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CASE REPORTS/CASE SERIES

Gas embolus and cardiac arrest during laparoscopic pyloromyotomy in an infant

Embolie gazeuse et arrêt cardiaque pendant une pylorotomie laparoscopique chez un nourrisson

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Abstract

Purpose *High volume tubing is used to deliver carbon dioxide during laparoscopic procedures. Failure to prime the tubing with carbon dioxide prior to abdominal insufflation may result in the delivery of nitrogen-containing air to the abdominal cavity. We report a case in which initial insufflation of laparoscopic gas resulted in immediate cardiovascular collapse requiring prolonged resuscitation. Persistent intracranial emboli following the arrest may have resulted from nitrogen contamination of the delivered gas.*

Clinical features *A 12-day-old female underwent laparoscopy for pyloric stenosis. During initial insufflation of the abdomen, the patient had an abrupt decrease in end-tidal carbon dioxide (CO_{2ET}) associated with bradycardia and pulseless electrical activity. Three hours after successful resuscitation and open pyloromyotomy, computerized tomography documented intra-arterial gas within the cerebral and hepatic circulations that resolved following hyperbaric oxygen therapy. Magnetic resonance imaging five days later revealed watershed infarcts in the right frontal and parietal regions. Nitrogen, an insoluble gas not easily eliminated from the body, was likely the gas present within the patient's circulation several hours after the event. It was unlikely carbon dioxide, which is a highly soluble gas that binds to hemoglobin and is rapidly buffered by the carbonic anhydrase system and excreted by the lung. Room air contamination of high volume insufflation tubing allows nitrogen to enter body cavities during endoscopic procedures.*

Conclusion *Persistence of emboli following endoscopic procedures suggests that the entrained gas is insoluble. Room air contamination increases the potential for catastrophic events during laparoscopy and other endoscopic procedures.*

Résumé

Objectif *Une tubulure de grand volume est utilisée pour fournir du dioxyde de carbone pendant les interventions par laparoscopie. À défaut de purger la tubulure avec du dioxyde de carbone avant l'insufflation abdominale, de l'air azoté pourrait être entraîné dans la cavité abdominale. Nous signalons un cas où l'insufflation initiale de gaz par voie intrapéritonéale a entraîné un collapsus cardiovasculaire immédiat nécessitant des manœuvres prolongées de réanimation. L'embolie intracrânienne persistante suivant l'arrêt cardiaque peut avoir été causée par une contamination par l'azote du gaz insufflé.*

Éléments cliniques *Une patiente âgée de 12 jours a été soumise à une laparoscopie en raison d'une sténose du pylore. Au cours de l'insufflation initiale de l'abdomen, il y a eu une brusque diminution de dioxyde de carbone télé-expiratoire (CO_{2ET}) avec une bradycardie et une activité électrique sans pouls. Trois heures après la réanimation et une pylorotomie ouverte, la tomodensitométrie a permis de documenter la présence de gaz intra-artériel dans les vaisseaux cérébraux et hépatiques, avec résorption suite à une oxygénothérapie hyperbare. Cinq jours plus tard, l'imagerie par résonnance magnétique a révélé des infarctus*

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jonctionnels dans les régions frontale et pariétale droites. Il est fort probable que le gaz présent dans la circulation de la patiente plusieurs heures après l'événement était de l'azote, un gaz insoluble que le corps élimine difficilement, et non du dioxyde de carbone, un gaz hautement soluble qui se lie à l'hémoglobine et est rapidement métabolisé par l'anhydrase carbonique et éliminé par les poumons. La contamination par l'air ambiant de la tubulure d'insufflation à grand volume a permis à l'azote de s'introduire dans les cavités de l'organisme pendant une intervention par endoscopie.

Conclusion *La présence d'une embolie persistante à la suite d'interventions par endoscopie laisse croire que le gaz insufflé est insoluble. La contamination par l'air ambiant augmente l'éventualité d'un événement catastrophique pendant la laparoscopie et d'autres interventions par endoscopie.*

Carbon dioxide is used as an insufflating gas during laparoscopic procedures because it does not sustain combustion, and its high solubility in blood decreases the complications associated with intravenous embolism. Historically, circulatory collapse associated with laparoscopic insufflation has been attributed to carbon dioxide embolism,[1-4] but the severity of some events is not consistent with intravenous carbon dioxide alone, and nitrogen in air may contribute to sustained hemodynamic decompensation and catastrophic outcomes. We report the case of a 12-day-old infant who suffered a massive embolism during laparoscopic pyloromyotomy, cardiac arrest, and successful resuscitation that included hyperbaric oxygen therapy for documented intracranial arterial gas emboli.

