Cerebral air embolism through a central venous catheter in the absence of intracardiac shunt

ABSTRACT
Central venous catheters are routinely placed on medically complex patients for a variety of reasons, including facilitating intravenous access in difficult intravenous (IV) access situations, accurate hemodynamic monitoring, large-volume resuscitation, medication administration, nutritional support, and continuous renal replacement. As with other invasive medical procedures, placement, maintenance, and discontinuation of central venous catheters introduces risk and potential complications. We report a case of bilateral cerebral infarct secondary to air embolism through the right internal jugular vein venous catheter in the absence of intracardiac shunt in a patient with ischemic colitis who underwent total abdominal colectomy.

Key words: Air embolism, central venous catheter, cerebral infarct

Introduction
Central venous catheters are routinely placed on medically complex patients for a variety of reasons, including facilitating intravenous (IV) access in difficult intravenous access situations, accurate hemodynamic monitoring, large-volume resuscitation, medication administration, nutritional support, and continuous renal replacement. As with other invasive medical procedures, placement, maintenance, and discontinuation of central venous catheters introduces risk and potential complications.

Complication rates for central venous catheters have been reported to be present in >15% of patients. Air embolism complications typically manifest with symptoms of pulmonary embolism. Blood flow from common central venous catheter sites (internal jugular, femoral, and subclavian veins) quickly reaches the right heart and the pulmonary vasculature.

The air passing through the pulmonary circulation is often resorbed and asymptomatic; however, a large enough volume can cause hypoxemia and cardiovascular collapse. Cerebral air embolism is an often fatal complication and one of the most serious complications of central venous catheters placement.

We report a case of bilateral cerebral infarct secondary to air embolism through the right internal jugular vein venous catheter in the absence of intracardiac shunt in a patient with ischemic colitis who underwent total abdominal colectomy.
Case Report

A 68-year-old male with a history of hypertension, peripheral vascular disease, congestive heart failure with a reduced ejection fraction of 30–35%, and methamphetamine use underwent total abdominal colectomy for ischemic colitis. The right internal jugular triple lumen venous catheter was placed intraoperatively. The patient failed extubation postoperatively due to acute encephalopathy. A computed tomography scan of the brain showed air in the cavernous sinus bilaterally, retro-orbital region, and pterygoid venous plexus [Figure 1], along with a 25 mm infarct in the right middle frontal gyrus and a 20 mm infarct in the left precentral gyrus [Figure 2]. The preoperative brain computed tomography shown Figure 3.

Postoperative transthoracic echocardiogram showed left ventricular thrombus, and there was no evidence of patent foramen ovale. The hospital course got complicated with severe septic shock secondary to an anastomotic leak and pelvic abscess. The patient passed away two weeks later.

Discussion

Cerebral air embolism is rare and carries a high morbidity and mortality risk. It can involve both the venous and arterial vasculature. Cerebral air embolism can occur through the retrograde transmission of air from the cerebral venous circulation into the arterial circulation, especially in the upright position during neurological and otolaryngological procedures. In the setting of intra-cardiac shunt, other scenarios of a paradoxical air embolism can occur such as intravenous injection of air through a central venous catheter, pulmonary artery catheter, or during procedures such as cardiac ablation, pacemaker placement, and bubble studies. Air embolism has been reported during endoscopic retrograde cholangiopancreatography through sphincterotomy using air for insufflation and also portal vein gases during bowel ischemia. Application of high positive end-expiratory pressure during acute respiratory distress syndrome can cause an arterial or venous air embolism. Deep-sea diving and rapid ascending are other risk factors which occur due to changes in air pressure.

The air embolism can also occur if the volume of air exceeds the ability of lung vascular to filter or if there is an intrapulmonary shunt.

The clinical manifestations of cerebral air embolism occur through two mechanisms; mechanical obstruction and the inflammatory response secondary to platelet activation and endothelial injury, which exacerbate the ischemic effect. These manifestations include shortness of breath, chest pain, arrhythmia, murmurs, hypotension, cardiac arrest, seizures, and ischemic event.
The incidence of air embolism secondary to central venous catheter insertion and fluid administration is rare and can be diagnosed clinically with acute onset of hypoxia, hypotension, seizures, or change in mental status. In addition to the signs and symptoms, using scan imaging and echocardiography can confirm the diagnosis.

Ultimately, cerebral air embolism is often an iatrogenic and preventable condition. Patients should be positioned appropriately during invasive line placement and removal. In addition, patients should be instructed to perform Valsalva maneuvers during line removal. Even with routine intravenous placement and fluid infusions such as in our patient, care must be taken to avoid accidental entry of air into the circulation. Transthoracic or transesophageal echocardiogram monitoring for venous air embolism should take place in surgical cases with a high risk of developing cerebral air embolism. In these high-risk procedures, anesthetic containing nitrous oxide should also be avoided as it can potentiate the development of air emboli. In mechanically ventilated patients, positive airway pressure should be appropriately minimized to prevent pulmonary barotrauma.

The management of cerebral air embolism is often challenging. It requires recognizing the mechanism of air entry, air aspiration through the central venous catheter, and the application of Durant’s maneuver, which is placing the patient in the left lateral and Trendelenburg decubitus position. This aims to trap air in the right atrium and ventricle, thus minimizing the entry of air emboli into the right ventricular outflow tract and pulmonary artery. Other modalities that can be used to decrease the air embolus size are endotracheal intubation, administration of 100% oxygen, and hyperbaric oxygen therapy. The higher the partial pressure of oxygen, the higher the solubility of oxygen in the blood.

In one series of 16 patients treated with hyperbaric oxygen, 50% of patients had complete recovery, 31% had partial relief, and 19% had no benefit with death in 12.5% of patients. Steroids administration has been shown to decrease air embolism-induced inflammation and subsequent cerebral edema; however, its routine use is still controversial.

Conclusion

This case highlights the importance of being vigilant during central venous catheter insertion and fluid administration to avoid air introduction into the systemic circulation. Early detection and treatment decrease the sequelae of cerebral air embolism.

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