BILATERAL TENSION PNEUMOTHORAX AND PNEUMOPERITONIUM DURING LASER PEDIATRIC BRONCHOSCOPY

Case Report and Literature Review

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Abstract

We describe a case of a fifteen month old girl with a previous history of partially repaired congenital heart disease, presented for diagnostic bronchscopic evaluation of mid-tracheal narrowing. Intraoperatively, the surgical team decided to perform a transbronchoscopic laser resection of a granulation tissue over the previously placed airway stent. After repeated airway manipulations, the patient developed intraoperative bilateral tension pneumothoracis as well as tension pneumoperitonium. These complications were recognized, diagnosed and promptly treated and patient made full recovery.

This paper presents a case details, reviews the literature about these life threatening complications and suggests ways to prevent poor outcomes.

Introduction

Pediatric tension pneumothorax may occur under several conditions: (a) spontaneous pneumothorax of the newborn which occurs in 1% of vaginal deliveries especially with difficult ones complicated by meconium aspiration. (b) neonates with respiratory distress syndrome which causes a

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diseased lung parenchyma vulnerable especially if mechanical ventilation and post-end-expiratory pressure is used, (c) congenital diaphragmatic hernia which results in non-compliant underdeveloped lung ipsilateral to the defect in the diaphragm but the more compliant contralateral lung is prone to barotrauma and pneumothorax. (d) Iatrogenic pneumothorax occurs as an inadvertent consequence of diagnostic or therapeutic procedures[1].

Intraoperative tension pneumothorax and or pneumoperitoneum is extermely rare and is potentially a life-threatening event. This case describes an intra operative bilateral tension pneumothorax and pneumoperitonium during bronchoscopic tracheal laser surgery in a 15 months old girl.

Case History

A 15 months old girl weighing 7.6 Kg was scheduled for trans-bronchoscopic laser surgery for tracheal narrowing.

During the preoperative assessment a complicated past medical history was noted. At age of 7 months, the patient experienced chest infection which was resistant to routine treatment. A chest CT scan revealed complex congenital heart disease. During this scan the left pulmonary artery was noted to be arising from the right pulmonary artery and was wrapped around the trachea causing tracheal obstruction with right lung hypoplasia. In addition, an echocardiogram showed small patent ductus arteriosus with dextro cardia. At that time, rigid bronchoscope was done which revealed a normal tracheo- bronchial tree except for small patch of narrowing of left main stem bronchus inlet.

At age of 8 months, an operative repair of the congenital heart was performed at another hospital. With the use of cardiopulmonary bypass, reimplantation of the left pulmonary artery was performed with division and ligation of the patent ductus arteriosus. The procedure was complicated post-operatively with bilateral pneumothorax which was managed with chest tube insertion and the patient was discharged home tracheostomized, which was removed later on and the patient did well after that.

At the age of 10 months, she developed fever, cough and shortness of breath without improvement on antibiotics. CT chest revealed a long tracheal narrowing (8 mm x 2.7 mm) extending from side to side of the trachea at the level of jugulum to the level of the carina with right lung hypoplasia for which tracheal stenting under fluoroscopy was done and the patient was discharged to the pediatric medical ward in a fair condition breathing spontaneously.

At this presentation, at an age of 15 months, the patient presented to the emergency room with respiratory distress, fever, wheezy chest, coarse crepitations, and desaturation on pulseoximetry (80-85%). Arterial blood gas on room air showed pH of 7.28, PO_2 63 mmHg, PCO_2 56 mmHg, HCO_3 21.mmol/l. Patient was intubated with 3.5mm endo-tracheal tube and was put on pressure controlled ventilation in the PICU. A-diagnostic bronchoscope was planned.

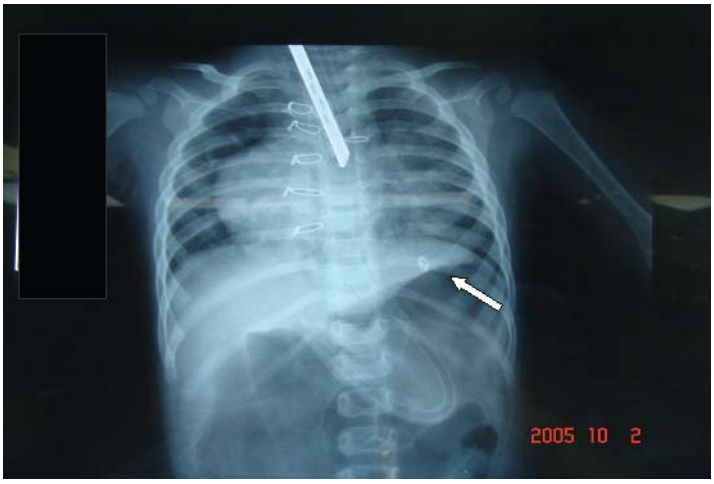
In OR, monitoring revealed HR: 160/min, NIBP: 90/50 mmHg and O_2 saturation: 94%. The endotracheal tube was connected to the anesthesia circuit with O_2 4 litres per minute, sevoflurane 4-5 vol %, following administration of IV atropine 140 mcg and propofol 2mg/kg with establishment of assisted manual ventilation. For airway assessment, the flexible fibroptic bronchoscope size 2.7 mm introduced through the ET tube 3.5mm discovered stoppage of ET tube at the level of obstruction (about 2 cm below the vocal cords) and it could not be pushed through this narrowing. The narrowing extended longitudinally up to the carina due to presence of granulation tissue over the stent lumen. A KTP laser was used for ablation of this obstruction using the apneic technique with intermittent IV dose of propofol (each = 7mg). During this stage of the procedure, the patient vital signs were stable (HR = 150/min, B/p = $^{80}/_{50}$ mmHg ad O_2 sat 94-97%).

