Clinical Reports

Carbon Dioxide Embolism Treated with Hyperbaric Oxygen

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We report a case of suspected carbon dioxide embolism occurring during laparoscopy. Among the sequelae was neurological dysfunction felt to be secondary to paradoxical embolization. The patient was treated with hyperbaric oxygen therapy. Hyperbaric oxygen should be considered when confronted with a clinically important gas embolism.

Carbon dioxide embolism is a rare but potentially devastating complication of laparoscopy or hysteroscopy.[1, 2] Embolic sequelae include cardiac arrest, pulmonary oedema, and neurological impairment due to anoxia or cerebral embolization.[3, 4] The following is a report of a patient who developed these complications after carbon dioxide insufflation for laparoscopy. The patient's neurological deficits rapidly improved following hyperbaric oxygen therapy. The case emphasizes the therapeutic potential of an increasingly available treatment for cerebral gas emboli.

Case report

A 38-year old, 157 cm, 52 kg woman was scheduled for laparoscopy and hysteroscopy as part of a diagnostic investigation for infertility. She had exercise-induced asthma which was well controlled with terbutaline and cromolyn sodium and also noted palpitation with exercise. She had had three previous uneventful general anaesthetics. Physical examination, urinalysis, haemoglobin, haematocrit, white blood cell and platelet counts were normal. The ECG showed sinus bradycardia; chest *x*-ray was not obtained.

Premedication included fentanyl 100 μg, droperidol 0.065 mg, and midazolam 1 mg IV. Anaesthesia was induced with thiopentone 250 mg and tracheal intubation facilitated by vercuronium 5 mg, and lidocaine 100 mg IV. Anaesthesia was maintained with isoflurane and nitrous oxide 60 per cent. Ventilation was controlled. Monitoring included electrocardiogram, blood pressure, capnography, apical stethoscope, pulse oximetry, and temperature. The patient was placed in the dorsal lithotomy position with head-down tilt. Hysteroscopy using 100 ml dextran 70 solution (Hyskon®) was uneventful. Carbon dioxide was then insufflated into the peritoneal cavity (gas pressure ≤ 1.59 kPa (12 mmHg) through a Verres needle. Prior to insufflation the blood pressure was 100/50 mmHg, heart rate 60 · min^{-1}, O_2 saturation 99 per cent, and end-tidal PCO_2 4.50 kPa (34 mmHg). Two minutes after beginning CO_2 insufflation, she developed ventricular fibrillation with no detectable pulse or blood pressure. End-tidal CO_2 fell to 1.19 kPa (9 mmHg).

Insufflation was stopped, nitrous oxide and isoflurane discontinued, and closed chest cardiac massage with manual ventilation (FiO_2 1.0) begun. Adrenaline 1 mg and lidocaine 100 mg were given IV followed by DC countershock (200 joules). Sinus tachycardia (130 beats · min^{-1}) and blood pressure (180/110 mmHg) returned two minutes later. End-tidal CO_2 tension increased to 4.64 kPa (35 mmHg). The pHa was 7.39, $PaCO_2$ 4.37 kPa (33 mmHg), and PaO_2 52.60 kPa (397 mmHg) (FiO_2 1.0). The procedure was terminated and neuromuscular blockade reversed with neostigmine 3.5 mg and glycopyrrolate 0.6 mg IV. Total operative fluid intake was Ringer's lactate 900 ml; blood loss was minimal.

The patient's trachea remained intubated and she was moved to the recovery room. Blood pressure was 90/40 mmHg, heart rate 120 · min^{-1}, and there were rales throughout both lungs. She did not respond to verbal command or open her eyes, but was agitated and had writhing purposeless movements of all extremities. The

Key words

CARBON DIOXIDE: embolism, insufflation; EMBOLISM: gas, carbon dioxide; HYPERBARIA: oxygen, chamber; SURGERY: laparoscopy.

