Carbon Dioxide Embolism During Pneumoperitoneum for Laparoscopic Surgery: A Case Report

Heather J. Smith, CRNA, MSN

Although rare, a carbon dioxide (CO₂) embolism is a potential complication of laparoscopic surgery. An embolism may occur during insufflation of the abdomen after incorrect placement of a Veress needle into a vascular organ or an intra-abdominal vessel. If the CO₂ embolism is not recognized, it can be rapidly fatal for the patient unless the patient receives treatment immediately. Therefore, anesthesia providers must be vigilant while monitoring, recognize when an embolism has occurred, and be able to provide effective management and treatment for their patient.

This case report describes a 34-year-old woman who underwent a suction dilation and curettage, followed by an exploratory laparoscopic procedure to examine her uterus. After placement of the Veress needle and insufflation of the abdomen, a CO₂ embolism developed that caused severe hypotension, bradycardia, and loss of end-tidal CO₂ tracing. The patient was treated quickly and aggressively with fluid administration and intravenous vasopressors. Because of rapid recognition and treatment the patient did not suffer any long-term adverse medical events.

Keywords: Abdominal insufflation, carbon dioxide embolism, laparoscopic surgery.

Laparoscopy is deemed a “minimally invasive” procedure that allows for exploration through an endoscope of the peritoneal cavity after insufflation of carbon dioxide (CO₂).1 During the laparoscopic procedure, distention of the abdomen may cause a number of physiologic disturbances for the patient as well as potential complications such as a CO₂ embolism. Although a CO₂ embolism is rare, if unrecognized, the embolism can be fatal for the patient unless the patient receives treatment immediately.2 Venous CO₂ embolism is often associated with profound hypotension, cyanosis, hypercarbia and hypocarbia, dysrhythmias, and potential cardiac collapse due to a “gas lock” in the vena cava and right atrium. When this occurs, there is a decrease in venous return and cardiac output, leading to circulatory collapse.3

Case Summary
A 34-year-old healthy woman was scheduled to undergo a suction dilation and curettage after a miscarriage at 11 weeks into her pregnancy. The patient presented with a normal health history and had the following surgical procedures in the past: knee arthroscopy, right ulnar shortening, and retinal reattachment as a child. She had no drug allergies and was not taking any medications. Her pregnancy history revealed a gravida 3, para 2. She weighed 50 kg and was 156 cm in height. Physical examination revealed a healthy female patient with lung sounds clear to auscultation, heart rate regular and in sinus rhythm, and Mallampati class II with normal neck extension. She had not had any adverse reactions to anesthetics in the past and had nothing to eat by mouth for 10 hours.

Preoperatively, midazolam (Versed), 2 mg intravenously (IV), was administered and the patient was taken to the operating room (OR) suite where she transferred herself to the OR table. Standard monitors were applied, which included a noninvasive blood pressure cuff, oxygen saturation (SpO₂) via pulse oximetry, end-tidal CO₂ (ETCO₂) monitoring, and electrocardiogram monitoring. The patient was preoxygenated with 8 L of oxygen via face mask, and induction ensued with 50 μg of fentanyl, 50 mg of lidocaine, and two 40-mg boluses of propofol IV. Ventilation was initiated via face mask with an oral airway in place. Sevoflurane administration was started to maintain a minimum alveolar concentration (MAC) of 1.0 to 1.2 with 1 L of oxygen and 1 L of air. Once an adequate level of anesthesia was achieved and adequate manual ventilation was secured, the patient began breathing without assistance and maintained an ETCO₂ percentage of 2.3 of sevoflurane. The patient demonstrated stable hemodynamics and ventilation throughout the entire dilatation and curettage.

Approximately 7 minutes into the procedure, the surgeon requested to convert the procedure from a dilatation and curettage to an exploratory laparoscopy. The surgeon reported that the depth of the surgical instrumentation used for the procedure might have been advanced too far. Therefore, an exploratory laparoscope was used to view the uterus and to ensure that inadvert-
tent puncture from the surgical instrument did not occur. The patient was given an additional 100 mg of propofol, 50 μg of fentanyl, and 35 mg of rocuronium IV. The patient was successfully intubated with a No. 7 endotracheal tube, and an 18 French oral gastric tube was placed. The patient’s stomach was suctioned and decompressed before beginning trocar insertion for the laparoscope. The patient was continued on a mixture of sevoflurane maintaining an end tidal of 2.3%, with 1-L flows of both oxygen and air. Controlled volume ventilation was initiated with tidal volumes of 375 mL at a respiratory rate of 12/min. Dexmethylasone, 4 mg, was given prophylactically for postoperative nausea and vomiting.

