CASE REPORT

Empyema Thoracis: A Rare Presentation

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SUMMARY

We present a case of spontaneous rupture into tracheobronchial tree of a large collection of pus in the chest during induction of anaesthesia. This event made active ventilation and oxygenation of the patient impossible till most of the pus was drained or suctioned. Prompt lateral positioning of the patient in this case with the diseased lung dependant is important to prevent the healthy lung from soiling.

Key words: Empyema thoracis; Hemithorax; Oxygenation.

INTRODUCTION

An empyema thoracis is simply a collection of pus in the pleural space. It is caused by an infection that usually originates in the lungs and spreads to the pleural space or it may spread from infected chest wounds and surgery, or a ruptured esophagus[1]. As the pus accumulates in the pleural cavity, it produces a pressure effect against the lungs, causing breathing difficulties and pain. The signs and symptoms of empyema thoracis vary according to the severity of the disease and the degree of ventilatory compromise e.g. fever, cough, fatigue, shortness of breath, chest pain and weight loss. When the empyema becomes severe, the abscess may burst and enter into the airways and/or spread to other tissues and organs such as the heart and brain[2]. It is with reference to these severe cases that we present this case of empyema thoracis, which burst open during attempt at ventilation after induction of the anaesthesia.

CASE REPORT

A ten year old girl weighing 25 kg was referred from NESCOM hospital with a prolonged history of severe degree of cough and a low grade fever. She had a past history of similar symptoms for which she had been diagnosed as pulmonary Koch's and had received anti-tuberculous treatment (ATT) for eight months in 2006. On readmission, the patient had a history of intermittent low grade fever, right sided chest pain and persistent productive cough. The patient was evaluated by the pediatrician and a diagnosis of empyema thoracis (right hemithorax) was made. (Fig 1-2)

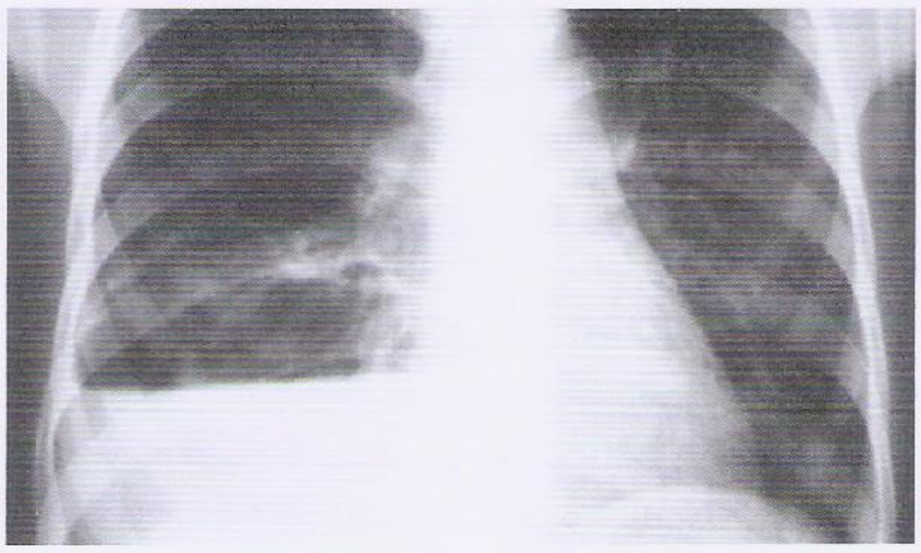

Fig 1 : Air fluid level in right hemithorax

Differential Count; Neutrophils 52%, Lymphocytes 42%, Monocytes 3%, Eosinophils 3%, Basophils 0%, ESR 52mm/hr

Pleural fluid: WBC 300/mm^3, total protein 0.9g/dl, Gram stain ve, AFB ve.

Sputum for c/s: Positive for growth of streptococci and E.coli

Other laboratory investigations were within normal limits.

