Acute pulmonary edema after low-level air embolism during craniotomy

Case report

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Acute pulmonary edema after a large air embolus occurring during neurosurgery is a recognized phenomenon. The authors describe the course of a 76-year-old man who presented with noncardiogenic pulmonary edema shortly after undergoing resection of a high convexity meningioma. Transthoracic Doppler sonography, however, showed no evidence of a large intraoperative embolus; the evidence for ongoing but low-magnitude air embolus included visualization of bone aspiration of irrigant before bone-edge waxing, transient intraoperative declines in end-tidal CO₂ tension, and an increase of the fraction of inspired oxygen to maintain adequate saturation after removal of the craniotomy flap. There was no hemodynamic instability noted. The airspace disease was self-limited and resolved on supportive treatment after approximately 1 week, as would be expected for pulmonary edema caused by a single large intravenous air embolus. The authors present this case as the first report of pulmonary edema resulting from low-level air embolus occurring during craniotomy. This situation may go unrecognized intraoperatively but can cause the same significant postoperative morbidity as larger, more easily identified air emboli.

KEY WORDS • craniotomy • pulmonary edema • venous air embolism

Noncardiogenic pulmonary edema seen after large air emboli occurring during neurosurgery is a recognized phenomenon and has been reported nine times previously, to our knowledge. This syndrome has been described as relatively acute pulmonary edema, believed to be due to increased pulmonary microvascular permeability or rupture of capillary integrity occurring within several hours of the air embolus. The course is self-limited and supportive care alone can successfully treat the problem. The reports of this entity, including the single report found in the neurosurgical literature, describe a single, large, demonstrable air embolus with hemodynamic instability as the precipitating event in the syndrome. We present a case of air embolus–induced pulmonary edema caused not by a single large bolus of intravenous air, but rather by prolonged low-level introduction of air into the venous system during intracranial surgery.

Case Report

This 72-year-old man presented for resection of a high parietal convexity meningioma on the left side. He first experienced symptoms of right-handed numbness and lack of control 3 years prior to admission. His lesion was imaged by computerized tomography (CT) and followed with serial scans. One month prior to admission he became unable to write or feed himself using his right hand. His balance and short-term memory were also impaired at that time. He denied difficulties with speech or motor control of the mouth, jaw, or swallowing.

History. His medical history was remarkable for a distant myocardial infarction and coronary artery bypass grafting 2 years prior to admission. Several months after the bypass surgery, he underwent aortic valve replacement. There was no known history of pulmonary disease. He was taking the medications captopril and phenytoin.

Examination. His general physical examination was entirely normal except for a Grade III–VI systolic ejection murmur and scars from his previous surgeries. Neurological examination revealed an alert mental status but inability to recall three of three objects after 5 minutes. His cranial nerves were grossly normal with the exception of slight right-sided facial weakness. Motor examination showed a right pronator drift and diffuse weakness in...
the right upper extremity. Sensory examination was unremarkable and his cerebellar function was intact. His deep tendon reflexes were symmetric and both toes were down-going on plantar stimulation.

His preoperative laboratory testing was unremarkable. Chest radiography performed 1 week prior to surgery showed clear lung fields. A CT scan revealed a large left-sided parietal extraaxial mass consistent with meningioma (Fig. 1). The patient refused MR imaging because of claustrophobia.

Operation. The patient underwent a left-sided fronto-parietal craniotomy in the supine/semisitting position. Endotracheal intubation went smoothly without evidence of aspiration. A central venous catheter was placed preoperatively and its position in the right atrium was confirmed by a chest radiograph. Continuous transthoracic Doppler sonography was used during the procedure. Anesthesia was maintained by a balanced nitrous–narcotic technique. The surgery was uneventful except for the intraoperative observation of aspiration of irrigant into the cut bone surfaces after craniotomy until bone waxing was completed. This was not associated with any change in the transthoracic Doppler sounds. There were two slow, transient decreases in end-tidal CO₂ from a baseline of 23 mm Hg to 17 mm Hg. These events were not accompanied by hemodynamic change. For the last 2 hours of the surgery, the patient required a fraction of inspired oxygen of 0.8 to 1 to maintain oxygen saturation above 90%. The patient was extubated uneventfully and taken to the recovery room postoperatively, where he was lethargic and some-

what difficult to arouse. He became hypotensive, requiring fluid boluses and vasopressor agents to maintain a systolic pressure above 120 mm Hg. Examination of his lungs revealed bibasilar crackles with decreased breath sounds bilaterally at the lung bases. His motor examination was unchanged from preoperative testing, with present but somewhat decreased right-sided motor strength. Arterial blood gas levels on 60% oxygen by face mask were a PO₂ of 89 mm Hg, a PCO₂ of 32 mm Hg, and a pH of 7.36. His osmolarity was 289. His central venous pressure in the recovery room was 1 cm H₂O. A chest radiograph showed new bilateral airspace opacities consistent with pulmonary edema. His electrocardiogram was unchanged; serial cardiac cardiac infarction over the next 24 hours.

