Hyperbaric treatment of arterial CO$_2$ embolism occurring after laparoscopic surgery: A case report.

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Reust RS, Diener BC, Stroup JS, Haraway GD. Hyperbaric treatment of arterial CO$_2$ embolism occurring after laparoscopic surgery: A case report. Undersea Hyperb Med 2006; 33(5):317-320. Arterial carbon dioxide (CO$_2$) embolism is a serious and sometimes fatal iatrogenic medical condition encountered in surgery. A thirty-five year old Caucasian female developed a CO$_2$ embolism during a laparoscopic appendectomy. After initial resuscitation and completion of the surgery, she was treated with hyperbaric oxygen (HBO$_2$) to help reduce the size of large CO$_2$ bubbles that had migrated into her left ventricle during surgery. The HBO$_2$ protocol was 2.8 atmospheres for 90 minutes, which resolved the problem with no adverse effects.

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INTRODUCTION

Arterial gas embolism can be a complication of invasive medical procedures and result in high morbidity and mortality. An arterial gas embolism can occur with the entry of gas directly into the systemic arteries or through the pulmonary veins(1,2). Two of the most common causes of gas embolism are pulmonary barotrauma induced from decompression during diving and iatrogenic embolism from surgical procedures(2). Several types of gas are utilized for surgical procedures including carbon dioxide (CO$_2$), helium, argon, nitrogen, and air. Because of its high solubility, CO$_2$ is rapidly absorbed from the peritoneal cavity and is less likely to cause embolism(3). CO$_2$ embolism is a rare complication of laparoscopic surgery occurring in approximately 0.0014-0.6% of cases, but its occurrence is associated with a mortality rate of approximately 28%(3).

Arterial gas embolism can present with collapse, loss of consciousness, apnea, and cardiac arrest(1,2,4). The gas that enters the systemic circulation is dissolved in serum and
tissue, and normally does not cause occlusive events. However, if the bubbles of CO₂ are large enough, they can cause occlusion of blood flow and can also cause mechanical irritation to the endothelium. Several studies have shown that HBO₂ is beneficial in reducing these obstructing bubbles.

We report a case of a patient that developed an iatrogenic carbon dioxide embolism during laparoscopic appendectomy who was successfully resuscitated and urgently treated with hyperbaric oxygen (HBO₂) therapy.

CASE

A thirty-five year old Caucasian female presented to a Tulsa, OK emergency department with sudden-onset of abdominal pain, fever, and rigors of 24 hours duration. The surgical consultant diagnosed an acute abdomen secondary to appendicitis. The patient had a history of endometriosis, cervical cancer, and gastritis. She was an employed mother of two and had a 20-pack year smoking history. The patient had a past surgical history consisting of a total abdominal hysterectomy with bilateral salpingo-oopherectomy and laparoscopic reduction of adhesions. Considering the patient’s clinical presentation and her elevated leukocyte count, she was urgently taken to the operating room for laparoscopic appendectomy.

Under general anesthesia with endotracheal intubation, a laparoscopic insufflation needle was inserted into the infraumbilical fold using a normal opening abdominal pressure of 6 mmHg using CO₂ to create the pneumoperitoneum. After insufflation of approximately 1 L CO₂, the anesthesiologists noted a sharp decrease in end tidal CO₂ via capnography consistent with a sudden increase in venous CO₂. Concurrently, the patient’s systolic blood pressure dropped from 120 to 70 mmHg. The physicians immediately suspected that carbon dioxide gas had entered the vascular system from the abdomen, abdominal insufflation was stopped, and the anesthesiologist noted a “mill-wheel” murmur on auscultation of the heart. The patient was placed in Durant’s position– left lateral decubitus with head down.

The patient’s cardiac rhythm changed from sinus to ventricular tachycardia to ventricular fibrillation. Advanced Cardiac Life Support (ACLS) was initiated, including defibrillation at 200 and 300 joules before obtaining a pulse and normal sinus rhythm. The patient remained in ventricular fibrillation for less than two minutes. It was noted preoperatively that the patient had mild hypokalemia at 3.2 mmol/L (normal 3.5-5 mmol/L), and although 40 mEq of KCl was administered in the intravenous fluids through a central line, hypokalemia may have contributed to the ventricular tachycardia. It is also possible that the patient had embolization of CO₂ into a coronary artery, which interfered with myocardial blood flow.

