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Pulmonary Edema after Catastrophic Carbon Dioxide Embolism during Laparoscopic Ovarian Cystectomy

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Laparoscopy is a surgical procedure used both for diagnosis and for various treatments. A rare but sometimes fatal complication of laparoscopy is pulmonary embolism with CO_2 resulting in pulmonary edema. During laparoscopic gynecological surgery in a 29-year-old woman who had previously undergone lower abdominal surgery, the end-tidal CO_2 suddenly increased from 40 mmHg to 85 mmHg and then decreased to 13 mmHg with hemodynamic deterioration. These events are characteristic of a CO_2 embolism. When this occurred, CO_2 insufflation was immediately stopped and the patient was resuscitated. The patient's condition gradually improved with aggressive treatment, but the clinical course was complicated by bilateral pulmonary edema. This case of pulmonary edema was soon resolved with supportive management. The formation of a CO_2 embolism during laparoscopy must be suspected whenever there is a sudden change in the end-tidal CO_2. In addition, the possibility of pulmonary edema should be considered when a CO_2 embolism occurs.

Key Words: Carbon dioxide, laparoscopy, pulmonary edema, pulmonary embolism

INTRODUCTION

Carbon dioxide (CO_2) is used as a distention medium during laparoscopy because it is more tolerable to patients than other gases. However, the insufflation of several liters of CO_2 along with an extreme Trendelenburg position can cause the following: rapid absorption of a large amount of CO_2 into circulation, impeded compensatory ventilation with high intraabdominal pressure, and decreased cardiac index with increased systemic and pulmonary vascular resistance. These occurrences can result in a rare CO_2 embolism, a possibly fatal complication of laparoscopy.[1]

The CO_2 embolism usually occurs during the insufflation of CO_2 or immediately after the completion of CO_2 insufflation,[2] but one should also consider the unexpected occurrence of a CO_2 gas embolism as late as a few hours after the patient has reached a stable cardiorespiratory status.[3,4] Sometimes, an air embolism is accompanied by pulmonary edema, which further complicates the management of the patient.[5,6] It is difficult to predict whether or not pulmonary edema will occur during the clinical course of a CO_2 embolism.

We report a case of a life-threatening CO_2 embolism with concomitant fulminant pulmonary edema that occurred approximately an hour after the completion of CO_2 insufflation.

CASE REPORT

A 29-year-old healthy woman who had previously undergone a cervical cerclage procedure for cervical incompetence was scheduled for laparoscopic gynecological surgery. The preoperative evaluation including chest PA was unremarkable (Fig. 1). Anesthesia was induced smoothly with tracheal intubation and was maintained with 1.5-2% enflurane, 3 L/min nitrous oxide, and 2 L/min O_2. The noninvasive blood pressure, electrocardiography (ECG), pulse oximetry, end tidal CO_2 ($ETCO_2$) and urinary output were monitored. The abdomen was insufflated with CO_2, and an intraabdominal pressure between 10 and 12 mmHg

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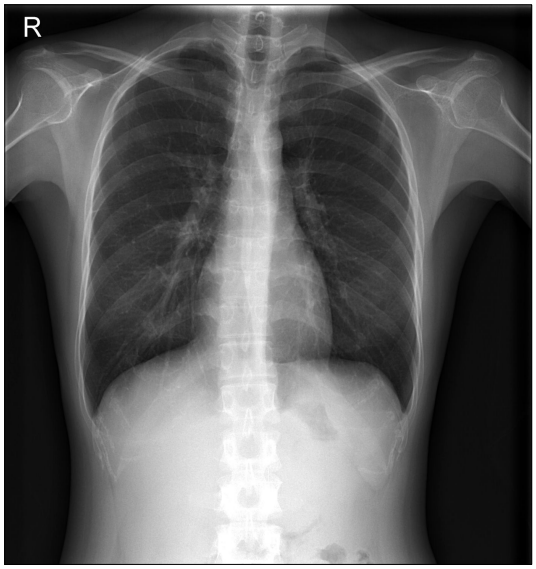

Fig. 1. Chest PA taken before the surgery shows no evidence of a definite abnormality.

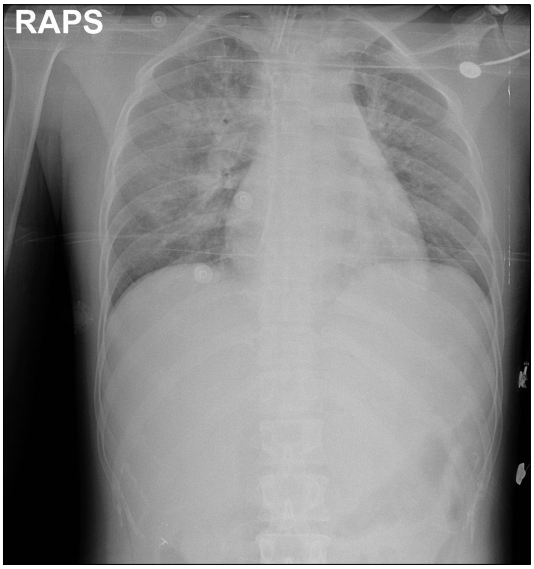

Fig. 2. Chest AP taken 40 minutes after the CO_2 embolism shows newly developing bilateral extensive consolidations in the lungs, indicating pulmonary edema.

was maintained with the laparoscopic machine (CO_2-aqua-purator 1611: WISAP: Germany), followed by the lithotomy and a 20 degree Trendelenburg head-down position. The surgery and anesthesia were uneventful for one hour with an arterial blood pressure (BP) of 100 - 120/70 - 100 mmHg, a heart rate of 70 - 102 bpm, $ETCO_2$ of 30 - 40 mmHg, and SpO_2 of 100%.

