

Re-insufflation after deflation of a pneumoperitoneum is a risk factor for CO₂ embolism during laparoscopic prostatectomy

-A case report-

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Although symptomatic carbon dioxide (CO₂) embolism is rare, it is recognized as a potentially fatal complication of laparoscopic surgery. Sudden hemodynamic instability could be a CO₂ embolism especially during insufflation. A 65-year-old man received laparoscopic prostatectomy for 5 hours under CO₂ pneumoperitoneum without any problem. After resection of prostate, it was stopped following deflation. Thirty minutes later, pneumoperitoneum was re-induced to continue the operation. Shortly after re-insufflation, the patient revealed hemodynamic instability suggested a CO₂ embolism; severe hypotension, tachyarrhythmia, hypoxemia, increased CVP, and changed end-tidal CO₂. Gas insufflation was stopped. He was managed with Durant's position, fluid and cardiotonics for 20 minutes. The residual was completed by open laparotomy. Re-insufflation, inducing gas entry through the injured vessels, might be a risk factor for CO₂ embolism in this case. The risk to the patient may be minimized by the surgical team's awareness of CO₂ embolism and continuous intra-operative monitoring of end-tidal CO₂. (Korean J Anesthesiol 2010; 59: S201-S206)

Key Words: Cardiovascular collapse, CO₂ embolism, Laparoscopic prostatectomy, Re-insufflation.

Laparoscopic prostatectomy was introduced in 1992, and the Montsouris technique, which is an advanced laparoscopic prostatectomy, has been widely used since 2000 [1]. In contrast to laparotomy, laparoscopic surgery has some benefits such as much less bleeding, less pain, a shorter hospital stay and a faster return to the normal life. However, the laparoscopic surgery requires creating a pneumoperitoneum (gas in the abdominal

cavity) using CO₂ gas to secure the visual field and space for surgery, which may cause very rare but serious complications such as CO₂ pneumothorax, pneumopericardium, pulmonary emphysema, pneumomediastinum, CO₂ embolism, etc. [2-7]. In particular, CO₂ embolism may lead to cardiovascular collapse and this can be fatal [2,3]. Herein, we report on our experience with a patient who developed sudden severe

Received: May 13, 2010. Revised: May 27, 2010. Accepted: July 6, 2010.

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cardiovascular collapse when re-insufflation of CO₂ gas was done after deflation of the pneumoperitoneum for laparoscopic prostatectomy, and we suggest that re-insufflation may be a causative factor for CO₂ embolism during laparoscopic surgery.

Case Report

A 65-year-old, 175 cm, 91 kg, male patient with prostate cancer was admitted for laparoscopic prostatectomy. He had an unremarkable medical history without any surgery. On the blood tests, the hemoglobin was 11.3 g/dl and the hematocrit was 35.1%. The liver function, renal function, blood coagulation time and bleeding time were all in the normal ranges. The electrocardiogram (EKG) was also normal, but simple chest X-ray showed bronchitis. On the pulmonary function test (PFT), the FEV₁ was 68% (2.74 L) and FEV₁/FVC was 79%.

For premedication, glycopyrrolate 0.2 mg was intramuscularly injected. The vital signs before anesthetic induction showed a blood pressure (BP) of 150/90 mmHg, a heart rate (HR) of 65/min, a respiratory rate (RR) of 16/min, a body temperature of 36.5°C and the oxygen saturation on pulse oximetry (SpO₂) was 96%. After pre-oxygenation with 100% oxygen, anesthesia was induced with thiopental sodium 250 mg and rocuronium 50 mg. Endotracheal intubation was then performed with an 8.0 mm inner diameter (ID) endotracheal tube.

