Fatal paradoxical air embolism during liver transplantation

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We describe a case of fatal paradoxical coronary air embolism during liver transplantation. The literature on the diagnosis and prophylaxis of paradoxical air embolism during liver transplantation is reviewed and discussed.

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Air embolism associated with orthotopic liver transplantation is a well known phenomenon which occurs during the dissection phase while performing vascular anastomosis (inferior vena cava, portal vein) or during liver reperfusion (insufficient hepatic wash out, leaks in vascular anastomoses). A specific problem is the possibility of passage of air bubbles from the right heart to the systemic circulation. Such a paradoxical air embolism can result in air being embolized to the terminal arterial bed of the brain or heart with severe consequences. We present a case of fatal paradoxical air embolism with coronary ischaemia during orthotopic liver transplantation. As far as we are aware, there are no previous reports of paradoxical coronary air embolism during this procedure.

Case report

A 26-yr-old woman with sclerosing cholangitis was admitted for orthotopic liver transplantation. There was no clinical evidence of cardiopulmonary disease in her clinical history or on physical examination. Arterial blood-gas values, chest x-ray, ECG, spirometry and transthoracic echocardiogram were normal. Anaesthesia was maintained with isoflurane in an air–oxygen mixture, with fentanyl, diazepam and pancuronium. Operative monitoring included pulse oximetry, end-tidal carbon dioxide concentration, radial artery cannulation to monitor arterial pressure and a pulmonary artery catheter.

Hepatic dissection was difficult because of the large size of the liver. Unexpectedly, an accidental tear of the suprahepatic inferior vena cava occurred. We observed an increase in pulmonary artery pressures and a decrease in end-tidal carbon dioxide concentration, heart rate and arterial pressure. The tear was sutured and the haemodynamic state was stabilized using atropine 0.5 mg and epinephrine 0.1 mg. We were not using positive end-expiratory pressure (PEEP). Central venous pressure varied between 6 and 10 mm Hg depending on liver manipulation.

Dissection continued and a few minutes later a second tear of the inferior vena cava occurred, allowing air entrainment: end-tidal carbon dioxide concentration and arterial pressure decreased and pulmonary artery pressures increased. The QRS complex of the ECG widened and ventricular tachycardia and fibrillation ensued. Resuscitation was initiated with epinephrine, 100% oxygen, cardiac defibrillation and closed chest cardiac massage. Air and blood (10 ml) were aspirated from the pulmonary catheter. The surgical bed was filled with saline solution and the inferior vena cava was clamped. The patient did not respond to resuscitation. Ventricular fibrillation alternating with asystole was commenced. Approximately 10 min after the second air embolism, an arterial blood sample showed metabolic acidosis with normal potassium and ionized calcium concentrations. We gave sodium bicarbonate, an infusion of lidocaine and bretylium, norepinephrine, and then hypertervented the lungs. After 60 min there was no spontaneous cardiac activity and so we decided to stop the resuscitation manoeuvres.

An autopsy showed well formed atrial and ventricular cavities and valves with a patent foramen ovale about 2 cm in diameter, coronary arteries without significant stenosis and local myocardial changes compatible with a recent ischaemic event, mostly in the anterolateral wall of the left ventricle.

Discussion

Embolism during orthotopic liver transplantation has been described, and has often been identified using transoesophageal echocardiography. Ellis and colleagues reported 16 patients who underwent orthotopic liver transplantation and observed echogenic material in the right ventricle of all patients using transoesophageal echocardiography.1 More-
over, this material moved into the left heart during reperfusion in 2 cases.1 Usually there are no clinical repercussions; even when massive embolism occurs, patients have been resuscitated successfully. Starzl and colleagues reported 48 patients who underwent orthotopic liver transplantation and developed neurological complications. Arterial air embolism was confirmed at the postmortem examination in two patients;2 Neither of these series reported evidence of air in the coronary vessels.

After the second embolism, we were surprised by the sudden appearance of arrhythmias and by the ineffectiveness of treatment. We speculate that the second embolism was bigger than the first, although the surgeons informed us that the tear was smaller and that they had clamped the inferior vena cava quickly. It was difficult to quantify the volume of air because increased pulmonary artery pressures and decreased end-tidal carbon dioxide concentrations were affected by clamping the cava, which reduced venous return. We did not have a transesophageal echocardiogram, which is a sensitive indicator for measuring the volume of the embolism.

Metabolic alterations can precipitate arrhythmias. Blood samples were analysed 30 min before the first venous air embolism, and 10 and 60 min after the second embolism, and showed normal potassium, ionized calcium and pH values.

A pre-existing cardiac abnormality may have affected the response to the embolism, but we found no preoperative evidence of an abnormal feature in this patient (from clinical history, ECG, transthoracic echocardiogram or chest x-ray). There was no evidence of idiopathic ventricular tachycardia (hypertrophy, fibrosis and septal sclerosis) or coronary artery disease at post mortem. Coronary vasospasm has been reported in a young patient during liver transplantation, although it was related to the cold graft being placed close to the right coronary artery.3

Therefore, we conclude that a paradoxical air embolism caused coronary obstruction: as little as 0.05 cm³ of air can cause a focal infarct.4 Paradoxical air embolism occurs when the air in the venous system or in the right heart passes through some intracardiac channel and reaches the arterial system. It is important to remember that a persisting patent foramen ovale exists in 10–35% of the population, and that the patent foramen ovale is the most common route for paradoxical air embolism.5 Venous air embolism causes immediate and intense vasospasm in the pulmonary circulation leading to increased right atrial pressure with the potential for flow through a patent foramen ovale.6 A patent foramen ovale is difficult to detect with transthoracic echocardiography6 and may not be ruled out even by a contrast transthoracic echo study. A patent foramen ovale was found at postmortem examination in our patient.

Air may also reach the arterial system through pathological dilatation of intrapulmonary vessels normally present in some people (but more frequent in end-stage liver disease).7 Hopkins, Waggoner and Barzilar found that 47% of patients with chronic liver disease had right-to-left shunting, as determined by contrast echocardiography.8 Eight of 27 paradoxical coronary embolism cases reviewed by Jungbluth and colleagues9 were caused by air, but an abnormal intracardiac defect was found in only three. Five air emboli involved the left coronary artery, as in our case, and this is most likely related to its anterocranial position.9 In the case presented by Ahmat and colleagues, the patient had a coronary air embolism without any intracardiac shunt in the postmortem examination, confirming that patients with liver disease may have less filtering protection against paradoxical air embolism.10

An intracardiac and/or intrapulmonary shunt may be detected using contrast transesophageal echocardiography, but this carries risks in patients with oesophageal varices and does not exclude all those at risk of paradoxical air embolism.11 When a patent foramen ovale is confirmed in a patient scheduled for posterior cranial fossa surgery, this procedure is better performed with the patient prone rather than seated. However, it is impossible to change either the surgical technique or the patient’s position during orthotopic liver transplantation if a patent foramen ovale is discovered. Some authors suggest surgical correction of a patent foramen ovale before posterior fossa surgery12 but cardiac surgery carries a high risk for patients with end-stage liver disease, and pulmonary shunts may also persist. Transcatheter occlusion of the septal defect may be considered in some cases with a patent foramen ovale.13

References
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