During irrigation and bleeding control, the $ETCO_2$ suddenly increased from 40 mmHg to 85 mmHg and then dropped to 13 mmHg. The BP decreased to 55/30 mmHg and a "mill-wheel" precordial murmur was heard on auscultation. A pulmonary CO_2 embolism was suspected. In response to these events, the inhalational anesthetics were discontinued, and the FiO_2 was changed to 100% while the CO_2 insufflation was stopped and the laparoscope was removed. At the same time, a 1 mg bolus of epinephrine was administered intravenously with a continuous infusion of 1 mg epinephrine and 4 mg norepinephrine in NS 100 mL while the IV fluid rate was increased. Despite these efforts, the BP and $ETCO_2$ were still low. Every 2 - 3 minutes, 1.0 mg of Atropine was administered, but the patient's heart rate decreased further and no beat was observed for approximately 15 to 30 seconds. Immediately, chest compression was initiated. After 2 - 3 minutes of compression, fine ventricular fibrillation was observed on the ECG, and defibrillator cardioversion (DC) at 200 J was delivered to the patient's anterior chest. The heart rate returned to sinus rhythm, but it was immediately converted to ventricular fibrillation. With another application of 200 J DC and 1% lidocaine 60 mg intravenously, the beat was converted to sinus tachycardia. However, the BP was still low at 55/28 mmHg.

The right subclavian vein was cannulated, and her hemodynamics improved gradually. Approximately 40 minutes after the incident, her BP was 130/80 mmHg, her heart rate was 135 bpm, her $ETCO_2$ was 30 mmHg, her SpO_2 was 100%, with her FiO_2 of 100%. At that time, chest radiography showed newly developing extensive bilateral consolidations in her lungs, suggesting pulmonary edema (Fig. 2). Twenty milligrams of Furosemide and 125 mg of methyl-prednisolone were administered intravenously, and her central venous pressure was maintained within 5 mmHg. In order to locate the hidden bleeding, the lower abdomen was incised and examined. But the bleeding was negligible.

A ventilator was applied to the patient in the intensive care unit, and supportive care was carried out to treat this case of pulmonary edema.

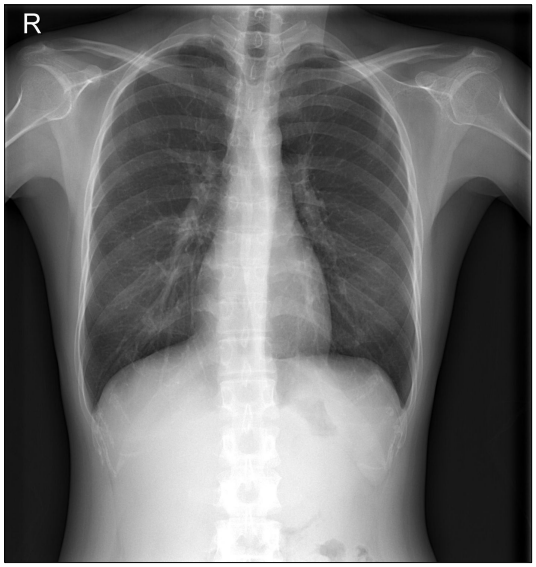

Fig. 3. Chest AP taken the morning after surgery shows clear lungs without an enlarged heart.

Three hours after admission to the intensive care unit, the ventilator was disconnected and her trachea was extubated. She was alert, and her BP was 110/70 mmHg, her heart rate was 95 bpm, her respiration rate was 20/min, and her SpO_2 was 99% with a facemask. The next morning, the chest AP showed clear lungs (Fig. 3).

DISCUSSION

The initial sign in this case included an abrupt and transient increase in the $ETCO_2$ to 85 mmHg, which likely was related to the acute increase in the CO_2 level in the vascular system followed by exhalation from the respiratory system.[7] Subsequently, there was a decrease in the $ETCO_2$ to 13 mmHg. In addition, there was a deterioration of the hemodynamic parameters with a decrease in the BP and heart rate, as well as a mill-wheel murmur consistent with the manifestation of a gas embolism. These manifestations can be explained by the increased CO_2 causing overcrowding in the right heart and the occluded right outflow tract of the heart, which was observed in the gas embolism.[8] However, Geissler et al.[9] have proposed 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 this case, the presence of the CO_2 in the right ventricle was the primary etiology causing cardiac dysfunction. It is believed that the immediate termination of laparoscopic gas insufflation blocked any further CO_2 entry into the vascular system, and the immediate resuscitative measures were useful in preventing any devastating events.

