

## **Paradoxical carbon dioxide embolism during laparoscopic right nephrectomy**

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### **INTRODUCTION**

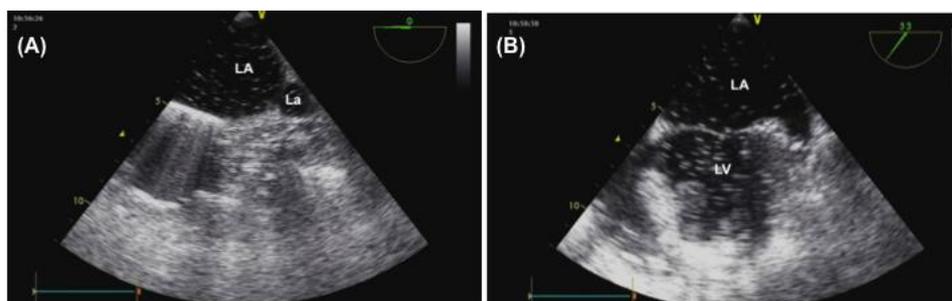
Gas embolism is defined by the presence of air bubbles in the arterial or venous circulation. Several medical and surgical procedures can lead to this pathology (1). Among those procedures, carbon dioxide (CO<sub>2</sub>) insufflation during laparoscopic surgery is responsible for gas embolism in 1 to 2 over 1000 procedures (2). CO<sub>2</sub> entry into the circulation is often due to accidental vascular or organ puncture, or to undetected vascular breach. Intraoperative signs guiding the diagnosis depend mainly on volume and speed of gas injection. Before induction of anesthesia, appropriate monitoring should be set up, including monitoring of oxygen saturation (SpO<sub>2</sub>) through pulse oximetry, non-invasive blood pressure monitoring (NIBP), electrocardiogram (ECG), and end-tidal carbon dioxide concentration (EtCO<sub>2</sub>). Indeed, desaturation, arterial hypotension, associated or not with bradycardia and brutal drop of end-tidal carbon dioxide concentration should immediately warn about eventual gas embolism. A patent foramen ovale (PFO) or a vascular shunt inside the lungs may be responsible for paradoxical carbon dioxide embolism with a fatal outcome, or severe subsequent permanent disabilities for the patient (3). We report here a case of a female patient who developed massive paradoxical carbon dioxide embolism during a laparoscopic right nephrectomy. The diagnosis was established rapidly using trans-esophageal echocardiography (TEE). Early resuscitation was successful, and the patient received hyperbaric oxygen therapy. She survived, although with mild neurological sequels.

### **CASE REPORT**

An 87-years-old woman (weight 61 Kg; height 160 cm) was admitted for the treatment of a right kidney tumor, discovered during the exploration of a hematuria. A laparoscopic right nephrectomy was scheduled. She had a history of chronic high blood pressure, atrial fibrillation, hypothyroidism, and hiatus hernia. She had already undergone other surgical procedures, including goiter resection and glaucoma. Her ongoing medications included eprosartan, sotalol, levothyroxine, and omeprazole. No allergies were mentioned by the patient.

On the day of surgery, monitoring encompassed SpO<sub>2</sub>, NIBP, ECG, and EtCO<sub>2</sub>. Induction of general anesthesia occurred intravenously using 10 µg of intravenous sufentanil 10 µg, 35 mg of ketamine, 80 mg ph lidocaine, 150 mg of propofol, and 50 mg of atracurium. Any unwanted events were recorded. The patient was positioned in the left-lateral position. After penetration of the Veress needle, CO<sub>2</sub> insufflation, at 12mmHg, provoked a fast drop in blood pressure from 150/90 mmHg to 60/40 mmHg. Tachycardia at 114/min, brutal drop in ETCO<sub>2</sub> from 38 mmHg to 20 mmHg, and decrease in SpO<sub>2</sub> 97% to 89% also occurred concomitantly. The surgeon was

immediately informed about the situation. CO<sub>2</sub> insufflation and surgical procedure were instantly interrupted. The patient was moved to a supine Trendelenburg position, and was given 100 % oxygen. A central venous catheter and a left radial arterial line were installed. Aspiration from the central venous catheter only brought blood without gas bubbles. Thereafter, TEE was performed and showed gas bubbles in the left atrium and ventricle (**Figure1A-B**).



