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CASE REPORT

# Suspected cerebral arterial gas embolism during a laparoscopic Nissen fundoplication

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## Abstract

We present the first case report known to us of a suspected cerebral arterial gas embolism (CAGE) leading to transient left-sided hemiparesis after a laparoscopic Nissen fundoplication. During the operation there was no evidence of hemodynamic compromise and the end-tidal carbon dioxide level and oxygen saturation had been within normal limits. Radiological studies and transesophageal echocardiography showed no abnormalities. We conclude that CAGE can occur during uncomplicated laparoscopic surgery even in the absence of demonstrable intracardiac shunts.



**Key words:** Paradoxical gas embolism; Arterial embolism; Laparoscopic Nissen fundoplication; Neurologic deficit; Laparoscopic surgery

**Core tip:** We present the first case report known to us of a suspected cerebral arterial gas embolism (CAGE) leading to transient left-sided hemiparesis after a laparoscopic Nissen fundoplication. During the operation there was no evidence of hemodynamic compromise and the end-tidal carbon dioxide level and oxygen saturation had been within normal limits. Radiological studies and transesophageal echocardiography showed no abnormalities. We conclude that CAGE can occur during uncomplicated laparoscopic surgery even in the absence of demonstrable intracardiac shunts.

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## INTRODUCTION

Carbon dioxide ($CO_2$) embolism is a well-recognized complication during laparoscopic procedures utilizing $CO_2$ insufflation for the establishment of a pneumoperitoneum[1]. The clinical presentation of $CO_2$ embolism ranges from a complete lack of symptoms to neurologic injury, cardiovascular collapse or even death depending on the rate and volume of gas entrapment[2]. $CO_2$ embolism can be fatal, yet the incidence during laparoscopic surgeries is varied. The true incidence is difficult to determine secondary to subclinical cases and the sensitivity of the detection of gas embolism by available monitors during procedures. Hong *et al*[3] report that the incidence of subclinical embolisms in laparoscopic radical prostatectomies is 17%. Usually, venous embolism manifests in the first few minutes after the start of the gas insufflation during initial establishment of a pneumoperitoneum and it is due to inadvertent venous cannulation with a Veress needle or gas absorption through open venous channels. However, in many cases no noticeable hemodynamic changes are noted since the pulmonary circuit may filter

or reabsorb the small bubbles of $CO_2$ without causing any embolic obstruction[4]. Conversely, in the presence of intracardiac shunts, such as a patent foramen ovale or septal defect, the $CO_2$ embolus can reach the left side of the circulation resulting in varied degrees of neurologic or vascular deficits depending on the location where the embolus lodges[5]. Such paradoxical $CO_2$ embolisms are extremely rare events, especially in the absence of intracardiac shunts. Nevertheless, they could have disastrous consequences.

Herein, we present the first case known to us of a suspected cerebral arterial gas embolism (CAGE) in the absence of intracardiac shunts that led to a transient left-sided hemiparesis after an uncomplicated laparoscopic Nissen fundoplication.

## CASE REPORT

A 22-year-old woman with gastroesophageal reflux disease unresponsive to maximal medical management and who failed lifestyle modifications was scheduled for a laparoscopic Nissen fundoplication. The patient also had a past medical history of Hashimoto's thyroiditis and Crohn's disease treated medically. Her past surgical history included two laparoscopic procedures: a bilateral ovarian cystectomy and a cholecystectomy, which had been uneventful. Four months prior to the laparoscopic Nissen fundoplication she had given birth to a healthy boy *via* a normal vaginal delivery.

