

Cerebral Air Embolism Following Removal of Central Venous Catheter

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ABSTRACT Cerebral air embolism occurs very seldom as a complication of central venous catheterization. We report a 57-year-old female with cerebral air embolism secondary to removal of a central venous catheter (CVC). The patient was treated with supportive measures and recovered well with minimal long-term injury. The prevention of air embolism related to central venous catheterization is discussed.

INTRODUCTION

Central venous catheters (CVCs) are used extensively in critically patients. They are commonly placed for hemodynamic monitoring, administration of medications, transvenous pacing, hemodialysis, and poor peripheral access. Complications can occur and are numerous. We describe a case of cerebral air embolism in a 57-year-old female as a complication of central venous catheterization and the treatment course. Additionally, we discuss methods to prevent air embolism related to central venous catheterization.

CASE REPORT

This is a 57-year-old female with a history of inflammatory bowel disease who was admitted to the hospital for revision of an ileostomy. While in the operating room, an extensive lysis of adhesions was undertaken as well as placement of a right internal jugular central venous catheter and nasogastric tube. The patient did well initially postoperatively but later developed an ileus requiring bowel rest and parenteral nutrition. The ileus resolved and discharge plans were made. The patient was prepared for discharge to home to include removal of her CVC. The patient was placed flat in bed for removal of the CVC as she did not tolerate the Trendelenburg position. The patient was instructed to exhale as the line was pulled out. Immediately as the central line left the skin, a sterile gauze dressing was put into place and pressure applied. Pressure was held by hand for 5 minutes. The patient was alert, oriented, and conversant at this time. After approximately 5 minutes, the dressing was taped in place. As the patient was assisted back into the sitting position, she complained of dizziness and slumped back into the bed. The patient was noted to have decerebrate posturing and difficulty breathing. Advanced life support measures were initiated. The patient was moved up in the bed and her airway stabilized. During the code, endotracheal intubation was established to help maintain the patient's

airway. No loss of pulse or changes in rhythm strip occurred during code procedures. Following the code and stabilization of the patient's airway, she was transferred to the Surgical Intensive Care Unit.

Spiral computed tomography (CT) of the chest and computed tomography of the abdomen and pelvis were undertaken with enteral and intravenous contrast. No pulmonary embolism or air embolism was visualized on computed tomography pulmonary angiography (CTPA) and no pathology was seen on CT of the abdomen and pelvis. The patient displayed some seizure activity later that day and a CT and magnetic resonance imaging (MRI) of the head were ordered which confirmed air embolism to the brain (Fig. 1). The patient was placed on 100% oxygen on the ventilator. Neurology and Cardiology were consulted. A transesophageal echocardiography revealed a patent foramen ovale (PFO). Neurologic examination over the next several days was consistent with severe cerebrovascular accident (CVA) and minor cerebral edema. Three days after initial insult, the patient began to show improvement on neurologic examination. Ten days after insult, she was discharged to a stroke rehabilitation facility tolerating a full liquid diet, breathing without the use of mechanical assistance, and communicating with a dry erase board. She is currently ambulating with a cane/walker and remains weak in the left lower extremity and profoundly weak in the left upper extremity. She has normal mental status.

DISCUSSION

Air embolism is a potentially catastrophic, though uncommon, event that occurs as a consequence of air entry into the vasculature. There are several times in the care of patients when an air embolism may occur. Surgery, barotrauma from mechanical ventilation, and central venous catheter access name only the most common causes.¹

In surgery, venous air embolism is most commonly a complication of neurosurgical or otolaryngological interventions because the incision is made above the heart by a distance greater than the central venous pressure. This is particularly true when a patient is in the sitting position and can occur 10–80% of the time. Most of these are clinically silent air

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FIGURE 1. Computed tomography of the head taken approximately 10 hours after initial incident demonstrating cerebral air embolus in right frontal lobe.

emboli.² Barotrauma, be it from high pressure ventilation or diving, also places patients at risk for air embolism. These are the result of violation of the pulmonary vascular integrity with alveolar rupture allowing communication between the vasculature and air.³

