CASE REPORT

Fatal carbon dioxide embolism and severe haemorrhage during laparoscopic salpingectomy

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SUMMARY

We report a case of fatal carbon dioxide embolism and severe haemorrhage during laparoscopic salpingectomy. A sudden decrease in end-tidal carbon dioxide concentration occurred after 1 h of operating time which, together with the clinical signs, suggested carbon dioxide embolism. Haemorrhage after pelvic venous injury was first noted after deflation of the pneumoperitoneum and resulted in potentiation of the adverse haemodynamic effects of massive gas embolism. Minimally invasive surgery involves more extensive tissue trauma and an increased duration of pneumoperitoneum compared with diagnostic laparoscopy and may increase the risk of serious complications. (Br. J. Anaesth. 1994; 72: 243-245)

KEY WORDS


Carbon dioxide embolism and major haemorrhage are uncommon, but potentially fatal complications of laparoscopic procedures. In gynaecological laparoscopies, gas embolism was reported to occur in eight of 50247 cases [1] and the incidence of major vascular injury was 0.64% in a review of 100000 cases [2]. Low morbidity and low incidence of serious complications have supported the view of the benign nature of laparoscopic surgery. The increasing popularity of minimally invasive techniques in gynaecological and general surgery may result in a greater incidence of complications. We describe a case in which the co-occurrence of two rare complications led to a fatal sequence of events.

CASE REPORT

A 36-yr-old woman was admitted to hospital with a history and signs consistent with the diagnosis of a right-sided ectopic pregnancy which was confirmed by ultrasound scan. Medical history and physical examination were unremarkable. Arterial pressure was 115/75 mm Hg and heart rate was 84 beat min⁻¹. Serum electrolyte and creatinine concentrations were within normal limits, haemoglobin was 12.5 g litre⁻¹.

The patient was scheduled for laparoscopic salpingectomy. After rapid sequence induction of anaesthesia and tracheal intubation, controlled mechanical ventilation was commenced using a circle system with carbon dioxide absorber. Anaesthesia was maintained with 60% nitrous oxide and enflurane in oxygen. Monitoring consisted of non-invasive arterial pressure, ECG, pulse oximetry, capnography and anaesthetic vapour analysis.

A pneumoperitoneum was produced uneventfully and sustained at pressures around 15 mm Hg. Laparoscopic inspection revealed a mass of 3 cm in diameter attached to the right Fallopian tube. No blood was visible and a right salpingectomy was subsequently attempted. The vital signs were stable, with oxygen saturation of 97%, end-tidal partial pressure of carbon dioxide 4.1 kPa and heart rate 86 beat min⁻¹.

One hour after commencement of the procedure, with the patient in a Trendelenburg position, the capnograph reading suddenly decreased to near zero values. This coincided with the surgeon’s use of a grasping forceps, which had been introduced through an infra-umbilical midline incision. Disconnection from the ventilator was excluded and auscultation of the chest revealed no abnormality. One minute later, arterial pressure became unrecordable, with impalpable carotid pulse, and a “mill-wheel” murmur was heard over the precordium. The ECG showed sinus tachycardia of 125 beat min⁻¹. Surgery was abandoned immediately. The patient was placed in a horizontal position; manual ventilation with 100% oxygen and external cardiac compression were commenced.

Blood was noted to be emerging from the trocar puncture sites and laparotomy was performed. Free blood was seen in the abdominal cavity, but the bleeding point could not be identified easily. Resuscitation was begun with colloid solutions, followed by blood transfusions until surgical haemostasis had been achieved. The measured blood loss was 4500 ml, which had been replaced with colloid solutions 2500 ml and blood 3600 ml.

