

TENSION PNEUMOTHORAX AT LAPAROSCOPIC GYNAECOLOGICAL SURGERY

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Pneumothorax after laparoscopy was first described in 1939 but it remains a rare complication and the incidence is reported as 0.01% to 0.4%.^{1,2} This can progress into tension pneumothorax which if not detected and treated early can result in cardiac arrest and death. The treatment ranges from simple close observation to invasive interventions like chest decompression with intercostal (IC) tubes but in an emergency needle thoracostomy may be life saving. This is a case of perioperative tension pneumothorax after pneumoperitoneum at a laparoscopic gynaecological procedure in a young healthy female with primary subfertility. Vigilant monitoring and early rational intervention prevented probable threat to life and she was able to undergo surgery with no further complications.

A 39 year old lady, ASA 1, was given a general anaesthetic for an elective laparoscopic dye test and adhesiolysis for primary subfertility. It was a routine general anaesthetic of benzodiazepine premedication, standard monitoring, an uneventful intravenous induction with propofol, pethidine and atracurium and endotracheal intubation with an internal diameter 7.5 mm cuffed tube. Anaesthesia was maintained with halothane (0.5%) in 2:1 nitrous oxide to oxygen via circle circuit and ventilated (volume control) with a tidal volume of 7ml/kg, PEEP 3 cmH₂O at 12 breaths per minute. She was placed on lithotomy 30° Trendelenburg position for a clear surgical field.

She was stable after the induction. Despite Carbon dioxide (CO₂) insufflation, there was no significant rise in intra-abdominal pressures initially (remained <6 mmHg) until almost 4 litres had been administered (normal average volume

required is 2 litres).³ Subsequent rise of intra-abdominal pressure to beyond 12 mmHg resulted in a concurrent drop of arterial saturation from 100 to 95%. This was soon followed by a tachycardia of 90-100 beats per minute and a drop of systolic blood pressure from initial 110 to 80 mmHg.

Vigilant monitoring led to the early detection in parameter change with immediate symptomatic therapy while looking for a cause. Senior help was called in while patient was manually ventilated on 100% O₂. Surgeons were informed and the procedure was temporarily halted while ephedrine boluses were given to improve blood pressure. Increased existing intravenous (iv) fluid infusion was augmented with a supplementary colloid via a second large bore cannula inserted into a large forearm vein.

Quick systematic examination of the patient revealed that there was a relative hyperinflation of the right hemithorax with reduced chest movements and a tracheal shift to the left. The percussion was hyper-resonant on the right side with minimal breath sounds without wheezing or crackles. Ventilator parameters showed an increased airway pressure from initial 15 to 27 cmH₂O for the same tidal volume ventilation. The endotracheal tube was in the original place of 18 cm incisor level.

On clinical grounds 'tension pneumothorax' was suspected and manual ventilation was started with reduced tidal volumes at a higher frequency while deflating the pneumoperitoneum to around 8 mmHg. This prevented further deterioration of the parameters. As haemodynamic instability persisted, needle thoracostomy was performed

with a large bore intravenous (iv) cannula (G17 changed over to a G14) and was connected to a 5 ml syringe barrel half filled with saline at the 2nd intercostal space at mid clavicular line. This resulted in initial 'whooshing' sound of air exit and subsequent copious bubbling. This improved saturation, haemodynamic parameters and the lung compliance. Thereafter surgery was continued with meticulous attention at keeping intra-abdominal pressures below 12 mmHg. As the patient was stable, insertion of an intercostal tube (IC) was deferred while preventing tension development with an improvised system of needle thoracostomy (G 14 iv cannula) connected to an underwater seal with an iv infusion set (Fig 1).

Thereafter surgery was completed without further complications but attempts at visualising the diaphragm to identify defects at laparoscopy failed. With pneumoperitoneum deflation and Valsalva manoeuvre, bubbling ceased and the subsequent chest examination was normal. Therefore patient was observed in the ICU without further interventions. Post operative chest x-rays taken at ICU showed fully expanded lungs and she was discharged from the hospital the following day.

Discussion

Laparoscopy necessitates gas insufflation of the abdomen to visualise its contents. Normal abdominal pressure ranges from 0-5 mmHg but raising it to 12-15 mmHg renders it adequate for surgical exposure at laparoscopy.³ Intra-abdominal pressures beyond this are avoided as this compromises respiration and oxygenation (diaphragmatic splinting and reduced lung compliance), haemodynamic stability (reduced venous return, increased afterload especially to right ventricle, direct cardiac compression) and microcirculatory shutdown (hepatosplanchnic flow compromise, renal hypoperfusion) which can be safely managed with simple fluid resuscitation.³ Beyond 25 mmHg the symptoms become detrimental needing immediate corrective measures and even vaso-active drugs to prevent onset of a vicious cycle of tissue hypoxia and permanent tissue damage.³

Tension pneumothorax at laparoscopy can occur either through breached diaphragm or the lungs.