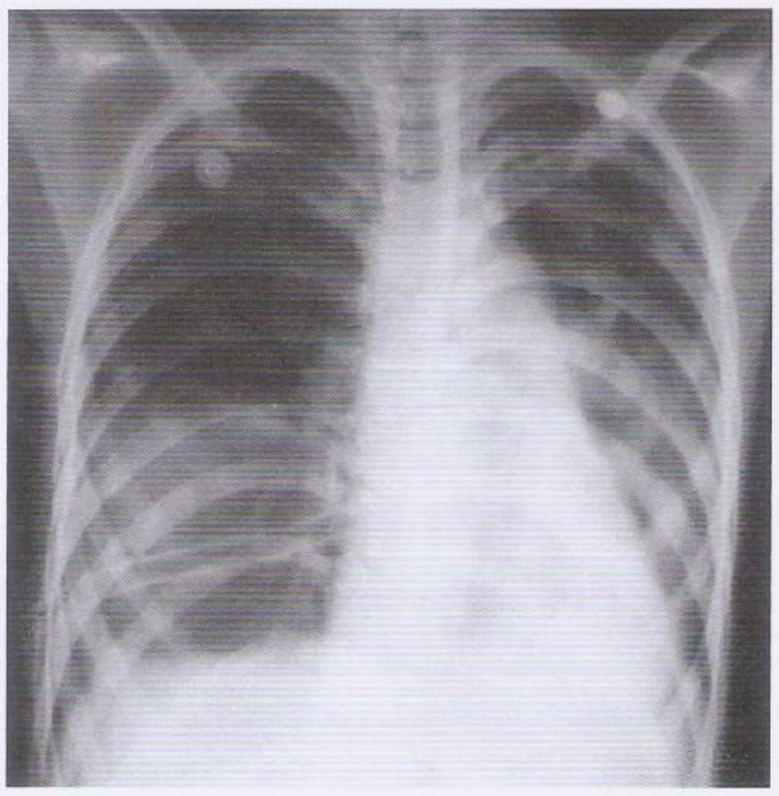

Fig 2 : Chest tube right hemithorax crossing midline to left side, left sided mediastinal shift, perihilar shadowing left lung.

CT scan was ordered and it suggested large air fluid cavity in right hemithorax with other possibility of a large lung abscess, bronchiectasis right lung, and carinal lymphadenopathy (Fig 4). The patient was referred to the pediatric surgeon for further management. The patient was seen by the pediatric surgeon and the surgery was planned.

The patient was premedicated with inj pyrolate 100µg and inj Decadron 4mg. Monitoring included pulse oximetry, NIBP, ECG, and $EtCO_2$. The patient was dyspnoeic while lying on the operating table, hence preoxygenation was done for more than 5 minutes. IV induction was carried out with inj propofol 100mg, relaxation was achieved with inj Acuron 15mg, and analgesia with inj Nalbin

