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CASE REPORT

When CO_2 goes wild – a tale of surgical emphysema, pneumothorax and a gas-filled adventure in laparoscopic hernia repair

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This case report describes a 69-year-old male patient who underwent laparoscopic inguinal hernia repair using a total extraperitoneal approach. Complications during the procedure were hypercarbia, surgical emphysema, and pneumothorax. Immediate interventions were necessary to address the rising end-tidal CO_2 levels, hypercarbia, and acidosis. The incidence of these complications may be higher than previously reported, particularly in extraperitoneal laparoscopic procedures, underscoring the importance of increased awareness among anaesthesiologists. This case report emphasises the significance of monitoring CO_2 inflation pressure, assessing subcutaneous CO_2 accumulation, and adjusting ventilation to enhance patient safety and promote the reporting of such complications in the future.

Keywords: surgical emphysema, hypercarbia, laparoscopic surgery, extraperitoneal, laparoscopic total extraperitoneal

Case report

A 69-year-old male presented with an irreducible inguinal hernia for an extraperitoneal laparoscopic repair. He had an American Society of Anesthesiologists (ASA) classification of II, weighed 80 kg and measured 190 cm (thus a body mass index of 22.16). He had a smoking history of 30-pack-years with no other comorbidities. Preoperatively he presented with mild bilateral expiratory wheezes and a hyperinflated chest with associated hyperresonance on percussion. He reported shortness of breath when walking up a hill which indicated a modified medical research council (mMRC) score of 1. To address the respiratory symptoms, he received salbutamol nebulisation four hours before the procedure, continuing for 16 hours. Cardiovascular and systemic examinations yielded unremarkable findings. His preoperative chest X-ray (CXR) displayed features of hyperinflation with clear lung fields, while the electrocardiogram (ECG) results were unremarkable. Informed consent for general anaesthesia was signed.

Upon arrival in the operating theatre his chest was clear. Standard monitoring, including ECG, noninvasive blood pressure (NIBP), pulse oximetry, end-tidal carbon dioxide ($ETCO_2$), anaesthetic gas concentration, nerve stimulation, and temperature measurement was initiated. Following preoxygenation, anaesthesia induction involved fentanyl (75 µg), lidocaine (100 mg) and propofol (150 mg), while orotracheal intubation was facilitated with rocuronium (50 mg), and anaesthesia was maintained using oxygen and isoflurane (minimum alveolar concentration of 0.9–1.1). Initially, volume-controlled ventilation was employed, with a tidal volume of 500 ml, respiratory rate (RR) of 8, fixed inspiration to expiration ratio (I:E) of 1:2.5, positive end-expiratory pressure (PEEP) of 5 cmH_2O peak pressure varying between 13 and 16 cmH_2O, and chest compliance of 77 cmH_2O. $ETCO_2$ ranged between 5 and 6.2 kilopascal (kPa), while minute ventilation (MV) varied from 3.8 to 4.4 liters/minute (l/min). He was placed in the Trendelenburg position after draping. We administered multimodal analgesia consisting of intravenous paracetamol (1 g), parecoxib (40 mg), morphine (4 mg), and ketamine (20 mg). Plasmalyte was administered at a rate of 1 ml/kg/h during surgery.

During CO_2 insufflation there was a progressive increase in $ETCO_2$ levels (5.0–7.2 kPa), which was compensated for by increasing the MV from 3.8 to 7.2 l/min. Peak pressure ranged from 24 to 29 cmH_2O and chest compliance was between 45 and 50 cmH_2O. The CO_2 insufflation pressure was initiated at 16 mmHg for the first five minutes and then reduced to12–15 mmHg for the remainder of the operation. Thirty minutes after insufflation, there was a rapid rise in $ETCO_2$ from 7.2 to 10.8 kPa, accompanied by an increase in systemic blood pressure, which was managed with additional opioids and increasing the depth of anaesthesia. The peak pressure remained stable and oxygen saturation (SO_2) was maintained. The surgical team was promptly informed about the rising $ETCO_2$ levels. The anaesthetic team increased the inspired fractional oxygen concentration (FiO_2) to 80%, suctioned the endotracheal tube (ETT), and evaluated the anaesthetic circuit and valves for potential obstructions. The ETT was clear and no circuit malfunction was detected. Auscultation revealed bilateral wheezes, leading to a provisional diagnosis of bronchospasm, which was managed with 8 salbutamol puffs administered through the ETT, along with dexamethasone (8 mg), ketamine (20 mg) and $MgSO_4$ (2 g). Upon palpation, extensive surgical emphysema was felt on both chest walls and the neck, with the left side being more affected. Considering the worsening condition, the decision was made to stop the insufflation and abort the procedure. $ETCO_2$ levels continued to rise, reaching a maximum of 12.2 kPa. Peak pressures remained stable with a maximum of 29 cmH_2O. To compensate for the increasing $ETCO_2$, MV was increased to a maximum of 9.1 l/min. Following the removal of surgical drapes, extensive surgical emphysema was observed bilaterally extending from the patient's pelvis to his neck. A point-of-care lung ultrasound (POCUS) was performed,

