

Carbon Dioxide Embolism during Laparoscopy

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Laparoscopy is a frequently used technique for sterilization and differential diagnosis of pelvic pain and ectopic pregnancy. Anesthesiologists and surgeons are aware of the rare, but potentially lethal, complication of gas embolism.¹ The following is a report of a carbon dioxide embolism during abdominal insufflation for laparoscopy in which the patient survived.

REPORT OF A CASE

A 22-year-old, 60-kg, 160-cm woman, gravida 4 para 3, was scheduled for dilatation and curettage after spontaneous passage of a 20-week fetus and placenta. The possibility of an accompanying appendicitis necessitated laparoscopy. The medical history had no significant abnormalities, and she had been anesthetized uneventfully for a previous vaginal delivery. Physical examination revealed localized tenderness over the right lower quadrant and the uterus was tender and 14 weeks in size. The remainder of the physical examination was normal. No heart murmur was noted. All laboratory results were normal, except for a white blood cell count of 19,500/mm³ and a urinalysis with 3+ ketonuria, 1-2 white blood cells, and 2+ bacteriuria.

No preoperative medication was given. Anesthesia was induced with thiopental, and endotracheal intubation was facilitated by succinylcholine. Anesthesia was maintained with nitrous oxide, halothane, and pancuronium. Ventilation was controlled with a tidal volume of 600 ml at a rate of 12 per min. Heart sounds were monitored with a precordial stethoscope. Electrocardiogram, temperature, and blood pressure also were monitored.

The patient was placed in the dorsal lithotomy position with a head down tilt. The curettage was uneventful. Then, a 1-cm incision was made at the umbilicus and a Verres needle was inserted into the peritoneal cavity without technical problems. Carbon dioxide, 3.5 l, was delivered through the needle at a rate of 1.0 l/min at an insufflating pressure of 10 to 20 mmHg.

Initial evaluation of the pelvic cavity through the laparoscope revealed the presence of purulent material. At this point, the laparoscop-

ist noticed that the peritoneal gas had disappeared, which was thought to be due to leakage around the trocar. Insufflation was started again and another 500 ml of carbon dioxide were delivered during the next 30 s. Hypotension and a weak radial pulse immediately resulted. Within one minute, the blood pressure could not be obtained and auscultation of the precordium revealed a "mill-wheel" murmur. The patient's face was dusky.

Insufflation was stopped immediately and nitrous oxide was discontinued. The patient was placed on her left side with slight Trendelenburg.² Controlled ventilation was increased to 800 ml at a rate of 12 per min with a Fi_{O_2} of 1.0, while an arterial catheter was inserted percutaneously into the left radial artery. The pH_a was 6.93, Pa_{CO_2} 103 mmHg, and Pa_{O_2} 44.3 mmHg. During the next 2 min, the blood pressure rose to 90/70 mmHg, and one minute later it was 110/80 mmHg. The right internal jugular vein was cannulated with a 16-gauge 30-cm long catheter. Twenty milliliters of foamy blood were aspirated from the central venous pressure catheter. Oscilloscopic display of the central venous pressure wave form tracked the position of the catheter tip from the pulmonary artery to the right ventricle to the right atrium during withdrawal. The central venous pressure was 14 mmHg. Most of the aspirated foam came from the right atrium.

The central venous pressure catheter was replaced by a flow-directed (Swan-Ganz) catheter. Pulmonary artery pressure was 40/20 mmHg, and pulmonary capillary wedge pressure was 18 mmHg. The patient's condition remained stable but a mill-wheel murmur still could be heard with a precordial doppler.

Anesthesia was maintained with fentanyl, halothane, and oxygen with controlled ventilation and PEEP of 10 cmH₂O. With the patient still in a slight left lateral tilt, the surgeons performed a laparotomy. A gangrenous appendix was found and removed. With a Fi_{O_2} of 1.0, the pH_a was 7.07, Pa_{CO_2} 74 mmHg, and Pa_{O_2} 205 mmHg. With increased minute ventilation, the pH_a was 7.30, Pa_{CO_2} 57 mmHg, and Pa_{O_2} 400 mmHg. The patient was transferred to the intensive care unit where analysis of arterial blood gases was normal. PEEP and controlled ventilation were discontinued gradually. After 1 h of spontaneous ventilation, the trachea was extubated. The postoperative course was uneventful and the patient had no sequelae.

DISCUSSION

Venous gas embolism during laparoscopy is a rare complication. In 1974, Phillips reported 15 probable carbon dioxide embolisms in 113,253 laparoscopy cases in a 1-year period.³ Six other cases of possible carbon dioxide embolism have been reported recently in the literature.^{1,4-7}

Unfortunately, criteria for diagnosis of gas embolism vary considerably in the literature, and, therefore, accurate data on incidence are difficult to obtain. It seems that the only absolute criterion for diagnosis of gas em-

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bolism is confirmation of gas bubbles in the vascular system at the time of surgery or autopsy.

Cardiovascular collapse during laparoscopy has been reported previously. Many mechanisms have been implicated, including dysrhythmias from hypercarbia and halothane anesthesia,⁸ perforation of a major vessel or viscera from direct injection of gas into the vascular system,⁹ excessive compression of the vena cava from increased intra-abdominal pressure,¹⁰ vasovagal reflex,¹¹ and unilateral or bilateral pneumothorax.¹² Hypercarbia results from hypoventilation or absorption of carbon dioxide from the peritoneal surface.¹³

Of the six cases reported above, evidence of vessel injury was noted in two of them.^{5,7} In two other cases, direct injection of gas through the fundus of the uterus was noted and may have resulted in the injection of gas into a vessel in the uterine wall. McKenzie suggested that carbon dioxide might enter the circulatory system when a vein has been ruptured because of the increased intra-abdominal pressure.¹⁴ Cardiovascular collapse was noted by Kleppinger, presumably secondary to carbon dioxide embolism through a bleeding mesosalpingeal vein.¹⁵ In our case, the sudden hypotension immediately following carbon dioxide insufflation and aspiration of foamy blood from the right heart strongly suggested carbon dioxide embolism. Carbon dioxide may have been introduced by intravenous injection, although no vessel injury was noted. The possibility of increased carbon dioxide absorption through necrotic appendiceal tissue is possible.

