Barotrauma during Anesthesia for Cardiac Catheterization


Barotrauma is a well-recognized complication of positive-pressure ventilation. The occurrence of air embolism as a complication is less frequent; however, it has been reported in neonates, traumatized patients, patients with adult respiratory distress syndrome (ARDS), and patients with status asthmatics. It also is a well-recognized problem in deep sea divers.

We present a case of pulmonary venous air embolism that occurred during general anesthesia and that was observed by cineangiography.

Case Report

The patient was an 8-year-old girl with Osler-Weber-Rendu syndrome who complained of shortness of breath. She had been well otherwise and had no history of respiratory disease. Physical examination revealed telangiectasias on her arms, chest, and face; mild clubbing of the extremities; and a 1/6 systolic murmur in her midthoracic region posteriorly. Her hemoglobin oxygen saturation while breathing room air was 77%. Initial cardiac catheterization during general anesthesia was uneventful. The heart was normal, but a large arteriovenous malformation (AVM) was present in the superior segment of her right lower lobe. The patient underwent thoracotomy and right lower lobectomy with removal of the lesion. Seven months later she underwent a second cardiac catheterization during general anesthesia. The procedure was uneventful, and multiple embolization coils were inserted into other small pulmonary AVMs. The shunt fraction was significantly reduced, and the patient was discharged home.

Three weeks later the patient returned for additional coil embolization of remaining pulmonary AVMs because of persistent dyspnea and cyanosis. General anesthesia was induced with midazolam 0.5 mg·kg⁻¹, ketamine 1.5 mg·kg⁻¹, sufentanil 20 μg·kg⁻¹, and pancuronium 0.1 mg·kg⁻¹. Her lungs were mechanically ventilated, and anesthesia was maintained with isoflurane in oxygen during the 4.5-h procedure. Multiple embolization coils were inserted in her right lung.

During angiography of one lung segment the cardiologist requested a sustained positive-pressure breath, which was administered manually. Immediately, a large amount of air was observed on the video screen to be entering the pulmonary venous and arterial systems and to reach the left atrium. Hypotension with severe bradycardia suddenly developed but responded to a crystalloid bolus of 12 ml·kg⁻¹ and atropine 0.01 mg·kg⁻¹. The end-tidal carbon dioxide tension was noted to have decreased. End-tidal nitrogen tension was not monitored. At the end of the procedure the patient was found to have a small pneumopericardium, but her condition was clinically stable. Her pupils remained small and reactive. She was discharged home with improvement in her dyspneic symptoms.

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Fig. 1. Air bubbles in the pulmonary vein moved centrally to fill the left atrium (arrow).
During the review of this case a previously unrecognized pulmonary venous air embolism was found to have occurred during an earlier cardiac catheterization. The air was seen entering from an AVM during injection of contrast simultaneously with administration of a positive-pressure breath. During this event the peak inspiratory pressure was not more than 30 cmH2O. There was no air in the central venous catheter, and air was not introduced with the contrast. The volume of air embolized was small and therefore had been overlooked at the time of injection.

Discussion

This case illustrates a serious complication of positive-pressure ventilation that has been accepted as occurring but that has never, to our knowledge, been witnessed. The possible reasons why air embolism occurred in this case should be considered.

Figure 1 shows multiple sites of air entry in the right middle lobe and air tracking centrally via the venous system to the left atrium. A first possible cause that must be excluded is that of air injected with the contrast. Three independent radiologists and one cardiologist, each experienced in angiography, reviewed the films. Nowhere was air seen in the catheter or in the contrast entering from the catheter. The application of a sustained positive-pressure breath with subsequent barotrauma is another possibility. This phenomenon has been widely reported in patients with ARDS in whom systemic venous gas embolism developed while their lungs were being mechanically ventilated. The presumed route of air escape is from the alveolar wall into the capillary, as was first described by Macklin and Macklin. Manni and Fulver, in a description of barotrauma in ARDS, discussed various contributing factors such as damaged lung parenchyma, which tethers open the blood vessels and makes them more susceptible to air leaks from the alveoli. This mechanism of venous air embolism was described by Gregory and Tooley in neonates with lung disease.

Our patient also sustained a small air embolus during the first postlobectomy catheterization. This embolus entered from an AVM in the left lower lung, where there were no coils, during a wedge injection (Fig. 2); there is clear radiologic evidence that it did not come from the contrast or catheter. The airway pressures were again monitored and were recorded to be less than 30 cmH2O, an airway pressure we have always assumed to be safe. A sustained positive-pressure breath of 30–40 cmH2O is the mechanism Benumof advocates for use in checking for an anastomotic leak after lobectomy. Many patients undergoing thoracotomy have longstanding respiratory disease with abnormal lung parenchyma, which theoretically may predispose them to pulmonary venous air embolism at lesser airway pressures.

AVM also must be considered as a contributing factor. Patients with AVMs are not known to be at higher risk for barotrauma. Structural abnormalities that might lead to pulmonary venous air embolism during positive-pressure ventilation have not been described in the pathology literature concerning pulmonary AVMs.

A recent review of barotrauma examined optimum modes of ventilation in patients with ARDS and suggested that peak pressures of 35–40 cmH2O place the alveolus at risk for overdistension. In ARDS, in which there is diffuse lung injury with patches of more normal lung, there are regional differences in lung compliance. There also may be regional differences in lung compliance in a patient with multiple pulmonary AVMs, multiple coils, a history of hemoptysis, or previous lobectomy. Significant abnormalities of pulmonary vasculature also may cause alveolar distention and, intuitively, would increase the risk of barotrauma, although this phenomenon has not been described in association with Osler Weber Rendu Syndrome.

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Another factor that may have contributed to the trauma was the use of embolization coils. We are unaware of specific warnings related to the ventilation of patients with these devices in situ. Newly placed coils might puncture a vessel wall and create a small bronchovenous connection; however, as stated earlier, this was not the case with this patient, nor has it been reported.

This case emphasizes that vigilance should be maintained during the ventilation of lungs of patients with underlying lung abnormalities, especially in cases in which there may be regional differences in lung compliance or in cases in which application of a sustained positive-pressure breath is required.

References


ERRATA

The Clinical Investigation by Voci et al. published in the November 1993 issue (Voci P, Bilotta F, Caretta Q, Chiarotti F, Mercanti C, Marino B: Mechanisms of incomplete cardioplegia distribution during coronary artery surgery: An intraoperative transesophageal contrast echocardiography study. ANESTHESIOLOGY 79:904-912, 1993) contained an error. On page 905, second paragraph (Anesthetic Technique), the fourth sentence should read, "Anesthesia was induced with fentanyl (30-35 μg (i.e., gamma)/kg), diazepam (0.25-0.5 mg/kg), and succinylcholine (1.5 mg/kg) and was maintained with fentanyl, droperidol, pancuronium bromide, and a 50% N2O in oxygen mixture."

The Laboratory Investigation by Harkin et al. published in the July 1994 issue (Harkin CP, Pagel PS, Kersten JR, Hettrick DA, Wartitter DC: Direct negative inotropic and lusitropic effects of sevoflurane. ANESTHESIOLOGY 81:156-167, 1994) contained an error. On page 162, table 4, the end-diastolic segment length (EDL) at 1.75 MAC sevoflurane was inadvertently omitted. This value is 15.5 ± 2.5 mm.