Anesthesia was maintained with O₂ and N₂O both at 1.5 L/min and sevoflurane at an optimal concentration, while vecuronium was intermittently administered to maintain adequate muscle relaxation. The partial pressure of end-tidal carbon dioxide (E_TCO₂) was continuously monitored from the induction of anesthesia, and it was 33 mmHg right after tracheal intubation. After endotracheal intubation, the gastrointestinal tract was suctioned using a Levin tube. A central venous pressure (CVP) catheter was inserted into the right subclavian vein and a 16-G catheter was placed in the left external jugular vein. A modified Allen's test was performed at the left radial artery and then a catheter was inserted to continuously monitor the arterial pressure.

After anesthetic induction, mechanical ventilation was performed with a tidal volume (TV) of 550 ml and a RR of 12/min. At that time, the BP was 120/70 mmHg, the HR was 58/min and there was a SpO₂ of 99%, a CVP of 8–9 mmHg, an E_TCO₂ of 30 mmHg, a peak airway pressure of 21 cmH₂O and a mean airway pressure of 17 cmH₂O. The arterial blood gas analysis (ABGA) showed normal findings and the EKG also indicated a regular sinus rhythm.

When the operational preparations were completed, the surgeon connected a trocar to a carbon dioxide gas pump and induced a pneumoperitoneum using CO₂ gas. The abdominal cavity pressure was maintained at 12 mmHg. After

the insufflation, the E_TCO₂ gradually increased to 40 mmHg; therefore, the RR increased to 14/min. The patient was placed in Trendelenburg's position at 25–30 degrees at the surgeon's request. At that time, the CVP was 13 mmHg, the peak airway pressure was 31 cmH₂O, the mean airway pressure was 23 cmH₂O, the E_TCO₂ was 34–35 mmHg and the SpO₂ was between 98 and 100%. Another ABGA one hour after the onset of operation showed a pH of 7.42, a PaCO₂ of 40 mmHg, a PaO₂ of 165 mmHg, a HCO₃⁻ of 25.9 mEq/L, a base excess of 1.4 mmol/L, a SaO₂ of 100% and a hematocrit of 32%.

It took 5 hours to completely remove the prostatic gland with five trocars under a pneumoperitoneum. After removal of the prostatic gland and local hemostasis was performed under laparoscopic surgery, the operation was interrupted due to the surgeon's unavoidable reasons. The patient was then repositioned from Trendelenburg's position to the normal position, and the pneumoperitoneum was deflated by ventilating two or three times a large volume of gas and the trocars were not removed from the abdominal cavity. After the deflation of the pneumoperitoneum, the peak airway pressure gradually decreased to 22 cmH₂O and the E_TCO₂ also gradually decreased to 30 mmHg. At that time, his BP was 110/70 mmHg, the HR was 65/min and the SpO₂ was 99%, which indicated that his vital signs were stable. Up to that moment, the total blood loss was less than 200 ml and the hematocrit was 31%.

About 30 minutes later, the patient was repositioned in the Trendelenburg position, and pneumoperitoneum with re-insufflation of CO₂ gas into the abdominal cavity was induced with keeping the intra-abdominal pressure less than 12 mmHg. During insufflation, there was no intra-abdominal bleeding and no other abnormalities detected on the viewing screen of the laparoscope. When the gas insufflation was close to the end, the patient's BP dropped suddenly from 120/80 mmHg to 60/30 mmHg, and HR increased sharply from 60/min to 100/min with tachyarrhythmia, including premature atrial contractures in a few seconds. Later, the HR returned to normal sinus rhythm with a rate of 78/min, and also the SpO₂ decreased from 99% to 92%. The peak airway pressure increased from 31 cmH₂O to 38 cmH₂O, and the E_TCO₂ also increased from 35 mmHg to 39 mmHg and the CVP increased from 13 mmHg to 18 mmHg. Manual ventilation was immediately performed using 100% oxygen, and the gas insufflation was stopped. Intravascular fluids were rapidly administered and ephedrine 8 mg was injected. The breathing sounds were clear on both lung fields and there were no secretions during endotracheal suction. His BP was not increased, and 2 doses of ephedrine 8 mg and phenylephrine 100 µg were additionally injected. However, his BP was not restored and it remained at 70/40 mmHg, and so dopamine was infused at 6 µg/kg/min. Under the suspicion of CO₂ embolism, he was moved into Durant's position (the left