If massive, carbon dioxide embolization can obstruct the pulmonary outflow tract, right ventricular failure may result. This can be controlled by placing the patient in a left lateral and head-down position to minimize the obstruction.² Carbon dioxide also has a direct effect on peripheral vascular resistance, producing the documented hypotension. When small carbon dioxide bubbles gain access to the pulmonary circulation, a transient increase in pulmonary artery, right ventricular, and right atrial pressures can be observed. This effect is diminished by the rapid solubility of carbon dioxide in blood and concomitant hyperventilation to promote carbon dioxide elimination. Since the diffusion capacity of nitrous oxide is lower than that of carbon dioxide, nitrous oxide will not increase the volume of the carbon dioxide embolism as it does with air. However, nitrous oxide must be discontinued to allow an $F_{I_{O_2}}$ of 1.0 to avoid hypoxemia from high alveolar carbon dioxide concentrations.

As carbon dioxide is highly soluble in blood, it is absorbed rapidly from the bloodstream. This accounts for the rapid reversal of the clinical signs of carbon dioxide embolism with treatment. These characteristics must be considered in the selection of an insufflation

agent in laparoscopy. Nitrous oxide is slightly less soluble than carbon dioxide, and air is five times more lethal. In angiocardiology, carbon dioxide has been well tolerated in doses as high as 7.5 ml/kg, while injected doses of 0.25 ml/kg of air in cats have been lethal. In dogs, the dose equivalent of one liter of carbon dioxide in humans can be given before cardiac output is altered profoundly.¹⁶

Carbon dioxide embolism is an extremely rare complication of laparoscopy.³ However, it can be lethal.¹ For this reason, patients obviously should be monitored carefully during laparoscopy.¹³ During insufflation with carbon dioxide, end-tidal carbon dioxide has been shown to increase slightly.¹⁷ If carbon dioxide embolism occurred during laparoscopy, a further increase in the end-tidal carbon dioxide concentration would be expected. However, no such increase has been reported. End-tidal carbon dioxide monitoring obviously needs further investigation. A precordial doppler is a sensitive device, but the low incidence of gas embolization during laparoscopy may preclude its routine use.¹⁸

In conclusion, to minimize the chance of a carbon dioxide embolus during laparoscopy, we recommend restriction of carbon dioxide volume and pressure to three liters and 30 mmHg, respectively. Also, the blood pressure, electrocardiogram and heart sounds should be monitored carefully for early detection of carbon dioxide embolism.

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New Methods for the Performance of Unilateral Lung Lavage

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Massive bronchopulmonary lavage using a double-lumen endotracheal tube has become an accepted modality for the treatment of pulmonary alveolar proteinosis.¹⁻⁵

During single-lung ventilation, the non-ventilated lung is lavaged with tidal volumes of saline until return of sediment is minimal; the procedure is then terminated.

Massive bronchopulmonary lavage is not without hazard. During the initial lavage of a patient, complete gas exchange must be accomplished by a single severely compromised lung. Periods of tidal drainage are accompanied by reperfusion of the non-ventilated lung⁶ and potentially dangerous levels of hypoxemia. Drowning is prevented only by proper positioning of the endotracheal tube and adequate cuff inflation.

Recognizing these hazards, we have modified the published techniques of massive bronchopulmonary lavage to: (1) ensure correct placement of the double-lumen tube, and (2) investigate maneuvers to maximize gas exchange during unilateral lavage. Experience was gained during eight lavages performed on three patients with biopsy-proven alveolar proteinosis.

METHODS

Patient evaluations included history, physical examination, arterial blood-gas analysis, and pulmonary function testing. In addition, preoperative ventilation-perfusion scanning was performed with the patient in the supine position ($FI_{O_2} = 0.21$ to 0.30) using standard techniques.⁷

With the patient in the supine position having received 5-10 min of preoxygenation, anesthesia was induced by the administration of 3-4 mg/kg sodium thiopental, iv, in divided doses, and inhalation of 0.5-2.0% halothane or 0.5-3.0% enflurane (patient 3) via mask. Following the onset of surgical anesthesia, the patients were paralyzed with pancuronium (0.15 mg/kg, iv).

For all lavages we used the National Catheter Corporation left lung, double-lumen tube. With one exception, we used the largest size tube that could be passed atraumatically through the glottis. The correct position of the double-lumen tube was confirmed by passing a fiberoptic bronchoscope (Machida FBS 4T bronchoscope) down both lumens of the tube. Via the left lumen, the subcarina separating the upper and lower lobes was clearly visible. The volume of air in the left endobronchial cuff was adjusted until the left endobronchial lumen just began to invaginate due to the external cuff pressure. Via the right lumen, a clear view of the carina was observed as well as the upper surface of the left endobronchial balloon.

The tracheal cuff was inflated until slight to moderate tension was palpated in the pilot balloon. We confirmed complete separation of the two lungs by clamping the tube connected to one lung proximal to the open suction port. A length of tubing was attached to the suction port, and the free end of this tube was submerged under water. When the ventilated lung was inflated statically to +50 cm H_2O , endobronchial cuff inflation was ad-

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