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Intraoperative management of a carbon dioxide embolus in the setting of laparoscopic cholecystectomy for a patient with primary biliary cirrhosis: A case report

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ABSTRACT

INTRODUCTION: Carbon dioxide (CO_2) embolism is a rare complication of laparoscopic cholecystectomy of which both surgeons and anesthesiologists must be aware. This paper presents a case of a CO_2 embolus that occurred in a patient with primary biliary cirrhosis (PBC) and discusses the possible correlation between these two events.

PRESENTATION OF CASE: Our patient with PBC presented with symptomatic biliary dyskinesia and was determined to be a good candidate for laparoscopic cholecystectomy. During this routine surgery a CO_2 embolus entered through the altered hepatic parenchyma and progressed to the heart leading to acute hemodynamic collapse. Rapid detection and management aided in the subsequent dissolution of the embolus and recovery of the patient.

DISCUSSION: In patients with PBC, pathological changes that have taken place in the liver may increase the risk of CO_2 embolism. Hepatic alterations that have been previously described include increased angiogenesis and vasodilation. Prior to the operation, the most appropriate method of monitoring should be determined for patients with known liver disease.

CONCLUSION: Both the surgical and anesthesia team must keep in mind the potential for CO_2 embolism during laparoscopic surgery. It is imperative that the medical staff be aware of the risks, signs, and subsequent management so this rare, but potentially fatal event can be managed appropriately.

1. Introduction

Although rare, CO_2 embolism is a dangerous complication of laparoscopy, with an associated mortality rate of 28%.[1] The mechanism of embolism is thought to be via intravascular injection of gas through direct needle or trocar placement, or a consequence of parenchymal injury in the presence of insufflation.[2] Emboli occur when a CO_2 bolus enters the local venous circulation, subsequently traveling through the inferior vena cava into the right atrium where a "gas lock" may occur, dropping the venous return and cardiac output, leading to hemodynamic collapse.[1] The absolute diagnosis depends on the detection of CO_2 emboli in the right side of the heart; however, due to its rapid elimination, it may be diagnosed based on physiological parameters.[2] The associated changes include a rapid decrease in the partial pressure of end-tidal CO_2 ($PETCO_2$), "tachycardia, cardiac arrhythmias, hypotension, increased central venous pressure",[2] presence of "millwheel" murmur, cyanosis, and electrocardiogram (EKG) changes.[2] Once detected, the management involves the release of pneumoperitoneum, repositioning of the patient into left lateral decubitus, steep head down position, and the discontinuation of anesthetic gases, allowing for ventilation of 100% oxygen (O_2). Hyperventilation is made necessary by the increase in physiologic dead space.[2] Rapid intervention with proper change in position in addition to hyperventilation should increase dissolution and cardiac output. Aggressive volume expansion may further reduce gas entry by elevating central venous pressure. A multi-orifice central venous line placement should be considered to allow aspiration of gas from the right atrium or ventricle, provide a quicker diagnosis, and significantly improve hemodynamic status by relieving "gas lock" in the right atrium or ventricle.[3] In the event of hemodynamic collapse, vasopressors should be used for hypotension, in addition to cardiopulmonary resuscitation as necessary.[2] We report the case of a 69-year-old female with PBC who developed a CO_2 embolus during laparoscopic cholecystectomy.

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2. Description

The patient, a 69-year-old Caucasian female with PBC, presented with postprandial epigastric and right upper quadrant pain. She also reported bloating and frequent bouts of nausea without emesis that had progressively worsened to daily occurrences over the past year. Her past medical history was significant for PBC controlled with Ursodiol. A hepatobiliary iminodiacetic acid (HIDA) scan revealed an ejection fraction of 11% with symptom reproduction upon cholecystokinin challenge. Due to biliary dyskinesia in the presence of worsening symptomatology, it was decided that she would likely benefit from a laparoscopic cholecystectomy.

