Life-threatening subcutaneous emphysema due to laparoscopy

INTRODUCTION

Laparoscopy is a common surgical technique used by surgeons in many specialities. From an anaesthetic point of view, it is imperative to understand the changes caused by this procedure to the patient's physiology. It reduces vital capacity, increases dead space, hypercapnia and acidaemia due to carbon dioxide (CO_2) absorption. Insufflation of the abdomen with CO_2 also makes it difficult to ventilate the patient due to high intra-abdominal pressures causing restriction of diaphragmatic excursion during ventilation and can potentially cause complications such as barotrauma and pneumothorax. Several gases have been used for insufflation. The ideal one should be non-toxic, inert, colourless, non-inflammable, soluble in blood and inexpensive.[1] Since CO_2 meets all those requirements, it is the most frequently used gas. The main problem with using CO_2 is the systemic absorption leading to hypercapnia. It is not always easy to deal with it, and anaesthesiologists not only have to prevent damage due to acidosis or altered gas exchange, but they also have to protect the lungs from high pressures required to effectively ventilate the patient. Studies have been published related to the best possible ways to avoid damaging the lungs, but this may be challenging in laparoscopic surgeries.[2]

One of the most feared complications in relation to CO_2 insufflation is subcutaneous emphysema as it could lead to pneumothorax or pneumomediastinum. We present the case of a patient who underwent laparoscopy for a hemicolectomy, complicated by subcutaneous emphysema leading to a 4-fold increase of CO_2 elimination, respiratory acidosis and haemodynamic instability.

CASE REPORT

A 50-year-old patient underwent laparoscopic hemicolectomy for diverticular disease. The patient, classified as American Society of Anesthesiologists physical status II and body mass index 21, showed no history of any relevant disorder or medications apart from being a regular marijuana user.

In addition to standard monitoring as per the recommendations of the European Society of Anaesthesiology and the Association of Anaesthetists of Great Britain and Ireland, a flow track system (Vigileo™) was used to measure the cardiac output and to guide fluid administration. Following general anaesthetic induction with propofol 200 mg, fentanyl 150 μg and rocuronium 50 mg, endotracheal intubation was performed with 8 mm internal diameter tube and mechanical ventilation was initiated. Sevofluorane was used as maintenance gas to maintain a minimum alveolar concentration (MAC) between 1.0 and 1.2.

Ventilatory settings were adjusted in volume control to achieve a tidal volume of 480 ml (6 ml/kg), positive end-expiratory pressure of 5 mmHg and respiratory rate (RR) of 14/min. Dynamic compliance read 70 mbar and driving pressure remained <14 mbar. Peak and plateau pressures reached 25 and 18 mbar. No recruitment manoeuvres were needed.

Initially, the patient was haemodynamically stable. However, 60 min after CO_2 insufflation, blood pressure (BP), heart rate (HR) and cardiac index (CI) increased over basal values; BP was 200/110 mmHg, HR was 105/min and CI was 4.8. A lighter plane of anaesthesia and pain was ruled out by ensuring a MAC of sevoflurane at 2.1 and providing adequate doses of intravenous analgesic agents.

Eventually, intravenous nicardipine was administered to control BP. Intra-abdominal pressure during pneumoperitoneum was consistently remaining between 13 and 15 mmHg.

Ventilator parameters were checked. Compliance, plateau and peak pressures remained unchanged; thus, pneumothorax or airway obstruction was not the most likely cause, as the ventilator did not show either a restrictive or compliance problem.

VCO_2 (CO_2 production) of 1205 ml/min was noticed and end-tidal CO_2 ($EtCO_2$) had risen from 40 to 62 mmHg. Minute ventilation was increased to 18 L (TV of 900 ml; RR of 20/min). Arterial blood gas showed a partial pressure of CO_2 ($PaCO_2$) of 76.7 mmHg and pH of 7.16. Subcutaneous emphysema was noticed on examining the patient's neck. The surgeon was notified and he noticed a subcutaneous leak from one of the four trocars used. Pneumoperitoneum was deflated and emphysema was drained with needles. All the parameters returned to baseline levels and surgery continued minimising pneumoperitoneum pressure without further complications. A further arterial blood gas analysis showed a pH of 7.35, $PaCO_2$ of 43.8 mmHg and partial pressure of oxygen of 368 mmHg on a fraction of inspired oxygen (FiO_2) of 0.5.

At the end of the surgery, keeping in view the potential airway compromise due to the subcutaneous emphysema, the patient was extubated after a cuff leak test. An airway exchange catheter remained in place for 20 min and subsequently removed, as the patient remained stable.

DISCUSSION

The incidence of grossly detectable emphysema associated with laparoscopy surgery ranges from 0.43% to 2.34%.[3] Several circumstances increase the risk of subcutaneous emphysema. Main predictors are $EtCO_2$ >50 mmHg, operative time >200 min, the use of six or more surgical ports, higher insufflation pressure (>15 mmHg), frailty and extra-peritoneal dissections, as was seen in our case, leading to CO_2 inflation into subcutaneous tissue.[4] Anaesthesiologists must be aware of these risk factors to take precautions and detect emphysema in the early stages.

Anaesthesia machines have option to measure VCO_2 This parameter is useful to detect increases in CO_2 production. It is related to pCO_2 as follows:

$PaCO_2 = 0.863 \times VCO_2/VA$. VCO_2 can increase as a result of several conditions. These include inadequate ventilation, excessive CO_2 production due to increased metabolism or release and external sources.

During laparoscopic surgery, CO_2 is absorbed by peritoneum; therefore, CO_2 levels in blood are high. This is readily compensated by assisting expiration from the lungs as it has high aqueous solubility and diffusibility. If alveolar ventilation is impaired, additional CO_2 load is not cleared, resulting in hypercapnia and acidosis and $EtCO_2$ elevation. This is usually corrected by increasing minute volume as much as 30%.[4,5]

If very high levels of $EtCO_2$ are noticed, palpating the patients' skin surface for emphysema and notifying the same to the surgeon is advisable.

Hypercapnia stimulates sympathetic nervous system, and hence the BP and HR increase. It also sensitises the myocardium to catecholamines, predisposing to cardiac arrhythmias.[6]

When subcutaneous emphysema appears, all the members of the team have to be notified. A blood gas analysis is mandatory to evaluate the grade of acidosis, electrolytes disorders and gas interchange. Other potential events such as pneumothorax or air embolism must be ruled out. FiO_2 of 100% must be delivered if oxygenation appears compromised.[7]

Drainage of subcutaneous gas has been reported with manoeuvres as described by Sucena *et al*.[8] At the end of surgery, airway assessment before extubation must be performed. A direct laryngoscopy and a leak test should be considered as well as an airway exchange device has to be kept ready for a potential airway compromise. Tracheal extubation should be postponed if unsure of airway patency post-extubation.[9,10]

CONCLUSION

Subcutaneous emphysema could lead to a life-threatening situation with haemodynamic instability, pneumothorax and pneumomediastinum. Management consists of increasing minute volume followed by early deflation of the pneumoperitoneum and decompression of the subcutaneous emphysema. Airway patency must be assessed before extubation.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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Access this article online	
Quick response code	Website: www.ijaweb.org
	DOI: 10.4103/0019-5049.179469

How to cite this article: Coronil AB, Sanchez-Canete AM, Bartakke AA, Fernandez JG, Garcia A. Life-threatening subcutaneous emphysema due to laparoscopy. Indian J Anaesth 2016;60:286-8.