The patient was brought to the operating room and standard American Society of Anesthesiologists noninvasive monitors (electrocardiogram, noninvasive blood pressure monitoring, oxygen saturation, capnograph, and temperature) were placed. After preoxygenation, the patient underwent a rapid sequence induction with propofol (200 mg) and succinycholine (100 mg) and the trachea was intubated. General anesthesia was maintained with air, oxygen, and sevoflurane. The patient was positioned with a beanbag on the operative table with both lower extremities placed in stirrups. Mechanical ventilation was adjusted to maintain the end-tidal $CO_2$ between 30-40 mmHg. The patient's abdomen was then prepped and draped. The Veress needle was inserted, a water drop test was performed, and the abdomen was insufflated at the rate of 3 L/min. The intra-abdominal pressure was built up to 18 mmHg according to recommendations set forth by Bhoyrul *et al*[6]. The introducer of the trocar was then removed and the laparoscope was reinserted into the abdominal cavity to inspect the entry area, ensuring that no intra-abdominal injuries were made upon entering the abdominal cavity. The patient was then placed in a sitting position to improve surgical exposure of the hiatus for the operation and the $CO_2$ insufflation pressure was decreased to 14 mmHg. The surgical and anesthetic course was uneventful. There were no episodes of sudden hypotension, bradycardia, arrhythmia, oxygen desaturation, or decrease in end-tidal $CO_2$ during the operation. The trachea was extubated without difficulty with the patient awake. In the recovery room, the nursing staff noticed the patient had a left eye ptosis and a left hemiparesis. A thorough neurologic assessment by a senior neurosurgeon in the recovery room demonstrated that the patient could not move her left upper arm and that she had 1/5 strength in her left lower extremity. Preoperatively, the motor strength in all extremities was 5/5. Deep tendon reflexes on the right were 2+ and were absent on the left side of the body. In addition to a diagnosis of possible $CO_2$ arterial embolism, our evaluation of this patient involved the differential diagnoses of a coagulopathy, intracardiac thrombus with subsequent embolus, cerebral vascular disease, and a hemorrhagic cerebral vascular event. A computed tomography (CT) scan was obtained within 1 h and showed no evidence of a stroke. The stroke team was informed and the patient was transferred by ambulance from the Veterans Administration Hospital to the University hospital in the immediate vicinity (one block away) for definitive care. Upon arrival to the emergency room (ER) of the University hospital, the patient was examined by the same surgical team who performed the laparoscopic procedure, by members of the stroke team, and by the ER attending physician. At this time, the patient had recovered the use of her left upper extremity to gain 3/5 strength and she had also improved her left lower extremity strength to 4/5. Motor strength on the right was unchanged at 5/5 and deep tendon reflexes on the right were 2+. Reflexes on the left side, which were initially absent, were found to be 1+.

A CT angiogram of the intracranial circulation failed to show any significant narrowing or obstruction to cerebral flow. Similarly, a hypercoagulable evaluation failed to show any hypercoagulable disorder. Finally, transesophageal echocardiography did not show any evidence of gas in any of the cardiac chambers or any demonstrable intracardiac shunt on color-flow Doppler imaging. The patient was admitted to the Neurological intensive care unit for monitoring and over a period 24 h she had complete resolution of her neurologic symptoms. She underwent a magnetic resonance imaging (MRI) on postoperative day two which was normal. Supportive treatment with postoperative pain control and fluid management were continued for the entire duration of the patient's hospital stay.

The patient was discharged on the third postoperative day with no residual weakness or deficits, tolerating a clear liquid diet without heartburn, regurgitation, or dysphagia. She was examined postoperatively at 2 wk and 2-mo follow-up in the Surgery and Neurology outpatient clinics. She continued to show no neurologic deficits. The patient has given written consent and has agreed to publication of this case report.

## DISCUSSION

CAGE is an exceptionally rare event during laparoscopic surgery. The clinical presentation of $CO_2$ embolism varies with respect to the rate of entry of the gas and the size of the embolus, which can increase with the use of

nitrous oxide[7]. $CO_2$ embolus can result in "gas lock" with obstruction to right ventricular outflow, ventilation and perfusion mismatch, cardiac arrhythmias, pulmonary hypertension, and cardiovascular collapse. The diagnosis of a $CO_2$ embolism can be revealed by the auscultation of a millwheel murmur with a precordial or esophageal stethoscope, decrease in end-tidal $CO_2$ noted with capnography, increased end-tidal nitrogen, decrease in oxygen saturation by pulse oximetry, electrocardiographic changes, Doppler ultrasonography, transesophageal Doppler, and transesophageal echocardiography[8]. In addition to cardiopulmonary and neurological symptoms seen with $CO_2$ embolus, patients with CAGE may also experience seizures, headaches, dizziness, and visual field defects.

