

Devastating Cerebral Air Embolism After Central Line Removal



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ABSTRACT

Background: Air embolism is a well-published complication arising from central venous catheter use. Literature and case studies provide information regarding clinical sequelae. Preventable mistakes still occur despite following what is considered appropriate protocol. This case report describes the neurological complications likely caused by a cerebral air embolism related to central venous catheter removal. **Case:** An 84-year-old man was admitted to the neuroscience critical care unit with acute stroke symptoms and seizures after removal of a central venous catheter. **Conclusion:** There is an abundance of literature describing best practice, complications, and treatment of venous air embolism associated with central line catheter use. Utilization of central venous catheters is increasing. With increased utilization comes the responsibility to improve commonplace knowledge and ensure that practice guidelines and protocols are dependable and consistent.

Case Report

An 84-year-old man was admitted to the intensive care unit with a primary diagnosis of severe sepsis due to community-acquired pneumonia. His medical history was significant for diabetes mellitus, coronary artery disease, and pulmonary fibrosis with resultant pulmonary hypertension. A right internal jugular central venous catheter (CVC) was placed to monitor central venous pressure for fluid treatment using Surviving Sepsis Campaign Guidelines (Dellinger et al., 2008). An echocardiogram was performed estimating the ejection fraction to be 65%–70% with right ventricular systolic pressure (RVSP) at 46.5 mm Hg and a right atrial pressure of 20 mm Hg; no bubble study was performed to assess for intracardiac shunt.

After fluid resuscitation, administration of antibiotics, and brief use of vasopressors, the patient improved and was transferred to the floor. Five days after his initial admission, he was seen by his physician and received instructions for discharge to home, and discharge orders were written. The registered nurse adhered to the institution's protocol for pulling out CVCs and followed all steps for removal: (1) placing patient's head of bed flat, (2) patient holding his breath, (3) application of direct pressure to the site, and (4) covering the site with gauze and tape. The patient was then allowed to get up and was

dressing himself for discharge. Several minutes later, the patient's call light went on. The clerk answered the call, and the wife stated that the patient was having a seizure. Nurses entered the room and found the patient lying across the bed with twitching of all extremities. The wife reported that the patient was using the urinal when he suddenly slumped over onto the bed.

The Acute Stroke Emergency Response Team (ASERT) was activated and entered the room to find the patient unresponsive. The ASERT followed protocol to assess the patient's situation and stabilize the patient for computed tomography (CT) scan as soon as possible. The patient's blood glucose was 140 mg/dl per bedside glucometer, blood pressure was 154/92 mm Hg, and heart rate was 97 beats/minute, with spontaneous respirations. One hundred percent oxygen via nonrebreather was applied with oxygen saturations of 99%, and administration of intravenous (IV) fluids was initiated. The Cincinnati Prehospital Stroke Scale, consisting of Face, Arms, Speech, and Time of onset (FAST), was utilized by the ASERT registered nurse. The FAST examination revealed abnormal face, abnormal arm movement, and abnormal speech. National Institute of Health Stroke Scale was performed by the neurovascular nurse practitioner, yielding a score greater than 10. The patient was unable to cooperate for a complete National Institute of Health Stroke Scale examination.

The patient was attached to a transport monitor and taken to radiology for a stat head CT scan. During transport, the patient became more alert and was able to respond with one-word answers and follow commands with his right extremities; however, he had left hemiplegia, left facial droop, and a right-gaze preference. Also noted were some rhythmic blinking of his eyes and twitching of his left cheek. A CT head perfusion study showed delayed mean transit time to much of

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the right hemisphere and mildly decreased cerebral blood flow in the right hemisphere as well. Then, CT angiogram was performed to assess the condition of the vessels. This showed no occlusions or stenosis of the cerebral or cervical vessels, with air bubbles being seen in the venous structures of the upper neck and skull base, including bilateral cavernous sinuses. He was immediately taken for magnetic resonance imaging (MRI) of the brain, which did not show any diffusion restriction to suggest acute ischemia. The MRI was performed at 90 minutes from onset of symptoms. It was decided that the patient was not a good candidate for recombinant tissue plasminogen activator (rt-PA) because of intermittent symptoms, seizure activity, his age, and concern that symptoms may have been related to an air embolism after CVC removal. Of note, the patient had a strict "Do Not Resuscitate" order that was recorded and confirmed by the patient's spouse.