Preoperative laboratory evaluation included a complete metabolic panel, complete blood count, prothrombin time/international normalized ratio, and an EKG which were all within normal limits with the exception of mildly elevated liver enzymes and alkaline phosphatase levels consistent with the chronic history of PBC. The patient reported a past surgical history significant for a laparoscopic hysterectomy without surgical or anesthetic complications. The anesthesia evaluation documented her as an American Society of Anesthesiologists (ASA) class II, with a Mallampati class II airway. Thus, she was determined to be a candidate for general anesthesia with general endotracheal intubation.

The patient was placed in a supine position on the operating table with standard monitoring in place. Anesthesia was induced with Versed, Fentanyl, Propofol, Robinul, Rocuronium, and maintained with Sevoflurane. After full muscle relaxation was attained, the patient was intubated with a size 7 endotracheal tube with no complications. The procedure began with an incision placed in the epigastrium, and the abdomen was entered under direct vision with a 5-mm Optiview trocar. Pneumoperitoneum was achieved with carbon dioxide gas insufflation maintained at a pressure of 15 mmHg. A second 5 mm trocar was then placed in the umbilical region. At this time, the monitor showed a drop in $PETCO_2$ from 40 mmHg to 7 mmHg and then to 0 mmHg. The anesthesia provider voiced concern about the possibility of CO_2 emboli and initiated immediate management. The pneumoperitoneum was evacuated and the patient was placed in both left lateral decubitus and reverse Trendelenburg. The lungs were auscultated and the patient was hyperventilated with 100% O_2. The subsequent blood pressure was recorded as 40/13, which prompted a 1 mg dose of epinephrine while an arterial line was placed for more precise blood pressure and arterial blood gas monitoring. The patient became hypoxemic with the lowest peripheral capillary oxygen saturation (SpO_2) recorded as 70. Following the IV dose of epinephrine, her blood pressure increased to 198/97. Within nine minutes from the initial $PETCO_2$ decrease, the SpO_2 returned to 100 and $PETCO_2$ increased to 32. At this time, the arterial pH was 7.244 and the partial pressure of carbon dioxide (pCO_2) was 50.2, suggesting respiratory acidosis and decreased perfusion consistent with a diagnosis of CO_2 emboli.

Following the placement of the trocar, the surgeon observed blood in both trocar sites, strongly suspecting an abdominal hemorrhage. The procedure was then immediately converted to an open laparotomy. Dark blood was visualized in the abdomen, along with a small laceration in the anterior left lobe of the liver that was not actively bleeding. Upon gross inspection, the liver was markedly enlarged, dark, and scarred, consistent with cirrhosis, and the gallbladder was observed to be massively distended. The gallbladder was then removed in its entirety, after which a small wedge of liver was removed using electrocautery and sent for biopsy. The patient remained stable throughout the remainder of the operation with no further complications. Following completion of the procedure, the patient was extubated and transported to the post anesthesia care unit in stable condition. In recovery, her pH was determined to be 7.337 and pCO_2 was 41.6, indicating improvement of the previous respiratory acidosis with dissolution of the suspected CO_2 emboli. Pathological analysis of the gallbladder specimen showed findings consistent with chronic cholecystitis. The liver biopsy findings were consistent with the history of PBC, showing evidence of cirrhosis, damage to small bile ducts, and fibrosis detected around occasional lobules.

After an uncomplicated recovery, the patient was discharged home on the third postoperative day with no sequelae or deficits from the hypoxia suffered during the cholecystectomy. Two weeks post-op the patient presented for follow-up and reported feeling markedly better than she had felt in years.