Table I: Case values

	ETC0₂ (kPa)	RR (B/min)	Vt (ml)	MV (l/min)	PAP (cmH_2O)	Compliance (ml/cmH_2O)	$PaCO_2$ (kPa)
Pre-laparoscopy	5.4	8	470	3.7	13	77	Not done
Laparoscopy	6.2	10	492	5.0	24	46	Not done
Emphysema	12.2	16	640	9.8	29	42	12.2
60 minutes after deflation and ICD insertion	5.8	12	566	6.8	19	71	8.13

ICD – intercostal drain; $ETCO_2$ – end-tidal carbon dioxide; RR – respiratory rate; Vt – tidal volume; MV – minute volume; PAP – peak airway pressure; kPa – kilopascal; $PaCO_2$ – partial arterial pressure of carbon dioxide

and absence of lung sliding was noted on the left side, while it remained present on the right side. Suspecting a pneumothorax due to significant left-sided surgical emphysema, absent air entry and lung sliding, a 28-frank intercostal drain (ICD) was inserted. Ventilation continued for another 30 minutes, reducing $ETCO_2$ levels to 7.9 kPa. An arterial blood gas (ABG) analysis showed respiratory acidosis (pH: 7.11, $PaCO_2$: 12.2 kPa, PaO_2: 18 kPa, base excess: -0.4, lactate: 1.1, bicarbonate (HCO_3): 22.2 mmol/l). The $PaCO_2$ to $ETCO_2$ gradient was 4.3 kPa. (Refer to Table I for a summary of important case values.)

An urgent mobile chest X-ray (CXR) was performed, which confirmed significant surgical emphysema present in bilateral chest walls. The ICD placement was correct, and no residual pneumothorax was observed. Ventilation was continued for an additional hour, lowering $ETCO_2$ levels to 5.8 kPa. Repeated ABG analysis showed improvement in respiratory acidosis (pH: 7.23, $PaCO_2$: 8.13 kPa, PaO_2: 18.7 kPa, base excess: -1.4, lactate: 1.1, HCO_3: 22.4 mmol/l). The $PaCO_2$ to $ETCO_2$ gradient decreased to 2.3 kPa.

He was successfully extubated and provided oxygen via a 40% face mask, reaching a Glasgow Coma Scale (GCS) score of 15/15. He was then transferred to a high care facility for further monitoring. A morning review indicated haemodynamic stability and a decrease in $PaCO_2$ to 5.63 kPa. He was extensively counseled on the events, and the ICD was removed on that day with a new elective surgical date provided for open hernia repair.

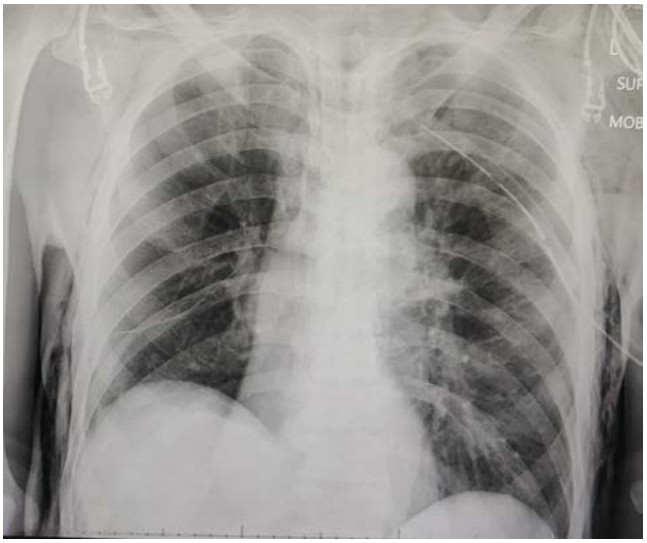

Figure 1: Mobile chest X-ray – after intercostal drain insertion

Discussion

Our patient presented for laparoscopic hernia repair (total extraperitoneal (TEP) approach), which is increasingly preferred due to its benefits such as improved postoperative recovery, reduced pain, higher patient satisfaction and faster return to daily activities.[1,2] His only risk factor was chronic smoking (30-pack-year).

