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# Acute Pneumothorax After Induction of General Anaesthesia

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## Abstract

pneumothorax Acute occurring under general anaesthesia with positive pressure ventilation especially when using nitrous oxide can rapidly become life-threatening tension pneumothorax. As the diagnosis is often difficult or delayed in this situation, a high index of suspicion and clinical examination aided by monitors are necessary for the diagnosis, which may be confirmed by radiography or ultrasonography. Immediate stoppage of nitrous oxide with prompt release of the pneumothorax by needle intercostals aspiration or drainage is needed to save the life of the patient. This is a case report of an un-anticipated acute pneumothorax which occured soon after the induction of general anaesthesia, which was diagnosed and managed successfully in time.

Keywords:AcutePneumothorax;GeneralAnaesthesia; Nitrous Oxide.

### Introduction

Pneumothorax is air in the pleural cavity which can occur unexpectedly under general anaesthesia. Diagnosis may be difficult or delayed due to the limited access to chest and the non-specific early clinical signs. Positive pressure ventilation and use of nitrous oxide can make it into a tension pneumothorax quickly. Hence early detection and prompt management is necessary to save the life of the patient.

## **Case Report**

A 52 year old housewife presented with neck pain radiating to the left upper limb of 3 months duration. MRI scan showed subluxation of the C5-C6 cervical vertebrae with radiculopathy, (Figure1) for which she was posted for anterior cervical discectomy and fusion. She was weighing 60 Kg, with no other significant medical problems. She had a short neck with cervical collar and airway was of modified Mallampatti Class III. Routine investigations including chest radiograph and electrocardiogram were normal.



**Fig. 1:** MRI scan of neck showing subluxation of C5-C6 cervical vertebrae

In the operation theatre baseline heart rate 86/ min, blood pressure 130/84 mm Hg and SpO<sub>2</sub> 100% on room air were recorded. Intravenous premedication was given with glycopyrrolate 0.2 mg, midazolam 1 mg, ondansetron 4 mg and fentanyl 100µgm. After preoxygenation, general anaesthesia was induced with propofol 120 mg followed by lignocaine 100 mg and succinvlcholine 100 mg. Endotracheal intubation was done by 7 mm cuffed flexometallic tube with the help of bougie and bilaterally equal air entry was confirmed.

Capnograph showed normal tracing with end-tidal carbon dioxide (EtCO<sub>2</sub>) of 34mm Hg. Vecuronium 4 mg was given intravenously to maintain neuromuscular blockade and anaesthesia maintained with IPPV using oxygen, nitrous oxide and isoflurane using workstation in volume control mode at a rate of 12/min, tidal volume 500ml, I:E ratio 1:2, PEEP 3 cm H<sub>2</sub>O. Intravenous infusion of dexmedetomidine at a rate of 20

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µgm per hour was started for analgesia.

Within 5 minutes, there was sudden drop in SpO<sub>2</sub> to 88%, EtCO<sub>2</sub> to 28 mm Hg with tachycardia of 134/min and drop in BP to 110/74 mm Hg. Immediately nitrous oxide and isoflurane was stopped and manually ventilated with 100% oxygen. The reservoir bag was feeling tight with markedly diminished air entry over the left side. As the clinical condition was worsening the surgical procedure was deferred and patient was transferred to Intensive Care Unit with ambu bag ventilation using oxygen enriched air. Clinical reassessment showed hyper-resonant left hemithorax with obliterated cardiac dullness and absent air entry. The patient was put on mechanical ventilator in volume control mode with 100% oxygen at a rate of 12/min, tidal volume 500ml, I:E ratio 1:2, PEEP of 0 cm H<sub>2</sub>O with peak airway pressure limited to 24 cm H<sub>2</sub>O. Chest radiograph showed massive pneumothorax on the left side with mediastinal shift to the right (Figure 2). A 12 lead ECG taken was unremarkable, except for tachycardia.



Fig. 2: Chest radiograph showing pneumothorax on the left side with mediastinal shift to the right

Immediately intercostal drainage (ICD) tube was inserted into the left pleural cavity and connected to underwater seal, which showed air bubbling, following which the clinical condition improved. Emergency fiberoptic bronchoscopy was done, which did not detect any airway injuries.

In three hours, the vital status improved,  $SpO_2$  picked up to 95%, and air entry was better. Repeat

radiograph confirmed the position of ICD tube and showed re-expansion of the lungs. Patient was extubated after six hours and ICD was removed after three days, when the lungs were fully expanded, as confirmed by radiograph. The patient was discharged from ICU with advice for High Resolution Computerized Tomography (HRCT) before taking up for surgery later.

#### Discussion

Pneumothorax occurring under general anaesthesia is life-threatening as it can rapidly turn into a tension pneumothorax. During positive pressure ventilation air enters into the closed pleural cavity, causing collapse of the lungs, which is worsened by the rapid diffusion of nitrous oxide causing mediastinal shift resulting in cardiovascular collapse. Prompt diagnosis and urgent insertion of intercostals drainage tube is often needed to save the life of the patient.

Pneumothorax during anaesthesia can occur due to rupture of bullae, violent cough causing airway tear, alveolar rupture or rupture of esophagus. Iatrogenic causes may be due to application of high peak inspiratory pressure (>40 cm H<sub>2</sub>O), large tidal volume and high positive endexpiratory pressure (PEEP). Trauma during intubation, overinflation of ETT cuff, stylet protruding beyond ETT and movement or coughing during intubation or extubation are other contributing factors. In patients with reduced lung compliance, IPPV can produce barotraumas even without application of high airway pressures. Surgical complications or diagnostic procedures like central venous cannulation or brachial plexus block can also result in pneumothorax.

Signs of pneumothorax like fullness of hemithorax, absent breath sounds, distended neck veins with tracheal shift are often difficult to detect under general anaesthesia. However, tachycardia, hypotension, narrowed pulse pressure and arterial desaturation should raise the suspicion especially when airway pressures are raised. With small pneumothorax, clinical signs are minimal and chest radiograph or ultrasongraphy may be inconclusive. CT scan can differentiate pneumothorax from bullous lung disease. In some cases, HRCT of the thorax may be needed for the correct diagnosis.

*Management* : Small pneumothorax in a spontaneously breathing patient without significant

breathlessness and hemodynamic instability can be observed for spontaneous resolution. As nitrous oxide diffuses into air, it should be immediately stopped and high concentration of oxygen has to be given which can speed up the resolution of the pneumothorax by reducing the partial pressure of nitrogen in the pulmonary capillaries increasing the absorption of air from the pleural cavity.

In those with minimal signs, aspiration can be tried using a cannula inserted into the pleural cavity in the fourth inter-costal space. If aspiration is unsuccessful or in symptomatic patients, intercostals drainage tube should be inserted and connected to underwater seal and retained until bubbling of air has ceased. Follow up with chest radiography is needed for confirmation of the position of the tube and for lung re-inflation before removal of the ICD.

## Conclusion

Critical incidents often occur in anaesthetic practice in un-anticipated situations, which can be

devastating and life threatening. Pneumothorax is one among them, in which early diagnosis and prompt management can often save the life of the patient.

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