CASE REPORT

Cardiac Arrest due to Carbon Dioxide Embolism During Laparoscopic Gynecologic Surgery of a Patient with Previous Abdominal Surgery: A Case Report

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Catastrophic carbon dioxide (CO₂) embolism is a rare, but potentially life-threatening, the complication of laparoscopic gynecologic surgery. We report the case of a healthy 53-year-old woman who developed CO₂ embolism and cardiac arrest during laparoscopic surgery. She had a history of two cesarean sections and had extensive peritoneal adhesions. After placement of the trocar and insufflation of CO₂, end-tidal CO₂ dropped from 35 to 15 mm Hg, and the patient had a cardiovascular collapse. In this patient, CO₂ embolism was diagnosed on the basis of a sudden decrease in end-tidal CO₂, hypotension, and hypoxemia. The patient was managed quickly and aggressively. The patient recovered completely following the treatment for CO₂ embolism, with no cardiopulmonary or neurological sequelae. There is an increased risk of catastrophic CO₂ embolism during laparoscopic gynecologic surgery in patients with previous abdominal surgery. Therefore, the surgeon and anesthesiologist should remain vigilant to promote early detection of CO₂ embolism.

Keywords: Carbon dioxide; Embolism; Laparoscopy; Case report

INTRODUCTION

Laparoscopic surgery is widely accepted as an alternative to open surgery for the management of various benign gynecologic diseases. Compared to laparotomy, laparoscopy is associated with less pain and bleeding because of smaller incisions, as well as a shorter recovery time [1]. However, there are concerns about the potential serious complications of laparoscopy, including puncture of hollow viscera and major vessels, hemorrhage, subcutaneous emphysema, pneumomediastinum, pneumothorax, and gas embolism [2,3]. Gas embolism is a rare but life-threatening complication of laparoscopy, with a mortality rate of 28.5% [4].

Gas insufflation is usually used during laparoscopic surgery for accurate visualization and manipulation. Carbon dioxide (CO₂) is typically used to create a pneumoperitoneum. Intravascular injection of CO₂ may result from needle or trocar placement into a vessel, or gas insufflation into an abdominal organ. This complica-

tion usually develops during the induction of pneumoperitoneum, particularly in patients with previous abdominal surgery [3].

We report a case of cardiac arrest associated with CO₂ embolism during laparoscopic gynecologic surgery, in a patient with a history of abdominal surgery.

CASE REPORT

A 53-year-old female patient was scheduled for laparoscopic supracervical hysterectomy because of menorrhagia. Her surgical history included two cesarean sections. The patient did not have any significant medical history or drug allergies, and her blood tests, electrocardiogram, and chest radiography were normal.

Propofol was used to induce anesthesia, and rocuronium was used to facilitate endotracheal intubation. The patient was mechanically ventilated, and anesthesia was maintained with desflurane mixed with 30% air and oxygen. After general anesthesia had

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been induced, the patient was changed from the supine to lithotomy position. Due to extensive adhesions, the surgeon attempted trocar insertion twice. Pneumoperitoneum was achieved with CO₂ gas insufflation, and the intraabdominal pressure was maintained at 12 mm Hg. Shortly thereafter, the end-tidal CO₂ dropped from 35 to 15 mm Hg, the oxygen saturation decreased from 99% to 85%, the heart rate decreased to 45 beats/min, and profound hypotension (blood pressure: 69/25 mm Hg) developed, followed by an unrecordable blood pressure. And a few second later, ventricular fibrillation developed. The surgeon was advised to stop the surgery, and CO₂ insufflation was stopped. Simultaneously, the patient was ventilated with 100% oxygen and hydrated with fluids. External cardiac compression was begun, and 1 mg of epinephrine was injected twice. In addition, electric cardioversion was performed. Five minutes after the initiation of external cardiac massage, spontaneous circulation returned. The right radial artery was cannulated, and a central venous catheter was inserted into the right internal jugular vein. Aspiration from this catheter did not obtain gas. Transesophageal echocardiography (TEE) did not find evidence of gas bubbles or interventricular septal shift toward the left ventricle. An arterial blood gas analysis shortly after the event revealed the arterial pH 7.162 and partial pressure of carbon dioxide (PCO₂) 66.9 mm Hg, partial pressure of oxygen (PO₂) 176.2 mm Hg, HCO₃ 23.4 mEq/L, arterial oxygen saturation (SaO₂) 98.7%, and fraction of inspired oxygen (FiO₂) was 1.0, suggesting respiratory acidosis and decreased perfusion, consistent with CO₂ embolism. Because of transient improvements with epinephrine boluses, norepinephrine and dopamine infusion was started. A repeat arterial blood gas analysis showed a pH of 7.264, PCO₂ of 48.5 mm Hg, PO₂ of 448.9 mm Hg, HCO₃ of 21.5 mEq/L, SaO₂ of 98.7%, and FiO₂ of 1.0. The patient recovered from the cardiac arrest, but remained hemodynamically unstable. Therefore, she was transferred to the intensive care unit (ICU) with continuous vasopressor infusion, and subsequently managed by the ICU physician.