CO_2 is the ideal gas for creating a pneumoperitoneum because it is non-toxic, colourless, soluble in the blood, easily expelled from the body, non-flammable, and inexpensive.[2] Complications such as subcutaneous emphysema, pneumomediastinum and pneumothorax may occur with the TEP approach.[2,3] During simple laparoscopic surgery, CO_2 production increases by less than 50% above the basal metabolic rate and then plateaus after 10 minutes; to compensate for the CO_2, pulmonary elimination increases by 15–80%, which is generally well tolerated.[3] However, with extraperitoneal surgery, as in our case, CO_2 production steadily increases instead of plateauing, reaching 65% above basal levels at 60 minutes.[3]

The patient developed subcutaneous emphysema, manifesting as palpable crepitus beneath the skin, and a pneumothorax; both are recognised complications associated with laparoscopic procedures. The reported incidence of subcutaneous emphysema ranges from 0.43% to 2.3%, pneumothorax 0.03%, and pneumomediastinum 1.9%.[1-7] These complications are more frequently observed during extraperitoneal laparoscopic surgery.[2-4] The actual incidence of these complications may be higher than that reported for extraperitoneal laparoscopy. Studies involving the use of a valveless trocar and dynamic pressure system have shown a significantly elevated rate of subcutaneous emphysema at 16.4%, which is 6.3 times higher than the reported rate. Pneumomediastinum occurred at a rate of 3.9%, double the reported rate, and masked pneumothorax had a complication rate of 0.9%, 2.3 times higher than the reported rate.[8,9] This leads to an overall complication rate of 21.24%, which is 4.3 times the total reported rate.[8,9] Unfortunately, these complications are underreported by anaesthesiologists, and the association between rising $ETCO_2$ and subcutaneous emphysema may often go unnoticed.[1,3,7] In our case, significant surgical emphysema developed, which can lead to an increase in the pulmonary CO_2 elimination rate of up to 340% and is more likely to complicate with pneumomediastinum and pneumothorax.[2-4] Based on the increased likelihood of a pneumothorax in the presence of significant surgical emphysema, it is entirely

possible that lung sliding was absent on POCUS, and there was a pneumothorax present. The decision to insert an ICD was driven by a convergence of factors: heightened barotrauma risk in undiagnosed chronic obstructive lung disease (COPD) during positive pressure ventilation (PPV), diagnostic ambiguity surrounding the extensive surgical emphysema, particularly pronounced in the left chest, and lateral asymmetry evident on ultrasound findings. While acknowledging that, based on the literature review, the insertion of the ICD might not have been required due to the likelihood of spontaneous CO_2 reabsorption and a low risk of barotrauma, our approach was driven by diagnostic uncertainty and risk mitigation. The observed asymmetry, combined with unilateral absent lung sliding on POCUS, prompted consideration of a pneumothorax, which led us to proceed with the ICD insertion. Our decision was primarily guided by patient safety, the imperative to prevent a potential tension pneumothorax, and the broader context of managing the clinical situation. In managing pneumomediastinum, while aligning with general principles applied to pneumothorax, particular attention should be directed towards potential complications involving mediastinal structures, such as a pneumopericardium necessitating vigilance for cardiovascular compromise and consideration of appropriate interventions.

Risk factors for subcutaneous emphysema include prolonged operative time, increased age, elevated end-tidal carbon dioxide levels, and increased number of operative ports.[2]

The surgical team elected to use an elevated insufflation pressure (> 12 mmHg) to enhance visibility and create an efficient surgical workspace for managing the large hernia. This decision was aligned with the procedure's need for visualisation and manoeuvering. The higher pressure is a risk factor for the development of subcutaneous emphysema.[2] The proposed mechanism for pneumomediastinum and pneumothorax is that CO_2 traverses from the abdominal to thoracic cavity through the aortic and oesophageal hiatuses, pleuroperitoneal hiatus or Bochdalek's foramen, congenital diaphragmatic defects, or inadvertent falciform ligament injury. Additionally, extraperitoneal carbon dioxide can migrate via the retroperitoneal space and dissect along the fascia transversalis and endothoracic fascia anteriorly to enter the mediastinum.[1] The later mechanism is more common in extraperitoneal laparoscopy and would be the likely mechanism in our patient.

The clinical significance of subcutaneous emphysema is related to its effects on hypercarbia and acidosis. Intraoperative hypercarbia most commonly occurs due to anaesthesia circuit or ETT malfunction, depleted CO_2 absorber, and rapid absorption of CO_2 from pleura and subcutaneous tissue into the circulation.[2] Hypercarbia leads to increased heart rate, systemic blood pressure, central venous pressure, cardiac output, cerebral blood flow, decreased peripheral vascular resistance, and pulmonary vasoconstriction due to the release of epinephrine and norepinephrine.[2,3] A $PaCO_2$ level of 18 kPa is equivalent to a minimum alveolar concentration of 0.5 for CO_2 anaesthesia, which can result in hypoxaemia.[3] The patient did not have significant cardiovascular risk factors but intraoperatively he was hypertensive and tachycardic in relation to the rising CO_2, which was attributed to pain and was treated with additional opioids. Prolonged exposure to elevated $PaCO_2$ levels above 11 kPa can cause systemic effects, peaking after 25 minutes and taking approximately 100 minutes to return to baseline, necessitating prolonged ventilation and subjecting the patient to prolonged cardiac stress.[10] He was ventilated for a prolonged period of time to allow for the reduction of CO_2.

