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Cerebral Air Embolism Following the Removal of a Central Venous Catheter in the Absence of Intracardiac Right-to-Left Shunting

A Case Report

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Abstract: Air embolism following central venous catheter (CVC) removal is a relatively uncommon complication. Despite its rare occurrence, an air embolism can lead to serious outcomes. One of the most fatal complications is cerebral air embolism.

We report a case of cerebral air embolism that occurred after the removal of a CVC in a patient with an underlying idiopathic pulmonary fibrosis, subcutaneous emphysema, pneumomediastinum, and a possible intrapulmonary shunt. Although the patient had a brief period of recovery, his condition deteriorated again, and retention of carbon dioxide was sustained due to aggravation of pneumonia. Despite full coverage of antibiotics and maximum care with the ventilator, the patient died about 5 weeks after the removal of the CVC.

We suggest that strict compliance to protocols is required even while removing the catheter. Furthermore, additional caution to avoid air embolism is demanded in high-risk patients, such as in this case.

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Abbreviations: AVM = arteriovenous malformation, CT = computed tomography, CVC = central venous catheter, FEV = forced expiratory volume, FVC = forced vital capacity, ICU = intensive care unit, IJV = internal jugular vein, IPF = idiopathic pulmonary fibrosis, PAE = paradoxic air embolism.

INTRODUCTION

Central venous catheters (CVCs) are frequently used for purposes such as accurate hemodynamic monitoring, efficient administration of medication and nutritional support, and renal replacement therapies. Despite its usefulness, the insertion, maintenance, and removal of a CVC bear the possibilities of various risks to the patient. It has been reported that CVCs are associated with complications in >15% of patients.[1] The majority of air embolism cases related to CVCs are cases of pulmonary embolism. This can be easily expected when considering the normal venous circulation, which flows from the large vein into the right heart and is subsequently pumped into the pulmonary vasculature. Though gas entering the pulmonary circulation may pass by unnoticed, it may also result in arterial hypoxemia and cardiac failure.[2] However, one of the most fatal complications is cerebral air embolism. We report a case of cerebral air embolism that occurred after the removal of a CVC in a patient with an underlying idiopathic pulmonary fibrosis (IPF), subcutaneous emphysema, pneumomediastinum, and a possible intrapulmonary shunt.

CASE REPORT

A 65-year-old man presented with lower abdominal pain and fever of 5 days' duration. The patient was initially treated at another hospital with propacetamol hydrochloride, and after subsequent investigations, including a computed tomography (CT) scan of the abdomen, intravenous ceftriaxone was administered for the possible diagnoses of peritonitis, abdominal abscess, and inflammatory bowel disease. He was transferred to our hospital afterwards, and his vital signs upon arrival were blood pressure of 97/72 mm Hg, heart rate of 114 beats/min, respiratory rate of 24 breaths/min, and temperature of 36.5°C. After clinical evaluation and reexamination of the CT scan previously taken, acute appendicitis with peritonitis was suspected.

One month prior, the patient was diagnosed with IPF that required lung transplantation and had been treated with prednisolone and *N*-acetylcysteine. Preoperative pulmonary function testing revealed forced expiratory volume 1/forced vital capacity (FVC) of 79%, FVC of 34%, and FVE1 of 31%. Subcutaneous emphysema in the neck, both suprascapular area and axilla, was noted on plain chest radiograph, which was already known to the patient, along with the pneumomediastinum. Due to his underlying lung condition, postoperative admission to the intensive care unit (ICU) was inevitable for close monitoring and possible ventilation support. Placement of CVC was to the right internal jugular vein (IJV) and was also undertaken prior to the operation.

The patient underwent laparoscopic appendectomy, which revealed severe soiling and adhesion of the small and large bowels. The perforated appendix was forming an abscess pocket and adhered to the pelvic lateral wall. Drainage of the pus and massive irrigation was carried out and the appendiceal base was ligated. A drain was placed in the pelvic cavity and right paracolic gutter. The total surgery lasted about 1 hour and a half.

