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Paradoxical air embolism detected by transesophageal echocardiography during hepatic resection

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Introduction

Venous air embolism (VAE) during anesthesia is classically recognized to occur in patients undergoing intracranial procedures in the sitting position. Recent clinical reports suggest that VAE may also occur during hepatic resection performed without opening the inferior vena cava (IVC) in the supine position [1,2]. This clinical report demonstrates paradoxical air embolism diagnosed by transesophageal echo cardiography (TEE) during hepatic resection.

Case report

A 68-year-old man weighing 41 kg was scheduled for right anterior segmentectomy of the liver for hepatocellular carcinoma. He had a 20-year history of medically controlled hypertension and a 10-year history of chronic hepatitis. Preoperative examination revealed a decreased creatinine clearance of 53 ml·min^{-1} and restrictive pulmonary disorder (percent vital capacity of 64%). The latter change was secondary to a right upper lobectomy performed 35 years previously for pulmonary tuberculosis and subsequent pleural adhesion.

The patient received atropine (0.5 mg) and hydroxyzine (25 mg) preoperatively. Anesthesia was maintained with nitrous oxide (50%) and isoflurane (0.3%–2%) in oxygen. Muscle relaxation was achieved with vecuronium bromide, and the lungs were mechanically ventilated. Routine monitoring included electrocardiogram, pulse oximetry, partial pressure of end-expiratory carbon dioxide ($P_{ET}CO_2$), bladder temperature, and invasive arterial pressure via radial arterial cannula. Hepatic resection using an electrocautery was started after occluding the main portal vein and common hepatic artery (Pringle's maneuver) to reduce blood loss. Each vascular occlusion lasting 10 min was followed by a 3-min period of reperfusion by releasing the clamp. The resected surface of the liver was covered with a wet sponge during reperfusion.

Resection of the liver proceeded uneventfully until the end of the third vascular occlusion where there was a moderate decrease in arterial pressure from 100/56 mmHg to 78/50 mmHg with minimal blood loss of 100 g. Intravenous administration of ephedrine (8 mg) and release of the vascular clamp effectively restored blood pressure. However, immediately after the resection was resumed by occluding the hepatic vasculature, there was a sudden drop in arterial pressure to 42/24 mmHg with a concomitant decrease in heart rate to 55 bpm. These changes were associated with elevation of the ST segment on precordial ECG, suggesting coronary vasospasm. The surgical procedure was halted and treatment for coronary spasm was initiated with bolus administration of dopamine and nicardipine followed by nitroglycerine infusion. At the same time, nitrous oxide and isoflurane inhalation was discontinued and anesthesia was maintained with a small dose of midazolam and fentanyl thereafter. Although the ST changes were normalized in several minutes, hypotension (systolic blood pressure of 70 mmHg) persisted despite treatment with a dopamine and dobutamine infusion. Central venous pressure at this time was 10 mmHg and there was no sign of active bleeding. To evaluate left ventricular function and to exclude venous air embolism, transesophageal

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echocardiography (TEE) was performed. There were no abnormalities of left ventricular wall motion. However, a large number of air bubbles were clearly noticeable in both the right and left side of the heart, indicating paradoxical air embolism (Fig. 1).

Volume resuscitation of 1.5 l was performed in the next half hour to increase venous pressure, and resection was then resumed. Immediately after clamping the hepatic vasculature, a sudden drop in arterial pressure with ST elevation occurred again. At the same time, a shower of air bubbles entering the ventricles was noticed on TEE (Fig. 2). This was followed by ventricular fibrillation and cardiac standstill. Closed chest cardiac massage, epinephrine (0.5 mg i.v.), electrocardioversion, and lidocaine infusion successfully converted the rhythm to the normal sinus pattern. Blood pressure was maintained with a norepinephrine infusion (0.2 mcg·kg^{-1}·min^{-1}) thereafter. After insertion of a pulmonary arterial catheter, an additional 1.5 l of infusion was given in the next 1 h for the total blood loss of 500 g while monitoring pulmonary arterial wedge pressure (PCWP). Initial readings of pulmonary arterial pressure (PAP) and PCWP were 27/11 (17) mmHg and 10 mmHg, respectively. Despite aggressive volume chal-

