Venous Air Embolism, A Possible Cause of Acute Pulmonary Edema

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Venous air embolism is known to cause acute pulmonary edema in animal experiments, yet it has never been directly implicated clinically. In this case report we present a patient who developed acute pulmonary edema following documented air embolism during surgical removal of a tumor in the right cerebellar-pontine angle in the sitting position.

REPORT OF A CASE

A 13-year old girl was admitted with progressive hearing loss in the right ear, dizziness, and unsteady gait. Four years earlier, she had had a frontal craniotomy and a meningioma had been removed, leaving her with a slight right hemiparesis. On the present admission, a mass in the right posterior fossa was diagnosed. A right ventriculo-peritoneal shunt was inserted two weeks prior to exploration of the posterior fossa.

On examination, the patient was alert, with mild right hemiparesis, hemoglobin 13.7 g/100 ml, hematocrit 42.3 per cent, blood pressure 120/80 torr, pulse rate 110/min, weight 44 kg, height 150 cm.

The patient was premedicated with atropine, 0.6 mg and pentazocine, 60 mg, and anesthesia was induced with thiopental, 175 mg, followed by pancuronium, 6 mg. The trachea was intubated with an 8-mmuffed armored tube. Anesthesia was maintained with halothane, 0.5 per cent, and N₂O-O₂ 50:50, and the patient mechanically ventilated.

The left radial artery was cannulated, a central venous pressure catheter was inserted through the right antecubital vein, and the position of the catheter tip was checked by x-ray. A Doppler transducer was placed on the right side of the sternum where the maximum flow signal was heard, and its correct position verified by rapid injection of 5 ml physiologic saline solution through the central venous pressure line. After positioning the patient, a positive end-expiratory pressure (PEEP) of 5 cm H₂O was instituted.

Three hours from the start of the operation, after exposure of a highly vascular meningioma, a rush of air in the right heart was detected by the Doppler transducer. Within 20 seconds this was followed by a sudden drop of blood pressure to 40 torr. The EKG showed many premature ventricular contractions, and a continuous murmur was heard in the heart through the esophageal stethoscope.

The diagnosis of air embolism was made, positive end-expiratory pressure was increased to 10 cm H₂O, and the patient was ventilated with 100 per cent oxygen. No air could be removed through the central venous pressure catheter. Methoxamine, 2 mg, was given iv, after which the blood pressure recovered to 100 torr systolic. Arterial blood-gas values obtained shortly thereafter were pH 7.63, P_o₂ 23 torr, and P_CO₂ 253 torr at F_iO₂ 1.0. Later, attempts to decrease PEEP to the original 5 cm H₂O were followed by a flow of air in the heart detected by the Doppler transducer. Therefore, PEEP was kept at 10 cm H₂O for the rest of the operation.

Central venous pressure stayed stable at 7–10 cm H₂O throughout the procedure. The estimated blood loss was 1,300 ml, which was replaced, and a total of 1,700 ml crystalloids (1,500 ml dextrose-Ringer’s lactate solution; 200 ml Ringer’s lactate solution) was given. Neither mannitol nor furosemide was administered intraoperatively.

At the end of the procedure, muscle relaxants were reversed. The patient became responsive and began to breathe spontaneously through the endotracheal tube. However, shortly thereafter, she showed signs of respiratory distress. Respiration became labored and pink froth was coming through the endotracheal tube. Diffuse moist rales were heard in both lungs. Arterial blood-gas values of blood obtained in the operating room at F_iO₂ 1.0 were pH 7.29, P_o₂ 30 torr, and P_CO₂ 33 torr. Controlled respiration was reinstated and the patient was transferred to the intensive care unit. Chest x-ray showed massive bilateral pulmonary edema. There was no clinical sign of increased intracranial pressure, such as bradycardia, papilledema, or hypertension. Primary cardiac failure was excluded by low central venous pressure, normal size of the heart, and the absence of tachycardia.

In the intensive care unit P_aO₂ was maintained between 24 and 28 torr. An end-expiratory pressure of 7–10 cm H₂O was used at F_iO₂ 1.0. The patient was given furosemide, 20 mg, iv, and fluids were re-
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stricted to 40 ml/hr in addition to hourly replacement of urinary output.
Over the next 12 hours, the patient became fully conscious and her pulmonary compliance was markedly improved, as evidenced by a decrease in the peak inspiratory pressure necessary to maintain adequate alveolar ventilation from 40 to 24 cm H₂O.
At F₁₅₀, 0.5 Pa₅₀ was 90 torr.