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pupils reacted sluggishly to light and were 1 mm in diameter. There were no focal neurological deficits. During spontaneous breathing pHa was 7.23, $PaCO_2$ 5.70 kPa (43 mmHg), and PaO_2 6.10 kPa (46 mmHg) (FIO_2 0.5). Chest *x*-ray showed diffuse pulmonary oedema. Her lungs were mechanically ventilated with oxygen and 5 cm H_2O positive end-expiratory pressure (PEEP). A further decrease in blood pressure to 70/40 mmHg was treated with Ringer's lactate 1,500 ml and dopamine 5–15 $\mu g \cdot kg^{-1} \cdot min^{-1}$. The vital signs stabilized but neurological abnormalities persisted. She was transported to a hyperbaric oxygen chamber, located in our emergency department, 140 minutes after laparoscopy. Midazolam and pancuronium bromide were used to control agitation before and during treatment. She underwent compression to three atmospheres for 30 minutes, followed by 2.5 atmospheres for 60 minutes (FIO_2 1.0).

Upon transfer to the Intensive Care Unit, she was lethargic, had persistent writhing movements of the trunk and extremities, but intermittently opened her eyes to speech and responded appropriately to questions by nodding her head. While receiving intermittent mandatory ventilation, rate $16 \cdot min^{-1}$ and 5 cm H_2O PEEP, the PaO_2 was 8.61 kPa (65 mmHg) (FIO_2 1.0). A pulmonary artery catheter revealed mean pulmonary artery pressure 26 mmHg, pulmonary artery diastolic pressure of 16 mmHg, pulmonary capillary wedge pressure 12 mmHg, and cardiac output 5.2 litres $\cdot min^{-1}$. The PaO_2 increased to 12.72 kPa (96 mmHg) (FIO_2 0.5) as PEEP was incrementally increased to 15 cm H_2O. Blood pressure and cardiac output were maintained by volume infusion and dopamine 10–12 $\mu g \cdot kg^{-1} \cdot min^{-1}$. Twelve hours after laparoscopy she was awake, alert, and responded appropriately to three-step verbal commands.

Cardiovascular and pulmonary function improved markedly during the first postoperative day. Ventilator rate, PEEP, and dopamine infusion were steadily reduced. A 3.5 L diuresis was accomplished using furosemide. Pulmonary oedema cleared and the patient's trachea was extubated on the second postoperative day. The remaining hospital course was uneventful and the patient had no sequelae.

Discussion

This case demonstrates the major consequences of venous gas embolization-cardiovascular collapse and noncardiogenic pulmonary oedema. The patient's neurological impairment was compatible with cerebral gas embolism; a diagnosis that with the rare exception of finding intraarterial gas at autopsy is presumptive.[5] Thus, as in underwater diving accidents, we assumed that cerebral embolism had occurred and immediately transferred the patient to a hyperbaric chamber. Although the impact of hyperbaric therapy is uncertain, we believe the case emphasizes how a readily accessible chamber can broaden therapeutic options.

In addition to laparoscopy and hysteroscopy, other causes of venous gas embolism encountered in anaesthetic practice include surgical procedures in which the operative site is above the level of the heart (e.g., sitting craniotomy, hysterectomy), venous cannulation, haemodialysis and diagnostic procedures such as arthrography.[6–10] Underwater diving accidents provide the best known instance of cerebral gas embolization.[11] Venous gas bubbles can enter the arterial circulation via direct pulmonary capillary passage, pulmonary arterialvenous anastamoses or intracardiac shunts including probe patent foramen ovale.[12] The probe patent foramen ovale is a particularly important route since it is estimated to occur in 20–30 per cent of the population.[13] In addition to venous sources, direct placement of gas into the arterial circulation can occur during cardiac surgery, cardiopulmonary bypass or angiography.[14, 15]

Cardiovascular collapse following gas embolization results mainly from obstruction to right ventricular outflow, direct mechanical blockade by a bubble or by the development of pulmonary hypertension in response to pulmonary embolization. The decrease in end-tidal CO_2[7] and sudden onset of collapse points to a massive embolism which initially obstructed blood flow proximal to the blood alveolar interface (presumably in the heart).