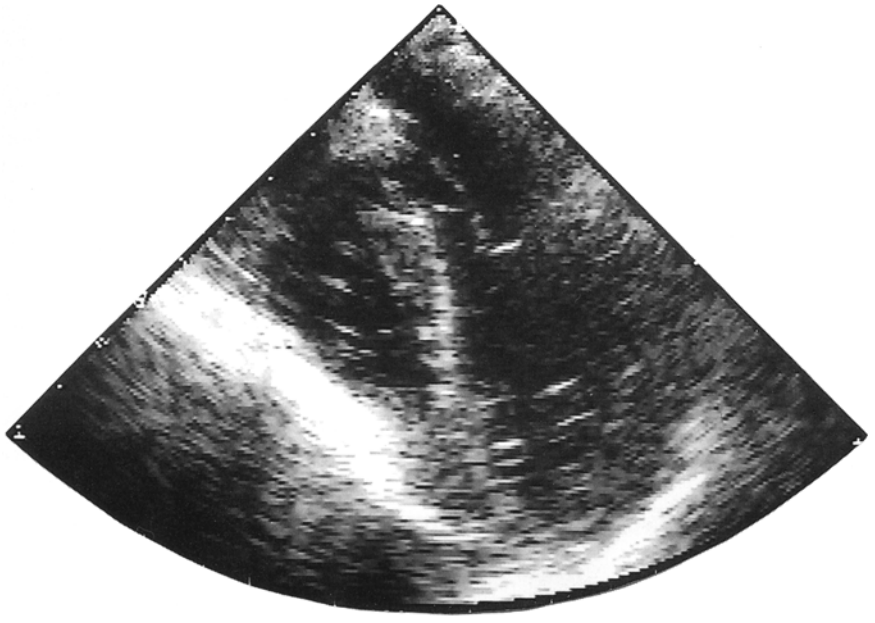

Fig. 1. Four-chamber view of transesophageal echocardiography demonstrating high-echoic, fine particles in both ventricles

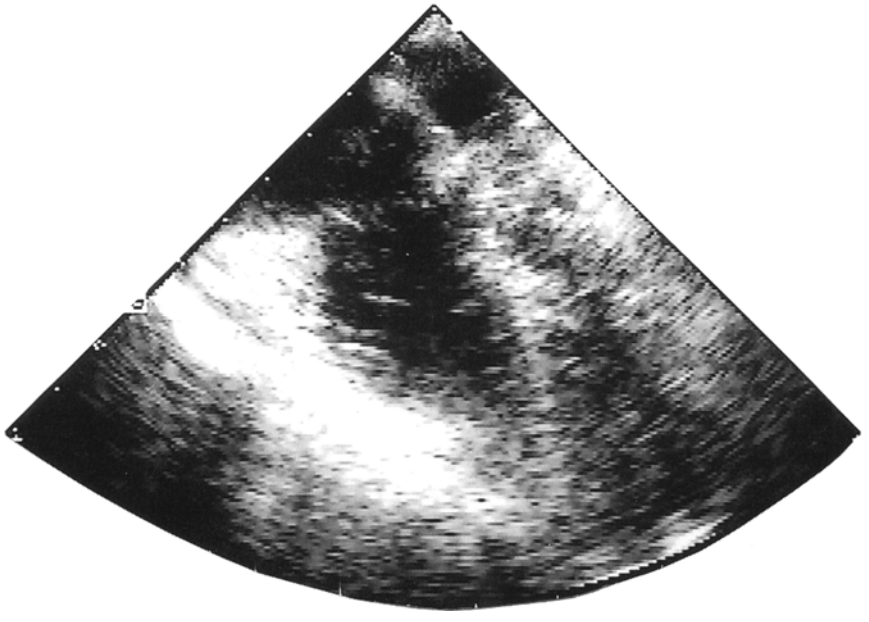

Fig. 2. Four-chamber view of transesophageal echocardiography as in Fig. 1. Shower of air bubbles is prominent in left ventricle

lenge and administration of catecholamines, the systolic blood pressure and heart rate stabilized to 60–70 mmHg and 100 bpm, respectively. The decision was made here to complete hepatic resection without occluding the hepatic blood supply to avoid air entrainment from the resected surface of the liver. The subsequent surgical procedure and anesthesia were accomplished without any difficulty. Body temperature at the end of surgery was 33.2°C. Overall blood loss amounted to 1000 g, and urine volume was 200 ml. The total volume of infusion amounted to 6500 ml, including 1000 ml of colloid solution.

Postoperatively, the patient was transferred to the intensive care unit, and mechanical ventilation was continued. Blood pressure gradually recovered while the body temperature was being increased with a warming blanket. As a consequence, administration of inotropes was able to be slowly tapered. The patient regained consciousness approximately 5 h after the end of anesthesia without any neurological deficit. His postoperative course was uneventful except for bilateral pleural effusion which did not require specific therapy, and the patient was transferred to the ward 4 days after the operation.