Case description

The patient's parents gave consent to publish this case. A term 12-day-old female neonate weighing 3.6 kg presented to an outside hospital with a 36-hr history of projectile vomiting and an inability to tolerate oral feedings. She had syndactyly of the fourth, fifth, and sixth toes of the right foot, but no other known congenital anomalies. She was admitted for volume resuscitation and evaluation of suspected pyloric stenosis, confirmed by ultrasound after transfer to Children's Hospital of Wisconsin. Preoperative electrolytes were within normal limits, and urine output was adequate following intravenous hydration.

In the operating suite, standard monitors were utilized throughout the case, including airway gas analysis using Raman spectroscopy. The patient's stomach contents were evacuated with an orogastric tube prior to induction of anesthesia. Her trachea was intubated with a 3.5 mm uncuffed endotracheal tube following a modified rapid sequence technique using propofol 7 mg and rocuronium 4 mg. Anesthesia was maintained with isoflurane in an air/oxygen mixture (inspired O_2 fraction 0.36) and ventilation was controlled. Approximately 30 min after induction of anesthesia, a laparoscopic trocar was placed. At the onset of carbon dioxide insufflation, the patient coughed. The end-tidal carbon dioxide partial pressure (CO_{2ET}) decreased from 38 mmHg to 22 mmHg and then increased to 27 mmHg. Within two minutes, however, there was no detectable CO_{2ET}. The patient was mottled and ashen. Chest compressions were initiated, and sequential doses of epinephrine were administered both via the peripheral intravenous cannula and the endotracheal tube for bradycardia and pulseless electrical activity. Pressure controlled ventilation with 100% oxygen was maintained through the resuscitation efforts. Direct laryngoscopy confirmed endotracheal tube position and pneumothorax was ruled out. Differential diagnosis of the cardiac arrest focused on inadequate preload from hemorrhage or gas embolism creating outflow tract obstruction. The surgeon reported a small extraperitoneal laceration of the umbilical vein. Open laparotomy failed to demonstrate any additional source of bleeding. Femoral venous and arterial catheters and near-infrared spectroscopy probes were placed during the resuscitation. Spontaneous circulation and ventilation returned 36 min after the initial loss of CO_{2ET} and blood pressure. Transthoracic echocardiography demonstrated a structurally normal heart with a patent foramen ovale. Echolucencies were noted in the hepatic circulation, confirming the diagnosis of gas emboli. Pyloromyotomy and closure of the abdominal incision were performed following the resuscitation.

Three hours after the arrest, computed tomography (CT) scans of the patient's head and body revealed multiple discrete foci of intra-arterial gas adjacent to the lateral ventricles (Figure 1) and in the pulmonary and hepatic circulations. The patient was treated with a series of hyperbaric oxygen treatments according to the US Navy Dive Protocol Table 6/6A.[A] A CT scan after the treatment confirmed resolution of the intracranial air. Additional supportive measures included moderate hypothermia to 33-34°C, deliberate hypercapnia, and barbiturate, benzodiazepine, and narcotic sedation for seizure prophylaxis and reduction of cerebral and systemic metabolic demands for oxygen during the reperfusion stage of ischemic injury. Although magnetic resonance imaging demonstrated watershed infarcts in the right frontal and parietal regions,

[A] U.S. Navy Diving Manual, chapter 21, available from URL: http://www.supsalv.org/pdf/DiveMan_rev6.pdf (accessed April 2010).