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After being admitted into the ICU, the patient was transferred to the general ward on the same day with full recovery of consciousness and vital signs in normal range, and showed satisfactory recovery from the surgery. Although discharge was being considered, the removal of the CVC was carried out on the seventh postoperative day. For the procedure, the patient's head was placed flat on the bed, and he was asked to practice holding his breath after inspiration several times. Although the patient held his breath, direct pressure was applied to the site, and the central line was removed. Compression was carried out long enough for the bleeding to stop. An occlusive dressing was done afterwards. The complete procedure was executed according to the standard protocol. About a minute later, his hands and feet curled up, and his body twisted to the left in a seizure-like motion while oxygen saturation level fell below 80%. After applying 10 L of oxygen via oxygen mask, saturation improved to 95%–99%, and his blood pressure and pulse rate was in normal range. CT angiogram of the brain revealed air bubbles along the sulci in the right frontal area, cavernous sinus, and superior sagittal sinus as well as an area of low attenuation in the right frontal lobe, suggesting cerebral air embolism (Figure 1). Hyperbaric therapy was recommended but was not available in our institution or others as well. Therefore, high-flow oxygen therapy was continued. There was little improvement in his consciousness, and the patient remained drowsy and stuporous. Left-side weakness, rigidity, pain on the right lower limb, and right deviation of both eyes could be seen. Because of oxygen desaturation, consistent tachypnea of 30 to 50 breaths/min, and intermittent episodes of myoclonic seizure on the right side, the patient was readmitted to the ICU. Mechanical ventilation was applied during the patient's ICU stay. Transthoracic echocardiogram showed no visible intracardiac shunt, but several bubbles were seen in the left heart after 5 beats during agitated saline study, suggesting the possibilities of an intrapulmonary shunt. Brain magnetic resonance imaging revealed a recent infarct with diffuse swelling and gyral enhancement in the right hemisphere. Another recent infarct in the left frontal and occipital lobes were also noted (Figure 2). Electroencephalography was performed, and its features suggested a structural lesion in the right hemisphere and partial nonconvulsive status epilepticus arising from the right hemisphere, especially in the frontocentral region.

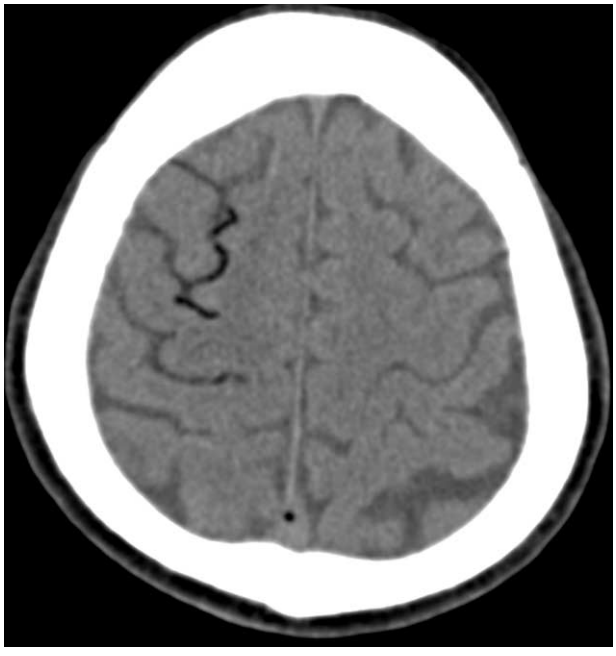

FIGURE 1. Brain CT angiogram showing air bubbles along the sulci in the right frontal area and superior sagittal sinus. CT = computed tomography.

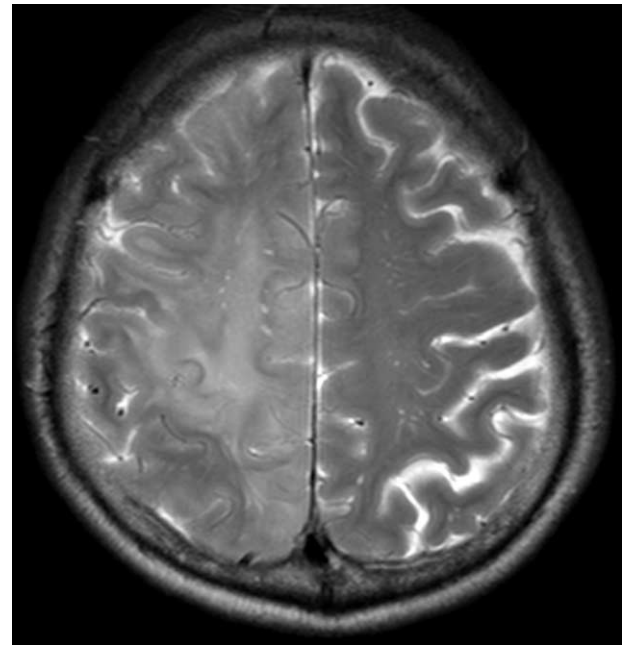

FIGURE 2. Brain MRI that showing signs of recent infarct with diffuse swelling and gyral enhancement in the right hemisphere. Recent infarct in the left frontal and occipital lobes is also seen. MRI = magnetic resonance imaging.