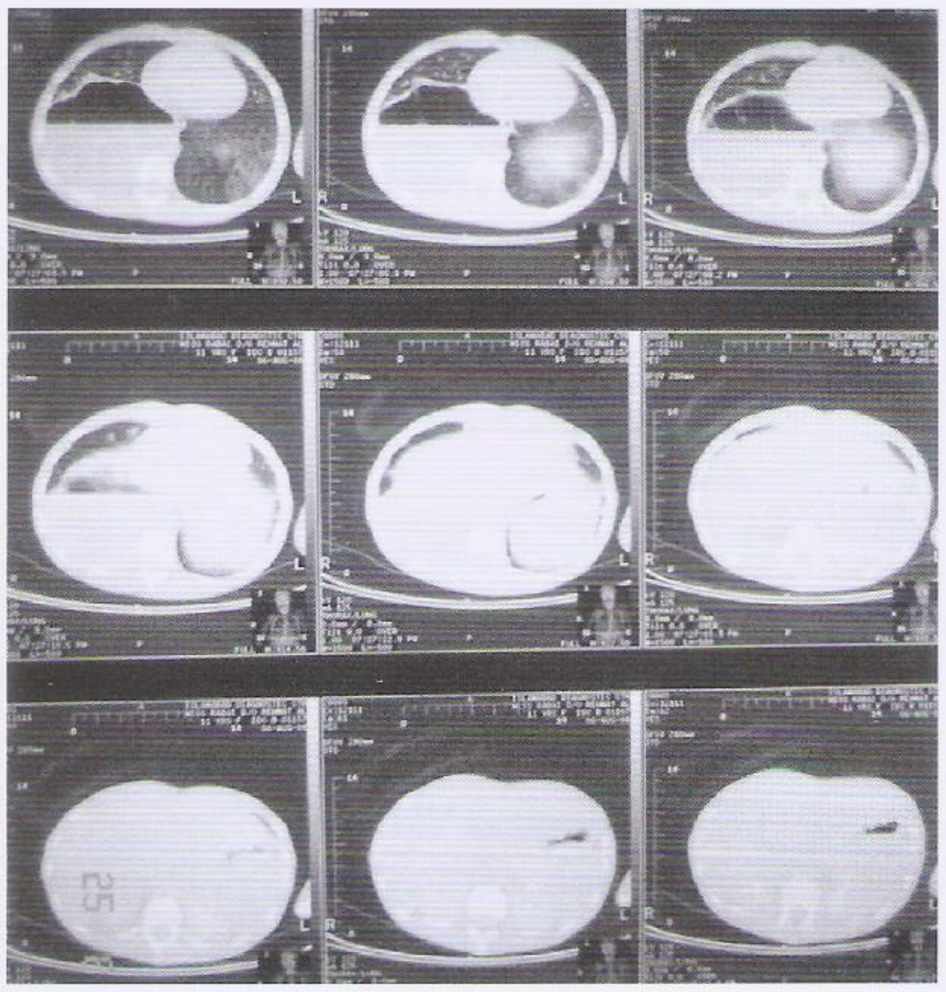

Fig 3 : Large cavity right hemithorax with air fluid level, intracavitary adhesions possibly due to hydropneumothorax/pyopneumothorax.

2mg with inj Gravinate 10mg IV. Once the anesthesia was deepened and the relaxant given, mask ventilation could not be carried out. There was no chest movement and no CO_2 trace on capnography. The SpO_2 which was in mid 90s with 100% inspired oxygen began to fall. When even after repositioning of the head and neck the ventilation could not be carried out satisfactorily, immediate laryngoscopy was performed for intubation. When the mouth was opened there was a sudden gush of pus from the oral cavity and the larynx could not be visualized. Patient was immediately placed in the right lateral position with head down. Suction was done during which there was continuous outpouring of pus and the SpO_2 began to fall. An endotracheal tube (ETT) was immediately passed through which the pus poured out like a tap. The suction catheter was now passed through the ETT and continuous suctioning was down. Ventilation at this stage was impossible. After some time the amount of pus pouring out reduced, so the anaesthesia circuit was attached and the ventilation with 100% oxygen started. The total amount of pus drained was about 400-500 ml. Once

the ventilation was successful, the saturation began to improve upto 98-99% and the process of anaesthesia was continued with a low level of sevoflurane in oxygen and nitrous oxide. The patient was reviewed by the pediatric surgeon on the operating table. Chest intubation was done with underwater seal drainage and the thoracotomy was postponed.

At the culmination of the surgery, the muscle relaxation was reversed and the patient was given a trial of spontaneous breathing. The patient was able to maintain her arterial saturation on room air so was extubated. Postoperatively the patient was shifted to pediatric ICU and put on inj Fortum and inj amikacin, analgesics, ATT, and regular chest physiotherapy. Her condition began to improve gradually and on the fifth postoperative day the chest tube was removed and the chest physiotherapy continued. The patient was active and was discharged to be managed in the out patient department by the pediatricians.

DISCUSSION

The term empyema is used to describe the presence of pus in the pleural space. The pus may be as thin as serous fluid or so thick that it is difficult or impossible to aspirate even through a wide-bore needle. Microscopically neutrophil leucocytes are present in large numbers. The causative organism may or may not be isolated from the pus. An empyema may involve the whole pleural space or only part of it ('loculated' or 'encysted' empyema) or most variably unilateral. The investigations necessary for its diagnoses are fluid and a pleural biopsy sent for bacteriological and histological examination.