In the case of central venous catheterization, air embolism can be a serious complication. The air embolism can occur at any time when the catheter is being inserted, while the catheter is in place, or at the time of catheter removal. When inserting the catheter, venous air embolism may occur from failure to occlude the needle hub or the catheter until it is capped.⁴ The entry of air will be more likely if the patient experiences a reduction in the central venous pressure (CVP), such as with deep inspiration or upright positioning.⁵ Once the catheter is in place, air may enter the vasculature from fracture or disconnection of the catheter connections. This is believed to be responsible for the vast majority of air emboli associated with CVC.⁶⁻⁸ Upon removal of the CVC, air emboli may occur from decreases in the CVP as a result of standing or deep inspiration.^{11,12} A persistent catheter tract may be present from long-standing CVC, allowing air to enter the vasculature after a dressing has been placed.⁵ Any number of these factors may exist over the time that a patient requires a CVC.

A pulmonary air embolism will result if the air embolus remains in the venous system. For air emboli to enter the cerebral vasculature, it must enter into the arterial system. It is important to point out that several studies and case reports have demonstrated retrograde flow of air in the venous system. This seems to be a very rare cause of cerebral air embolism.⁹ The air embolism may pass to the arterial system by

one of several other mechanisms. Paradoxical embolization may occur through a septal defect or through a PFO.¹⁰ Air may pass from venous to arterial through pulmonary arterial-venous malformations. For large air emboli, the pulmonary capillaries may incompletely filter the air, allowing the air to move from vein to artery. Once the air has crossed into the arterial system, damage occurs when the bubble moves to an end organ and causes ischemia.

The location of the CVC insertion site has been studied to determine if it plays a role in complications. Heckmann, et al.⁸ reviewed cerebral air embolism as a complication in 26 patients in 2000. He found that of the 26 catheters, 12 were placed in the subclavian veins, 8 were placed in the internal jugular veins, and 6 did not specify the placement site. Looking at this evidence, one could justify that the subclavian veins are more likely to result in cerebral air embolism if a complication occurs. However, the vast majority of CVC are placed in the subclavian veins. Because of the rarity of the complication of cerebral air embolism, it is difficult to assess whether the placement site predisposes a patient to suffer an air embolism. At either site, the ideal location of the catheter tip is in nearly the same location near the confluence of the vena cava and the right atrium; however, because the site of skin penetration is slightly higher with internal jugular catheters, air would be more likely to entrain because of the increased pressure gradient between the atmosphere and the venous system of the neck.

The effect of the air embolus on the patient is dependent upon the rate and volume of air introduced into the circulation. In a canine model, gas entering the venous system overwhelms the ability of the lungs to filter at a rate greater than 0.30 mL/kg per minute.¹³ At rates greater than this, arterial air emboli and tissue ischemia resulted. Large, rapid boluses of air are not as well tolerated compared to slow infusions of small amounts of air. An estimated fatal dose in humans is 300–500 mL of gas introduced at a rate of 100 mL/s. This can occur through a 14 gauge catheter with a pressure gradient of 5 cm H₂O.¹⁴ Minor cases of air embolism are believed to occur frequently and are minimally symptomatic. In a review of patients who suffered a cerebral air embolism as a complication of central venous catheterization, the mortality rate was 23%.⁸

Diagnosis of a cerebral air embolism requires a high level of suspicion. Any neurologic symptoms surrounding the insertion, use, or removal of a central venous catheter must be investigated. Diagnostic tests helpful in identification of a cerebral air embolism include arterial blood gas analysis, echocardiography, chest radiography, and computed tomography.⁸ Computed tomography or magnetic resonance imaging may or may not show the presence of air in the cerebral vasculature, especially if there is a delay in imaging.¹⁵

Treatment of cerebral air embolism requires early diagnosis of the condition. If the initial insult is witnessed, then the patient can be placed head down lying on their left side, known as Durant's maneuver.⁸ This keeps any air trapped within the

TABLE I.

