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Fatal Air Embolism after Gastrointestinal Endoscopy

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Although intraoperative venous air embolism (VAE) has been described in many settings, the conditions surrounding the case of VAE during gastrointestinal endoscopy have not previously been described.

REPORT OF A CASE

The patient, an infant girl, originally presented for medical care at age 1 month with persistent hyperbilirubinemia and was found to have biliary atresia. At age 5 weeks, the patient underwent a Kasai procedure (hepatopertoenterostomy), and her condition improved. Liver biopsy at that time revealed a mild degree of hepatic cirrhosis. Over the subsequent 3 months, the patient experienced multiple bouts of ascending cholangitis. On the current hospital admission, the 4-month-old patient's bile drainage had diminished significantly, and the surgeon wished to visualize the hepatoportal enterostomy site with a gastroscope.

In the operating room, monitors (including pulse oximeter) were applied, and the induction of anesthesia began by inhalation of nitrous oxide (70%) and oxygen (30%) via a mask with increasing concentrations of halothane. After atropine (0.04 mg) and succinylcholine (10 mg) were administered iv, nitrous oxide (N₂O) was discontinued. After 100% oxygen had been administered for 1 min, the trachea was intubated, and bilateral auscultation of the chest demonstrated clear and equal breath sounds. Vital signs were stable at this time with an arterial blood pressure of 90/40 mmHg, a normal sinus rhythm at 120/bpm, and 100% oxygen saturation of hemoglobin. Anesthesia was maintained with 2% halothane and 60% N₂O in oxygen. About 4 min after the gastroscope was inserted into the stoma, the peripheral oxyhemoglobin saturation dropped to 60% and the heart rate decreased from 140 to 70 bpm within 15-30 s. Almost immediately thereafter, peripheral pulses were not palpable, and the ECG showed ventricular fibrillation.

Cardiopulmonary resuscitation was begun, and the femoral artery was exposed by surgical dissection in order to obtain arterial blood for gas analysis. Air bubbles were noted in the arterial cannula as blood was withdrawn. The patient never regained a viable heart rhythm or arterial blood pressure.

At autopsy, massive air embolus was determined to be the cause of death. Air was found in the right atrium, right ventricle, and in a large hepatic vein in the area of the porta hepatis. A patent foramen ovale was noted, and air was present in the coronary arteries.

On closer examination of the hepatopertoenterostomy, the pathologist found a large hepatic vein just under the enterostomal surface of the liver. Examination of serial sections of tissue revealed that the hepatic vein approached and was almost contiguous with the denuded surface of the liver. The medical examiner's report also included an evaluation of the gastroscope. This gastroscope was capable of infusing air or water by operating one switch. By occluding the end of a button with light pressure, the operator would infuse air at 8-9 cc/s. If the button were completely depressed, water would be infused at a rate of 0.3 cc/s.

DISCUSSION

The effects of VAE may be minimal or clinically significant depending on the amount of air infused, its rate of infusion, and the location of the embolus. When an open vein is higher than the level of the heart with a pressure gradient of approximately 5 cm H₂O, the patient is at risk for VAE.¹ VAE is commonly reported during neurosurgical procedures in the sitting positions.² It has also been described during Cesarean section,³ liver transplantation,⁴ and total hip replacement.⁵ However, a review of the literature suggests that a case similar to ours has not been reported.

Studies in dogs revealed different physiologic responses to VAE depending on the rate and quantity of air infused.⁶ Slow infusion of air caused predictable cardiopulmonary changes, including, in order of increasing air infusion, a characteristic "gasp," ECG changes (peaking of P waves, later ST segment depression), increased central venous pressure, increase in heart rate, decrease in peripheral resistance, and eventually the presence of a "mill wheel"

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murmur. When the air was injected as a bolus, the pathophysiological responses were different. First, a brief period of hyperpnea was followed by a period of apnea; central venous pressure increased with relatively small boluses of air. The greater the bolus of air, the greater the heart rate increase and the greater the decline in arterial blood pressure. On necropsy, animals that died after slow air infusion had air dispersed throughout the cardiopulmonary circulation, whereas animals dying after rapid injection had air primarily in the right side of the heart.⁶

The use of nitrous oxide (N_2O) may influence the pathophysiology and progression of VAE.² Because N_2O is 34 times more soluble in blood than nitrogen, N_2O shifts rapidly into air-filled spaces.² Animal studies have shown that the administration of N_2O decreased the volume of intravenous air needed to cause death.¹² In a different study, the cardiovascular responses of VAE were magnified in those dogs ventilated with nitrous oxide (50%) and oxygen (50%) compound to dogs ventilated with 100% oxygen.¹³

Biliary atresia is a disease characterized by progressive fibrosis of the extrahepatic biliary tree. The diagnosis is usually made during the first month of life.⁷ The etiology of the disease is unknown, but the pattern of parenchymal and duct cell injury are similar to those of neonatal hepatitis.⁸ In 1959, Kasai and Suzuki introduced the hepatoportoenterostomy. Although there are many variations, the essence of the procedure involves excision of the fibrous remnant of atretic ducts¹⁰ at the level of the porta hepatis.¹¹ After exposing the patient's hepatic ducts, the surgeon performs an end-to-side portoenterostomy with a 40-cm loop of jejunum.¹⁰

In the case presented, the patient was admitted with a diagnosis of recurrent cholangitis after a Kasai procedure. Air was infused by gastroscope into the bed of the porta hepatis, and the patient died of a massive air embolus. The pathologist could not establish a direct connection between the hepatoportoenterostomy site and a vascular structure. We propose that air under pressure dissected across diseased liver tissue into a large hepatic vein lying just below the denuded liver surface.

In the future, perhaps gastroscopes should be inspected to insure that they infuse saline, not air. An extension of this thought would involve the manufacture of endoscopes with separate, distinct controls to infuse air and saline and with limits on the rates of infusion. Furthermore, anesthesiologists might keep in mind the potential for air embolism in adults, as well as children, undergoing endoscopic procedures, especially in those patients with prior surgery on the gastrointestinal tract.

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