Postoperative Course. The patient was managed overnight with pressor agents and unsuccessful attempts at diuresis. The following morning he required intubation for progressive worsening in arterial oxygenation. A pulmonary arterial catheter was placed and pulmonary capillary wedge pressure was persistently measured below 10 cm H₂O. Echocardiography showed no evidence of cardiac dysfunction. A course of antibiotic medication was begun to avert the possibility of aspiration pneumonia, although ultimately all sputum cultures returned with no growth. Initially, the patient required a fraction of inspired O₂ of 0.6 and positive end-expiratory pressure of 7.5 cm H₂O to maintain adequate oxygenation. He also required intravenous volume repletion and multiple pressor agents to maintain satisfactory arterial pressure. By postoperative Day 4, the patient’s oxygenation and hemodynamic status had improved, and he was weaned from pressors and extubated. His neurological status returned to baseline by postoperative Day 6. A chest radiograph showed improvement in bilateral airspace disease by that time. He was released from the intensive care unit on postoperative Day 7 with good respiratory function and improved neurological status. On postoperative Day 12 he was discharged home with improved right-sided hand and arm function; he was able to feed himself with both hands. His respiratory status was at baseline. At follow-up evaluation 1 month later, he showed no evidence of any long-term sequelae from his pulmonary process and his neurological examination now showed complete recovery of the right upper-extremity motor function.

Discussion

We report the case of a 76-year-old man who presented for resection of a high convexity meningioma. His course was complicated intraoperatively by low-level intravenous entrainment of air leading to postoperative pulmonary edema. His pulmonary status gradually improved with supportive care, and he was eventually discharged home without sequelae.

The observation of acute pulmonary edema occurring after intravenous entrainment of air is well recognized. The cases described in relation to neurosurgical procedures have generally occurred after large boluses of intravenous air that caused hemodynamically significant changes. Certainly, craniotomy performed in the sitting or semisitting position, as in the present case, increases the

Fig. 1. Axial computerized tomography scanning in a 76-year-old man after administration of intravenous contrast material showing the lesion before surgery. The scanning demonstrates a dural-based tumor high on the convexity.
Air embolism and pulmonary edema

risk of intravenous air aspiration. Our case is novel in that air aspiration appeared to occur at a low enough level that it did not cause changes in transthoracic Doppler sounds or hemodynamic instability. Rather, the best intraoperative evidence for ongoing aspiration included transient decreases in end-tidal CO₂, and an increasing requirement for a high inspired percentage of oxygen in the setting of observed aspiration of irrigant into cut bone surfaces.

Of note, transthoracic Doppler sonography was uninformative for the intraoperative diagnosis of air embolism in this case and no attempt was made to aspirate air from the indwelling central venous catheter. Presumably, the amount of air was insufficient to cause adequate turbulence to be heard by Doppler monitoring. This point is significant for the present case, in that the diagnosis of air embolism was not recognized intraoperatively. Had the air embolism been recognized, appropriate changes in surgical and anesthetic technique may have prevented the somewhat complicated postoperative course. Considerations that may have affected the patient’s recovery include continued mechanical ventilation postoperatively, until satisfactory gas exchange had been documented, and use of 100% O₂ rather than N₂O as an anesthetic carrier gas to decrease the size of embolized air bubbles.

The exact series of events noted in the present case has been modeled in sheep. Pfitzner, et al., have shown that sustained low-level air embolism produces hypoxemia prior to the development of pulmonary edema. The ensuing syndrome of clinical pulmonary edema mimics that seen with larger boluses of intravenous air.

The differential diagnosis of pulmonary edema in the postoperative neurosurgical patient is broad but can be divided into the general categories of cardiac failure from a primary cardiac event, fluid overload, or another noncardiac cause of pulmonary injury. The categorical diagnosis can easily be made by evaluation of pulmonary capillary wedge pressure. Once a noncardiogenic source of edema is recognized, the diagnosis of air embolism should be considered along with the other more common causes of noncardiogenic pulmonary edema in this setting, such as neurogenic pulmonary edema or fluid overload.

In the present case, the absence of increased intracranial pressure at surgery and the initially low postoperative central venous pressure allowed us to rule out quickly these other possible causes of pulmonary edema.

The cause of pulmonary edema after venous air embolus has been extensively studied and is likely due to an induced leaky capillary state in the pulmonary microvasculature. There is evidence that neurogenic pulmonary edema and pulmonary edema induced by air emboli are caused by increased wall stress resulting in structural damage to pulmonary capillaries. The mechanism of injury may be high intravascular pressures that are often transient and undocumented. Air embolism from the venous bed results in mechanical obstruction of the pulmonary outflow tract of the right ventricle. Turbulent flow is created around the air bubbles, which enhances platelet aggregation and fibrin formation. A cascade of physiological abnormalities is initiated, not unlike those seen in other types of pulmonary embolism, which results in marked pulmonary vasoconstriction. Under such acute stress, the capillaries can develop disruptions of some or all the layers of the alveolar barrier, resulting in pulmonary edema. Initially, Starling equilibrium is disturbed and fluid flows to the interstitium. At higher pressures the alveolar pores are stretched, allowing larger molecules to pass. With further disruption, large proteins and cells can freely move into the interstitium. Ultimately, the disruption causes frank hemorrhage. Microscopically, the damage seen in animal models of venous air embolism is identical to that seen in animal models of edema induced by supraphysiological intravascular pressures.

An alternative pathway of injury to the pulmonary vascular endothelium after air emboli may be toxic free radical damage. Vascular permeability caused by intravenous air emboli is dramatically reduced by superoxide dismutase, a free radical scavenger enzyme. Prevention of this pulmonary vascular bed damage by treatment with an antioxidant molecule, such as megadose administration of methylprednisolone, may be an intriguing approach to preventing the pulmonary edema expected after known air embolus.

The most interesting aspect of the present case is the observation that an unrecognized low level of intravenous air aspiration without hemodynamic sequelae or Doppler changes can produce life-threatening pulmonary edema. Awareness of this entity as a cause of pulmonary edema in the presence of low left-sided pulmonary pressures, coupled with the knowledge that supportive care will allow spontaneous resolution, should guide perioperative evaluation and treatment. Clearly, prevention of air emboli through optimal positioning and rigorous intraoperative monitoring remains the best approach to this problem.

References


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