Risks	Prevention	Diagnosis	Treatment
Patient Risks	Correct dehydration before placement, if possible		Immediately execute Durant's maneuver if air embolism is suspected
Long-term placement of CVC	Keep all connections closed during placement and while catheter is in place	New neurologic symptoms in a patient with a CVC	Oxygen therapy
Failure to occlude needle hub	Inspect catheter for cracks or broken seals daily	CT/MRI of the head	Hyperbaric oxygen therapy
Decreased CVP during placement or removal	Have patient valsalva or maximally inhale during removal		Supportive care

heart away from the outflow of the right ventricle and may help to reduce or dislodge the blockage of the vasculature by the air bubble. The patient should be placed on oxygen therapy. The efficacy of hyperbaric oxygen therapy has been debated. Some studies have demonstrated improvement in patients with early hyperbaric oxygen therapy while others have not shown a statistically significant difference between oxygen therapy and hyperbaric oxygen therapy. All of these studies are limited by the small sample size of patients with cerebral air embolism.^{8,16} The best treatment of cerebral air embolism remains prevention of the condition. Prevention can begin even before a CVC is placed. Correcting any dehydration before the procedure will increase central venous pressure, decreasing the gradient that is necessary for air embolism to occur. During placement of a central venous catheter, occlusion of the needle hub and the catheter can prevent air embolism.⁵ Also, ensuring to keep all the connections tight and to keep all unused hubs closed and locked when not in use will prevent air embolism from the catheter.⁶ Inspecting the catheter each time that the patient is seen can help to assure that this is achieved. When removing the catheter, placing the patient in a supine or Trendelenburg position will increase CVP. Having the patient valsalva during removal will also increase CVP. There is debate in the literature over whether the patient should be exhaling during removal of the CVC.¹¹ Some have suggested that the patient might inhale unexpectedly at the shock of having the line removed. Patients can be asked to maximally inhale, which would increase CVP and prevent the patient from inhaling more when the line is removed.⁵ Immediately after removing the catheter, an impermeable dressing should be applied to the site and pressure should be held.¹² The length of time that pressure should be held has not been standardized. Most texts concerning central venous catheters suggest a time period of 1 to 5 minutes.¹⁷ Erring on the long end of this time period will help to ensure that any otherwise patent catheter tracts have a chance to close. Even if a patent catheter tract exists, using an impermeable dressing, such as gauze with antibacterial cream, will prevent any air from entering the vasculature.¹² Table I illustrates the prevention, diagnosis, and treatment of central venous catheter air embolism.

In the case of our patient, the cerebral air embolism most likely occurred through the gauze dressing through a patent

catheter tract into the internal jugular vein. The dressing placed was not impermeable and placing a barrier, such as bacitracin, on the gauze may have helped to prevent the incident. Once in the venous system, the air most likely crossed through her pulmonary vasculature through an arterial-venous shunt; however, it may have crossed to the arterial system at the heart through her small PFO. Though the incident was witnessed, the patient was not immediately placed into Durant's maneuver. She was treated with 100% oxygen therapy once placed on the ventilator, which will help to ameliorate ischemic brain damage.

Following the treatment of this patient, new standards were instituted at our facility for the removal of CVC. Patients are placed in Trendelenburg position. The dressing covering the CVC is removed and any sutures holding the line in place are removed. The patient is instructed to maximally inhale and then the line is removed. An occlusive dressing is placed immediately over the site. This dressing may consist of regular gauze with bacitracin ointment applied or petroleum gauze. Pressure is held with the patient in Trendelenburg position for 5 minutes. Following this time, a plastic dressing that completely locks out air (i.e., an Opsite or Tegaderm) is applied. Then, the patient may be taken out of Trendelenburg. This checklist is to be followed and documented with each CVC removal.

Cerebral air embolism is an exceptionally rare complication to the common procedure of central venous catheterization. Whenever a CVC is used, a high clinical suspicion must be present in any of these patients who display any neurologic symptoms. Prevention, as well as early diagnosis, may decrease morbidity and mortality.

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Case Report

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