Respiratory acidosis and subcutaneous emphysema during laparoscopic cholecystectomy

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A case is presented of a healthy 69-yr-old woman who underwent elective laparoscopic cholecystectomy under general anaesthesia. As surgery proceeded she developed hypercapnia (arterial blood $PaCO_2$ = 100 mmHg) and a related respiratory acidosis (arterial blood pH 7.07). The cause was attributed to subcutaneous insufflation and absorption of CO_2, directly related to the surgical pneumoperitoneum.

Une patiente bien portante de 69 ans est opérée en chirurgie réglée pour une cholécystectomie par laparoscopie. Pendant l'intervention, elle présente de l'hypercapnie ($PaCO_2$ = 100 mmHg) associée à une acidose respiratoire (pHa = 7,07). L'étiologie de cet incident est attribuable à une insufflation percutanée avec absorption de CO_2 causée par un pneumopéritoine d'origine chirurgicale.

Laparoscopic surgery is being performed in many surgical facilities and is growing in popularity. Recently the advent of laparoscopic cholecystectomy (LC) has given anaesthetists new challenges and many complications have been described.[1-7]

This report illustrates the diagnosis and management of an unusual case of intraoperative respiratory acidosis occurring during laparoscopic surgery and considers some practical issues related to anaesthesia for laparoscopic surgery.

Case report

A 69-yr-old woman was admitted to hospital for LC. Medical history indicated that she had essential hypertension which was well controlled with enalapril and hydrochlorothiazide. She denied symptoms of cardiac disease. However, previous echocardiography demonstrated mitral valve prolapse without regurgitation. She was a previous smoker who had stopped many years earlier. She had hypothyroidism which was compensated by thyroid hormone replacement. On physical examination no abnormalities were detected except for a midsystolic click heard at the apex, with no evidence of regurgitant murmur. Chest *x*-ray, ECG, CBC, and electrolytes were within normal limits.

The patient received premedication with lorazepam 1 mg and ranitidine 150 mg *po* with 125 ml of water two hours before surgery.[3] Intraoperative monitors included ECG, automatic BP, axillary temperature probe, pulse oximetry, $P_{ET}CO_2$ and volatile gas concentrations were measured by a Nelcor 2000 monitor. Muscle relaxation was monitored with a peripheral nerve stimulator.

Anaesthesia was induced breathing 100% oxygen, with fentanyl 100 mg, followed by thiopentone 250 mg and muscle relaxation was produced with vecuronium 5 mg. The trachea was intubated and anaesthesia was maintained with N_2O/O_2 and isoflurane (0.5–0.75% inspired concentration). The lungs were ventilated using a Narcomed 2A anaesthetic machine with a Bain circuit with a tidal volume of 600 ml, at 12 bpm and I:E ratio of 1:2. The patient received one litre of Ringer's lactate solution in the first hour of surgery.

Within a few minutes of induction of anaesthesia she developed sinus bradycardia and a decrease in systolic blood pressure from 140 mmHg to 105 mmHg which lasted about 30 sec. The heart rate returned to 60 bpm after atropine 0.3 mg *iv* was given. The SpO_2 and $P_{ET}CO_2$ remained at 99% and 32 mmHg respectively, and the temperature remained constant at 35°C. The surgeon inserted the CO_2 insufflation needle periumbilically but required three attempts to achieve an unobstructed flow of CO_2 into the peritoneal cavity at a rate of 2 to 3 $L \cdot min^{-1}$ maintaining a pressure <13 mmHg. The patient was maintained in Fowler's position.

Key words
CARBON DIOXIDE: absorption, hypercarbia;
COMPLICATIONS: hypercapnia, emphysema;
SURGERY: laparoscopy.

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Fifteen minutes later, the $P_{ET}CO_2$ was noted to have increased from 32 mmHg to 45 mmHg and SpO_2 was 99%. As the surgery proceeded without difficulty the $P_{ET}CO_2$ continued to increase considerably but without haemodynamic instability, oxygen desaturation, or pyrexia. Peak inspiratory airway pressures were <25 mmHg throughout anaesthesia. In response, the ventilator settings were modified to increase tidal volume to 800 ml (16 ml · kg^{-1}), respiratory rate to 20 bpm and N_2O/O_2 to 4/2 L · min^{-1} from 3/1.5 L · min^{-1}. Other causes for hypercapnia were sought and ruled out including, ventilator or circuit leaks, malpositioned endotracheal tube, CO_2 embolus, pneumothorax, malignant hyperthermia and airway obstruction.

Approximately 50 min after induction of anaesthesia, the $P_{ET}CO_2$ was 60 mmHg. Nitrous oxide was discontinued and O_2 delivered at 5 L · min^{-1}. The patient's neck was distended and some periorbital swelling was noted. Palpation revealed crepitus consistent with subcutaneous emphysema. The surgeon was asked to discontinue CO_2 insufflation but felt he could not complete the procedure, which was approaching completion, by doing this. A few more minutes were granted to maintain pneumoperitoneum. An immediate chest *x*-ray ruled out the presence of pneumothorax and pneumomediastinum. Appropriate endotracheal tube position was confirmed radiologically and the presence of gas in the chest wall and neck was evident.

The $P_{ET}CO_2$ climbed to a maximum of 85 mmHg in spite of rapid manual hyperventilation with 100% oxygen. Arterial blood gas analysis revealed pH 7.07, PCO_2 100 mmHg, PO_2 311 mmHg, HCO_3 28 mmol · L^{-1}, and TCO_2 31 mmol · L^{-1} indicating an acute respiratory acidosis.