Central venous catheters were inserted via the right subclavian and right internal jugular veins, but...
several attempts to aspirate gas were unsuccessful, as was attempted intracardiac aspiration. The capnograph continued to read zero values and the pattern of electromechanical dissociation changed to asystolic cardiac arrest. Arterial blood-gas analysis revealed pH 6.9, $P_{CO_2}$ 5.8 kPa, $P_O_2$ 28.3 kPa, standard bicarbonate 7.7 mmol litre$^{-1}$ and base excess $-22.9$ mmol litre$^{-1}$. Oxygen saturation was 98.2% and haemoglobin concentration 7.7 g litre$^{-1}$. Asystole persisted and resuscitation was abandoned 50 min after the initial event.

Postmortem examination, which was performed 36 h after death, showed extensive retroperitoneal haematoma without injury to major pelvic vessels. Massive gas embolism could not be excluded as a cause of death, although gas emboli were not detected in the heart or pulmonary circulation.

**DISCUSSION**

Carbon dioxide embolism occurs most frequently in the initial phase of laparoscopic procedures. Intravascular gas insufflation after inadvertent puncture of intra-abdominal blood vessels, accidental insertion of the trocar into the liver before laparoscopic cholecystectomy and direct transuterine gas injection have been reported as causes of carbon dioxide embolism [3-5]. Rupture of pelvic and abdominal veins as a consequence of increased intra-abdominal pressure or surgery have been suggested as possible aetiological factors [6,7]. Major haemorrhage is a rare complication of laparoscopic tubal sterilizations. Signs of acute blood loss, a large retroperitoneal haematoma and minimal intraperitoneal bleeding are found in the majority of cases. In contrast with arterial haemorrhage, injury to venous blood vessels may be concealed by the tamponading effect of the pneumoperitoneum [8].

The clinical manifestation of gas embolism is determined by the volume of gas and its rate of entry into the venous circulation. A bolus gas embolism creates a "gas-lock" in the right atrium, obstructing the pulmonary outflow tract and resulting in reduced venous return with subsequent decrease in cardiac output [9]. Smaller gas bubbles may enter the pulmonary circulation, causing pulmonary hypertension, right ventricular failure and non-cardiogenic pulmonary oedema [5]. Rapid injection of gas is more likely to form a gas-lock, whereas slow air entrainment (as in neurosurgery) results in air entrapment in pulmonary vessels.

In contrast with embolism in neurosurgery, subclinical embolism during laparoscopic procedures seems to be uncommon [10]. Alterations in heart sounds and heart murmurs may precede other manifestations and are ominous signs of impending gas embolism. Precordial and oesophageal stethoscopes and Doppler apparatus have been suggested for monitoring heart sounds [11,12].

This case history demonstrates that gas embolism can occur at any time during the procedure as a consequence of an open venous channel and a pressure gradient between abdominal cavity and the venous system [13]. Vascular injury may precede gas embolism, even though bleeding may not become apparent until the pneumoperitoneum has been deflated. Minimally invasive surgery requires an increased duration of pneumoperitoneum and is inevitably associated with a greater degree of micro- and macrovascular injury, which may increase the risk of serious complications.

The sudden decrease of end-tidal partial pressure of carbon dioxide, associated with mill-wheel murmur, loss of cardiac output and sinus tachycardia on the ECG were highly suggestive of massive gas embolism. Failure to aspirate gas via the central venous catheter or by direct cardiac puncture may have been suggestive that the gas embolus was trapped in the pulmonary artery. Placing the patient in a left lateral, head-down tilt position proved impractical with a laparotomy and external cardiac massage in progress.

Carbon dioxide is highly soluble in blood. Prolonged cardiopulmonary resuscitation and massive fluid replacement would have enhanced this process and may explain why gas emboli were not identified at postmortem examination, which was not performed until 36 h after death.

The management of gas embolism consists of cessation of gas insufflation, release of the pneumoperitoneum and placing the patient in the left lateral decubitus position. Aspiration of gas should be attempted, although in animal experiments this method has been demonstrated to be no more effective than the left lateral position [10]. Emergency thoracotomy and internal cardiac massage should be considered and a laparotomy should be performed if vascular injury is suspected. Concurrent massive haemorrhage aggravates the adverse haemodynamic effects of gas embolism and complicates effective resuscitation.

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