Radiologic evaluation is not always conclusive in the diagnosis of CAGE. CT scans can distinguish CAGEs from cerebral infarcts or hemorrhages; however, the distinction may be elusive[9]. MRI may show injured tissue with a fluid collection; yet, again, this is not reliable especially if the patient has mild symptoms[10].

The treatment of CAGE is similar to the treatment of a $CO_2$ embolus. However, the treatment of a patient with CAGE may include the transfer to a hyperbaric oxygen chamber if the patient is stable; placing the patient in the supine position; anticonvulsant medications; and lidocaine[11]. Furthermore, distinct from the treatment of venous gas embolism in which the patients are placed in the Trendelenburg and left lateral decubitus position, patients with CAGE should be placed in the supine position to avoid gas bubbles flowing toward the head and to prevent cerebral edema[12]. However, under general anesthesia with stable cardiopulmonary signs, small cerebral arterial emboli may go undetected until neurological signs are apparent after the emergence from anesthesia, as we encountered in this patient.

The exact cause of CAGE in this case remains unknown. The rapid elimination of $CO_2$ due to the high solubility of $CO_2$ in blood as well as a reduction in $CO_2$ insufflation pressures contributed to the transient nature of this patient's symptoms and the elusiveness of medical studies. The blood/gas solubility of $CO_2$, nitrous oxide, dissolved oxygen, and nitrogen are 0.60, 0.45, 0.024, and 0.013 mL/mL solvent with 100% gas at 17 degrees Celsius respectively. Furthermore, since the blood solubility of nitrous oxide and $CO_2$ are similar, discontinuing nitrous oxide will not reduce the size of a $CO_2$ embolus as it would an air embolus[13].

Intraoperatively, a more urgent intraoperative transesophageal echocardiogram would have been indicated in the setting of hemodynamic compromise[14]. Such a hemodynamic collapse did not occur in this patient perhaps because, as shown by Huang *et al*[15], the gas was released in time without formation of a fatal pulmonary gas lock. Although during the operation we had no evidence of bleeding or vascular injury due to the placement of the Veress needle, we postulate that the initial intra-abdominal pressure of 18 mmHg might have been high enough for the $CO_2$ to enter and bypass the pulmonary circuit and that, at the same time, was kept brief enough to avoid a pulmonary gas lock. In fact, Eiriksson *et al*[16] demonstrated that high intra-abdominal pressures (16 mmHg) during experimental laparoscopic liver resection in swine reduced bleeding but increased the risk of gas embolism.

Paradoxical embolisms have been demonstrated in patients without intracardiac defects. Bedell *et al*[17] presented a case of a patient in the sitting position undergoing occipital artery to posterior inferior cerebellar artery bypass who developed paradoxical gas embolism in the absence of any intracardiac defect. Bedell *et al*[17] demonstrated transesophageal echocardiographic evidence for the transpulmonary passage of gas from the right to the left side of the circulation. The event confirmed the validity of a 50-year-old theory that attributed a precise pathogenic role to arteriovenous connections, called "*sperrarteries*", within the pulmonary vasculature. According to this theory, the *sperrarteries* bypassed the pulmonary parenchyma and were thought to serve as rapid conduits for absorbed venous air to travel to the arterial side of the circulation[18].

In conclusion, we report, to our knowledge, the first case of suspected CAGE occurring during a laparoscopic Nissen fundoplication causing transient neurologic symptoms. We conclude that CAGE can occur during uncomplicated laparoscopic surgery even in the absence of demonstrable intracardiac shunts and that such an event might be prevented by keeping the $CO_2$ insufflation pressure set at 15 mmHg with a slow flow rate during the creation of the pneumoperitoneum[19].

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