The patient was extubated and the vasopressor infusion was discontinued on the first postoperative day. She was moved to the general ward on postoperative day 4, and discharged on day 13 without any cardiopulmonary or neurological sequelae.

Written informed consent was obtained from the patients for the publication of this report. As this case was not a clinical trial, no ethical approval was required.

DISCUSSION

CO₂ is an inexpensive gas that is more soluble in blood compared to air, oxygen, and N₂O. CO₂ is rapidly eliminated from the body and is non-combustible. Rapid elimination of CO₂ increases the safety margin in cases of accidental intravenous injection [5]. The definitive diagnosis of CO₂ embolism depends on the detection of CO₂ emboli in the right side of the heart; however, due to the rapid elimination of CO₂, other physiological parameters must be used for diagnosis [6].

CO₂ embolism may be asymptomatic but can lead to neurologic injury, cardiovascular collapse, and even death. The presentation of CO₂ embolism depends on the speed and volume of the embolized gas that enters the blood [7].

A previous study reported that adhesions, formed as a result of visceral or parietal peritoneum injury, were found in 94% of patients after laparotomy [8]. Therefore, patients with a history of abdominal surgery are at risk for adhesions [4]. In our patient, previous cesarean sections caused the severe intraabdominal adhesions, which may have been responsible for the accidental intravascular injection of CO₂.

Embolism occurs when gas or other particulate matter enters the systemic venous system. End-tidal CO₂ is the most important parameter for early detection of CO₂ embolism. A rapid decrease in end-tidal CO₂ may be caused by obstruction of the pulmonary vasculature by emboli, which expands the ventilatory dead space and is a reliable indicator of CO₂ embolism [9]. However, other causes, such as air embolism and thromboembolism, should be excluded [5]. In our patient, CO₂ embolism was diagnosed on the basis of a sudden decrease in end-tidal CO₂, accompanied by hypotension and hypoxemia.

As mentioned previously, CO₂ is more soluble in blood compared to air and nitrous oxide. Therefore, CO₂ will not remain in the gas form for a long time. The lethal dose of air is approximately five times lower than that of CO₂. Therefore, air embolism is far more dangerous than CO₂ embolism [4]. In addition, CO₂ embolism must be differentiated from thromboembolism, because the composition of embolic materials and treatment regimens differ.

TEE is the most sensitive technique to diagnose gas embolism. However, disadvantages of this technique include its high cost, technical complexity, invasive nature, and the need for an experienced anesthesiologist [10]. In addition, laparoscopic surgery is commonly performed even in hospitals without TEE, which is not

routinely recommended during laparoscopy. Furthermore, CO₂ embolism may be missed on TEE because of rapid elimination of CO₂. Therefore, it may not be possible to detect gas bubbles unless TEE is prepared before insufflation, as in our case.

When a massive CO₂ embolism is suspected, the anesthesiologist must advise the surgeons to terminate the surgery, and administer 100% oxygen to correct the hypoxemia and reduce the size of the embolus. Gas insufflation should be stopped, and the entrained gas should be eliminated if possible. The patient should be placed in the left lateral decubitus/steep head down (Durant) position. If these measures are not effective, a central venous catheter may be introduced to aspirate the gas. In our case, we attempted to aspirate the gas bubbles through the central venous catheter, but this failed. Norepinephrine significantly improves ventricular performance without constricting the pulmonary or renal circulation [9].

In previous report, surgery was safely performed after treatment for CO₂ embolism [4]. However, our patient was hemodynamically unstable. Therefore, surgery was terminated and she was transferred to the ICU. Because there was no definitive evidence of CO₂ embolism, the ICU physician managed the patient as in pulmonary thromboembolism, i.e., with heparin and tissue plasminogen activator. Pulmonary angiography was performed to exclude the possibility of pulmonary thromboembolism but there was no evidence of thromboembolism.

In conclusion, there is an increased risk of catastrophic CO₂ embolism during laparoscopic gynecologic surgery in patients with a history of abdominal surgery. Therefore, the surgeon and anesthesiologist should remain vigilant to promote early detection of CO₂ embolism. In addition, communication with other healthcare personnel, including the ICU physicians, is essential for optimal

management of these patients.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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