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Cerebral Arterial and Venous Air Embolism Following Removal of Percutaneous Sheath Introducer

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Abstract: Cerebral air embolism after removal of central venous catheter (CVC) is a rare complication but can lead to fatal outcomes. We report a rare case of both cerebral venous and arterial embolism occurring in a patient with underlying scleroderma-related interstitial lung disease (SSc-ILD) and pulmonary hypertension following removal of percutaneous introducer sheath for pulmonary artery catheterization. We discuss the mechanisms, pathophysiology, management and prevention of cerebral air embolism.

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Introduction

Central venous catheters are used extensively in critical care for hemodynamic monitoring, medication administration, hemodialysis and transvenous pacing. Cerebral air embolism is a rare, but potentially fatal complication of central venous catheterization. Venous air emboli can paradoxically enter the arterial circulation through a patent foramen ovale leading to cerebral air embolism and cause neurological compromise. We report a case of cerebral arterial and venous air embolism that occurred after removal of central venous catheter in a patient with interstitial lung disease secondary to systemic sclerosis and severe pulmonary hypertension. In addition, we discuss methods to prevent air embolism related to central venous catheterization.

Case report

A 57-year-old male with a history of interstitial lung disease (ILD) secondary to systemic sclerosis presented to the pulmonary clinic with progressive dyspnea. He had been on supplemental oxygen at 2 liters per minute, but his requirements had been rapidly increasing over the past few weeks. He denied any fever, chills, productive cough or chest pain. Due to worsening hypoxia, he was admitted for further workup. He was on high flow nasal cannula at 20 liters per minute in the medical intensive care unit. Computed tomography (CT) of the chest showed a pattern consistent with usual interstitial pneumonia with lower lobe predominant bilateral reticular opacities in the posterobasilar distribution, volume loss, traction bronchiectasis and honeycombing. No consolidations, effusions or ground glass opacities were noted. Transthoracic echocardiography showed dilated right ventricle with depressed systolic and diastolic function, abnormal septal motion, elevated right ventricular systolic pressure at 84 mm Hg. No interatrial shunting was visualized on colour Doppler. Right heart catheterization was done at bedside which showed mean pulmonary artery pressure of 43 mm Hg, pulmonary capillary wedge pressure 10 mm Hg, cardiac output 3.8 liters per minute, cardiac index 1.9 l/min/m^2, systemic vascular resistance 2803 dynes×s/cm^5 (35 Wood units), pulmonary vascular resistance 695 dynes×s/cm^5 (8.7 Wood units) which was consistent with severe group 1 pulmonary hypertension.

Patient was started on ambrisentan and tadalafil in addition to prednisone and mycophenolate for ILD. He responded well to therapy with symptomatic improvement and decrease in oxygen needs to 2 liters per minute. The patient was prepared for discharge with removal of Swan Ganz catheter. He was placed in supine position in bed as he did not tolerate the Trendelenburg position. The patient was instructed to hum as the sheath introducer was pulled out. A sterile gauze dressing was applied and taped in place. The patient was alert and conversant

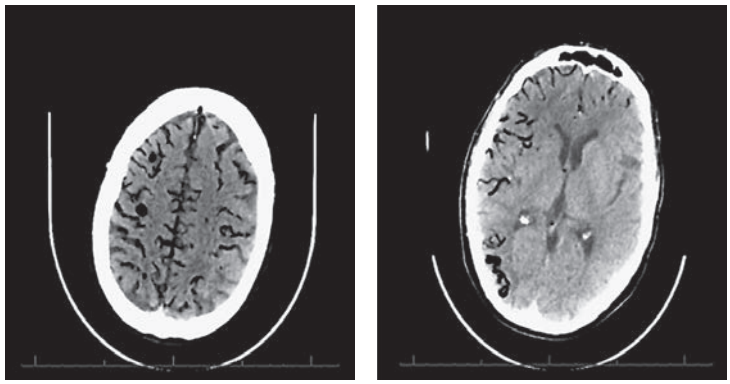

Figure 1 – Computed tomography of head showing extensive air in the vascular territory overlying both cerebral hemispheres. Gas is seen predominantly within the cerebral veins, superior sagittal sinus and right cavernous sinus. Several pockets of gas are seen in the right frontal region which may represent gas within focally dilated cerebral veins or adjacent sulci.

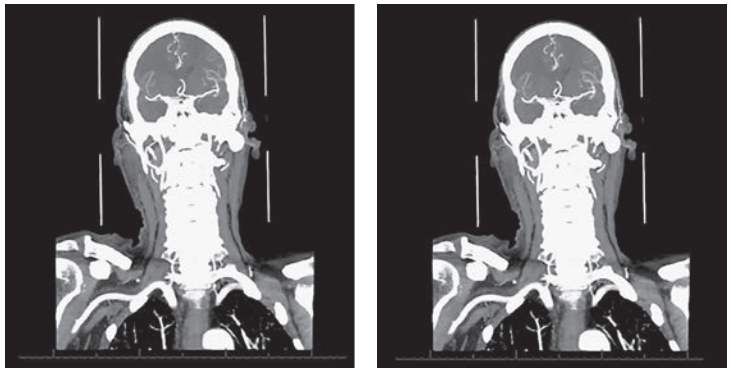

Figure 2 – Computed tomography angiography of head and neck (maximum intensity projection images) showing decreased arterial density in the upper right cerebral arteries and, to a lesser extent, upper left cerebral arteries indicating decreased perfusion. This would suggest air within distal small arteries.

at the time being assisted into the chair. Within a few minutes, he was noted to be unresponsive with horizontal nystagmus and generalized tonic clinic seizure activity. CT of the head was done immediately afterwards showing air within the cerebral veins, superior sagittal sinus and right cavernous sinus (Figure 1). CT angiography of the head showed decreased arterial density in the upper cerebral arteries (right > left), suggestive of air within distal small arteries (Figure 2). Patient was placed on 100% oxygen in Trendelenburg position. Bedside echocardiogram showed dilated right ventricle (RV), normal left ventricular ejection fraction with small amount of bubble artifact in the left atrium and left ventricle. Despite supportive measures, patient did not improve and passed 12 hours after the insult. Autopsy

showed multifocal subacute microinfarcts in occipital lobe and cerebellum, focal subarachnoid hemorrhage in the occipital region, and hypoxic ischemic changes in the hippocampus, cerebellum, and occipital lobe.

