Title: A Rare Cause of Intraoperative Neonatal Cardiac Arrest

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ABSTRACT BODY:

Introduction

Circulatory collapse secondary to venous gas embolism during peritoneal carbon dioxide insufflation for laparoscopic procedures is well known. We describe a case of neonatal cardiac arrest during attempted laparoscopic repair of duodenal atresia. The cardiac arrest occurred prior to peritoneal insufflation and thereby initially obscured the diagnosis. However, emergency echocardiography was performed during the resuscitation and revealed the mechanism of the arrest. The poster will describe the case and discuss the risks of laparoscopic procedures, with special emphasis on the effects on neonatal physiology.

Case Report

An ex-36 week gestation one day old male was scheduled for laparoscopic repair of duodenal atresia that had been diagnosed prenatally by ultrasound. He was delivered uneventfully by cesarean section following a non-reassuring fetal heart trace. He weighed 2 kg., had physical features of trisomy 21, and the rest of his history and physical examination were unremarkable. His vital signs and laboratory results were normal.

Following standard monitoring and preoxygenation, he underwent rapid-sequence intravenous induction with cricoid pressure, propofol and rocuronium. His trachea was intubated uneventfully on the first attempt with a 3.0 endotracheal tube, and anesthesia was maintained with Sevoflurane in oxygen and air, and a small dose of fentanyl (1mcg/kg). A second intravenous catheter was placed uneventfully, and the patient was prepared for surgery.

3-4 minutes following surgical incision and insertion of the umbilical trocar, the end-tidal CO2 suddenly decreased from 26 to 6 mmHg and was accompanied by a loss of the pulse oximetry trace, ST segment depression and a decrease in the heart rate from 135 to 100/minute. The surgeons were immediately asked to release the insufflation pressure, but to our surprise, they indicated that they had not yet insufflated the peritoneal cavity. Concomitant with this exchange, the Sevoflurane was discontinued, the patient was hand ventilated with 100% oxygen while the integrity of the gas delivery system was checked, and the patient's lungs were auscultated to rule out pneumothorax or endotracheal tube displacement. All the intravenous lines and catheters were checked for sources of air entrainment, but nothing obvious was detected. The surgeon was asked to inspect the abdomen for trauma, which he did following partial insufflation, but this was negative. The surgeons reported discoloration of the abdominal wall, and the drapes were immediately removed. The child's skin was profoundly mottled, with large purplish-black patches on the trunk, head, and all extremities. Cardiopulmonary resuscitation (CPR) was immediately commenced with chest compressions, atropine, and several epinephrine boluses. In the absence of a diagnosis, the cardiology team was consulted for emergent echocardiography; they arrived less than 10 minutes later and transthoracic echocardiography demonstrated large amounts of air in the right ventricle, pulmonary arteries, aorta, and liver tissue, sufficient to obscure cardiac anatomical details (Fig 1). Over the next 10 minutes of CPR, gradual improvement in the end-tidal CO2 level and S-T segment depression was accompanied by the reappearance of a palpable pulse and the pulse oximeter waveform. Ventricular systolic function was noted to be normal following CPR despite mild right heart dilatation. Venous blood gas revealed hypoxemia and a profound metabolic acidosis that was treated with boluses of crystalloid and sodium bicarbonate. The surgeons, meanwhile, upon withdrawing the umbilical trocar, noticed a small amount of active bleeding from the stump of the umbilical vein. The vein was ligated, and the abdomen closed. Following stabilization of the vital signs with an epinephrine infusion and placement of a central venous catheter, the patient was transported to the pediatric intensive care unit (PICU) for elective mechanical ventilation and further management. Despite the improvement in the mottling of the patient's head and trunk, all four extremities of his limbs remained purplish-black at the time of transfer to the PICU.

The patient had seizure-like activity overnight, but electroencephalography was negative for seizures. A brain MRI was performed the next day and was normal. His extremities gradually regained color by the following

morning with no tissue loss. A repeat echocardiogram the next morning demonstrated a patent ductus arteriosus with left-to right flow and no residual air. He remained intubated and underwent successful open repair of duodenal atresia two days later. Karyotyping confirmed the diagnosis of trisomy 21.

Following extubation, the baby had no signs of neurological impairment and was discharged home uneventfully 10 days later. At follow-up in clinic one month later, he was developing normally, feeding well, and had no obvious residual effects from the intraoperative cardiovascular collapse.

Discussion

Massive venous air embolism (VAE) with paradoxical embolism via the foramen ovale and the ductus arteriosus was responsible for this patient's cardiovascular collapse. The source of entrainment was probably the bleeding stump of the umbilical vein noted at the site of insertion of the umbilical trocar. Identification and ligation of bleeding vessels following laparoscopic trocar insertion should be carried out promptly to prevent air entrainment before peritoneal insufflation.