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Massive Venous Air Embolism during Orthotopic Liver Transplantation

MARIE CSETE PRAGER, M.D.,* GEORGE A. GREGORY, M.D.,† NANCY L. ASCHER, M.D., PH.D.,‡
JOHN P. ROBERTS, M.D.§

Although venous air embolism is cited as a complication of orthotopic liver transplant, there is no documented case reported of air embolism after adequate perfusion (flushing) of the liver. This case is the first such documentation of massive air embolism during liver transplantation.

CASE REPORT

A 40-yr-old 65-kg woman with chronic non-A, non-B hepatitis was admitted for liver transplantation. Four years prior to admission she had an episode of bleeding from esophageal varices, and soon thereafter underwent an end-to-side portocaval shunt. She had no history of significant cardiopulmonary disease.

Immediately prior to surgery her coagulation studies were: PT 14.8 s, PTT 40.7 s, fibrinogen 88 mg/dl, platelets 78,000/mm^3. Her hematocrit was 28%. Arterial blood gas while breathing room air was P_{O_2} 102 mmHg, P_{CO_2} 31 mmHg, *p*H 7.44. Pulmonary function tests revealed FEV_1 of 2.7 l and FVC of 3.4 l. Electrocardiogram and echocardiography were within normal limits.

In anticipation of large blood requirements because of her coagulopathy and prior portocaval shunt, two 14-G iv catheters, a 20-G radial artery catheter, a pulmonary artery catheter (*via* 8.5-Fr introducer), and a right internal jugular 14 Fr USCI® venous cannula were inserted. This last catheter was connected directly to a rapid transfusion device.

Rapid sequence induction of anesthesia using 3 mg d-tubocurare, 275 mg thiopental, and 100 mg succinylcholine was followed with 5 mg midazolam and 15 mg morphine sulfate. Anesthesia was maintained with isoflurane and muscle paralysis was accomplished with pancuronium.

Vital signs were stable throughout the dissection phase of surgery. Veno-venous bypass is not used in our institution, so that at the time the liver vasculature was clamped, there was an expected decrease in systemic blood pressure as well as central filling pressures. The patient required dopamine at 10 $\mu g \cdot kg^{-1} \cdot min^{-1}$ to maintain a systolic blood pressure of 90 mmHg during the anhepatic stage, which lasted 50 min. Prior to removing the suprahepatic caval clamp the portal venous clamp was opened, the liver was perfused, and the venous effluent allowed to drain through a small defect in the incomplete infrahepatic anastomosis. Adequacy of hepatic perfusion was assessed visually: the liver was perfused until it reached a uniform color (about 45 s). The portal clamp was then reapplied, closure of the anterior wall of the inferior vena cava was completed, and the portal, suprahepatic, and infrahepatic clamps were removed.

Within 2 min after removal of the vena cava clamps the CVP was noted to increase suddenly from 13–30 mmHg. Pulmonary arterial diastolic pressure rose simultaneously from 9–24 mmHg. The surgeons noted hepatic congestion, consistent with right ventricular failure. Systemic arterial pressure decreased and was treated with two neosynephrine boluses (100 μg each); dopamine was increased to 18 $\mu g \cdot kg^{-1} \cdot min^{-1}$. An attempt was made to remove fluid from the central circulation by allowing blood from the internal jugular catheter to drain from the patient into a heparinized collection bag. The patient's systemic blood pressure decreased with this intervention, and so the drained blood was returned to the patient. An arterial blood gas during the acute event showed a P_{O_2} 72 mmHg, P_{CO_2} 53 mmHg and *p*H 7.17. At the time hemoglobin saturation was 100% by oximetry. (During the anhepatic phase ABG showed P_{O_2} 312 mmHg, P_{CO_2} 32 mmHg, *p*H 7.35 with the same ventilator settings with gas flows of 2 l O_2 and 2 l air.) The patient was treated further with sodium bicarbonate, $CaCl_2$, and the FIO_2 was increased to 1.0.

The above data were interpreted to be diagnostic of air embolism, and an attempt was made to aspirate air from the CVP and PA ports of the pulmonary arterial catheter. No gas was withdrawn. Over the next 30 min, during which inotropic and metabolic support was maintained, the patient's vital signs improved and the acidosis resolved. The patient ultimately recovered uneventfully from this event.

After the case the computer record of end-tidal gases from the mass spectrometer was retrieved. This record, shown in figure 1, documents a marked and sudden decrease in end-tidal CO_2 and an equally abrupt increase in end-tidal N_2, occurring within 2 min of vena cava unclamping, confirming the diagnosis of massive venous air embolism.

