Massive Paradoxical Air Embolism After Orthotopic Liver Transplantation

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Citation

Abstract
Systemic venous air embolism is a serious complication in patients with chronic liver disease. Intrapulmonary arteriovenous shunting can permit air emboli to pass into the systemic circulation. We report the case of a patient who suffered a paradoxical air embolism after successful liver transplantation as a result of the separation of the two component parts of a pulmonary artery catheter introducer.

INTRODUCTION
Paradoxical systemic air embolism (PAE) is defined as an arterial air embolus originating from a venous source passing through an intracardiac or intrapulmonary right-to-left shunt. Air embolism is a rare complication of invasive monitoring of the right heart and of central venous cannulation. However it is particularly dangerous in patient with cirrhosis, because 15–45% of these patients have pulmonary abnormalities including intrapulmonary shunting caused by pulmonary vascular dilatation and arteriovenous communication. In these patients, paradoxical emboli can occur during air embolism even if intracardiac abnormalities are not present. We report a case of paradoxical air embolism that occurred 2 days after successful liver transplantation.

CASE REPORT
A male patient 44 years old weighing 63 kg was admitted to the SICU after a liver transplantation. His perioperative course in the intensive care unit was uneventful. 2 days after operation the pulmonary artery catheter (PAC OPTIQ SVO2/CCO Abbott Laboratories, North Chicago, IL60064) was removed, and its sheath was left in place. After 3 hours from catheter removal; the patient started to complain from severe chest pain, with abrupt decrease of SpO2 from 100% to 70 %, after few seconds systolic arterial blood pressure decreases from 120 mm Hg to 70 mm Hg, and heart rate fell from 80 beats/min to 50 beats/min. The patient was immediately intubated with an 8.0 mm I.D. endotracheal tube, and ventilated with 100% oxygen. During intubation the attendant physician noticed that the hemostatic valve of pulmonary artery catheter sheath was leaking, which was immediately removed. Pressure was applied to the insertion site. Blood gases were done and revealed that PaO2 was 100 mm Hg with FIO2 1.0. Noradrenaline was started on 0.2 ug/kg/min and his blood pressure became 120/60. ECG was done and was normal. Transthoracic Echocardiography was done and revealed massive air bubbles in right, left sided chambers, and aorta. Figure (1,2)

Figure 1
Figure 1: Apical four chamber view with air shadows in both right and left ventricle
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**DISCUSSION**

Numerous case reports of venous air embolism (VAE) related to central venous cannulation exist in the medical and surgical literature, and several reviews of the pathophysiology and management of air embolism have been published. This particular venous air embolism occurred when the two component parts of a pulmonary artery catheter introducer became separated. The driving pressure for air entrainment is the gradient between atmospheric and intrathoracic vascular pressure. A negative intrathoracic venous pressure is more likely to occur with deep inspiration, hypovolemia, and in the upright position.

PAE occurs when the air in the venous system or in the right heart passes through an intracardiac channel and reaches the systemic circulation. A persistent patent foramen ovale exists in 10–35% of the population and is the most common route for PAE. However, air may also reach the systemic circulation through pathological dilatation of intrapulmonary vessels normally present in some people (but more frequent in end-stage liver disease). In the normal individual, pulmonary parenchymal precapillary and capillary blood vessels have a diameter of 8–15 mm. However, in patients with chronic liver diseases, intrapulmonary shunting can occur by intrapulmonary vascular dilatation (up to 160 mm) at the precapillary level, or by direct arteriovenous communications. In this case, No right-to-left intracardiac shunt could be detected by colour flow echocardiography. These findings suggest that the source of PAE was from dilated pulmonary vasculature and not from an undiagnosed intracardiac shunt.

The entry of gas into the aorta causes the distribution of gas bubbles into nearly all organs. Small emboli in the vessels of the skeletal muscles or viscera are well tolerated, but embolization to the cerebral or coronary circulation may result in severe morbidity or death. In our case there was no evidence of myocardial ischemia, however neuralgic deficit persist until patient death. CT scan was not done in our case to confirm the diagnosis due to hemodynamic instability. However, pathologic changes are sometimes very subtle and not well visualized on CT. The patient developed hemoconcentration with rising hematocrit from 24% to 31% which may be direct consequence of the extravascular shift of fluid into the injured tissues.

The primary goal of treatment is the protection and maintenance of vital functions. If necessary, cardiopulmonary resuscitation should be performed, since not only venous but also primary arterial gas embolism may
lead to serious impairment of the cardiovascular system. Oxygen should also be administered; at as high a concentration as possible. Administration of oxygen is important not only to treat hypoxia and hypoxemia but also to eliminate the gas in the bubbles by establishing a diffusion gradient that favors the egress of gas from the bubbles. Hyperbaric oxygen is the first-line treatment of choice for arterial gas embolism, however we could not initiate such therapy as it was not available in our center, and there was a problem involved in the long distance transport of our patient to a specialized treatment center. We started lidocaine therapy to provide cerebral protection. Although the results of clinical studies of lidocaine for the treatment of arterial gas embolism are not yet available, studies in animals suggest that lidocaine may be beneficial.

The patient developed a clinical picture consistent with ARDS. This is a known complication of VAE. The shock state caused by acute right ventricular outflow tract obstruction and obstruction of the pulmonary arterioles by the air microemboli may cause activation of polymorphonuclear leukocytes, liberation of chemical mediators, and capillary injury, leading to ARDS.

In conclusion when two-pieces pulmonary artery catheter introducer are used it is essential that connection between the individual components is securely tightened.

Figure 3
Figure 3: P-A chest X-ray shows bilateral lung infiltrate.

References
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