Discussion

Development of venous air embolism during hepatic surgery is generally preceded by an accidental injury of the IVC or hepatic veins. Under such circumstances, hypovolemia secondary to massive bleeding precipitates the entry of a large volume of air into the venous circulation via the injured great vessels. However, in the present case, an abrupt drop in blood pressure occurred in the absence of great vessel damage or bleeding. Hatano et al. [2] reported a similar case of VAE during hepatectomy where they suggested that air can be drawn inward via small hepatic veins open to the atmosphere by the Venturi effect. During hepatectomy, the IVC is invariably compressed and, as a result, the venous pressure of the constricted portion of the IVC can become subatmospheric if blood flow through the narrowed portion is high [2]. In the present case, the hepatic vascular supply was interrupted by Pringle's maneuver while the hepatic vein was not clamped. In such a situation, since there is no blood inflow, hepatic venous pressure is lower than the pressure without Pringle's maneuver and, consequently, a larger volume of air is entrained. The fact that we did not note further hemodynamic alterations or air entrainment during the resection without Pringle's maneuver indicates that lowered hepatic venous pressure was the factor most responsible for the development of VAE.

Various diagnostic tools have been applied to anesthetic management in cases where the possibility of VAE is considered to be high, for purposes of quick detection and accurate treatment. These monitors include a Doppler device, $P_{ET}CO_2$, and pulmonary arterial pressure. Although a correctly positioned precordial Doppler device has been shown to be the most sensitive method for detecting embolic air [3], it is not useful during cases when an electrocautery is used because of radio frequency interference [4]. Furthermore, stable attachment of the Doppler probe to the chest is technically difficult during abdominal surgery. A decrease in $P_{ET}CO_2$ is widely regarded as of the early signs of VAE [5]. However, an abrupt decrease in venous return or cardiac output may also affect $P_{ET}CO_2$. Occlusion of the hepatic vasculature invariably decreases venous return from the splanchnic vascular bed and consequently decreases cardiac output and $P_{ET}CO_2$. In the present case, blood pressure decreased by 15–20 mmHg and $P_{ET}CO_2$ decreased from 38–39 mmHg to 34 mmHg during vascular occlusion. After the release of occlusion, these two variables returned to the preocclusion level with in 2–3 min. Furthermore, as noticed in our patient during the catastrophic episode, it is hard to interpret whether decrease in $P_{ET}CO_2$ is the cause or the result of cardiac event.

Continuous monitoring of pulmonary arterial pressure (PAP) is recommended for both detection and treatment of VAE [6,7]. An increase in PAP precedes the decrease in systemic arterial pressure during VAE. Experimentaly, as little as 0.25–0.5 $ml \cdot kg^{-1}$ of air can be detected by the changes in PAP [6]. Compared to PAP measurement, TEE provides several advantages for the diagnosis of VAE. It is less invasive and placement can be quickly and easily performed. Furthermore, TEE offers quantitative information of the biventricular functions as well as visual confirmation of air entry. Concerning the sensitivity for detection of air bubbles, Furuya et al. [8] have shown that, in experimental air embolism, the threshold volume of air was 0.02 $ml \cdot kg^{-1}$ for TEE compared to 0.05 $ml \cdot kg^{-1}$ for the Doppler device. In our case TEE provided us not only a differential diagnosis of VAE and coronary vasospasm but also the presence of paradoxical air embolism. The latter information cannot be obtained by any other diagnostic tools.

In the present case, a shower of air bubbles was clearly identified in both right and left side of the heart, suggesting the presence of right to left shunt. Simultaneous elevation of ST segment on precordial ECG indicates the occurrence of cororary air embolism. Although we could not detect any anatomical intracardiac shunt postoperatively by Doppler echo cardiography, a functionally patent foramen ovale might have been present intraoperatively. Increased right atrial

pressure secondary to massive air retention in the right ventricle could have pushed air bubbles into the left atrium via the foramen ovale, which is reported to be patent in 20%–30% of normal adults [9]. Conversely, air bubbles might have passed through the pulmonary circulation via intrapulmonary shunt, which would have been increased by alteration of the pulmonary vasculature secondary to tuberculosis or chronic hepatitis [10,11] and by the vasodilating action of isoflurane. Furthermore, by using TEE, Vik et al. [12] have confirmed in anesthetized pigs that the threshold value for the breakthrough of air bubbles through the pulmonary circulation was related to the infusion rate of air into the right ventricle. They also noted that breakthrough was prominent in animals with a dramatic decrease in systemic blood pressure. It is reasonable to assume that similar phenomenon may have occurred in the present case.

In conclusion, we believe that TEE should always be applied in anesthetic management where the possibility of VAE is high. We also emphasize that TEE should be performed immediately whenever unexpected circulatory change is encountered intraoperatively.

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