**Figure 1: Intraoperative TEE shortly after the occurrence of gas embolism. TEE shows gas bubbles (A) in the left atrium (LA), left auricle (La) and (B) in the left atrium (LA) and left ventricle (LV).**

It confirmed the diagnosis of gas embolism, but failed to demonstrate FOP. Hypotension was treated first using boluses of ephedrine, and a noradrenaline continuous infusion thereafter. In the meantime, aggressive fluid loading was completed using 500mL of crystalloids (Plasmalyte®) and 1500 mL of colloids (Gelofusine®). A dobutamine continuous infusion was also initiated to improve myocardial contractility, and allow fractionation of gas bubbles into the heart's chambers. All patient parameters returned to normal and she was allowed to wake up. Her trachea was extubated in the recovery room, and the patient was then moved to the post anesthesia care unit. When she recovered, she was disoriented and presented a right upper limb motor deficit. Cerebral tomodensitometry (TDM) did not show any gas bubbles. The patient was transferred to intensive care unit of another hospital, where she received 5 sessions of hyperbaric oxygen therapy. Complementary exams made at that time revealed a regular sinus rhythm on the ECG, atheroma without stenosis on the supra aortic arterial trunk on a doppler ultrasound, no PFO on TEE, and recent multiple ischemic lesions spread into the two hemispheres on a cerebral magnetic resonance imaging. Neurological examination 2 weeks later found a right lower limb motor impairment, graded at 4/5, with Babinski sign. The diagnosis of a stroke following a gas embolism and low cardiac output was retained. After a month of rehabilitation, the patient was discharged home.

## Discussion

CO<sub>2</sub> insufflation during laparoscopy is essential to provide enough room to the surgeon for performing the surgical procedure. CO<sub>2</sub> is the most commonly used gas, because of its high solubility in the blood. In case of embolism with CO<sub>2</sub>, the

consequences are therefore less severe than with helium or nitrogen (4). Pure oxygen or air is not suitable for pneumoperitoneum, because of the high risk of combustion in the presence of monopolar cautery or laser. CO<sub>2</sub> insufflation can cause gas emboli if the Veress needle goes through the liver, the spleen, or within a vessel. A non-detected arterial breach can also lead to gas emboli, particularly when the insufflation pressure is higher than 15 mmHg. In our patient, insofar as symptoms developed rapidly, gas embolism was probably due to inadvertent Veress needle insertion into the liver. However, laparoscopic control to objectify such inadvertent liver lesion was not performed. TEE confirmed gas embolism, nicely showing numerous gas bubbles in the left heart. This explains why aspiration on the central venous catheter did not drain gas bubbles. Paradoxical CO<sub>2</sub> embolism is a very rare event. It only occurs in patients with right to left intracardiac such as PFO. PFO is present in 10 to 30 % of the general population (5,6). In our patient, no PFO was observed, either by the intraoperative TEE, or later. Lungs are physiologic filters for gas bubbles. They prevent gas passing from the right side of the heart to the left. However, Yahagi and colleagues (7) have shown that a big volume of gas, as well as the use of some anesthetic agents, such as the volatile anesthetic agents, could interfere with the ability of the pulmonary filter to play its role. This can lead to the right to left crossing of gas bubbles within the lungs, and may explain why massive gas embolism did not provoke ventricular defusing and cardiac arrest. Paradoxical gas embolism, which always originates from the heart, often leads to the spreading of gas towards the peripheral arterial circulation. This event can damage any organ, particularly the brain, and heart, causing stroke or myocardial infarct. Seven hours after the incident, our patient presented a confused state and upper limb motor deficiency. This prompted us to perform cerebral TDM that revealed normal. Even if TDM had been performed earlier, it would not have necessarily been able to evidence gas bubbles, because they are only observed in 8 cases out of 13 (8). Therefore, normal TDM cannot eliminate the diagnosis. Despite receiving 5 sessions of hyperbaric oxygen therapy, the patient remained with a motor deficiency in her lower limb. Blanc and colleagues (9) have shown that hyperbaric oxygen therapy treatment has to be provided as soon as possible. Indeed, to improve outcome, this treatment should ideally be given 3 to 6 hours after acute venous or arterial gas emboli. Our patient should have probably been transferred earlier for hyperbaric oxygen therapy, without waiting for clinical signs of neurological impairment. Indeed, massive paradoxical gas embolism was already evidenced.

## **Conclusion**

Gas embolism is one of the well-known intraoperative complications that can occur during laparoscopic surgery. A fast diagnosis based on three points such as hypotension, desaturation with abrupt drop in ET<sub>CO</sub><sub>2</sub>, and an immediate resuscitation process with the interruption of the insufflation significantly increase the chances of survival. Paradoxical gas embolism does not exclusively requires a PFO, but can also occur Within the lungs. As soon as paradoxical gas embolism is identified, hyperbaric oxygen therapy treatment should be given in order to improve the neurological outcome of those patients.

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