Following laser surgery, a pointed nasal swab soaked with mitomycin mounted over a forceps cup was applied to the ablated raw areas. This swab was lost inside the airway distal to previously narrowed site. Intra-operative chest radiograph was performed, during rigid bronchoscopy, which showed the swab to be within the left lower lung parenchyma (Fig. 1). Shortly after

the surgical team tried to retrieve it with several trials.

Fig. 1

Intraoperative protable x-ray during rigid brochoscopy showing dextocardia, evidence of previous sternotomy, the intra-parenchymal foreign body (arrow)



Minutes later, the patient experienced rapidly progressive O_2 desaturation (O_2 sat 30-40%), bradycardia (HR 70-80 bpm), blood pressure and expired carbon dioxide were unrecordable, and the skin was mottled allover. Patient was immediately re-intubated with ET tube 2.5 mm and on manual ventilation with O_2 100% 4 LPM, high resistance for manual bagging was immediately obvious. On quick general inspection, the chest was noted to be bulged immobile bilaterally, and clinical percussion revealed a hyper-resonance all over the chest. On auscultation, there was distant amphoric breath sounds. Tension pneumothorax was clinically suspected. This was followed by a rapid insertion of 18 G needle in both second right and left intercostal spaces in the mid clavicular line with a gush of air through needles. O_2 saturation started to increase (90-94%), HR increased spontaneously to 160/min, BP was now recorded at $^{60}/_{30}$ mmHg, and End-Tidal CO_2 reached 60mmHg. Manual bagging was started to effectively inflate the chest easily.

This temporary improvement was followed by progressive abdominal enlargement with hyper resonance on percussion (Fig. 2-A).

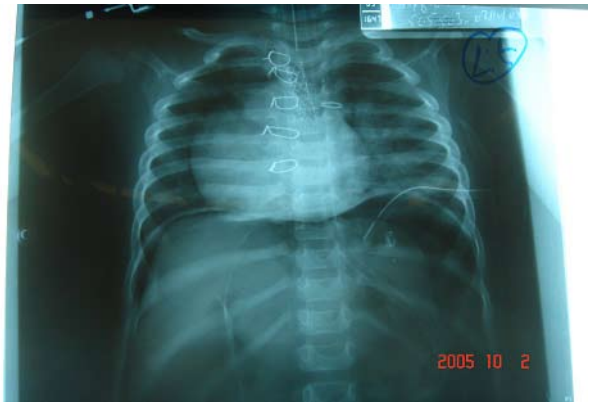
Fig. 2A

Intraoperative photograph showing acute abdominal distension

Intra-operative fluoroscopy confirmed the diagnosis of bilateral tension pneumothorax and pneumoperitonum with a ribbon shaped mediastinum and air under the diaphragm. X-ray of chest and abdomen (Fig. 2-B) confirmed the clinical diagnoses.

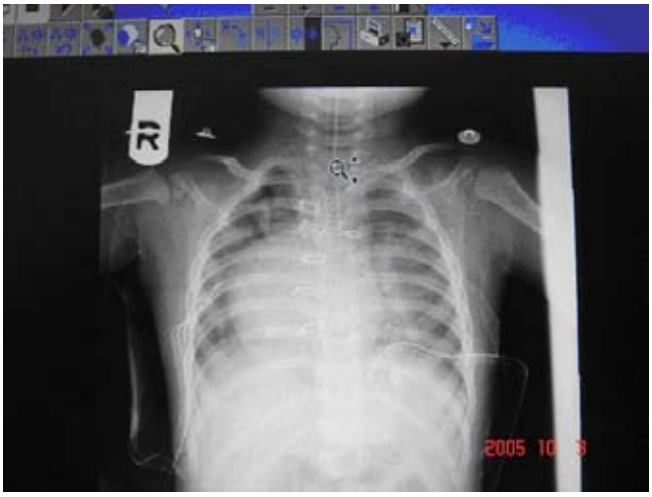
Fig. 2B

Follow-up intra-operative portable x-ray showing bilateral chest tubes and acute pnuemoperitonum



Bilateral chest tubes were inserted immediately in the 5th intercostal space midaxillary line and connected to a draining system. The pediatric surgeon decided to insert a peritoneal tube (actually chest tube 5Fr) at the level of the umbilicus on the right side and jet of air also was detected. At this time O_2 sat reached 100% and the rest of the vital signs were back to baseline levels (Fig. 3).