Pulmonary oedema is a well described consequence of venous gas embolism. The oedema is usually nonhydrostatic in nature as reflected by our patient's normal pulmonary capillary wedge pressures. Increased pulmonary capillary permeability occurs through leukocyte aggregation and activation as well as platelet activity.[16–18]

The manifestations of cerebral gas embolism can be quite variable and include sudden death, coma, seizure, visual disturbance, confusion, personality change, aphasia, vertigo, headache, and focal sensory or motor deficits.[11,19] Once in the brain, gas bubbles produce ischemia by direct vascular obstruction, platelet and red cell aggregation, and increased capillary permeability.[20] The ensuing vasogenic oedema can lead to elevations in intracranial pressure which may be responsible for cases in which symptoms or neurological deficits have recurred despite initially successful therapy.[21]

Hyperbaric oxygen therapy (HBO) is considered the treatment of choice for cerebral gas embolism.[10, 14, 19, 22] Hyperbaric compression reduces bubble size (one-third of original volume at three atmospheres) thus restoring blood flow and limiting the detrimental effects of a gas–blood interface. Other potential beneficial effects include a reduction in intracranial pressure and increased tissue oxygenation via diffusion.[23, 24] In air embolism, a

large gradient for nitrogen is created between the bubble and surrounding tissue promoting resorption.[25] Evidence demonstrating HBO's beneficial effect in cerebral gas embolism comes largely from underwater diving accidents.[11, 19, 21, 22] Reports of successful hyperbaric treatment for iatrogenic gas embolism have been limited. One of these involved carbon dioxide embolism complicating urethral surgery. The patient was resuscitated from an asystolic cardiac arrest but remained comatose with flexor responses to pain. Following hyperbaric treatment at three atmospheres for 90 minutes, the patient regained consciousness and suffered no permanent neurological damage.[26]

The recompression regimen was chosen based on published recompression schedules modified for the pressure limitations of a monoplace chamber and the presumption that carbon dioxide embolism could be more readily treated by a reduced compression schedule than an air embolism (Dr. J. Smith, personal communication). There are no controlled clinical trials evaluating the efficacy of the various recompression schedules in gas embolism.[27]

The complications of HBO include barotrauma, particularly to the ear and sinuses; pulmonary oxygen toxicity; decompressive sickness, "the bends"; seizures and fire.[27] In addition to hyperbaric oxygen, additional treatment includes basic respiratory and cardiovascular support. The head down position has been recommended to prevent further bubble migration to the brain, but it is not clear that this potential benefit outweighs the risks of worsening cerebral oedema.[28] Heparin therapy and measures to reduce cerebral oedema and intracranial pressure such as hyperventilation, mannitol, lidocaine and steroids have been used but a clear benefit has not been demonstrated.[29]

The reduced acquisition costs of monoplace versus walk-in hyperbaric chambers has led to a marked increase in the number of hyperbaric facilities in the USA. Concern has been raised about the indications for their use, cost and safety.[27] Cerebral gas embolism, however, remains an entity for which HBO has proven efficacy and is of lifesaving value. Anaesthetists are likely to be involved in caring for those patients at greatest risk for gas embolization. If cerebral gas embolism is suspected, they should strongly consider using hyperbaric treatment, particularly if a chamber is available in-hospital.

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Résumé

On rapporte le cas où une embolie au CO_2 est possiblement survenue lors d'une laparoscopie. Parmi les séquelles on nota une dysfonction neurologique possiblement secondaire à une embolisation paradoxale. Le patient fut traité avec de l'oxygène hyperbare. Les anesthésistes doivent considérer l'utilisation de l'oxygène hyperbare lorsque confronté avec une possibilité clinique d'embolie gazeuse.