The patient was admitted to the neuroscience critical care unit for further monitoring. His breathing was labored after MRI and he was given a small dose of IV furosemide (Lasix) to relieve his respiratory distress. He had complex partial seizures that were treated with lorazepam (Ativan) and levetiracetam (Keppra). By electroencephalogram, he had ongoing status epilepticus, which was terminated approximately 22 hours after the onset of symptoms. He maintained a decreased level of consciousness with sporadic ability to follow commands on his right side only. Speech consisted of moaning and groaning with no eye opening (Glasgow Coma Scale 7–9). Troponin I, creatine kinase-MB, and B-type natriuretic peptide were found to be elevated 48 hours after the event (3.12 ng/ml, 8.3 ng/ml, and >4,000 pg/ml, respectively). A trans-thoracic echocardiogram with bubble study was performed. Ejection fraction was 25%–30%, with no evidence of intracardiac shunt and an estimated RVSP of 64 mm Hg. A cardiology consultation diagnosed ischemia due to decreased myocardial oxygenation in the setting of underlying cardiomyopathy from chronic pulmonary disease with elevated right-heart pressures.

His breathing slowly became more labored, and he continued to require 100% oxygen. At Day 3, with no improvement in mental status, the wife felt that the patient would not want to continue in his current state. The wife requested withdrawal of oxygen and medical support, except for medications to keep him comfortable. His oxygen was turned down, and IV morphine was given for comfort measures. He passed away with his family at his bedside.

Discussion

Iatrogenic air embolism can be the most serious complication of CVC removal (Ely et al., 1999). The

A serious complication of CVC use, air embolism can occur during insertion, removal, tubing or cap changes, or accidental disconnection.

incidence of iatrogenic air embolism is difficult to determine because it may occur without symptoms. Air embolism can result from central line insertion, tubing or cap changes, accidental disconnection, cracked tubing or caps, infusion of air, or removal of the central line (Wittenberg, Richard, & Conrad, 2006). Certain companies recommend an obturator (dust cap) over the hemostasis valve to prevent air embolism (Darovic, 2002). Other causes of air embolism are neurosurgery, cardiovascular surgery, orthopedic surgery, lung transplant, liver transplant, obstetrical or gynecological procedures, dental implants, blunt or penetrating traumas, lumbar punctures, insufflation for arthroscopic or laparoscopic procedures, positive pressure ventilation, and childbirth (Fink, Abraham, Vincent, & Kochanek, 2005; Wittenberg et al., 2006).

Venous air embolism (VAE) occurs from air entering the venous system via the CVC itself or the track from removal of the CVC (Fink et al., 2005). Air is transported to the pulmonary arteries, hindering gas exchange and causing pulmonary hypertension. This can result in right ventricular strain and, ultimately, cardiovascular collapse (Fink et al., 2005; Wittenberg et al., 2006). A large enough bolus of air can create an obstruction to the outflow of blood from the right ventricle, essentially forming an airlock (Wittenberg et al., 2006). It is postulated that in this patient's case, an airlock occurred briefly, resulting in retrograde flow of air from the jugular CVC site to the cavernous sinuses, as evidenced by CT scans. This could have occurred easily with the patient's underlying pulmonary hypertension, which significantly increased during this acute event (RVSP of 64 mm Hg). Interestingly, the patient's symptomatology reflects a right hemispheric injury, with the CT scan demonstrating decreased transit time and cerebral blood flow to the right hemisphere; however, there was no evidence of air in the arterial system. Small amounts of air in the blood are generally absorbed by the circulation (Wittenberg et al., 2006), and air absorption is hastened by administration of high-flow oxygen (Fink et al., 2005). This likely contributed to lack of visible arterial air bubbles as seen on the head CT.