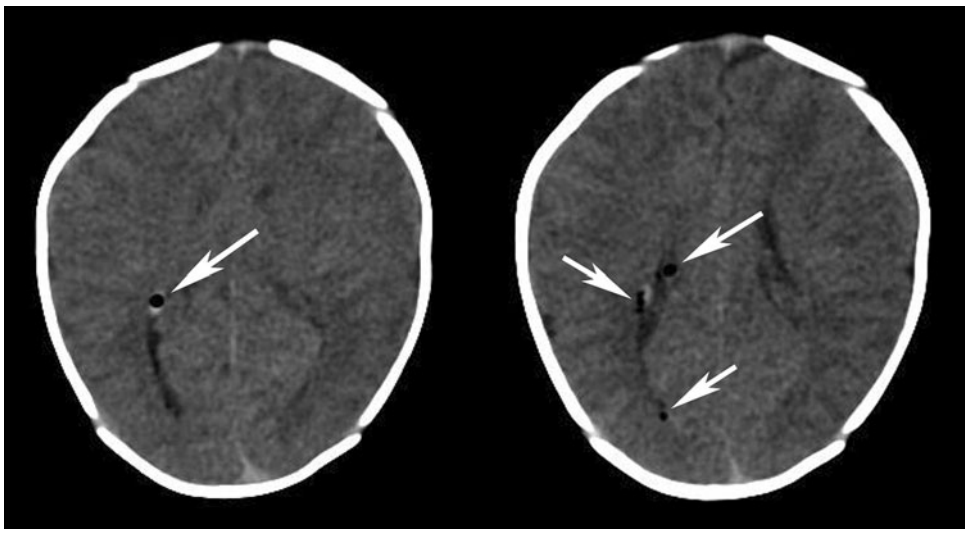

Fig. 1 Cranial computed tomography (CT) scan. Axial cut at the level of the lateral ventricles demonstrates multiple small foci of gas

the patient's neurological examination was normal at the time of discharge nine days following the arrest. More than two years after the event, the patient continues to thrive and meet developmental milestones.

Discussion

This case report shows the potential for catastrophic cardiovascular collapse associated with gas emboli during laparoscopic procedures in neonates. Although most studies demonstrate shorter hospitalizations and time to oral feeds when comparing laparoscopic and open techniques for pyloromyotomy, a 2005 analysis of hospital data[5] demonstrated a skewed distribution of costs for patients undergoing laparoscopic procedures, prompting the authors to propose that such charges may result from a small number of serious complications.

This case supports their hypothesis. Persistent fetal cardiovascular anatomy increases the risk of gas emboli during laparoscopic procedures, particularly when a periumbilical or umbilical approach is employed. Our patient suffered a small extraperitoneal laceration of the umbilical vein. The surgeons elevated the abdominal skin to facilitate placement of a needleless sheath under direct vision through the umbilical stump. During attempted insufflation of the peritoneum, an abrupt reduction in end-tidal CO_2 and pulseless electrical activity occurred. This episode was probably due to gas entering the central circulation via the umbilical vein and ductus venosus in sufficient volume to obstruct the right ventricular outflow tract or pulmonary vessels. Elevated right atrial pressures facilitated entry of gas into the systemic circulation via the foramen ovale.

In a recent report of embolism during laparoscopic repair of duodenal atresia in a one-day-old infant,[6] the loss of end-tidal CO_2 and subsequent cardiovascular collapse occurred *in association with* placement of the umbilical trocar but *before* insufflation of carbon dioxide. As in our case, bleeding was found at the stump of the umbilical vein. The authors stated that there was no vertical gradient between the surgical site and the right atrium. However, the traction used during insertion of the laparoscopic tools may elevate a lacerated vessel within the umbilical stump several centimetres above the height of the right atrium, producing the necessary pressure differential required to allow entry of air in hypovolemic patients with attendant low right atrial pressures. Similar phenomena leading to fatal outcomes have been reported during lumbar laminectomy in older patients.[7,8]

There are two possible sources of nitrogen during laparoscopy: direct entry of room air through a lacerated extraperitoneal vessel and residual air within the endoscopic tubing entering the peritoneum during insufflation and potentially entering the circulation through a bleeding vessel anytime throughout the procedure. The tubing currently in use in our institution for laparoscopy has a volume of 230 mL. Priming the equipment with carbon dioxide prior to use is neither routine nor suggested by the manufacturer's instructions. There is no warning on any of the instruments that air may be introduced during insufflation. In infants, less than 100 mL of gas is sufficient to provide good visualization during laparoscopy. Therefore, all of the initial intraperitoneal gas in small patients may be air rather than carbon dioxide. In our case, the insufflator had a minimal detectable volume of 100 mL and did not record the volume of gas delivered.

