overhead surgical light and exposure to unintended high levels of visible and infrared radiant energy. It is remarkable that such a commonplace and seemingly benign device as the surgical light could cause a large, full-thickness burn that ultimately required plastic surgical repair. This case report underscores the need for vigilance on the part of the anesthesiologist.

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Profound Hypercarbia Late in the Course of Laparoscopic Cholecystectomy: Detection by Continuous Capnometry

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LAPAROSCOPIC techniques increasingly are being applied to intraperitoneal surgical procedures. Carbon dioxide insufflation into the peritoneal cavity is essential. We describe a case in which carbon dioxide insufflation caused profound hypercarbia late in the course of laparoscopic cholecystectomy. This sudden event was detected by continuous capnometry.

Case Report

An 82-yr-old woman was scheduled for a laparoscopic cholecystectomy after admission for jaundice. Medical history was significant for hypertension, treated with enalapril and hydrochlorothiazide. Past surgical and anesthetic history was noncontributory. Physical examination was remarkable only for jaundice. Laboratory evaluation and radiologic findings were consistent with extrahepatic cholestasis

secondary to gallstones. Noninvasive monitoring included an Ohmeda RGM 8250 pulse oximeter/capnometer (Madison, WI). Anesthesia was induced with 2 mg/kg propofol and 0.4 mg/kg atracurium intravenously. After tracheal intubation, anesthesia was maintained with 0.5–1.0% isoflurane, 50% N_2O, 50% O_2, and atracurium. During the 1st hour of anesthesia, the patient also received 2 mg midazolam and 100 μg fentanyl intravenously in divided doses. Sixty minutes after skin incision and carbon dioxide, insufflation was begun (75 min after anesthesia induction), end-tidal carbon dioxide was noted to have increased within 5 min, from 44 to 93 mmHg. Vital signs were stable at this time (temperature 35.0° C, pulse 72, blood pressure 130/70 mmHg, respirations with mechanical ventilation at a rate of 10 and 700 ml expiratory volume). Inspired carbon dioxide was noted to be zero. Hemoglobin oxygen saturation was 98% by pulse oximetry. An arterial blood gas revealed pH 7.02, partial pressure of oxygen 261, carbon dioxide tension 114, and bicarbonate 29.

Immediate hyperventilation by hand was begun (rate 20–25/min, expiratory volume 700–1,000 ml), and the surgical team was informed. At this time, the gallbladder was removed and peritoneal irrigation was accomplished. Several moments later it was noted that the tip of the insufflation port was no longer intraperitoneal. This was not discovered earlier because the camera had been inserted through the same port. The camera had been removed for adjustment and could not be reinserted through this port. After carbon dioxide insufflation was discontinued, end-tidal carbon dioxide returned to normal within about 10 min. Mechanical ventilation was instituted again (rate 10, expiratory volume 700 ml).

As the drapes were removed at the conclusion of the procedure, the patient was seen to have significant areas of subcutaneous emphysema over the trunk, thighs, and lateral abdomen. Anesthesia was discontinued, the effects of the neuromuscular relaxants were reversed, the trachea was extubated in the operating room, and the patient was taken to the recovery room awake without incident. A repeat arterial blood gas performed after 1 h in the post anesthesia care unit with the patient breathing room air revealed a pH of 7.37, partial pressure of oxygen 81, carbon dioxide tension 44, and bicarbonate 25. The remainder of the patient's postoperative course was unremarkable.

Discussion

This is the first reported case of acute profound hypercarbia occurring late in the course of a procedure, caused by carbon dioxide insufflation and first detected by capnometry. Carbon dioxide insufflation into the peritoneal cavity is known to increase arterial carbon dioxide tension, which may be managed by increasing minute ventilation to compensate for peritoneal carbon dioxide absorption.[1] There is evidence that this increasing arterial carbon dioxide tension stabilizes in approximately 40 min.[2] Profound hypercarbia secondary to initial subcutaneous placement of an insufflation trocar has been reported; however, subcutaneous emphysema was the initial finding.[3] The present case differs from the above observations in that subcutaneous emphysema apparently occurred late in the course of the procedure, was confined to areas concealed by surgical drapes, and was detected only after the hypercarbia was documented with continuous capnometry and confirmed with arterial blood gases.

Another possible explanation for the hypercarbia in the present case is carbon dioxide embolization. Carbon dioxide embolization during laparoscopy is a rare event.[4,5] However, several cases of purported carbon dioxide embolization during laparoscopy have been reported.[6-11,*] In all of these cases, severe hemodynamic instability was present. In addition, these reports noted "mill-wheel" murmurs,[7,9,10] pulmonary edema,[6,12,*] metabolic acidosis[6] and electrocardiographic abnormalities.[6-8,10,11,*] In the present case,

none of these findings were observed. In addition, there was no evidence of blood vessel perforation at the site of the insufflation port. Nonetheless the possibility of carbon dioxide embolization *via* a cutaneous blood vessel could explain our observations.

Capnometry has been demonstrated to be useful in detecting aberrations in circulation, respiration, carbon dioxide production/excretion, and anesthetic equipment problems, as well as an effective early warning detector of esophageal intubation, malignant hyperthermia, venous air embolism, and hypercarbia caused by carbon dioxide insufflation.[12] The present case not only demonstrates the utility of continuous capnometry in detecting carbon dioxide derangements during laparoscopic procedures but also serves to remind us that not all carbon dioxide derangements are related to changes in ventilation.

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