lateral decubitus position with a head-down tilt) [4] and blood was aspirated via the CVP catheter, but gas bubbles were not detected. About 15 minutes after the onset of hypotension, transesophageal echocardiography (TEE) was done at the middle of the esophagus for monitoring with a probe to secure a good view in all directions, but no gas bubbles were observed in the 4 chambers. A simple chest X-ray was taken to exclude the possibility of CO₂ pneumothorax, subcutaneous emphysema and pneumomediastinum, and there were no abnormalities other than atelectasis in both lower lobes. No subcutaneous emphysema was detected.

Approximately 20 minutes after the sudden drop in BP, his vital signs took a favorable turn: the BP was 100/60 mmHg, the HR was 85 /min and the SpO₂ was 99%. However, the peak airway pressure gradually increased to 39 cmH₂O and the E_TCO₂ increased to 40 mmHg. At the moment, the TV was maintained at 550 ml and the RR was kept at 14/min. ABGA showed that the pH was 7.30, the PaCO₂ was 49 mmHg, the PaO₂ was 116 mmHg, the HCO₃⁻ was 24.1 mEq/L, the base excess was -2.3 mmol/L and the SaO₂ was 98%. The hematocrit was 29% and the blood glucose was 166 mg/dl.

Forty minutes after the BP decline, his vital signs were maintained at the BP of 100/60 mmHg, a HR of 80/min, a CVP of 15 cmH₂O and an E_TCO₂ of 37 mmHg. At that time, the surgeon decided to perform laparotomy because the configuration of the patient's prostate gland and bladder neck was so abnormal that suturing of the incision site was difficult. Yet as the open surgery proceeded, massive bleeding developed, which required a transfusion of blood. ABGA during the transfusion showed a pH of 7.28, a PaCO₂ of 46 mmHg, a PaO₂ of 175 mmHg, a HCO₃⁻ of 21.6 mEq/L, a base excess of -5.1 mmol/L, a SaO₂ of 99% and a hematocrit of 27%.

For fear that an increase in the peak airway pressure and CVP could cause pulmonary edema, 10 mg of furosemide was intravenously injected after performing vesico-urethral anastomosis. The amount of urine was 500 ml within the first hour and this was followed by approximately 300 ml of urine output per hour. About 3 hours after the onset of laparotomy, the operation was completed. It took 9 hours and 15 minutes for the operation and 10 hours and 10 minutes for anesthesia. The total amount of infused fluid was 9,200 ml, the amount of transfused blood was 1,600 ml, the urine output was 1,080 ml and the estimated blood loss was 2,500 ml.

At the end of operation, the patient was transferred to the intensive care unit (ICU) with the endotracheal tube in place. The chest X-ray conducted one hour after transfer revealed a finding of mild pulmonary edema. With the patient under synchronized intermittent mandatory ventilation (SIMV), the peak airway pressure was 28 cmH₂O and the CVP was 13 cmH₂O. Three hours later, the peak airway pressure and CVP

decreased to 25 cmH₂O and 12 cmH₂O, respectively. Extubation was performed on the second postoperative day (POD 2), and he was transferred to the general ward on POD 4. On POD 10, he was discharged home without any related complications.

Discussion

We encountered an episode of sudden severe cardiovascular collapse during laparoscopic prostatectomy. After the pneumoperitoneum was transiently deflated and the surgery was stopped after removal of the prostate under laparoscopic surgery, when we tried to resume the surgery with creating a pneumoperitoneum by re-insufflation of CO₂ gas into the abdominal cavity, an abrupt severe cardiovascular collapse emerged. Two major pathogeneses have been described for the major causes of cardiovascular collapse and cardiac arrest during laparoscopic surgery: gas embolism caused by gas insufflations and the vagal reflex activated by peritoneal stretch [6].

