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BILATERAL PNEUMOTHORAX AND CARDIOGENIC SHOCK FOLLOWING RIGHT UPPER LOBECTOMY FOR LUNG CANCER IN A PATIENT WITH COPD [A CASE REPORT]

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Summary: COPD is a common respiratory problem amongst the masses and when these patients present for a surgical procedure, they may require high tidal volumes, generating high inspiratory pressures during one lung ventilation with an increased probability of lung injury, particularly in patients with lung carcinoma resulting in a bad prognosis. We report the case of a 68-year old, thin built, healthy looking gentleman, a known case of chronic obstructive lung disease [COPD], who developed subcutaneous emphysema, bilateral pneumothorax and cardiogenic shock after a right upper lobectomy performed for a non-metastatic small cell carcinoma of the lung. We believe that if the preoperative predictors of postoperative prognosis had been taken seriously, and option of non-operative treatment been given, may be we would have avoided the catastrophic end.

Key Words: COPD, Pneumothorax, Cardiogenic shock

A pneumothorax can occur in the presence of a pleural drain if there is a bronchopleural communication and the chest drain gets occluded (e.g. blood or kink) or if there is a malfunction with the

underwater seal drain apparatus. This may progress to a tension pneumothorax, especially if the patient is being ventilated with IPPV or PEEP. COPD can also contribute to surgical emphysema, pneumothorax and hypoxemia even without IPPV as occurred in our patient.

Case Report: A 68-year-old, 55 kg, thin built, 165cms tall, active man was admitted to the hospital for a right-sided upper lobectomy for a solitary pulmonary nodule in the right upper zone with no evidence of metastasis. He was a chronic smoker and a known case of chronic obstructive lung disease [COPD]

His preoperative assessment was normal, except for clubbing of all the fingers, bilateral expiratory wheeze, hypoxia [PaO2 69mmHg], hypercapnia. [PaCO2 53 mm Hg.] and FEV1 of <35%, on room air. After the bronchodilator therapy the FEV1 increased to 66%, FVC was: 41% and FEV1/FVC ratio was 49%. He had no evidence of metastasis.

The bronchodilators used during the preoperative and postoperative period were: Duolin respules [2.5ml] 6 hly [Salbutamol 2.5mg+ Ipratropium Bromide 0.5mg]; Seroflow 250mcg inhaler 2 puffs BD[Salmetrol 2.5mg + Flucicasone propionate 250mcg] and Tab. Deriphyllin Retard 150mg BD [Etophylline 115mg + Theophylline 35mg].

The patient underwent Rt. upper lobe resection under one lung ventilation, using Robertshaw's Lt. DLT [size 37], during general anesthesia using preset protocols for intra-operative monitoring and thoracic epidural analgesia. Care was taken to ventilate with low tidal volumes and low inspiratory pressures keeping in view the presence of COPD.

Intraoperatively and postoperatively after extubation, the patient maintained stable vitals. He was comfortable and pain free [VAS-3] at rest with an epidural Infusion of bupivacaine [0.125%] and fentanyl [10mcg/ml] infused @ 5 -7 ml/hr. The surgery lasted for two hours. Estimated blood loss during operation was approximately 400 ml.

Six hours after transfer to MICU, the patient developed respiratory distress along with restlessness. On examination the patient showed right-sided surgical emphysema [on the surgical side] up to the neck. Portable chest X-ray revealed right-sided pneumothorax. The thoracic surgeon replaced the rt. sided chest drains and placed another chest tube in the second intercostals space through a separate rt. sided incision under local anesthesia

Four hours later, the subcutaneous emphysema increased to involve the left side of the chest also. ABG showed severe respiratory acidosis.[Table-1] Repeat X-Ray chest showed a left sided pneumothorax for which a left sided chest tube was also inserted. The

patient was injected with a bolus dose of fentanyl for pain relief during chest tube insertion following which the patient had a respiratory arrest. He was intubated and ventilated.

Eight hours later the condition of the patient further deteriorated. He developed moderate hypotension for which large doses of vasopressors. [Dopamine and Nor-adrenaline] were needed to maintain a MAP of 70mmHg. In the mean time the epidural infusion had been stopped and patient was on intravenous fentanyl @25-50mcg/hr.

Bedside ECHO was done. It revealed a poor echo window, normal chamber size, lateral wall akinesia with hypokinesia of left ventricle segment, restriction of left ventricle filling and LVEF of 25 %. The cardiac enzymes were also elevated; suggestive of acute myocardial infarction with cardiogenic shock. The patient was started on heparin therapy and anti platelet drugs. An intra-aortic balloon pump [IABP] was also needed to maintain the cardiac output. The patient developed multiple organ dysfunction [MOD] and acute renal failure [ARF] for which he was dialyzed. His pupils became bilaterally dilated and fixed. He died two days after the surgical procedure.