After the surgical preparation and draping of the patient for the laparoscope, the Veress needle was inserted, and insufflation of CO₂ was initiated. Approximately 3 minutes after the initial CO₂ insufflation of the abdomen, the patient’s SpO₂ began to drop rapidly from 98% to 74%, her heart rate (HR) dropped from 74/min to 50/min, blood pressure (BP) dropped from 90 mm Hg systolic to 50 mm Hg systolic, and all etCO₂ tracings were noted to be at 0. The patient was quickly given 100% oxygen with manual ventilation. Insufflation of CO₂ was stopped, and a differential diagnosis of CO₂ embolism was made. Aggressive fluid resuscitation was initiated, and five 10-mg boluses of ephedrine were given. The patient was positioned in steep Trendelenburg position with a left lateral tilt. After approximately 2 minutes of supportive treatment with fluid resuscitation and vasopressor administration, the patient’s BP returned to 80 mm Hg systolic, HR was 90/min, etCO₂ was 29 mEq/L, and SpO₂ was 98%. Carbon dioxide insufflation was reinstituted, and a uterine rupture was visualized via the laparoscope. Additional surgical assistance was requested via the surgeon as the laparoscopic procedure was aborted and an emergent laparotomy was performed to repair the uterine wall. Because of the large blood loss visualized by the surgeon, estimated to be 2,000 mL, the patient was volume resuscitated with a total of 4 L of crystalloids, 3 U of packed red blood cells, and 1 L of 6% hetastarch in 0.9% sodium chloride (Hespan). Bolus injections of ephedrine, 10 mg, were administered, and an additional 16-gauge peripheral IV catheter was inserted. Mephérine, 0.2 mg intramuscular (IM), and carboprost (Hemabate), 250 μg IM, were administered to assist with uterine contractions. Once the uterine wall was repaired, the patient was hemodynamically stable. The patient was then weaned from mechanical ventilation, sevoflurane administration was discontinued, and the patient began breathing spontaneously with adequate tidal volumes and respiratory rate. The patient was extubated awake and able to move all extremities. She was transferred to the postanesthesia care unit (PACU). The patient was found to have a hemoglobin level of 10.4 g/dL with a hematocrit of 31.8% postoperatively. She was stabilized in the recovery room and then transferred to the intensive care unit for close monitoring and frequent assessments of hemoglobin and hematocrit values.

**Discussion**

The proposed mechanism of gas embolism includes “inadvertent intravenous placement of the Veress needle [and] passage of CO₂ into abdominal wall and peritoneal vessels during insufflation, or into open vessels on the liver surface during gallbladder dissection.” Initially, a profound increase in etCO₂ will occur, followed by a rapid decrease in etCO₂. The embolism leads to cardiovascular collapse and a reduction of pulmonary blood flow. If the gas volume is large enough, a “mill-wheel” murmur may be auscultated with the use of a precordial or esophageal stethoscope. However, the most sensitive means to detect gas emboli are precordial or transesophageal Doppler ultrasonography and transesophageal echocardiography. The treatment of a suspected CO₂ embolism during laparoscopy or any surgical procedure includes the suspension of CO₂ insufflation with deflation of gas in the abdomen. It is recommended that the patient should be placed in the left lateral decubitus position and steep Trendelenburg position to allow the gas to rise to the apex of the right ventricle and to prevent entry into the pulmonary artery. Hyperventilation with 100% oxygen to increase CO₂ clearance and to relieve hypoxemia and aggressive cardiopulmonary resuscitation should also be performed. See Table.

The initial sign of a CO₂ embolism that emerged in the patient presented in this case was the complete loss of an etCO₂ tracing. According to Lee et al., “[t]he formation of a CO₂ embolism during laparoscopy must be suspected whenever there is a sudden change in the end-tidal CO₂.” The drop in etCO₂ is due to a reduction in pulmonary blood flow caused by the embolism. In addition to the drop in etCO₂, the patient also experienced hemodynamic instability. Typically, it is believed that the hemodynamic compromise is due to a “gas lock” that occurs after high-pressure insufflation of CO₂. This gas lock created by the embolism travels to the vena cava and right atrium, which then decreases venous return and cardiac output. Geissler et al. give another proposed rationale for cardiac instability. They propose that the primary mechanism for cardiac dysfunction after venous embolism is increased right ventricular afterload and arterial hypotension, possibly with subsequent right ventricular ischemia, rather than right ventricular outflow obstruction by an airlock.