The radiological appearances may be indistinguishable from those of pleural effusion. When air is present in addition to pus [pyopneumothorax] a horizontal "fluid level" marks the interface of liquid and air if the film is taken in the erect position.[2:3]

An empyema follows infection of the structures surrounding the pleural space; an infection solely of the pleural space probably does not occur. In the lung, empyema common follows pneumonia but may also be associated with lung abscess and bronchiectasis.

In some communities, trauma is the most frequent cause of empyema. During the First World War, and the early part of the Second World War, empyema occurred in 25-30% of all thoracic casualties but decreased to 6% among thoracic casualties in Vietnam. The frequent finding of resistant organisms in empyema fluid that follows chest trauma suggests that this may be associated with poor techniques of chest intubation rather than a contaminated weapon. An empyema may be associated with infection below the diaphragm as a liver abscess (pyogenic or amoebic) or a subphrenic abscess. Interestingly, empyema does not result in sub-diaphragmatic infection, probably because of the cranial direction of the lymphatics and the negative intrathoracic pressure compared to the abdominal cavity.[4-7]

Mediastinal infection may also result in an empyema. The most common example is rupture of the esophagus. Mediastinitis an empyema may also follow infections of the head and neck.[8]

The patient with an empyema may have variable symptoms difficult to distinguish from those of the primary provocation, whether this be 'pneumonia', mediastinitis, subphrenic abscess, or posttraumatic haemothorax. The patient has a wide spectrum of symptoms that may be virtually asymptomatic or may be frankly toxic, depending upon the causal organisms, the volume of pus within the pleural space and the host defense mechanisms. Other features result from occupation of the pleural space: dyspnoea; diminished movement of a hemithorax; dullness to percussion; diminished breath sounds;[2] and, if the empyema is large, displacement of the mediastinum to the opposite side.

The patient with a bronchopleural fistula gives a distinctive history. Whenever he lies on the side opposite to empyema he coughs excessively and the volume of pus produced is frequently large. The patient with a pointing empyema characteristically has

a discharging sinus or a chest wall swelling that may be confused with an abscess.[9-13]

Any delay in the correct management of empyema may lead to prolonged hospitalization or morbidity: the latter the stage of the empyema, the more difficult and prolonged it is to treat. Too much reliance on antibiotics and aspiration rather than well established principles of surgical intervention e.g. decortication[4], pulmonary resection, empyectomy or thoracoplasty may in fact worsen the situation. The underlying cause of empyema should be treated at the same time as the empyema.

The problem encountered with our patient was that the patient was quite dyspnoeic preoperatively and could not be put in recumbent position. Even with preoxygenation with 100% oxygen, the SpO_2 remained in mid 90s. After the IV induction and relaxation the patient could not be ventilated and the larynx could not be visualized due to frank pus. First, it was contemplated that it was the vomitus and perhaps the patient had a full stomach. There was some difficulty in performing tracheal intubation and even after the correct intubation for moments ventilation could not be carried out due to massive outflow of pus through the ETT and the SpO_2 gradually began to fall. However this could be well managed by alternate catheter suction and intermittent ventilation with 100% oxygen. Then the ETT being clogged, had to be changed and good ventilation reestablished with improvement of SpO_2 to late 90s. It is not understood how and why the pus containing cavity got burst into some main bronchus during manual ventilation. Perhaps the bronchial wall was so eroded to remain just like a thin septum and with the force of pushed oxygen into it, got burst. In these cases it becomes impossible to ventilate as all the upper airways are full of pus. The best strategy is to place the patient in the lateral position with the diseased lung dependant and wait till the spontaneous flow of the pus followed by active suctioning has drained bulk of the pus from the airways. The vital signs must be carefully monitored during this period and all arrangements made to cater for severe hypoxia as well as the possible cardiac arrest.

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