Compared with conventional open surgery, laparoscopic surgery has benefits such as less tissue damage, less post-operative pain, a faster return to everyday living due to the short recovery time, less post-operative scarring, etc., and so laparoscopic surgery has been widely used in various areas. In most cases, in order to secure the visual field and space for surgery, a pneumoperitoneum is deliberately induced for the surgery. A pneumoperitoneum is achieved by insufflating the abdomen with gas such as carbon dioxide, nitrous oxide, helium, argon, etc. [5,6]. Among them, CO₂ is largely used because it is chemically inert, colorless, inexpensive, readily available and less explosive than air, and it also has such high solubility in the blood that there is less risk of embolism even though CO₂ is absorbed fast into the blood [5,6].

When gas is insufflated into the abdominal cavity, the abdominal pressure increases and the CO₂ in the abdominal cavity is absorbed into the blood stream, leading to hypercarbia. An increase in intraperitoneal pressure consequently reduces the venous return and ventricular preload. It also compresses the arterial vessels, so that the afterload and systemic vascular resistance increase, whereas the cardiac output decreases [5,6,8]. To minimize such hemodynamic changes, it is recommended to maintain the intraperitoneal pressure at 12 mmHg or less [6,9].

The risk of complications during laparoscopic surgery such as hypercarbia, subcutaneous emphysema, pneumothorax and pneumomediastinum may increase according to a longer duration of surgery, a higher E_TCO₂, a greater number of used trocars and an older age of the patient [10]. If an infusion flow rate reaches over 1 L/min or the intra-abdominal pressure passes over 15 mmHg during insufflation of CO₂ gas, then

complications seem to easily develop [5]. In other words, too fast an infusion rate of CO₂ with excessive pressure is likely to induce embolism, causing the gas lock phenomenon, which interrupts blood flow in the vena cava or the right atrium, and then it decreases the cardiac output and induces cardiovascular collapse [3,6]. About 60% of the symptomatic cases of CO₂ embolism occur during the early period of insufflations of CO₂ [6], while the majority of the episodes incur when trocars are improperly inserted into the abdominal cavity or they are directly inserted into veins or organs. Sometimes, gas embolism may develop without direct infusion of gas when the gas diffuses into the damaged vessels around the surgical site or the abdominal wall.

In the present case, sudden and serious hypotension occurred during the induction of pneumoperitoneum, which was performed to resume the operation. CO₂ embolism was suspected as the primary cause of the hypotension due to the diffusion of carbon dioxide into injured blood vessels that were damaged by the surgical operation. Other factors, such as hypovolemia, a vagal reflex caused by sudden peritoneal stretch, CO₂ pneumothorax, pneumomediastinum, pulmonary emphysema or air embolism could be considered as alternative causes. Considering that there was no bleeding caused by the operation until the development of hypotension and that the laparoscopic findings observed since the resumption of pneumoperitoneum did not detect any bleedings in the abdominal cavity, and that the CVP and urine output were properly maintained, hypovolemia was unlikely to be the cause. As for the peritoneal stretch-induced vagal reflex, it could also be ruled out as the cause because the HR during the hypotension was not low enough to be regarded as bradycardia, but it was maintained at 78/min, which was slightly higher than the peri-operative HR. Simple chest X-ray helped eliminate the possibility that the hypotension was induced by CO₂ pneumothorax, pneumomediastinum or pulmonary emphysema. Air embolism unusually can occur in open prostate surgery because of the affluent venous vascular plexus in the prostate gland and using Trendelenburg's position. However, in the present case, there was less chance that air entered into the blood circulation because the laparoscopic surgery created airtight conditions and the trocars still remained in the abdominal cavity even though the operation was suspended. Therefore, CO₂ embolism was suspected to be responsible in the current case for such phenomena as the abrupt onset of hypotension, the relative tachycardia following several occurrences of premature atrial contraction, the increase in the CVP, the increase in the E_TCO₂, the decrease in oxygen saturation (SpO₂), etc.