The manner in which CO_2 entered the vessels during the laparoscopic surgery is unclear. When a CO_2 embolism is encountered, it normally occurs at the beginning of or immediately after the completion of CO_2 insufflation.[2] In addition, the risk of an embolic episode decreases after the completion of the pneumoperitoneum because increased intra-abdominal pressure can cause the collapse of the injured vessel. In contrast, our case occurred more than an hour after the insufflation of CO_2.

There are several possible reasons for this embolism. First, the previous lower abdominal surgery might have caused peritoneal adhesions that could have ruptured during the forceful irrigation. This may have caused CO_2 to enter into the circulatory system through the injured vessels and then led to the CO_2 embolism.[2] Another possibility is inadvertent use of the laparoscopic machine. In our case, irrigating solutions were introduced into the intraperitoneal cavity by CO_2 pressure; an unnoticed empty irrigation bottle or a CO_2 mixed irrigation solution could have allowed for the entry of CO_2 into the open venous sinuses under pressure. The coincidence of the occurrence of CO_2 embolism with the irrigation and bleeding control in this case support this possibility. Additionally, CO_2 might have become trapped in the portal system after transperitoneal absorption and later might have been released into the circulation.[4] Finally, although direct injury to a vessel with a Veress needle might be possible, this possibility is less likely because the embolus occurred one hour after completing the pneumoperitoneum.

Although short-lived, the patient developed pulmonary edema in the operating room and required intensive therapy even after surgery in the intensive care unit. The cause of pulmonary

edema associated with laparoscopic surgery, especially with CO_2, is unclear. However, there are some reports of noncardiogenic pulmonary edema as a consequence of an air embolism.[5,6] In these types of permeability edema, nonmechanical factors, such as the release of superoxide dismutase from leukocytes, are known to be involved.[5,10] Moreover, severe pulmonary edema after a venous air embolism is often associated with massive fluid loss.[6] This is not believed to be the cause of this case of pulmonary edema even though much fluid volume had been administered during resuscitation. This administration of fluid might have helped to resuscitate the patient more rapidly.

In this case of pulmonary edema, steroid, mechanical ventilation with positive end expiratory pressure was required.[5] To avoid aggravation of the edema, infused fluid should be carefully controlled. In this case we managed the patient with mechanical ventilation and fluid control under the guidance of central venous pressure. Luckily, patient oxygenation was improved when the edema was discovered, so we avoided the use of positive end expiratory pressure, which can cause cardiac depression during treatment.

Recovery time is relatively short after such events. CO_2, the causative agent in this case, is highly soluble compared with other gases including helium, argon, nitrogen, oxygen, and air.[1] Furthermore, the bubbles of CO_2 gas rapidly traverse the vascular systems and disappear. These physiochemical characteristics of CO_2 may shorten the clinical course and contribute to a more rapid recovery.

According to this report, if a cardiac event occurs during laparoscopic surgery, one should consider a CO_2 embolism, even if the events occur after the completion of CO_2 insufflation. In order to prevent devastating events, preventive monitoring of the $ETCO_2$, SpO_2, and heart and lung sounds is essential. However, when it does occur, pulmonary edema should also be considered during patient management.

REFERENCES

1. Gutt CN, Oniu T, Mehrabi A, Schemmer P, Kashfi A, Kraus T, et al. Circulatory and respiratory complications of carbon dioxide insufflation. Dig Surg 2004;21:95-105.
2. Cottin V, Delafosse B, Viale JP. Gas embolism during laparoscopy: a report of seven cases in patients with previous abdominal surgical history. Surg Endosc 1996; 10:166-9.
3. Haroun-Bizri S, ElRassi T. Successful resuscitation after catastrophic carbon dioxide embolism during laparoscopic cholecystectomy. Eur J Anaesthesiol 2001; 18:118-21.
4. Root B, Levy MN, Pollack S, Lubert M, Pathak K. Gas embolism death after laparoscopy delayed by "trapping" in portal circulation. Anesth Analg 1978;57:232-7.
5. Clark MC, Flick MR. Permeability pulmonary edema caused by venous air embolism. Am Rev Respir Dis 1984;129:633-5.
6. Lam KK, Hutchinson RC, Gin T. Severe pulmonary oedema after venous air embolism. Can J Anaesth 1993; 40:964-7.
7. Butler BD, Hills BA. The lung as a filter for microbubbles. J Appl Physiol 1979;47:537-43.
8. Souders JE. Pulmonary air embolism. J Clin Monit Comput 2000;16:375-83.
9. Geissler HJ, Allen SJ, Mehlhorn U, Davis KL, Morris WP, Butler BD. Effect of body repositioning after venous air embolism. An echocardiographic study. Anesthesiology 1997;86:710-7.
10. Flick MR, Hoffel JM, Staub NC. Superoxide dismutase with heparin prevents increased lung vascular permeability during air emboli in sheep. J Appl Physiol 1983;55:1284-91.