Despite the rarity of the incidence, the commonest reason identified is the tracking of CO₂ into the mediastinum and subsequent rupture in to the pleural cavity.¹ Other less common routes of entry are around loose connective tissue around transdiaphragmatic structures (aorta, inferior vena cava and oesophagus), congenital defects or weak points with subsequent rupture due to increased intra abdominal pressures, or surgical trauma.^{1,4,5,6} Likewise the rupture of lung tissue like spontaneous bullae or barotrauma could also result in tension pneumothoraces.

To prevent lethal consequences, early detection with high degree of suspicion is very important. Arterial desaturation resulted by increased ventilation-perfusion mismatch and shunting and elevated airway pressures seem to be the earliest signs. With worsening intrapleural tension, there is compensatory tachycardia. The classical signs of ipsilateral chest hyperinflation with reduced movements, contralateral tracheal shift with reduced/absent breath sounds and hyper-resonant percussion note are usually obvious and should be checked in all suspected cases. Since tension pneumothorax is a life threatening emergency, urgent decompression is mandatory. Therefore the initial diagnosis is undoubtedly a clinical one needing immediate corrective measures.

As a practical tool in identifying the origin of the gas in the pneumothorax (thus the cause), CO₂ concentration analysis with a capnograph attached to the IC tube has been suggested.⁵ With end tidal tensions of CO₂ in the vented gases via IC tube, the origin is likely to be of ruptured lung whereas higher tensions are likely to be that of pneumoperitoneal, leaking via diaphragmatic defects.⁵ The importance of this differentiation is in the immediate and the subsequent management in which PEEP, Valsalva and large tidal volume breathing may help in lung re-expansion with intact lungs.⁵ In our patient it was clinically diagnosed as of diaphragmatic origin by exclusion and as the onset and the resolution was closely related to the induction of pneumoperitoneum but not the mechanical ventilation.

Initial symptomatic therapy such as urgent chest decompression and improvement of oxygenation (stopping N₂O, 100% O₂, deflation of abdomen,

small tidal volume ventilation, reversion of Trendelenburg position to reduce diaphragmatic splinting and needle thoracostomy), and early communication between the team members and seeking senior help averted the probable threat to life. Haemodynamic restitution with rapid fluid infusion for venous return improvement and vasoconstrictors (ephedrine) also became beneficial.

There are some controversies regarding the efficacy and the safety of the needle thoracostomy.⁷ But in a recent literature review it has been concluded that needle thoracostomy is as effective and safe as the IC tubes in tension pneumothorax as a life saving intervention.⁷ In this patient clear clinical judgement to proceed with close monitoring and continued chest decompression with an improvised system of needle thoracostomy with under water seal helped to prevent postponement of the procedure (Fig. 1). This improvised chest drain system was a normal intravenous infusion set connected to the G14 needle at 2nd intercostal space (ICS) at mid clavicular line with the cut end dipped in a bottle of saline (fluid height < height of the saline level in the bottle, roughly 5-8 cm) kept at the ground level (~1 m below patient). For chest decompression in an emergency small bore tubing is found to be as effective as large bore ones for pure pneumothoraces and it was clearly adequate in our case.⁸ Further interventions were not done as complete lung re-expansion was confirmed by post operative chest x-rays.

Conclusion

This is a case of unilateral tension pneumothorax diagnosed clinically in a young healthy patient with induced pneumoperitoneum. Probable portal of gas entry was stipulated as a congenital defect

of the diaphragm as the onset of pneumothorax was well before a significant rise in intra-abdominal pressure. The case highlights the importance of vigilant monitoring, high degree of suspicion of probable complications and expedient rational management in preventing possible catastrophies in anaesthesia.

References:

1. Kaushik R, Attri AK. Spontaneous pneumothorax A rare complication of laparoscopic cholecystectomy. *Ind J of Surg* 2004; 66(5): 294-6.
2. Kumar G, Singh AK. Pneumothorax during Laparoscopic Cholecystectomy. *Med J Armed Forces of Ind* 2007; 63: 277-8.
3. De Backer D. Abdominal compartment syndrome. *Crit Care* 1999; 3(6):103-4.
4. Abu-Rafea B, Vilos GA, Vilos AG, Hollett-Caines J, Al-Omran M. Effect of body habitus and parity on insufflated CO₂ volume at various intraabdominal pressures during laparoscopic access in women. *J Minim Invasive Gynecol* 2006; 13(3): 205-10.
5. Labow DM, Conlon KC. Pneumothorax after diagnostic laparoscopy. *Surg Endosc* 1999; 13: 935-6.
6. Ding Z, Zhou Q, Gui B. Spontaneous tension pneumothorax during laparoscopic cholecystectomy secondary to congenital diaphragm defects. *J Nanjing Med University* 2009; 23(2): 146-8.
7. Shah V, Butala B, Parikh B, Raval R. Bilateral pneumothorax as a complication of laparoscopy – A case report. *Ind J Anaes* 2003; 47 (1): 55-6.
8. Zehtabchi S, Rios CL. Management of emergency department patients with primary spontaneous pneumothorax: needle aspiration or tube thoracostomy? *Ann Emerg Med.* 2008; 51(1):91-100.
9. Paramasivam E, Bodenham A. Air leaks, pneumothorax, and chest drains. *CEACCP* 2008; 6: 204-9.