In a review of the current literature, it was found that laparoscopic procedures are performed for a variety of surgeries, including gastrointestinal, gynecologic, urologic, and vascular. Although a venous gas embolism is a rare complication of laparoscopic surgeries, (nearly 0.0014% to 0.6% of laparoscopic cases), it is associated with a 28% mortality rate. The literature review also
found that the incidence of gas embolism occurs more frequently with gynecological surgeries, specifically laparoscopic procedures associated with hysteroscopy.8

A gas embolism can occur after the insertion of the trocar or Veress needle as CO2 is inadvertently entrained into the vascular system through an open vessel upon insufflation. In this case it is believed that the uterine wall rupture, which occurred from the surgical instrumentation used during the dilatation and curettage, created an entry into the venous system for the CO2.

In addition to auscultation of a “mill-wheel” murmur, the use of transesophageal echocardiography (TEE) can be useful in detecting gas emboli. However, because of the buffering system in the bloodstream, CO2 is highly soluble, is rapidly absorbed, and diffuses readily across membranes.1 Therefore, it is believed that the CO2 embolism was rapidly dissolved and a TEE would not have been helpful.

Treatment of CO2 embolism includes the discontinuation of nitrous oxide. Although nitrous oxide does not enlarge the CO2 embolism as it does with an air embolism, it is essential to provide the patient with 100% oxygen. Insufflation should be stopped and an increase in the rate and tidal volume of controlled ventilation with positive end-expiratory pressure should be initiated to minimize the gas entrainment. If possible, the patient should be placed in left lateral decubitus and steep Trendelenburg positions. This allows the gas to become trapped in the apex of the right ventricle rather than the pulmonary outflow tract.2 If the patient has a central venous catheter, it is helpful for diagnosis and therapeutic aspiration of air. Treatment also includes supportive measures, with the administration of a large volume of fluids and vasopressors. This patient was not receiving nitrous oxide and the treatment included all previously mentioned measures, with the exception of the withdrawal of air from a central venous catheter.

Conclusion
The purpose of this case report was to review the signs and symptoms of a CO2 embolism and discuss the anesthetic implications if one occurs during the intraoperative period. As an anesthetic provider, it is essential to remember that CO2 embolism is a recognized risk during laparoscopic surgery. With the use of appropriate monitors and diligent observation of the patient, early detection and corrective treatment can occur rapidly.

REFERENCES

AUTHOR
Heather J. Smith, CRNA, MSN, is a staff nurse anesthetist at Northern Regional Hospital, Petoskey, Michigan. At the time this paper was written, she was a senior student at Oakland University, Beaumont Hospital Graduate Program of Nurse Anesthesia, Royal Oak, Michigan. Email: hjsmith@oakland.edu.

ACKNOWLEDGMENT
I thank Lisa Mileto, CRNA, MSN, and Anne Hranchook, CRNA, MSN, for their support and encouragement throughout graduate school and especially for their assistance throughout the editing process of this case report.

Table. Symptoms and Treatment of Carbon Dioxide (CO2) Embolism
(Adapted from Yao et al.1)

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sudden increase followed by rapid drop in end-tidal CO2</td>
<td>Immediate cessation of insufflations</td>
</tr>
<tr>
<td>Hypotension</td>
<td>Discontinuation of nitrous oxide to allow for 100% oxygen delivery</td>
</tr>
<tr>
<td>Hypoxia</td>
<td>Steep left lateral decubitus position</td>
</tr>
<tr>
<td>Cyanosis</td>
<td>Hyperventilation</td>
</tr>
<tr>
<td>Cardiac arrest</td>
<td>Placement of central venous access for diagnosis and therapeutic aspiration of air</td>
</tr>
<tr>
<td>Auscultation of a “mill-wheel” murmur with precordial and transesophageal Doppler ultrasonography</td>
<td>Cardiopulmonary resuscitation if necessary</td>
</tr>
</tbody>
</table>