Massive carbon dioxide embolism during pneumoperitoneum for laparoscopic adrenalectomy: A case report

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Introduction

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]. It has been widely used during many types of surgery. There are a number of advantages compared to open procedure which includes: decreased risk of bleeding, infection, and postoperative pain. However, there are also some complications associated with laparoscopic surgery. For example, the pneumoperitoneum may lead to decreased cardiac output and/or increased systemic vascular resistance. In addition, the Carbon dioxide pneumoperitoneum may cause respiratory acidosis and deep venous thrombosis [2].

Although CO2 embolisms are rare, they are potentially fetal if they are not diagnosed or treated immediately. This is especially true when the CO2 from the right heart flows into the left heart [3] Clinically, carbon dioxide embolism can present itself with systemic hypotension, dyspnea, cyanosis, tachycardia or bradycardia, arrhythmia or asystole, elevated pulmonary arterial pressure (PAP), elevated central venous pressure, hypoxemia and increased arterial partial pressure of carbon dioxide, increase or decrease end-tidal carbon dioxide tension [4]. There are case reports about cardiac arrest associated with CO2 embolism during laparoscopic surgery. We are reporting this case of potentially fatal complication of massive CO2 embolism during laparoscopic adrenalectomy at our institute. Even the patient had experienced a massive CO2 embolism during the surgery, he discharged without any complication by the twelfth day after the operation.
was introduced. During the surgery, we began with a high flow of CO₂
intra-peritoneal insufflation to achieve an intra-abdominal pressure
of 15 mmHg.

During the initial 75 minutes, the patient’s vital signs were
stable and the procedure was performed smoothly. However, when
attempting to remove the adrenal gland and adrenal tumor which was
tightly adhered to the IVC, the surgeon accidentally nicked the large
inferior vena cava which resulted in a large IVC tear of about 2×2 cm.
The bleeding from the torn IVC was temporary compressed by the
guaze. However, we suspect that a massive amount of CO₂ entered
the vessel via the IVC broken hole. Taking into account the difficulty
of performing exploratory laparotomy due to previous hepatic tumor
excision related intra-abdominal adhesion, the surgeon tried to sow
the torn IVC by the use of laparoscope and to control the bleeding
via carbon dioxide (CO₂) insufflation pressure. However, we suspect
that a massive amount of CO₂ entered the vessel via the IVC broken
hole while the surgeon sow the torn IVC. They spent about 60 minutes
on repairing the IVC tear. Blood pressure was stable during the IVC
tear insult but dropped slightly after the torn IVC repaired. Systolic
blood pressure during the insult was kept at 100~130 mmHg, pulse
70~90/min, airway pressure was maintained at 35mmHg which may
be induced by the laparoscope and SPO2% was 100%. However, the
end tidal CO₂ gradually elevated from 35 mmHg to 53 mmHg. The
ventilator respiratory rate was then set higher to wash out CO₂.

We then inserted TEE to evaluate the severity of the CO₂ embolism
while the surgeon tried to fix the IVC tear. Through the TEE image,
we noticed that the right atrium and right ventricle were totally white
out and full of air (Figure 1). About 20 minutes after IVC tear, the TEE
image also revealed that the left heart also had scattered air embolisms
(Figure 2). We then inserted a CVC via the right internal jugular vein,
to attempt to drain the CO₂ through the CVC, which failed. Sewing the
fragile IVC tissue proved difficult, so we consulted cardiac surgeons
who were then able to repair the torn IVC. The total massive CO₂ influx
time was about 60 minutes with continuous 15 mmHg CO₂ insufflation
pressure. And the total blood loss was about 1250 ml.

The drop-in blood pressure caused by the IVC tear was restored by
low dose norepinephrine intermittent bolus (5mcg) and blood
transfusion. In total, we infused 2u of pRBC, 2u of whole blood and
12 u of platelet. The blood gas 1hr after the insult showed respiratory
acidosis with PH 7.01, PCO₂ 129%, PO₂ 75% (FiO₂ 86%), SatO₂ 83%. Pulmonary CO₂ embolism induced hypercapnia and hypoxemia was
under impressed. We suspected pulmonary edema because of the
presence of pink bubbled sputum. SPO₂ % was also gradually decreased
to 93%. The blood gas 3 hours after the insult showed PH 7.1, PCO₂
82.9%, PO₂ 186% (FiO₂ 85%), SatO₂ 99%. Post-surgery, the patient was
transferred to the ICU with mechanical ventilation to receive further
care.

