

CASE NOTE

Paradoxical carbon dioxide embolism during laparoscopic unroofing of a recurrent nonparasitic liver cyst

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Carbon dioxide (CO₂) embolism is a rare but severe complication of laparoscopic surgery. It has been reported during laparoscopic cholecystectomy and hepatic resections.¹⁻³ In open hepatic surgery, patients with intrapulmonary or intracardiac arteriovenous communications (i.e., patent foramen ovale [PFO]) are at risk for paradoxical air embolism, as air can pass into the systemic circulation.^{4,5} We report the rare case of a patient with a paradoxical CO₂ embolism (PCO₂E) with elevated end-tidal CO₂ concentrations due to an undiagnosed PFO occurring during the laparoscopic unroofing of a congenital liver cyst.

CASE REPORT

A 41-year-old otherwise healthy woman presented with a recurrent congenital cyst of the liver in segment II and III, which caused upper abdominal pain. Ten years earlier she had undergone surgery for a ruptured liver cyst at the same location. One year later, an infection developed within the recurrent liver cyst, which was treated with repeated percutaneous drainage.

We maintained general anesthesia with 2% sevoflurane in oxygen (FiO₂ = 0.3), supplemented with sufentanil, and performed ventilation in a volume-controlled mode at a positive end-expiratory pressure of 5 cm H₂O. We measured end-tidal CO₂ concentration (FE'_{CO_2}), arterial blood gas (pCO₂, pO₂) and hemoglobin oxygen saturation (SpO₂) throughout.

During surgery, we limited the insufflating CO₂ pressure to 12 mm Hg. Owing to the previous surgical interventions, we had to perform considerable adhesiolysis to completely expose the liver. Thereafter, we started resecting of the cystic roof with an adjacent rim of hepatic parenchyma using the harmonic scalpel. After 3 hours of surgery, there was a sudden increase in FE'_{CO_2} (from 31 to 70 mm Hg) followed by a deterioration of hemodynamic functions: tachycardia from 75 to 140 beats/min, a decrease in systolic blood pressure (from 125 to 60 mm Hg) and a decrease in SpO₂ (from 98% to 80%). These clinical signs suggested that a CO₂ embolism had developed. We immediately stopped intra-abdominal CO₂ insufflation and deflated the pneumoperitoneum. We started the patient on mechanical ventilation with 100% oxygen and stabilized hemodynamic functions with intravenous administration of dobutamine and noradrenaline. Thereafter, a transesophageal echocardiograph (TEE) showed an enlargement of the right ventricle, gas bubbles in both ventricles and a so far unknown PFO. Owing to gas embolism, a right-to-left shunt appeared through the PFO. Fifteen minutes after onset of symptoms, there was a slight decrease in FE'_{CO_2} to 55 mm Hg, but arterial pCO₂ concentration was still elevated (105 mm Hg). After 30 minutes of resuscitation, hemodynamic and respiratory functions stabilized (heart beat 85/min, SpO₂ 98%, systolic blood pressure 115 mm Hg, FE'_{CO_2} 45 mm Hg, pCO₂ 53 mm Hg). We were able to wean the patient off inotropic and vasopressor

support as well as FiO_2 . A second TEE demonstrated recovery of right ventricular function. We converted the operation to open laparotomy without any further complications. The patient recovered completely without any sequelae.

DISCUSSION

Today, the laparoscopic unroofing of the cyst wall is considered the treatment of choice in cases of solitary or multiloculated benign and congenital cysts of the liver. Since the laparoscopic approach necessitates a pneumoperitoneum with a positive intra-abdominal pressure, gas may gain access into the venous circulation. The most common cause is inadvertent injection of CO_2 into a large vessel or solid organ during initial insufflation using a Verres needle.^{1,2} Moreover, CO_2 or argon gas can be insufflated into cut vessels at the dissected parenchyma (i.e., during laparoscopic liver resection).³ In this case report, we demonstrate that CO_2 embolism may also occur during laparoscopic unroofing of a congenital liver cyst when the cystic roof is excised with an adjacent rim of hepatic parenchyma to allow a wide opening of the cyst. Apparently, CO_2 embolism can also be caused by cut small hepatic veins.

Another extraordinary aspect of our patient's case was the occurrence of a PCO_2E through a previously undiagnosed PFO. Usually, carbon dioxide embolism during laparoscopic surgery and in patients without pre-existing arteriovenous shunts is characterized by a deterioration of hemodynamic functions and decreased SpO_2 and FE'_{CO_2} .¹⁻³ The latter is due to a "gas-lock" within the right ventricle, which interrupts the outflow into the pulmonary arteries.

In case of a paradoxical air embolism during open hepatic surgery, where air passes into the systemic circulation by way of arteriovenous communication, the same alterations with decreased FE'_{CO_2} have been described.^{4,5} In contrast to those reports, we observed a markedly increased FE'_{CO_2} during PCO_2E . As documented by intraoperative TEE, the PFO caused a serious right-to-left shunt with an overflow of CO_2 into the systemic circulation. Probably as a result of this arterial pCO_2 overload within the systemic circulation, the FE'_{CO_2} increased as well despite blockade of the right ventricular outflow.

Considering the outlined complication of a PCO_2E during laparoscopic surgery and the possible consequences to the patient, the preference for using CO_2 as insufflating gas should be emphasized owing to its short-lasting effects in case of embolism.

Competing interests: None declared.

References

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