The symptoms of CO₂ embolism vary according to the amount of gas absorbed into the blood and the inflow rate of gas into

the blood. With the rapid influx of a large amount of gas, a gas-lock takes place, leading to a significant decrease in the cardiac output and an increase of the physiological dead space due to a large amount of effluent gas into the lung. Severe embolism may cause hypotension, bradycardia, arrhythmia, an increase in the mean pulmonary artery pressure, an increase in the CVP, a change in the Doppler wave form and heart sounds, millwheel murmur, cyanosis, excessive right heart strain seen on EKG, an increase in the peak airway pressure, a change in the E_TCO₂ (an increase in the early stage and a decrease in the late stage), etc. Sometimes, it may induce pulmonary edema or sudden death [6,8].

The E_TCO₂ in CO₂ embolism can display an unusual biphasic change [11]. The E_TCO₂ can increase at the early stage of embolism because CO₂ flowing into the blood is emitted through the lung, but it can decrease at the latter stage as the cardiac output decreases and physiological dead space increases with the elapse of time. Yet in the present case, the E_TCO₂ showed a slow increase, but there was no decrease in the latter stage. It might mean that the rate of development or the volume of embolism was not high enough to induce a severe decrease of cardiac output or a big increase of the physiological dead space. Furthermore, while the cardiac output did not decrease significantly, the hypotensive condition lasted for a long time in our patient. The reason for the prolonged hypotension was guessed that the volume effect of the embolism exceeded the rate of embolism formation. Because CO₂ has high solubility in blood, a large amount of absorbed CO₂ may be dissolved into the blood rather than inducing gas-lock or being emitted through the lung, resulting in systemic hypercarbia, which may induce prolonged hypotension by dilating the blood vessels and reducing the peripheral vascular resistance.

The symptoms and risks of gas embolism are different not only according to the gas influx volume and rate, but also for the different kinds and solubility of the gas [5,6,12]. Even a small amount of helium or argon gas embolism can be lethal, while symptoms are rarely seen with a large amount of CO₂ embolism due to its high solubility. Derouin et al. [13] reported that 60% of the patients without any abnormalities of their vital signs revealed CO₂ shadows on the right atrium and ventricle by TEE when the patients had undergone laparoscopic cholecystectomy. In a study by Park et al. [14] when making observations, via TEE, of air shadows in the right atrium of 20 patients who had undergone laparoscopic cholecystectomy, emboli were detected in 4 patients of 20 patients during insufflation of CO₂ gas during dissection of the gallbladder. Their results would support that laparoscopic surgery using pneumoperitoneum is always vulnerable to the possibility of CO₂ embolism.

Monitoring devices such as TEE and a Doppler probe attached to the epigastric/precordial region or the esophagus, and a pulmonary trunk catheter are well-known for their high sensitivity to detect gas embolism, while SpO_2 and E_tCO_2 are useful for the early detection of hypoxia and embolism as well as for estimation of the embolus. For making the definite diagnosis of embolism, TEE is the most sensitive among them. It is 5 to 10 times more sensitive than transthoracic Doppler-echocardiography, enough to be able to diagnose paradoxical embolism due to its capacity to visualize even the left heart. Paradoxical embolism can be caused by the formation of a right-to-left shunt through a patent foramen ovale (PFO), which is detected in 20–30% of the population of normal adults. It is known that TEE can detect even as little as 0.5 cm^3 of air and the detected air can be aspirated by a CVP catheter in 50% of the cases. Yet in the present case, we could not detect any CO_2 shadows on TEE. For these reasons, it is plausible to assume that the amount of CO_2 running into the blood was small or the CO_2 already was dissolved into the blood, or that the CO_2 migrated from the heart into the pulmonary vessels. Considering that TEE was not used directly after the onset of hypotension, but it was used more than 15 minutes after it developed, and that the hemodynamic change and E_tCO_2 decrease were not significant, the CO_2 was more likely to have been dissolved into the blood.