The abdominal trocars were withdrawn after removal of the gallbladder, allowing CO_2 to escape from the peritoneal cavity which was followed by a dramatic reduction of the $P_{ET}CO_2$. Mechanical ventilation was continued postoperatively after the addition of vecuronium 1 mg and midazolam 2 mg with 100% O_2 5 L · min^{-1}, RR 12 bpm and V_T 800 ml. Twenty minutes after release of the pneumoperitoneum the $P_{ET}CO_2$ was 51 mmHg, SpO_2 99% and temperature 35°C. After another 20 min the patient was awake and coughing. Neuromuscular blockade was reversed with atropine 0.3 mg and edrophonium 25 mg. The trachea was extubated after adequate reversal with the patient breathing spontaneously, achieving normocarbia and adequate oxygenation, $P_{ET}CO_2$ 42 mmHg and SpO_2 99%. She was discharged to the recovery room becoming more alert, breathing adequately and showing a marked reduction of her subcutaneous emphysema. The patient proceeded to have an uneventful recovery breathing oxygen by mask at 5 L · min^{-1}. Repeat arterial blood gas analysis revealed pH 7.35, PCO_2 46 mmHg, PO_2 140 mmHg, HCO_3 26 mmol · L^{-1}, and TCO_2 27 mmol · L^{-1}. She was discharged home the next day with no sequelae.

Discussion

This case of a healthy woman who underwent elective laparoscopic surgery illustrates a previously unreported complication, extensive subcutaneous CO_2 emphysema resulting in hypercarbia and acute respiratory acidosis. The surgery and anaesthetic commenced in a routine manner until significant rise in $P_{ET}CO_2$ was noted. After other causes of hypercarbia had been ruled out the cause of the hypercapnia was attributed to subcutaneous CO_2 insufflation.

Previous intraoperative anaesthetic complications have been described including hypotension, hypothermia, nausea and vomiting and oxygen desaturation. Hypercarbia ($PaCO_2$ increasing by up to 10 mmHg) has been noted during gynaecological laparoscopy where high insufflation pressures are used (20 to 40 mmHg). Due to the lower insufflation pressures during LC the $PaCO_2$ and $P_{ET}CO_2$ increases are less.[4,5] It is recognized that ventilatory settings must be adapted to handle the excess burden of CO_2 to the body to preserve normal acid-base milieu.[6]

A working differential diagnosis of the hypercarbia was quickly formulated and other causes ruled out by checking the integrity of the anaesthetic circuit, the position and function of the endotracheal tube, confirming adequate air entry into the lungs. Auscultation of the heart for a mill wheel murmur was done to help rule out pulmonary CO_2 embolus. Radial artery blood gas sampling confirmed good oxygenation and considerable hypercarbia as indicated by the noninvasive monitors. The ABG also confirmed a respiratory acidosis with a normal bicarbonate level to exclude metabolic causes of acidosis. This and the lack of hyperthermia or haemodynamic instability ruled out malignant hyperthermia.

One can only speculate that in this case the CO_2 tracked subcutaneously and was absorbed resulting in acute hypercarbia and respiratory acidosis. One suspects that the difficult insertion of the insufflating needle may have created a tunnel from the peritoneal cavity to the subcutaneous tissues which created a conduit of low resistance allowing CO_2 to collect and be absorbed. During the case the surgeon looked for any obvious site for creation of a fistula but was unable to identify any unusual defects from within the peritoneal cavity or externally over the abdominal wall. The trocars and needles were returned to the manufacturer for inspection of material or mechanical flaws, but none were found.

Anaesthetic management involved discontinuation of N_2O to prevent further distention of the subcutaneous

space, and 100% oxygen (100 ml · kg^{-1}), increased tidal volume and respiratory rate were used to achieve hyperventilation. According to Spoerel and Bain this flow will achieve mild hyperventilation.[7] In retrospect, the fresh gas flow should have been increased further to be more effective. A CO_2 absorber on the circuit might have proved most useful but was not available. Although the insufflation was carried out in the usual fashion with CO_2 flow 2–3 L · min^{-1} on an automatic demand mode using pressures of <13 mmHg, considerable CO_2 accumulation occurred making its elimination difficult. However, the amount of CO_2 absorbed was insufficient to create conditions for pulmonary CO_2 embolus.

Recommendations to be considered in managing dramatic CO_2 absorption during laparoscopic surgery might include: (1) routine and repeated examination of the abdominal wall for subcutaneous gas accumulation; (2) discontinuation of N_2O as it may exacerbate the tracking of subcutaneous gas; (3) use a CO_2 absorber; (4) increase of ventilation to eliminate CO_2; (5) immediate deflation of the pneumoperitoneum and cessation of CO_2 insufflation will dramatically improve the situation and is indicated if the patient is unstable; (6) efforts to rule out other causes of hypercarbia such as inadequate ventilation, pulmonary CO_2 embolus, malignant hyperthermia, pneumothorax and consider subcutaneous CO_2 emphysema.

In conclusion, an unusual case is presented of respiratory acidosis during laparoscopic cholecystectomy, which was most likely caused by CO_2 absorption secondary to severe CO_2 emphysema. This entity should be considered in the differential diagnosis of hypercarbia during laparoscopic surgery and appropriate measures should be taken to eliminate carbon dioxide accumulation.

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