Fig. 3
Postoperative chest x-ray showing resolution of the bilateral pneumothoraces and the pneumoperitoneum



The patient was transformed to the PICU and was put on pressure controlled ventilation. Chest and peritoneal drainage tubes were removed after 6 days and patient was discharged to the ward after 10 days in a good general condition and normal arterial blood gas analysis.

Discussion

Pneumothorax in the mechanically ventilated patient may present as an acute cardiopulmonary emergency and if unrecognized and untreated, it will progress to cardiovascular collapse.

In one report of 74 patients, the diagnosis of pneumothorax was made in 45 (61%) patients on the basis of hypotension, hyperresonance,

diminished breath sounds, and tachycardia[3]. The mortality rate was 7% in these patients receiving the immediate clinical diagnosis. In the remaining 29 patients, diagnosis was delayed between 30 minutes and 8 hours and 31% of these patients died of pneumothorax. Other series of barotraumas in the setting of mechanical ventilation have reported mortality rates of 58% to 77%[4-7].

Tension pneumothorax is lethal if diagnosis and treatment are delayed. The diagnosis should be made clinically at the OR table for the patient receiving mechanical ventilation who shows a sudden desturation and cardiovascular collapse. The diagnosis may be a problem in both the anesthetized patient and patient with bilateral tension pneumothorax, which maybe more protective of the mediastinal structures and may lessen the impact on cardiac output[8]. This is what probably happened in this case which experienced severe desaturation (O_2 sat: 30%) without drop of HR below 80/min and BP was 50/30 mmHg but with the occurrence of pneumoperitonium (known by progressive enlargement of the abdomen), the blood pressure started to be un-recordable due to further impedance of the venous return.

In this case several precipitating factors were suspected as leading events to the development of the noted complications: These were related to the patient, such as the right hypoplastic lung detected by CT chest, the bilateral pneumothorax experienced after the previous cardiac surgery period, the post-stenotic retained bronchial and bronchiolar secretions and the non-obliterated foramena of Bochdalek and Morgagni which were the channel of air escape from thorax to the peritoneal cavity causing pneumoperitonium. Other suspected precipitating factors were iatrogenic, which might be due to the pattern of manual ventilation during transportation from the PICU to the OR and intraoperatively, airway injury by the laser surgery and the use of the cup forceps searching for the missed pointed nasal swab.

Pressure in the pleural sac is usually subatmospheric throughout the normal respiratory cycle, averaging -9mmHg during inspiration and -5 mmHg during expiration. Because of lung elasticity, pressure in the airway

is positive during expiration (+3 mmHg) and negative during inspiration (-2 mmHg) due to diaphragm downward and chest wall outward movements. Thus, in normal breathing, airway pressure is greater than pleural pressure throughout the respiratory cycle.

Airway pressure may be increased markedly with coughing or strenuous exercise; however, pleural pressure rises concomitantly so that the transpulmonary pressure gradient is usually not substantially changed. When there are rapid fluctuations in intracthorasic pressure, however, a large transpulmonary pressure gradient occurs transiently[8]. In this case, bronchial and bronchiolar obstruction by the post stenotic secretions, resulted in airtrapping, with substantial increase in the transpulmonary pressure gradient. The alveolar walls and visceral pleura maintained the pressure gradient between the airways and pleural sac but in the presence of the positive pressure ventilation, the pressure gradient was transiently increased and alveolar rupture occurred and air entered the interstitial tissues of the lung and pleural space resulting in pneumothorax.

The right hypoplastic lung could not accommodate the positive pressure ventilation and barotrauma occurred to the ipsilateral hypoplastic lung and the contralateral one due to over inflation and this may explain the bilateral pneumothorax developed. By measuring the static lung compliance of the patient (normally, 50-80 mL/cm H_{20}) before transport to OR, the ventilating technique during transport should be adjusted. If this patient who had low compliant lung due to both retained bronchial and bronchiolar secretions and right lung hypoplasia was subjected to either large tidal volume breaths, it would cause barotrauma or to very rapid breaths, it would cause air trapping with subsequent alveolar rupture. In this critical situation it would be difficult to follow the recommended ventilatory pattern during the whole transport time for this patient with poor compliant lung[9].

The laser induced injury was suspected to be an associated cause of several cases of pneumothorax and subcutaneous emphysema during laryngeal laser surgery caused by unobserved laser damage[10,11].

Pneumoperitoneum followed the decompression of intrathoracic air

into the abdomen. The presumed mechanisms might be either escape of pressurized air through a diaphragmatic hiatus from the induced pneumo mediastinum into the retroperitoneum, into the bowel mesentry, and through the subserosal surface of the bowel with subsequent leak into the peritoneal cavity. Or, air forcing its way from the pleural sac to the peritoneum through the foramena of Bochdalek and Morgagni[12].

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