As soon as CO_2 embolism is suspected, the insufflation of CO_2 should be discontinued and the pneumoperitoneum should be deflated, while the patient should be positioned to the Durant position (head-down tilt and left lateral decubitus position) [4]. The Durant position allows air bubbles to migrate towards the apex of the heart, which prevents the formation of a gas-lock by gathering up air into the right ventricular apex. Therefore, when a left lateral decubitus position is difficult to apply, it would be helpful to use the head-down tilt position alone. Anesthesiologists should stop using N_2O and they should alleviate the hypoxia and hypercarbia by performing mechanical controlled hyperventilation using 100% O_2 . Simultaneously, central venous aspiration of blood intimately mixed with air-bubbles can be performed to withdraw gas directly from the heart. The patient's vital signs should be promptly improved by applying adjuvant therapy such as administering fluid and inotropics. In our case, ventilation with 100% O_2 was performed, the patient was placed in Durant's position, air was aspirated and fluid and inotropics such as ephedrine, phenylephrine, dopamine, etc. were administered immediately at the onset of symptoms. Due to excessive embolism, if the patient's cardiovascular collapse is not restored and the patient does not respond to the adjuvant therapy, then a cardiopulmonary bypass (CPB) should be performed [7,15]. Diakun [7] reported a case of severe CO_2 embolism that was encountered during laparoscopic surgery for the excision of

the uterine septum using a hysteroscope. Sudden arrhythmia was rapidly followed in turn by cardiac arrest, an increase of the E_tCO_2 , a decrease of oxygen saturation and an increase of the peak airway pressure. They administered atropine, bicarbonate, epinephrine and calcium and they applied defibrillation four times for resuscitation. There were no responses, and the cardiac rhythm did not respond to all these measures. Then CPB was performed with internal cardiac massage and the patient was successfully resuscitated.

In the present case, we analyzed the risk factors for CO_2 embolism. The most highly possible cause was that a large amount of CO_2 had flowed into the blood through the injured blood vessels that were damaged by the surgical operation, which was caused by the re-induction of the pneumoperitoneum to resume the stopped operation after removal of the prostate gland under laparoscopic surgery. Furthermore, the possibility of a gas insufflation rate over to 12 mmHg and that of the patient being placed into the Trendelenburg position seem to be other risk factors that are responsible for CO_2 running into the damaged vascular plexus of the surgical site. Kypson and Greenville [15] also reported an episode of CO_2 embolism that developed after gas re-insufflation during a coronary artery bypass graft (CABG). After they harvested the saphenous vein using an endoscope and they completed the CPB, they tried to re-insufflate the CO_2 gas in order to confirm hemostasis on the surgical site of the saphenous vein. Then, a serious CO_2 embolism occurred and they had to perform CPB again to resuscitate the potentially fatal cardiovascular collapse. Therefore, it should be emphasized that re-insufflation of CO_2 gas after the deflation of pneumoperitoneum is a strong risk factor for CO_2 embolism because a large amount of gas might flow into the blood through damaged blood vessels. In addition, Cottin et al. [2] reported two deaths out of seven patients with gas embolism that developed during laparoscopic surgery. They indicated that a previous history of having received intra-abdominal surgery could be a potential risk factor for gas embolism due to the possibility of vascular injury.

In summary, we experienced a case of severe cardiovascular collapse when we tried to resume the stopped operation by re-inducing a pneumoperitoneum with CO_2 gas during laparoscopic prostatectomy. Through the current case, it came to our attention that physicians are apt to overlook the potential risks of severe cardiovascular collapse during laparoscopic surgery even though laparoscopic surgery is widely employed. That is to say, it should be noted that the laparoscopic surgery with CO_2 -induced pneumoperitoneum is not free from the risk of CO_2 embolism. Therefore, surgeons should refrain from re-inducing pneumoperitoneum after deflation during laparoscopic surgery, and they should pay closer attention to

the patient's choice for operation and assess the surgical risk factors such as a previous history of intra-abdominal surgery and also properly manage the intra-abdominal pressure. In addition, anesthesiologists should be thoroughly prepared for the rare, but potentially lethal complications of CO₂ embolism.

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