3. Discussion

In patients with biliary dyskinesia as determined by an abnormal ejection fraction on HIDA scan, laparoscopic cholecystectomy has been shown to be beneficial in reducing pain and associated symptoms in the majority of patients.[4] However, the procedure is not without risks, one being the formation of CO_2 emboli. This risk may be increased in patients with PBC for the following reasons: Fibrosis associated with PBC increases resistance to blood flow, resulting in parenchymal hypoxia, which in turn upregulates angiogenesis.[5] This increased blood flow is essential for the delivery of nutrients, oxygen, and inflammatory mediators in these areas.[5–7] Immunohistochemical studies in liver samples from patients with PBC demonstrated increased levels of vascular endothelial growth factor (VEGF), a key mediator of angiogenesis, along with other angiogenic factors such as Angiopoietin-1 and Angiopoietin-2, in response to hypoxia.[5] It has also been shown that the inflamed portal tracts in PBC have increased vasculature as compared to controls.[8] Additionally, patients with PBC have increased nitric oxide levels, which may allow for the vasodilation required in early stages of angiogenesis.[5,9] Similar mechanisms of angiogenesis and increased expression of VEGF have been characterized in other biliary tract diseases.[6] The relationship between fibrosis and angiogenesis not only exists in primary biliary cirrhosis, but has been observed in chronic liver diseases such as hepatitis B, hepatitis C, and alcoholic liver disease.[10] Increased angiogenesis and increased levels of nitric oxide, a potent vasodilator, could provide a more direct pathway for CO_2 to enter the liver upon laceration and allow for the embolism to travel more readily through the altered vasculature into the inferior vena cava and up to the heart.

In our patient with probable cirrhotic changes, the operative risk may be increased during abdominal entry due to hepatic enlargement, parenchymal friability, increased angiogenesis, and dilation of blood vessels. Vigilant monitoring should be employed to reduce the time it takes for quick repositioning and hyperventilation to avoid circulatory collapse, thus hastening dissolution and recovery. Intraoperative monitoring at our facility includes blood pressure, pulse oximetry, EKG, and capnography. In this case, the diagnosis of CO_2 embolus was made based on the documented rapid decrease in $PETCO_2$, hypotension, and hypoxemia. Under the current standard, a rapid decrease in $PETCO_2$ is an acceptable indicator of CO_2 embolism, although other causes should also be considered, such as mechanical error, position change of endotracheal tube, and pneumothorax.[11] In order to reduce the differential and allow for more rapid intervention, transesophageal echocardiography (TEE) can be used to diagnose CO_2 emboli. Mann and colleagues[12] found that TEE was more sensitive, more rapid, and allowed for the detection of smaller CO_2 emboli in controlled porcine experiments. It also has been found that TEE can be placed with relative ease following anesthetic induction, with placement maintained throughout the entire procedure.[13] Due to the need for rapid detection and management, TEE would likely need to be placed following anesthesia and prior to insufflation.[2] While TEE has merit for detection and

response, there are additional considerations for its use. TEE may be too sensitive and can distract the anesthesiologist due to increased amounts of sub-clinical emboli that do not require intervention.[14] The use of TEE is not absolute or intended for every patient, but is an option that can be considered should a patient be evaluated to be a higher risk for CO_2 emboli, such as in this case. However, vigilance is the number one safeguard for detection of CO_2 emboli, and the anesthesia provider must always be aware of this potential complication and be prepared to act accordingly.

4. Conclusion

It is vital that both surgeons and anesthesiologists be aware that CO_2 emboli can occur during laparoscopic surgery. Knowledge of the risks, signs, and management of this complication allows for rapid detection and response. It is also worth noting that there may be an increased risk of CO_2 embolus in patients with PBC in addition to other chronic liver diseases that result in fibrosis of the liver. Increased monitoring may be employed to quickly respond to a complication should it occur.

Conflict of interest

The authors report no conflict of interest pertinent to this work.

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Ethical approval

Written informed consent was obtained from the patient for publication of this case report. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Author contributions

Amy Cadis, literature review, research and writing; Chelsea Velasquez, literature review, research and writing; Mark Brauer, literature review, writing, editing; Bruce Hoak, writing, editing.

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