It is proposed that the patient also had paradoxical air embolism related to a physiological pulmonary or cardiac right-to-left shunt. Cardiac right-to-left shunts may be intermittent because of briefly elevated right-sided cardiac pressures that cause right atrial pressure to be higher than left atrial pressure, creating a patent foramen ovale (PFO) (Carey, Boltax, Dickey, & Finkelstein, 1999; Cheng, 1976). Although the echocardiogram showed no evidence of intracardiac shunt, elevated right ventricular pressure leading to high atrial pressures could create a temporary opening of the PFO, only to close when the right ventricular pressure decreased to equal the pressure of the left atrium; however, the patient had significantly elevated right-heart pressure when the echo was performed and still showed no PFO.

Another possible cause of paradoxical air embolism is a pulmonary right-to-left shunt, which may occur with an arterial-venous malformation (AVM). Due to this patient's underlying lung pathology and extensive pulmonary testing, a prior CT chest angiogram was done, which showed no evidence of AVM, allowing us to rule out pulmonary AVM as a cause. However, anatomic anomaly is not necessary for a pulmonary right-to-left shunt to occur. Large quantities of abrupt venous air infusion can result in acute increases in pulmonary capillary pressures, causing incomplete filtering of air because air bubbles can easily be distorted, resulting in pulmonary right-to-left shunting (Fink et al., 2005; Murphy, Harford, & Cramer, 1985; O'Dowd & Kelley, 2009; Seeburger et al., 2009).

Air in the cavernous sinus does not explain this patient's symptoms of dense left hemiplegia, comatose state, and seizures. The internal carotid arteries course through the cavernous sinuses bilaterally, the carotid sympathetic plexuses, and the oculomotor cranial nerves (III, IV, and VI), with a portion of the trigeminal nerve segments (V1 and V2) passing through the wall of the cavernous sinuses (Kattath & Pula, 2009). With injury of these areas, one would expect to see more of a focal symptomatology than exhibited by the patient.

The right cerebral hemisphere may be more susceptible to paradoxical embolization due to anatomy. The first branch from the aorta is the innominate artery, which separates into the right subclavian and right common carotid artery. The right common carotid then divides into the right internal carotid artery and external carotid artery (Osborn, 1994). Plaque in the right internal carotid artery could have worsened right hemispheric cerebral perfusion pressure during this patient's transient flow problems; however, a CT angiogram of the neck showed no significant stenosis.

Notably, this case report reveals that the nurse reviewed and followed the procedure for discontinuing a catheter sheath introducer line, which did not include covering the site with an air occlusive dressing. Lack of an air occlusive dressing may have led to this patient's devastating air embolism and eventual death. There was a disparity between the procedure for CVC removal and introducer catheter removal. The same technique should be used for removal of any type of CVC. It is critical that all policies, procedures, and protocols align and are trustworthy.

Evaluation for acute stroke was initiated promptly upon the patient's change in status. Treatment for possible VAE could have also been initiated simultaneously. The immediate treatment for VAE should include preventing further air entry by occluding the site; turning the patient to the left lateral position with the head down, if possible; maintaining high-flow oxygenation; and providing critical care to stabilize and maintain vital signs (Heckman et al., 2000). Consideration should be made concerning hyperbaric oxygen (HBO) treatment. The efficacy of HBO is questioned because there have been no randomized controlled trials and benefits versus risk must be determined (O'Dowd & Kelley, 2009). For this patient, HBO as a treatment for air embolism was not discussed.

Conclusion

According to Stoker (2006), "Each year an estimated six million central venous access devices (CVAD) are placed in the United States, and this number continues to increase as the population ages" (p. 14). The most disturbing concept of air embolism from CVCs is that it is completely avoidable (Drewett, 2000; Mennim, Coyle, & Taylor, 1992). Case reports are plentiful and prevalent in nursing and medical literature, far more than had been anticipated by the authors. The literature carefully describes the pathophysiology, evidence-based standards, and treatment of air embolism. The need for improved education has also been described by many. There is an unacceptable lack of awareness about safe care of patients with CVCs. With increased utilization comes responsibility to improve commonplace knowledge and ensure that practice guidelines and protocols are dependable and consistent.

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