A Rare Case of Cerebral Air Embolism Caused by Pulmonary Arteriovenous Malformation After Removal of a Central Venous Catheter

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Summary: Cerebral air embolism following central venous catheter (CVC) removal is extremely rare. We report a case of cerebral air embolism with loss of consciousness after removal of CVC caused by pulmonary arteriovenous malformation (PAVM). Computed tomography revealed air bubbles in the internal carotid arteries along the sulci in the cerebral hemispheres, as well as a PAVM. The cerebral air embolism was treated with hyperbaric oxygen and intravenous thrombolytic therapy, and transcatheter embolization of the PAVM was performed. When inserting/removing CVC in a patient with a small PAVM, treatment of the PAVM, irrespective of its size, could prevent the type of complication that occurred in our present case.

Key words cerebral air embolism, central venous catheter, pulmonary arteriovenous malformation

INTRODUCTION

A central venous catheter (CVC) is often used for the management of parenteral nutrition, administration of anti-tumor drugs, plasmapheresis, and hemodialysis [1]. Most cases of air embolism associated with CVC use involve pulmonary embolism [2]. Although cerebral air embolism is rare, it is one of the most fatal complications of CVC use. Here we report an extremely rare case of cerebral air embolism that was caused by pulmonary arteriovenous malformation (PAVM) after CVC removal.

CASE REPORT

A 61-year-old woman with a hepatic neuroendocrine tumor underwent hepatectomy of the right lobe of the liver. Preoperatively, a 12-gauge triple-lumen CVC was inserted via the right internal jugular vein under ultrasound guidance with local anesthesia. The CVC was removed with the patient in the supine position on postoperative day 6, with manual compression. Despite a normal blood pressure, the patient experienced a sudden loss of consciousness (Glasgow coma scale: 3), 5 min after CVC removal. Computed tomography (CT) revealed areas with air attenuation along the sulci in both cerebral hemispheres with

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Abbreviations: CT, Computed tomography; CVC, central venous catheter; MRI, magnetic resonance imaging; PAVM, pulmonary arteriovenous malformation.

greater attenuation on the right side (Fig. 1). In addition, air bubbles were detected in the bilateral internal carotid arteries (Fig. 2A, B).

Diffusion-weighted magnetic resonance imaging (MRI) revealed cortical areas in the right frontal and parietal lobes as well as the left cerebellar hemisphere with restricted diffusion near the air. MRI suggested a cerebral air embolism (Fig. 3). Ninety minutes after CVC removal, the patient regained consciousness, but

demonstrated left-sided paralysis. The cerebral air embolism was treated with hyperbaric oxygen and intravenous thrombolytic therapy, which resulted in complete recovery from paralysis. Transesophageal echocardiography revealed no visible intracardiac right-to-left shunting, such as that observed with an atrial septal defect. Preoperative CT had revealed a PAVM in the middle lobe of the right lung (Fig. 4). Subsequent pulmonary arteriography confirmed the

Fig. 1. Brain computed tomography revealed air bubbles in considered cortical veins that were examined (arrows) in both cerebral hemisphere.

Fig. 3. Brain magnetic resonance imaging (diffusion-weighted- imaging) revealed restricted diffusion in the cortical area (arrows) near the air embolism seen observed on the brain computed tomography.

A

B

Fig. 2. (A, B) Brain computed tomography revealed air bubbles in bilateral internal carotid arteries (arrows).

Fig. 4. CT revealed a PAVM in the middle lobe of the right lung (arrow).

presence of the PAVM (feeding pulmonary artery diameter, 1.8 mm) (Fig. 5A). Considering that the patient was scheduled to undergo further CVC for chemotherapy we treated the small PAVM by transcatheter coil embolization to avoid complications, such as cerebral air embolism, infarction, or abscess formation.

Pulmonary arteriography also confirmed the complete absence of anatomic shunting immediately after the feeding pulmonary artery and sac embolization (Fig. 5B). There were no complications related to endovascular treatment.

DISCUSSION

Cerebral air embolisms are uncommon complications of CVC removal. However, the complications can be severe, with a reported mortality rate as high as 23% [3].

The reported incidence of CVC-associated air embolism ranges from 0.1% to 2% [4].

Cerebral air may originate from the CVC through a retrograde mechanism directly and/or by paradoxical embolus formation via right-to-left shunting [5].

A

B

Fig. 5. (A) Digital subtraction pulmonary angiogram revealed a simple type of pulmonary arteriovenous malformation in the middle lobe of the right lung.

(B) A pulmonary arteriogram confirmed the complete absence of the shunt immediately after the feeding pulmonary artery and sac embolization.

TABLE 1.
The procedure for the removal of central venous catheters to prevent air embolism

Place the patient in the Trendelenburg or head-down position.
Remove the catheter only after the patient takes a deep breath and holds it.
Apply pressure to the catheter insertion site for 5 min.
Apply an airtight dressing to the catheter insertion site.

Intracardiac right-to-left shunting is frequently associated with paradoxical air embolism [1]. The cause of CVC-related cerebral air embolism in our case was believed to be right-to-left shunting due to a PAVM. We reached this conclusion for three reasons. First, brain CT revealed air bubbles in the bilateral internal carotid arteries. Second, the CVC was removed in the supine position with manual compression. Third, the PAVM was located in the anterior part of the lung (segment 5) under supine position.

Intravascular air can lead to both arterial and venous infarcts [6]. In our case, MRI revealed multiple infarcts in the cortical area. Other studies have reported similar MRI findings in cases with cerebral venous air embolism [6, 7]. Cerebral infarcts are often observed in the cortical area near air bubbles [6]. Mechanisms that lead to air embolism ischemia are blood flow obstruction, vasospasm, and thrombus formation due to platelet activation [6]. The blood-brain barrier is compromised by the migration of air bubbles to the arteries and arterioles because of endothelial damage [8, 9]. Furthermore, air bubbles cause air embolisms and inflammatory reactions, such as margination and activation of leukocytes, resulting in cerebral edema or secondary ischemia [10]. In many cases, right-to-left shunting is frequently associated with a paradoxical air embolism [1]. The most common mechanism for right-to-left shunting is through a patent foramen ovale [11]. Although not as frequent as intracardiac right-to-left shunting, numerous cases have been reported where transpulmonary passage has been suggested to be the cause of a paradoxical air embolism [12]. PAVMs are abnormal vascular structures that most often connect pulmonary arteries to pulmonary veins while bypassing the normal pulmonary capillary bed, resulting in intrapulmonary right-to-left shunting [13]. A paradoxical air embolism across a PAVM has been suggested as a cerebrovascular complication. Usually, a symptomatic PAVM or one with a feeding artery diameter of ≥ 3 mm, regardless of the symptoms, should be treated with transcatheter embolization or surgical intervention [14]. Smaller feeding arteries are often left untreated. However, neurological complications

have also been described in patients with small PAVMs that were left untreated; there is no evidence to support a 3 mm feeding artery diameter as the critical size that can potentially result in complications. It has not been possible to stratify risks according to the size of the feeding artery [14]. We recommend carefully following the appropriate protocol when removing a CVC to prevent air embolism (Table 1). We conclude that careful CVC removal can avoid air embolisms. When inserting/removing a CVC in a patient with a small PAVM, treatment of the PAVM should be considered, irrespective of its size, to avoid the complications seen in this case.

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