On the second postoperative day, the trachea was extubated, but the patient's condition rapidly deteriorated again. The trachea was reintubated and mechanical ventilation immediately resumed. Effective tracheostomy was performed on the fifth postoperative day; the patient was weaned during the following few days. The tracheostomy closed and the patient was discharged from the hospital in the third postoperative week.

Six months later, the patient was again seen, because of recurrence of the tumor, and was scheduled for re-exploration of the right cerebellar pontine angle. The anesthetic management and monitoring were exactly the same as in the previous procedure, except that positive end-expiratory pressure was kept at 10 cm H₂O throughout the operation. The amount of blood replaced was 1,100 ml; crystalloids, 3,000 ml.

The vascular tumor was successfully removed. No air embolism was detected during the second operation, at the end of which the trachea was extubated without pulmonary complication.

DISCUSSION

Venous air embolism is a known hazard during surgical procedures on the posterior fossa done with the patient in the sitting position. Its cardio-vascular sequelae (hypotension, arterial hypoxemia, and reduction in end-expiratory CO₂) are thought to be due to mechanical obstruction of the right ventricular outflow tract by air. Experimental air infusion in dogs produced similar symptoms and consistent increases in pulmonary arterial pressure. Clinical application of the latter observation proved to provide a sensitive early method of detecting venous air embolism.

Animal experiments also showed that injection of lethal or near-lethal doses of air caused variable pulmonary edema. This was associated with severe arterial hypoxia due to: 1) overperfusion of normally ventilated alveoli; 2) anatomic shunting through the pulmonary arteriovenous communications and through bronchial and pulmonary veins. This transient hypoxemia may cause endothelial damage, changing capillary permeability. Also, the increased pulmonary arterial pressure and the reflex venospasm, together with the overperfusion of the unobstructed pulmonary vascular bed, lead to increased pulmonary capillary pressure and development of pulmonary edema.

Although pulmonary edema was demonstrated to be a possible complication of venous air embolism in animal experiments, the incidence of such in man has been attributed to the concomitant neurologic condition of the patient.

This neurogenic pulmonary edema has always been described in association with CNS conditions invariably associated with increases in intracranial pressure leading to intense sympathetic stimulation, increases in blood pressure, systemic vasodilatation, and pulmonary venous hypertension. These, in turn, cause acute left ventricular failure and pulmonary edema, which has also been shown in experimental animals.

Chandler et al. reported the occurrence of acute pulmonary edema in a neurosurgical procedure using local anesthesia with the patient in the sitting position, possibly due to air shown by fluoroscopy to be streaming in the dural sinuses toward the base of the skull.

Tateishi described the development of bilateral homogeneous opacities in lung fields in the immediate postoperative period following exploration of the posterior fossa. Although a total of 1.003 ml of air was aspirated via a central venous pressure catheter during operation, he did not correlate the postoperative pulmonary changes with the presence of air.

Gordon reported arterial hypoxemia and intra- and postoperative pulmonary complications in four of eight patients operated upon in the sitting position. The surgical manipulation was ruled out as a possible cause of pulmonary lesions. Although Gordon thought the complications might be due to air embolism, he did not use any special means for detecting air embolism during operation.

In our case, air embolism was definitely demonstrated, not only by clinical signs, but by the precordial Doppler transducer, although we could not remove air through the central venous pressure catheter.

There was no evidence of increased intracranial pressure after the operation, and the patient had no neurologic deficits when the
signs of pulmonary edema were noticed. Also, the patient did not have any pulmonary complication during or after a similar neurosurgical procedure performed a few months later, in which no air emboli in the heart were detected. As we pointed out, the only change in our anesthetic technique for the second operation was the continuous use of relatively high PEEP (10 cm H2O), which might have played an important role in prevention of air embolism.

In our opinion, there is evidence that acute pulmonary edema following neurosurgical procedures done with the patients in the sitting position could be caused not only by increased intracranial pressure, but also by venous air embolism.

REFERENCES