The awareness of these associated risk factors is crucial to prevent complications related to laparoscopic procedures. Preventive strategies include monitoring CO_2 inflation pressure (< 12 mmHg), regular examination and palpation of the abdominal and chest wall for subcutaneous gas accumulation, adjusting ventilation to maintain acceptable $ETCO_2$ levels, ruling out other causes of subcutaneous emphysema and acute hypercarbia.[3,5,6]

To address rising intraoperative $ETCO_2$ levels, recommended approaches include increasing FiO_2 to 100%, increasing MV, reviewing the circuit, ETT, and CO_2 absorber, performing an ABG, excluding pneumothorax, decreasing intra-abdominal pressure, and ruling out other causes of elevated $ETCO_2$.[2-4]

The incidence of such complications is potentially higher than previously reported, emphasising the need for increased awareness among anaesthesiologists. Lessons learnt from this case include the importance of monitoring CO_2 inflation pressure, regularly assessing and palpating the abdominal and chest wall for subcutaneous gas accumulation, and adjusting ventilation to maintain acceptable $ETCO_2$ levels. When encountering rising intraoperative $ETCO_2$, it is crucial to investigate potential causes, such as pneumothorax, and promptly implementing appropriate interventions. Furthermore, healthcare professionals should be aware of the risks associated with extraperitoneal laparoscopic procedures, which may lead to higher rates of subcutaneous emphysema, pneumomediastinum and pneumothorax.

By sharing this case report and highlighting the potential complications and preventive strategies, we aim to increase awareness, improve patient safety, and encourage further reporting of surgical emphysema and related adverse events. Vigilance, proactive management and interdisciplinary collaboration are essential to minimise the occurrence and impact of these complications in future cases.

Conflict of interest

The authors report no conflict of interest.

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Ethical approval

Informed consent from the patient was obtained for the publication of this case report.

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References

1. Wallace L, Leigh Y. Pneumomediastinum and pneumothorax: a rare complication of laparoscopic total extraperitoneal inguinal hernia repair. J Surg Case Rep. 2023;2023(3):rjad146. https://doi.org/10.1093/jscr/rjad146.
2. Ott DE. Subcutaneous emphysema-Beyond the pneumoperitoneum. J Soc Lapar Surg. 2014;18(1):1-7. https://doi.org/10.4293/108680813X13693422520882.
3. Critchley LAH, Ho AMH. Surgical emphysema as a cause of severe hypercapnia during laparoscopic surgery. Anaesth Intensive Care. 2010;38(6):1094-100. https://doi.org/10.1177/0310057X1003800622.
4. Wolf JS, Monk TG, McDougall EM, McClennan BL, Clayman RV. The extraperitoneal approach and subcutaneous emphysema are associated with greater absorption of carbon dioxide during laparoscopic renal surgery. J Urol. 1995;154(3):959-63. https://doi.org/10.1016/S0022-5347(01)66943-X.
5. Aldakhil SK, Tashkandi AA, Al Harbi MK, Al Shehri A. Subcutaneous emphysema and hypercarbia as a complication of laparoscopic procedure: case report. J Surg Case Rep. 2020;2020(3):rjz415. https://doi.org/10.1093/jscr/rjz415.
6. Klopfenstein CE, Gaggero G, Mamie C, Morel P, Forster A. Laparoscopic extraperitoneal inguinal hernia repair complicated by subcutaneous emphysema. Can J Anaesth. 1995;42(6):523-5. https://doi.org/10.1007/BF03011692.
7. Saggar VR, Singhal A, Singh K, Sharma B, Sarangi R. Factors influencing development of subcutaneous carbon dioxide emphysema in laparoscopic totally extraperitoneal inguinal hernia repair. J Laparoendosc Adv Surg Tech. 2008;18(2):213-6. https://doi.org/10.1089/lap.2007.0089.
8. Herati AS, Atalla MA, Rais-Bahrami S, et al. A new valve-less trocar for urologic laparoscopy: initial evaluation. J Endourol. 2009;23(9):1535-9. https://doi.org/10.1089/end.2009.0376.
9. Hillelsohn JH, Friedlander JI, Bagadiya N, et al. Masked pneumothorax: risk of valveless trocar systems. J Urol. 2013;189(3):955-9. https://doi.org/10.1016/j.juro.2012.08.244.
10. Yoshida H, Kushikata T, Kabara S, et al. Flat electroencephalogram caused by carbon dioxide pneumoperitoneum. Anesth Analg. 2007;105(6):1749-52. https://doi.org/10.1213/01.ane.0000290293.13532.60.