A right internal jugular catheter was inserted and bubbles aspirated from the right heart. Oxygen was increased from 50% to 100%. A cardiology consultant obtained a transthoracic echocardiography in the operating room and demonstrated bubbles on both sides of the heart originating from the pulmonary veins (Figure 1). The cardiologist did not identify an intra-cardiac shunt and felt that an extra-cardiac arteriovenous shunt was responsible for bubbles to appear in the left heart so quickly and in such a large quantity. Neurological assessment could not be performed under anesthesia and muscle relaxation. The HBO₂ on call team was notified and mobilized to prepare for treatment.

Once the patient was stabilized, the surgeon decided to complete the emergent appendectomy via a midline incision. A gangrenous appendix was removed, and the abdomen inspected for trauma. No visceral
or vascular injury by the insufflation needle was found. Following surgery the patient was extubated and exhibited no neurological problem other than residual sedation from the anesthetic. Vital signs were stable with a blood pressure of 150/70 mmHg and an O₂ saturation of 99% on 6 L oxygen. A repeat echocardiogram was not performed. Concerns of CNS gas emboli prompted a brain CT, which was negative.

The sudden ventricular tachycardia in a young, otherwise healthy patient raised the strong suspicion that the large bubbles in the heart may have occluded a coronary artery. The decision was made to transfer the patient to the HBO₂ chamber and treatment with HBO₂ at 2.8 atmospheres was initiated 30 minutes after the initial arrest in surgery for 90 minutes to prevent further cardiac events.

After HBO₂ treatment, a repeat echocardiogram was performed that showed clearance of the CO₂ that had entered the patient’s circulation to obstruct arterial blood flow (Figure 2). A neurologist reviewed the case and determined that the patient had no focal neurological deficits and recommended no further studies. A myocardial perfusion imaging scan was performed which was normal. The patient recovered fully and suffered no permanent adverse effects.

**DISCUSSION**

The gas bubble formation in our patient was not caused by rapid decompression as seen with divers, but rather by the introduction of a gas into the patient’s blood stream. Other medical procedures have been documented to cause gas embolism including: angiography, carotid endarterectomies, Cesarean section, craniotomies, laparoscopic surgery, and open heart procedures(2). CO₂ is not considered an inert gas, and usually is readily dissolvable into serum(3). Studies have shown that CO₂ is 6.5 times less than air to cause physiological disturbances when utilized for insufflation, and is the preferred gas for procedures such as laparoscopic insufflations and CO₂ angiograms(3).

Very small CO₂ bubbles in the circulation usually do not cause serious problems, whereas small air or nitrous oxide bubbles may cause death because of their poor solubility(3). CO₂ is much more tolerable than air because it is more than 25 times more soluble in blood than nitrogen(10). But, bubbles of CO₂ large enough to obstruct capillary or arterial blood flow...
represents a serious risk of tissue infarction, and bubble size can be an important factor related to adverse outcomes. When CO₂ embolism occurs during surgical procedures, approximately 60% of symptomatic cases occur during the initial insufflation as experienced in our patient(3).

The lungs are usually an efficient bubble filter, and venous gas emboli do not require therapy unless hemodynamic compromise or arterial gas embolism are present(3). In these instances, treatment with HBO₂ is utilized, but is less well studied than with decompression illness. In this case, HBO₂ was utilized to decrease the bubble volume and size by raising the ambient pressure and by creating systemic hyperoxia to facilitate gas egress from the bubble(1,2). The benefit of HBO₂ is greatest when initiated within several hours of air embolism onset(6). The standard HBO₂ treatment of arterial air embolism is US Navy Treatment Table 6, which recommends recompression to 60 FSW (2.8 ATA) at a descent rate of 25 feet per minute (3,7). This table keeps the patient at depth for a total of 285 minutes(2,7).

It was felt that our patient required urgent treatment to avoid more cardiovascular complications, and access to a multiplace chamber capable of following Treatment Table 6 was not available. The available hyperbaric unit had the capability to dive to 2.8 ATA, but was not able to allow for air breaks. Because of this, we were limited to 90 minutes of treatment to avoid CNS oxygen toxicity. This HBO₂ regimen eliminated the CO₂ that entered our patient’s bloodstream and caused obstruction of arterial flow. As this case was being written up, another report of CO₂ cerebral gas embolism was published(8). Those authors utilized 3 ATA HBO₂ for 90 minutes with no adverse effects and return of baseline neurological status in their patient(8).

CONCLUSION

In a patient with CO₂ arterial embolism, monoplace HBO₂ therapy at 2.8 ATA for 90 minutes proved to be a viable treatment. Whether this regimen is optimal for CO₂ embolism cannot be determined from this case. HBO₂ was utilized because of a possible coronary occlusive event related to a large volume of intravascular CO₂ gas, which had caused of the onset of sudden ventricular tachycardia with potentially catastrophic consequences.

REFERENCES