DISCUSSION

Venous air embolism during liver transplantation is cited as a potential complication in many reviews of this procedure.[1] This potential complication is our justification for avoiding nitrous oxide during the procedure. There are no data available, however, on the true incidence of venous air embolism at the time of anastomosis of the donor liver. In addition, although air embolus is a commonly cited problem, there is only one case report of massive air embolus during liver transplant in the anesthesia literature. In that report the diagnosis of air embolism was not considered during the acute event and mass spectrometry data were not reported.[2]

The cause of the large venous air bolus during our case is not apparent since the surgeons felt their perfusion (flush) of the liver to be adequate. Donor livers are routinely perfused *in situ* with a preservation solution (usually

* Assistant Professor of Anesthesiology.
† Professor of Anesthesiology and Pediatrics.
‡ Professor of Surgery.
§ Assistant Professor of Surgery.

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Address reprint requests to Dr. Prager: Department of Anesthesia, University of California, San Francisco, 513 Parnassus Avenue, S-436, San Francisco, California 94143-0648.

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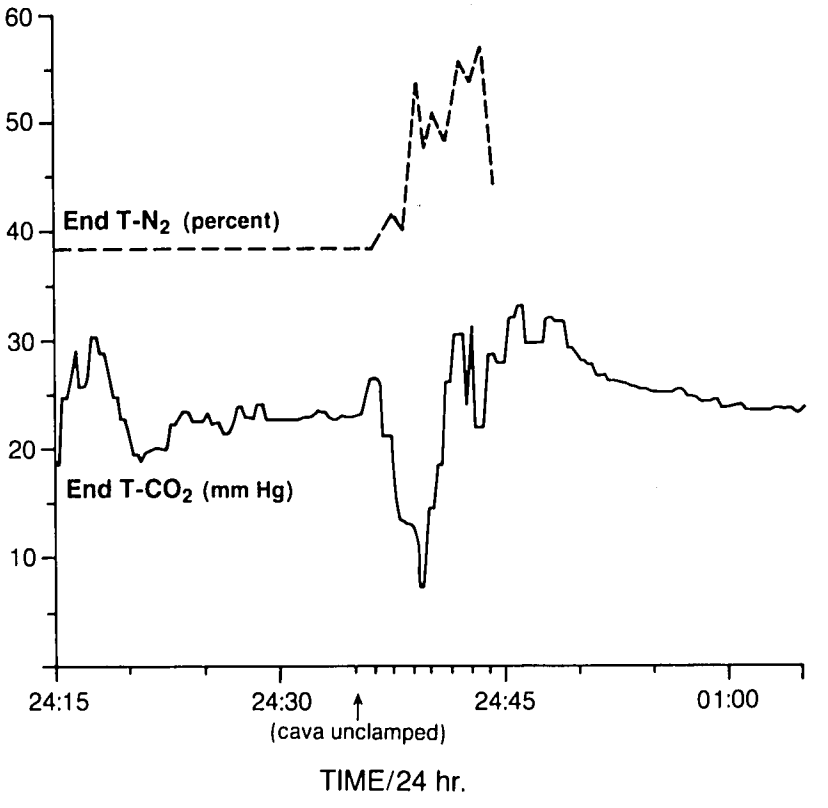

FIG. 1. Plot of end-tidal nitrogen and carbon dioxide levels over time. Note the sudden decrease in end-tidal CO_2 and sudden increase in end-tidal N_2 immediately following release of cava clamps.

UW solution) *via* the portal vein and hepatic artery. Later, when the liver is placed in the recipient and the portal vein anastomosis is complete, the liver is perfused *via* the portal vein, such that air plus preservation solution are drained *via* the open (incomplete) infrahepatic inferior vena cava anastomosis. This maneuver is performed in order to prevent entry of potassium-rich preservation solution and air into the recipient circulation.

In the earlier case report[2] a clamp from the infrahepatic inferior vena cava was not reapplied after flushing, and this error led to entrainment of venous air. In our case all clamps were appropriately applied. Other external sources of venous air have been reported during liver transplant or resection. These include air entry from a peristaltic pump[3] or air embolism associated with veno-venous bypass.[4] In our institution, all tubing used is made of clear materials and was examined for air. No source of air in our peripheral, pulmonary artery, or central transfusion tubing was found. Veno-venous bypass was not used during the case. The patient was completely paralyzed and therefore not entraining air by respiratory efforts. There was no leak in the anesthesia circuit or airway.

A recent case report of fatal pulmonary embolism during liver transplant[5] highlights another rare cause of acute right heart failure during liver transplant. During both venous air embolism and pulmonary thromboembolism, end-tidal CO_2 concentrations would be expected to decrease. Only venous air embolism, however, is associated with the increase in end-tidal N_2 seen in our patient.