Preliminary studies at our institution suggest that at least 400 mL of carbon dioxide is required to fully eliminate nitrogen from the tubing. Thus, the greatest risk of air emboli exists for the smallest patients shortly after initial

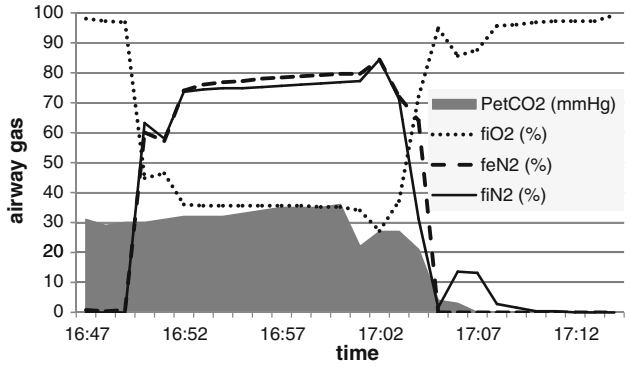

Fig. 2 Inspired and expired gas data prior to cardiorespiratory arrest following laparoscopic insufflation. Note that the expired nitrogen fraction (feN_2) exceeds the inspired (fiN_2) fraction for 13 min prior to the arrest. $PetCO_2$ = end-tidal CO_2 partial pressure; F_iO_2 = inspired oxygen fraction

insufflation when the intraperitoneal nitrogen concentration is likely to be high. The presence of an expired to inspired nitrogen pressure gradient can be used to diagnose air embolism.[9] In our case, *expired* nitrogen exceeded *inspired* nitrogen concentration for 13 min leading up to the arrest. This coincided with two attempts at insufflation of the abdomen (Figure 2). The increase in expired nitrogen suggests an additional source of the gas, i.e., the peritoneum. Air embolus in awake patients is associated with cough,[10] although the mechanism for this reaction is unclear. Other possible explanations for coughing at incision include light anesthesia, irritation of the carina from a malpositioned endotracheal tube, and intrinsic reactive airway disease, none of which can be ruled out in our patient.

Carbon dioxide is rapidly cleared from the circulation due to its high solubility and the binding and buffering capabilities of blood. Arterial injection of carbon dioxide for angiographic procedures is safe in patients with reduced renal function or iodine allergy and is rarely associated with complications.[11] In piglets, continuous intravenous injection of carbon dioxide at clinically relevant pressures of 5 and 10 mmHg is well tolerated for as long as five minutes. However, some animals experience hemodynamic compromise and death at insufflation pressures of 15 mmHg and 20 mmHg.[12] The mean lethal intravenous dose of carbon dioxide in a pig model approaches 50 $mL \cdot kg^{-1}$,[13] approximately ten times the lethal intravenous dose of air.[14]

Although sporadic reports of death in humans associated with massive carbon dioxide embolism do exist,[15] the possibility that nitrogen is the lethal contaminant within the insufflation tubing must be considered. Most cases are diagnosed by auscultative or hemodynamic changes and loss of end-tidal CO_2, followed by successful resuscitation efforts.[1,2,4] Management includes removal of the carbon dioxide source, intravascular volume replacement, inotropic support, and if necessary, chest compressions. Hyperbaric oxygen therapy may be required if there is persistent evidence of gas hours following an embolic event.

This case highlights the potential for serious neurological complications during laparoscopy and suggests that nitrogen may contribute to these complications. The risk of the umbilical approach in neonates is greater than for older patients who lack residual fetal vessels that may permit right-to-left shunting and paradoxical emboli. However, small volumes of air contamination may increase the risk of laparoscopy even in an adult with a patent foramen ovale. Purging insufflation tubing of air is necessary to reduce the risk of nitrogen-containing emboli. Effective cardiopulmonary resuscitation, hyperbaric oxygen therapy, and supportive measures to minimize ischemic brain injury may reduce long-term complications of arterial gas embolism.

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Conflicts of interest None declared.

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