Discussion: The incidence of early postoperative acute cardio respiratory failure and death after lung resections and lung cancer has been reported to be 1.2% – 1.85%[1,2]

COPD has been recognized to be a significant risk factor for development of respiratory-related complications like atelectasis, pneumothorax, pneumonia, bronchopleural fistula, empyema and acute respiratory distress syndrome[3]

Occasionally, the development of tension pneumothorax may be delayed for hours to days after the initial insult, and the diagnosis may become evident only if the patient receives positive-pressure ventilation.[2,3]

The occurrence of bilateral pneumothorax during the postoperative period in our patient who was a chronic heavy smoker may be relegated to the pre-existence of an emphysematous chest and COPD. Intraoperative IPPV, although instituted with guarded low tidal volume upto 250 ml might still have led to the hyperinflation of the alveoli that resulted in spontaneous pneumothorax.

Bilateral pneumothorax leads to the worsening of hypoxia and compromises the venous return. Researchers are still debating the exact mechanism of cardiovascular collapse after a pneumothorax. They believe that the condition develops from a combination of mechanical and hypoxic effects. In either event, decreasing cardiac output and worsening metabolic acidosis secondary to decreased oxygen delivery to the periphery induces anaerobic metabolism. If the

underlying problem remains untreated, the hypoxemia, metabolic acidosis, and decreased cardiac output lead to cardiac arrest and death.

Severe obstructive pulmonary disease also predisposes the patients to the development of prolonged air leak [PAL] after radical upper lobectomy.[4] Acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) after pulmonary resection in males over 60 years of age who undergo resection for lung cancer has been reported to be 3.9%.[5] ALI along with myocardial infarction seems to be the cause of death in our patient.

Table 1: Arterial blood gases of the patient during preoperative, intraoperative and postoperative periods

	pH	**PCO2 (mmHg)**	**PO2 (mmHg)**	**HCO3**	**SaO2**
Preoperative [FIO2-0.21]	*7.44*	*53*	*69*	*36*	*90%*
Intraop. *[OLV]	*7.24*	*62*	*68*	*26.3*	*90%*
Intraop. **[DLV]	*7.34*	*50*	*167*	*27*	*99%,*
1st P.O. 2hrs. [Nasal O2 @2L/min]	*7.34*	*50*	*170*	*26*	*100%*
1st P.O. at 6hrs	*7.05*	*79*	*129*	*17.3*	*97%*
1st P.O. at 8hrs	*6.99*	*66*	*99*	*23.9*	*94%*
1st P.O. at10hrs	*7.02*	*110*	*98*	*21*	*94%*
2nd P.O. day	*7.06*	*110*	*55*	*21*	*79%*

OLV: during one lung ventilation, **DLV: during double lung ventilation, P.O: postoperative

Implications: COPD patients may require high tidal volumes, generating high inspiratory pressures during one lung ventilation with an increased probability of lung injury, particularly in patients with lung carcinoma. "Understanding the pathogenesis of lung damage may enable the anesthesiologist to modify the process and decrease the incidence and severity of the problem"[6]

Conclusion: It was unfortunate that the patient did not recover from the progressive events following the lobectomy.

On retrospective analysis and review, we believe that there were preoperative predictive values [as mentioned below] for poor postoperative outcome but both the anesthesiologists and the surgical team seemed to ignore them unknowingly because the tumor mass was small and appeared to be easily resectable.

Moreover, the patient was lean and thin, not dyspnoic, non-diabetic and non-hypertensive. He had quit smoking a few months back and looked very comfortable at rest and walking at level.

Preoperative predictors of poor outcome were:

FEV1 : <35%
PaO2 on room air : 69%
PaCO2 on room air : 53 mm Hg.
Stair Climbing <2 flights: Signifying VO2, max. between 10-15ml/kg/min.

It appears that this patient had theoretically destroyed his lungs from a lifetime of smoking. The radiological picture of the lungs was, however not very remarkable of COPD. The positive pressure ventilation in a patient with such poor lungs during anesthesia and after bilateral pneumothorax posed a threat of hypoxemia. Additionally, it was likely that decades of smoking had left the patient with a vulnerable heart, which was not discovered preoperatively. The combination of a heart and lungs destroyed by smoking, the surgical insult of a lobectomy, and the post-operative stress of mechanical ventilation following spontaneous rupture of an emphysematous bulla on the surgical side and suspected tension pneumothorax on the other side was more than the patient could handle.

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