Transesophageal echocardiography and precordial Doppler ultrasound are considered the most sensitive monitors of venous air embolism.[6] Neither monitor was used in this patient. We are unwilling to use transesophageal echocardiography in patients with esophageal varices, but will consider its use in transplant patients with documented absence of varices. It is possible that Doppler monitoring could be beneficial in a setting where ongoing entry of air is detected. Since release of the cross-clamp is a one-time event, however, the sensitivity of the Doppler is unlikely to change the clinical course or treatment.

An increase in pulmonary artery pressure, though less sensitive than Doppler in the diagnosis of venous air embolism, has been shown to be proportional to the severity of venous air embolism.[7] End-tidal nitrogen is also a quantitative measure of entrained venous air.[8] Both mass spectrometry and pulmonary artery pressure monitoring were useful in establishing a diagnosis in this case.

Venous air embolism is particularly worrisome in patients with chronic liver disease who may have extensive intrapulmonary arteriovenous shunting.[9] Certainly nitrous oxide is contraindicated during the period of vascular unclamping because of the real potential for venous air release from the donor liver.

This report clearly documents a case of massive venous

air embolism during liver transplantation, despite adequate perfusion of the donor organ prior to unclamping of the inferior vena cava. The case is presented to alert anesthesiologists to prepare for this potentially fatal problem at the time of vascular unclamping.

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Venous Air Embolism during Surgical Manipulation of a Femoral Bone Cyst

JEFFREY M. RUSHEEN, M.D.,* DORA HSU, M.D.,† CHINGMUH LEE, M.D.,‡ MAURICE LIPPMANN, M.D.§

Venous air embolism has been reported as a complication of many types of surgery, but is perhaps best known as a complication of the sitting position for posterior fossa neurosurgery. We report here an unusual case of air embolism in a child occurring as a result of surgical manipulations of a femoral bone cyst.

Case Report

The patient is a 3-yr-old male who was in his normal state of good health until 2 days prior to admission when he jumped from a step of a bus and subsequently was unable to walk because of left leg pain. Physical examination on admission to the hospital revealed: BP 92/50 mmHg, HR 88/min, RR 20/min, T 97.0° F, height 98.1 cm, and weight 13.4 kg. The remainder of the physical exam and laboratory tests were normal. X-ray of the left lower extremity revealed lucency of the proximal femur with a questionable nondisplaced fracture.

The patient was admitted for the treatment of a proximal left femur cyst and to rule out a pathological fracture. Plans were made by the orthopedic service to aspirate the cyst and to inject a steroid into the cyst, followed by treatment with a hip spica cast.

In the operating room, monitors initially consisted of a pulse oximeter, precordial stethoscope, BP cuff, and ECG. Anesthesia was induced with halothane, nitrous oxide, and oxygen *via* mask, after which the trachea was intubated. An iv infusion of D5 in 1/4 normal saline was administered. End-tidal CO_2 (ET_{CO_2}), esophageal temperature, and breath sounds were also monitored. Maintenance of anesthesia consisted of halothane 1% and N_2O (60%) in O_2 *via* a semiclosed circle system, supplemented with iv atracurium for relaxation. Thirty-five minutes after surgery began, ET_{CO_2} suddenly decreased from 30 mmHg to 17 mmHg, then to 4 mmHg. Although breath sounds were still equal bilaterally, auscultation revealed a mill-wheel murmur. Oxyhemoglobin saturation (Sp_{O_2}) which had been 100% decreased to 69%. Blood pressure decreased from 110/52 to 80/40 mmHg and the heart rate decreased from 148 to 102 per min.

The above changes coincided with the injection of air into an 18-G spinal needle that had been inserted into the proximal left femur through a direct lateral approach. A second 18-G needle was also placed distally to the first. In an effort to collect a fluid specimen from the cyst, the surgeons had aspirated one needle or the other. At one point, they injected air of unknown quantity into one needle in an attempt to aspirate fluid from the other. A total of 3 cc of serosanguinous fluid was finally aspirated from the cyst.

Venous air embolism from the left femur was suspected and the surgeons were so informed. Nitrous oxide and halothane were discontinued, and the patient was given atropine 0.2 mg iv. The Sp_{O_2} increased from 69 to 95%, then to 100% within 5 min. No additional air was injected by the surgeons. The ET_{CO_2} increased from 4 to 35 mmHg, the heart rate from 102 to 162 per min, and the blood pressure from 80/40 to 115/45 mmHg. After the patient regained hemodynamic stability, anesthesia was changed to isoflurane 0.5–1% in O_2, supplemented with alfentanil totalling 100 μg.

* Resident.
† Fellow.
‡ Professor and Chairman.
§ Professor.

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Address reprint requests to Dr. Lippmann.

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