The patient did not show much progress and was treated with antiepileptic drugs and sedatives but still had occasional episodes of seizure and frequent tachypnea, especially when tapering of drugs was attempted. After a brief period of recovery, the patient's condition deteriorated again, and retention of carbon dioxide was sustained due to aggravation of pneumonia. Despite full coverage of antibiotics and maximum care with the ventilator, the patient died about 5 weeks after the removal of the CVC.

DISCUSSION

Among many threats that a CVC may impose, air embolism is a relatively uncommon complication. Despite its rare occurrence, an air embolism can lead to rather serious outcomes and deserves attention from medical practitioners.

Less frequent are cases of cerebral air embolism, which can be explained as emboli obstructing small cerebral arteries causing subsequent neurologic symptoms. In order for cerebral embolism to occur from gas originating from CVCs, gas introduced to the venous circulation must somehow be carried into the systemic arterial circulation. This can be made possible by any means of bypassing the pulmonary capillaries and its normal filtering capacity, a mechanism often called paradoxical embolism.[2–4]

Intracardiac right-to-left shunting (usually through a patent foramen ovale) was the first that suggested explanation for the paradoxic air embolisms (PAEs) and still remains frequently

associated with many PAE cases.[3,5,6] Therefore, an intracardiac shunt was instantly suspected as the cause of the PAE in our case, and to confirm its existence, saline contrast echocardiography was carried out. Despite the initial assumption, the test result showed signs of a suspicious intrapulmonary shunt. Though it was not a definite diagnosis, we view it as a strong candidate for the main mechanism of air embolism.

Saline contrast echocardiography is a technique widely used to detect right-to-left intracardiac shunts and intrapulmonary shunts. After an agent with microbubbles, such as agitated saline solution, is injected intravenously, the appearance of microbubbles in the left heart is detected if any kind of shunt is present. If the bubbles appear in the left atrium within 1 or 2 cardiac cycles after being seen in the right atrium, an intracardiac shunt is suspected. If the arrival to the left atrium is delayed to 4 to 8 cycles after being in the right atrium, as is our case, an intrapulmonary shunt is much more probable.[5,7]

Although not as frequently reported as intracardiac right-to-left shuntings, there are a number of cases in which transpulmonary passage was suggested as the mechanism behind PAEs, which was usually confirmed through echocardiography and sometimes upon autopsy results.[3,6] There are several explanations for the possibility of transpulmonary gas passage, including pulmonary arteriovenous malformations (AVMs). Most pulmonary AVMs are hereditary and related to hereditary hemorrhagic telangiectasia.[4] On the other hand, according to canine studies, certain factors might compromise the ability of pulmonary capillaries to filter out gas emboli, allowing it to pass through the capillary bed. These factors include large volumes of air and the use of vasodilators or anesthetic agents.[6] Another study suggests the existence of inducible intrapulmonary arteriovenous anastomoses in healthy individuals.[5] Lovering et al[5] explains that these intrapulmonary arteriovenous anastomoses stay closed at rest but can open during exercise. Its patency can also be affected by oxygen tension and body positioning. Increased pulmonary artery pressure might possibly act as another factor associated with the functional opening of an anatomic shunt.[3] Accordingly, underlying lung conditions that can cause pulmonary hypertension, such as IPF is like in our case, deserves further research regarding their association with transpulmonary air passage.

The patient's preexisting upper body subcutaneous emphysema also deserves attention, especially because the embolism occurred despite following the protocol. There is a possibility that the air originating from the subcutaneous emphysema entered through the IJV puncture site due to forceful direct pressure. Even if this were true, any kind of right-to-left shunting is still required for a PAE to have happened.

Furthermore, regardless of the origin of the gas, we cannot completely rule out the possibility of a retrograde venous cerebral air embolism. It refers to air in the central venous circulation moving in the opposite direction of the venous blood flow, eventually reaching the cerebral vasculature. This has been proved probable through an experimental model and was considered as the mechanism behind a few cases of cerebral air embolism.[8–10]

The fact that air embolism occurred during intentional removal of a CVC adds more rarity to our case. Air embolism in this situation is less well known compared with cases associated with insertion or accidental removal of the catheter. The removing of the CVC is carried out very quickly, leaving little chance for air to enter.[11] Nevertheless, considering the devastating results possible in air embolism, either pulmonary or cerebral, strict compliance to protocols is required even while removing the catheter. As seen in this patient, however, complication in patients at high risk may occur despite following protocol. Thus, we suggest additional caution to avoid air embolism in high-risk patients.

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