Paradoxical Air Embolism in the Absence of an Intracardiac Defect*

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A 58-year-old man experienced paradoxical air embolism with passage of air from the systemic venous to the systemic arterial circulation with subsequent stroke and death. No intracardiac shunt was present. Pulmonary fibrosis concomitant with severe pulmonary arterial hypertension appears to have been responsible for the air traversing the pulmonary capillary bed. This unusual outcome of a complicated central venous catheterization must be borne in mind and guarded against in similar patients.

(CHEST 1991; 99:754-55)

The systemic and pulmonary venous and arterial systems are being accessed increasingly for a myriad of invasive diagnostic and therapeutic procedures. As a result, unusual forms of iatrogenic morbidity and mortality present themselves. Such a case in which a "routine" central venous catheterization was complicated by air embolism, subsequent paradoxical embolism with resultant stroke, and death is described herein. The paradoxical embolism occurred in the absence of an intracardiac shunt, and the possible mechanisms of this are discussed.

Case Report

A 58-year-old man was admitted to the hospital with idiopathic pulmonary fibrosis. History included dermatomyositis and hepatitis in the distant past. The patient was initially treated with prednisone and azathioprine (Imuran) with a substantial improvement, but during the last six to seven months, he had worsening shortness of breath. The patient was found to have tricuspid regurgitation, rightsided heart strain, and pulmonary hypertension. He was scheduled for a three-day serial drug testing program. Invasive monitoring was accomplished using an intra-arterial catheter as well as a pulmonary arterial thermocatheter (Swan-Ganz). Pulmonary artery pressure was 85/40 mm Hg (with mean of 60 mm Hg), pulmonary capillary wedge pressure was 3 mm Hg, and cardiac output 5.8/min. Pulmonary vascular resistance was 877 dynes*cm⁻².

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before treatment with diltiazem and 493 dynes*cm⁻² after.

The Swan-Ganz catheter was removed at the end of the study. The obturator was not returned to the sheath which was still in place. The patient was sitting and a large "sucking" sound was heard, and within ten minutes the patient developed left hemiparesis. An emergency echocardiogram revealed air within all cardiac chambers, especially the right ventricle (Fig 1). An intracardiac right-to-left shunt with paradoxic embolism to the cerebral arterial circulation was suspected. However, this intracardiac defect could not be documented echocardiographically. The patient was too unstable for transfer to a facility with a hyperbaric chamber. The patient manifested a large right middle cerebral artery distribution stroke and suffered severe hypoxic encephalopathy. Complications thereafter included sepsis and disseminated intravascular coagulopathy. The patient died of a cardiac arrest four days after the catheterization.

At autopsy a large recent right cerebral hemorrhagic infarct was found, with subtentorial herniation and changes of severe anoxic-ischemic encephalopathy. The cerebral vasculature was unremarkable, and the findings from the neuropathologic examination were compatible with air embolism. The lungs had extensive parenchymal fibrosis especially of the lower lobes and basal segments of the upper lobes, consistent with the effect of dermatomyositis. There were changes of marked pulmonary artery hypertension with medial hypertrophy and intimal hyperplasia of smaller pulmonary arteries, marked dilatation of large pulmonary arteries, marked right-sided heart dilatation and hypertrophy, and changes of marked chronic passive venous congestion of visera. The terminal event was a severe aspiration bronchopneumonia probably precipitating a cardiorespiratory arrest. Careful examination of the great vessels of the heart and lungs, of the pulmonary parenchyma, and of the heart revealed no pulmonary arterial-systemic arterial defect. The puncture site in the jugular vein, through which catheterization took place, was identified and was locally uncomplicated.

Discussion

Air embolism is most often venous, but may be arterial and encountered in the settings of head and neck trauma and surgery, obstetric and gynecologic procedures, pneumothorax, decompression sickness, positive pressure ventilation, and a number of cardiothoracic procedures such as needle biopsy and open cardiac surgery. Venous air embolism is also a hazard of the use of central venous catheter systems. The upright position and cyclical negative pressure within the catheters can result in embolization of air into the arterial system, with fatal consequences.
pressures produced within the thorax during respiration facilitate the rapid entry of air through any puncture defect in the central veins or through any defect in the infusion systems placed in these veins. Such venous emboli, if rapid and/or massive in total volume, cause functional obstruction of the pulmonary capillary bed, pulmonary arteries, and the right ventricular outflow tract. The pulmonary circuit is normally capable of filtering smaller volumes without untoward effect or with reversible changes such as pulmonary vasoconstriction and edema. In the systemic arterial system, air emboli impact in end-arteries and cause infarcts. Venous air or other emboli may cause such infarcts if they become paradoxic emboli, that is, are allowed passage by some means from the systemic venous/right heart system to the left heart/systemic arterial system. Such passage is virtually always considered to be via an intracardiac defect, congenital or acquired, and most often via a patent foramen ovale.

Careful examination in the patient described revealed no evidence of such an intracardiac defect or other shunt. Cases of paradoxic air embolism without a shunt are exceedingly rare, and such a complication after diagnostic catheterization has not been previously reported (to our knowledge). Two possible means by which air might reach the systemic arterial circulation suggest themselves. Air may pass across the pulmonary capillary bed or air may pass through pulmonary arteriovenous anastomoses in the lung which are not grossly or microscopically appreciable using standard methods.

There has been controversy regarding the existence of such physiologic anastomoses in normal lungs. Earlier studies performed, for instance, embolizing glass spheres into the pulmonary artery and later recovering them in pulmonary veins, were used as evidence that anastomoses existed. However, morphologic methods and other studies have failed to support their existence. It has been suggested that recovery of glass spheres in venous blood may have been artifactual due to overdilution of the pulmonary capillary bed by the high perfusion pressures needed in the experiments or due to operative contamination.

Interestingly, pulmonary arteriovenous anastomoses of varying sizes have been demonstrated in relation to syndromes of congenital arteriovenous malformations and may occur in some forms of liver abnormalities. These anastomoses have not been demonstrated roentgenographically or pathologically in patients with pulmonary fibrosis, although bronchopulmonary anastomoses have. This latter circuit, pulmonary artery to bronchial vein to pulmonary vein, is hypothetically an alternate route for emboli. Its potential in this patient was perhaps increased. These channels would have been dilated with the severe pulmonary artery hypertension this patient manifested.

This patient's paradoxic air embolism, however, may have been more simply accomplished by transpulmonary passage of the air over the pulmonary capillary bed. This is known to be possible and experimentally conditions that favor this transmission are those that produce pulmonary artery dilatation, including oxygen toxicity, the administration of vasodilators, and volume overload.

The pulmonary arterial hypertension in the patient described herein likely facilitated or potentiated passage of air, pulmonary artery to vein, whether it was via the pulmonary capillary bed or by anastomotic circuits. Patients similar to ours may be at increased risk for paradoxic air embolism if venous embolization occurs. That such catastrophic outcome may complicate a routine venous catheterization must be borne in mind, even in those patients without obvious intracardiac or other shunts, and not otherwise thought of as at risk.

**Acknowledgments:** The authors acknowledge the assistance of Dr. J. M. Kay, Professor of Pathology, McMaster University, Hamilton, Ontario, Canada, for his help with the discussion. The authors thank Ms. J. Vaile for her help preparing the manuscript, and Ms. M. Campagna for her help with the pathologic examination.

**References**