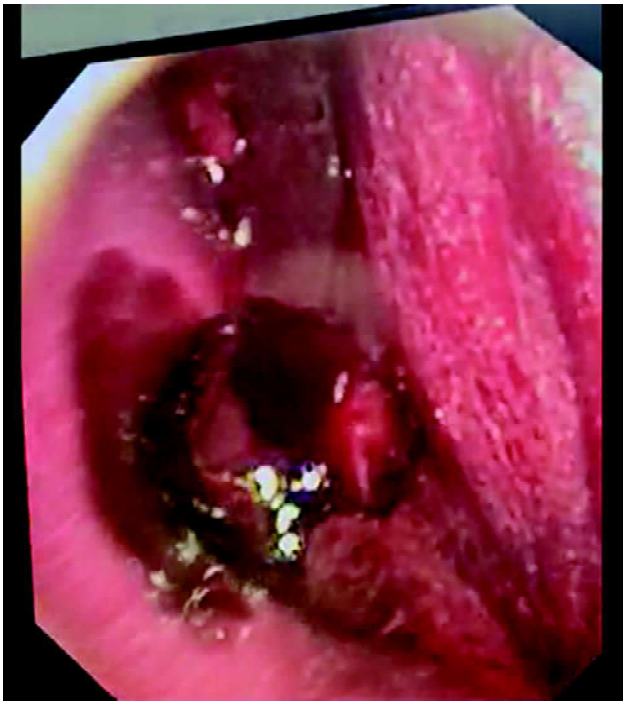

Fig. 6: *Post-evacuation and drainage of the loculated collection.*

Discussion

Haemothorax can be grouped into traumatic and spontaneous haemothorax, the former contributing to majority of cases. Within the spontaneous group, it can further be classified into vascular, coagulopathy, neoplastic and rarest of all are infectious. The differential diagnosis of a loculated haemopneumothorax occurring in a fissure should include an infected bulla; cavitary lung carcinoma, emphysema with congestive heart failure and tuberculosis[7].

RH can undergo progressive organisation over several days to become an empyaema or fibrothorax. Failure to evacuate the haemothorax may be due to malposition or poor drainage of the chest tubes, which can be influenced by the experience of the clinician. Empyaema can occur as a complication of RH due to primary or secondary bacterial contamination and can originate from broncho tracheal lesions, esophageal injuries, penetrating injuries, long-standing clotted thoracostomy tube, and postsurgical exposure[6].

The need for surgical drainage has been considered an important end-point in previous studies on pleuro-fibrinolysis; the latest meta-analyses concluded that fibrinolysis alone may prevent the requirement for surgical intervention[8].

Studies involving non-trauma patients have demonstrated the efficacy of intrapleural fibrinolysis in the management of a variety of complex pleural processes. In his study Carmen *et al*, divided patients in two groups – Group I: alteplase 20 mg in 20 mL saline every 24 h (or alteplase 10 mg in 20 mL saline every 24 hr) and Group II: urokinase 100,000 IU in 20 mL saline every 24 hr; showed that a maximum dose of 10,000 IU urokinase was far superior than alteplase[8].

Intrapleural fibrinolytic therapy with streptokinase has been used in clotted haemothorax with a success rate of 91% to 93%[9-10]. The early initiation of fibrinolytic therapy, before the development of severe pleural adhesions, may lead to a more effective pleural drainage as has been demonstrated in an experimental study[11] and in a study by Boures *et al*[12].

Unfortunately, there is no agreement on where and how intrapleural fibrinolysis should be incorporated into a treatment plan, and not all studies support its effectiveness in treating clotted haemothoraces. For the treatment of post-traumatic retained haemothorax, Ozgur *et al*[13], compared VATS with intraplerual fibrinolysis which was unable to show any clear dominance of VATS except possible shorter hospital stay.

However, Stile *et al* have shown intrapleural tPA is both safe and effective and should be included in the physicians' armamentarium for treating traumatic retained haemothoraces and recommend using intrapleural tPA in patients who present late, or in patients with low physiologic reserves.

Conclusion

Loculated haemothoraces may not always be because of trauma or malignancy, a differential for tubercular infection should always be kept in mind for such patients. A RH should also be dealt with through thoracoscopic means to avoid further fibrosis and empyaema formation.

Appropriate use of fibrinolytics and proper framework for the same has to be established and implemented where VATS is not readily available.

References

1. Charlie strange. Hemothorax. *Seminars in Respiratory and Critical Medicine* 1995; 16: 4.
2. Patrini D, Panagiotopoulos N, Pararajasingham J *et al*. Aetiology and management of spontaneous haemothorax. *J Thorac Dis* 2015; 7: 520-26.
3. Mahoozi H, Volmerig J, Hecker E. Modern management of traumatic hemothorax. *J Trauma Treat* 2016; 5: 1-5.
4. Cohen NS, Braig Z, Collins JN. Prevalence and management of post-traumatic retained hemothorax in a level 1 trauma center. *Am Surg* 2018; 84: e369-e371.
5. Inci I, Ozçelik C, Nizam O *et al*. Thoracic trauma in the elderly. *Eur J Emerg Med* 1998; 5: 445-50.
6. Zeiler J, Idell S, Norwood S, Cook A. Hemothorax: A Review of the Literature. *Clin Pulm Med* 2020; 27: 1-12.
7. Marin M, Kofler. Loculated Hemopneumothorax of a Major Fissure in Patients with COPD and Associated Pleural Disease. *Chest* 1990; 97: 87.1-76.
8. Nie W, Liu Y, Ye J *et al*. Efficacy of intrapleural instillation of fibrinolytics for treating pleural empyema and parapneumonic effusions: a meta-analysis of randomised control trials. *Clin Respir J* 2014; 8: 281-91.
9. Inci I, Ozcelik C, Ulkii R *et al*. Intrapleural fibrinolytic treatment of traumatic clotted hemothorax. *Chest* 1998; 1: 160-5.
10. Jerjes-Sa´nchez C, Ramirez-Rivera A, Elizalde JJ *et al*. Intrapleural fibrinolysis with streptokinase as an adjunctive treatment in hemothorax and empyema: a multicenter trial. *Chest* 1996; 109: 1514-9.
11. Kimbrell BJ, Yamzon J, Petrone P *et al*. Intrapleural thrombolysis for the management of undrained traumatic hemothorax: a prospective observational study. *J Trauma* 2007; 62: 1175-8.
12. Strange C, Baumann MH, Sahn SA, Idell S. Effects of intrapleural heparin or urokinase on the extent of tetracycline induced pleural disease. *Am J Respir Crit Care Med* 1995; 151: 508-15.
13. Samancilar O, Akçam I, Kaya SO, Ozturk O. The Efficacy of VATS and Intrapleural Fibrinolytic Therapy in Parapneumonic Empyema Treatment. *Ann Thorac Cardiovasc Surg* 2018; 24: 19-24.
14. Stiles, Drake, Helmer *et al*. Evaluation of chest tube administration of tissue plasminogen activator to treat retained hemothorax. *The Amer J Surg* 207 (6): 960-3.