During the ICU admission, the ECG showed S1Q3 without
ST-T change. Acute respiratory distress syndrome (ARDS) was also
diagnosed via chest radiography and clinical presentation. Low dose
norepinephrine and dopamine continuous infusion were administered
for SIRS reaction but were then gradually tapered off in 2 days.
Neurological examinations showed mild weakness with muscle power
of 4+ in the patient’s left limbs during the first day after the surgery but
these symptoms dissipated by the second day after the operation. By
the second day the patient was under a stable enough condition to be
extubated and transferred out of the ICU into the general ward by the
fifth day. He was discharged by the twelfth day after the surgery without
any residual complication.

Discussion

Carbon dioxide is the most widely used insufflation gas. Most
serious cases of CO₂ embolism reported in the literature occurs during
the beginning of the procedure, usually due to the misplacement of the
Veress needle either directly into a vein or parenchymal organ. Lesser
amounts of CO₂ may also enter circulation through openings in injured
vessels, as seen in our case, which explains the late onset of the carbon
dioxide embolism [4].
Rapid entrainment of a large volume of gas can lead to the formation of large emboli, which may lodge in a large central vessel and potentially lead to cardiovascular collapse [5]. In our case, the surgeon was facing the dilemma of whether to transform into open surgery. Considering previous hepatic tumor excision related intra-abdominal adhesion, exploratory laparotomy to approach the bleeder would be also difficult [6]. Instead, we tried an adventurous method: we controlled the IVC bleeding via the carbon dioxide (CO₂) insufflation pressure, at the same time, the surgeon sow the torn IVC by the use of laparoscope. A necessary condition for the development of gas embolism is the presence of an open vein with a lower pressure in the vein than the surrounding pressure [7]. Nevertheless, it is because the little higher pressure of the CO₂ gas insufflation, the bleeding rate was not so fast to be life-threatening. But the pressure of CO₂ gas insufflation can’t be too high to make fetal amount of CO₂ embolism.

Fortunately, our patient did not develop cardiovascular collapse even encounter this massive CO₂ embolism. It may because the slow entrainment of the CO₂ and the high solubility of CO₂ (20:1 to oxygen and 25:1 to nitrogen) [8]. Mayer, et al. described a mortality of 60% at a continuous intravenous CO₂ infusion rate of 1.2 mL/kg/min, which is equivalent to a rate of 72 mL/min for a 60 kg person. That volume is only 5% of the volume of carbon dioxide that may be infused into a vein, intentionally cannulated by a Veres needle, in one minute at a low-flow rate [9]. Our effort to keep the balance of IVC venous pressure and surrounding CO₂ insufflation pressure during the episode may prevent a massive fetal CO₂ embolism and a fetal bleeding rate.

About 20 minutes after IVC tear, the TEE image also revealed scattered air embolisms in the left heart. The foramen ovale didn’t open on the TEE image. We suspect that the left heart gas emboli may have been caused from pulmonary capillary diffusion when large amounts of air entered the vena cava. Post-surgery, the patient developed neurologic signs of left limbs weakness but was able to rapidly recover in 2 days. The rapid improvement maybe attributed to the high solubility of CO₂.

Treatment of CO₂ embolisms consists of the immediate cessation of insufflation and the release of the pneumoperitoneum, steep head-down and left lateral decubitus position. And the discontinuation of nitrous oxide and to ventilate with 100% oxygen. In addition, adequate hydration and the placement of a CVC to aspirate CO₂ is required. Our patient was already in the head-down and left lateral decubitus position as it was required for surgery. However, the insufflation of CO₂ didn’t cease in our patient, because the surgeon needed to control the bleeding through applying pneumoperitoneum pressure.

In conclusion, our patient’s prognosis was good despite having gone through a massive CO₂ embolism event. The patient’s relatively stable vital signs and possibly intra-abdominal adhesion were the reasons why we did not transfer the laparoscope to exploratory laparotomy nor immediately cease CO₂ insufflation. In addition, the rapid improvement from the neurological symptoms maybe attribute to the high solubility of CO₂. Hence, we hypothesize that the decision of whether to cease the pneumoperitoneum immediately when CO₂ embolisms occur, may depend on the patient’s hemodynamics. In admission, it is important to find the balance between venous and CO₂ insufflation pressure to avoid massive bleeding or fetal amount of CO₂ embolisms. And that the neurological tolerance of CO₂ emboli may be much higher than that of other gases.

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