Discussion

Air embolism is an uncommon, but potentially lethal complication that occurs as a consequence of air entry into the vasculature. The reported incidence rate is 0.03–2% (Nagai et al., 2014). It can be classified into venous and arterial air embolism. Venous air embolism occurs when air enters the systemic venous circulation and travels to the right ventricle and pulmonary circulation causing interference with gas exchange, pulmonary hypertension, right ventricular strain, cardiac arrhythmias, and eventually cardiac failure (Muth and Shank, 2000). Arterial air embolism occurs when air enters the arterial circulation. It is a more serious occurrence and can cause ischemia in any organ with poor collateral circulation. The most common etiologies of air embolism include surgery, trauma, vascular interventions, barotrauma from mechanical ventilation (Muth and Shank, 2000) or as a result of paradoxical embolism.

Air embolism can occur during central venous catheter (CVC) insertion, manipulation, or removal. During catheter insertion, venous air embolism may occur from failure to occlude the needle hub and/or catheter, fracture or detachment of catheter connections (Inamasu et al., 2001). The risk is increased if the patient is hypovolemic with reduced central venous pressure (CVP). Increased negative intrathoracic pressure due to deep inspiration or upright positioning of the patient reduces CVP to below atmospheric pressure putting patient at risk for air entering very rapidly into the venous circulation (Pronovost et al., 2004).

Air embolization may lead to three outcomes: delivery to pulmonary circulation, to the systemic circulation via anatomic right to left shunts, or retrograde ascension to the cerebral venous system. Migration of emboli to the pulmonary circulation causes rise in pulmonary arterial pressure and increased resistance to right ventricular outflow which in turn causes diminished pulmonary venous return. This causes decreased left ventricular preload, diminished cardiac output and, ultimately, systemic cardiovascular collapse (Durant et al., 1947).

Paradoxical air emboli, such as cerebral arterial air emboli, are caused when air that has entered the venous circulation manages to enter the systemic arterial circulation and causes end-artery obstruction. The mechanisms by which this can occur can be broadly divided into intracardiac or intrapulmonary right-to-left shunts. Intracardiac shunts include passage of air via patent foramen ovale, atrial or ventricular septal defects. Elevated pulmonary arterial pressure due to venous air embolism may lead to an elevated right atrial pressure exceeding the left atrial pressure, causing right-to-left shunt of air through a patent foramen ovale, septal

defect or functional right-to-left shunt due to severe pulmonary hypertension. Intrapulmonary shunting of air bubbles can occur through pulmonary arteriovenous malformations. In some cases, venous gas may enter the arterial circulation by overwhelming the ability of the pulmonary circulation to filter out gas emboli. Animal studies suggest that a large bolus of gas (20 ml or more) or small continuous amounts (11 ml per minute) introduced into the venous system may generate intraarterial bubbles (Muth and Shank, 2000).

Cerebral venous air embolism can also result from retrograde flow of air against the direction of blood flow as it enters the venous circulation. Gas bubbles rise in blood due to their lower specific gravity (Chuang et al., 2019). This depends on bubble size, central vein diameter, cardiac output and position of patient's head above the heart. In veins above the level of the heart, atmospheric pressure is higher allowing air to enter the bloodstream and rise against gravity causing cerebral venous occlusion and infarction that does not follow typical arterial vascular distribution (Schlimp et al., 2014). Experimental studies have shown that retrograde venous air emboli can occur in certain settings, such as the patient being either supine or at least at an angle of 45 degrees to the horizontal plane, venous valve insufficiency, and increased right sided cardiac pressures (Schlimp et al., 2005; Fracasso et al., 2011).

Cerebral arterial embolism typically involves the migration of gas to small arteries, average diameter 30–60 micrometers. The emboli cause pathologic changes by obstructing end-arterial flow causing distal ischemia and cytotoxic edema. The surface of the bubble mechanically irritates the arterial endothelium and also generates an inflammatory foreign body response resulting in vasogenic edema and greater perfusion impairment (Mathieu et al., 2006).

Heckmann et al. (2000) studied cerebral embolism as a complication of CVC and found that subclavian vein puncture access was more likely to result in cerebral air embolism. This is possibly because the site of skin penetration is slightly higher than with internal jugular catheters hence air is more likely to entrain due to the increased pressure gradient between the atmosphere and the venous system of the neck (Heckmann et al., 2000). The overall mortality rate was 23% in these patients (Heckmann et al., 2000).

The rate and volume of air introduced also plays a role in the effect of the air embolus. Large, rapid boluses of air are more likely to result in complications than slow infusions of small amounts of air. The estimated fatal dose is 300–500 ml of air introduced at 100 ml/s (Ordway, 1974). This can occur through a 14-gauge catheter with a pressure gradient of 5 cm H_2O (Ordway, 1974). In canine models, gas entering the venous system at a rate more than 0.3 ml/kg per minute overwhelms the ability of the lungs to filter and results in arterial air emboli and tissue ischemia (Orebaugh, 1992).

Introducer catheters are large bore intravenous catheters that are combined with a valve side-port apparatus, which allows for penetration by a multi-lumen or pulmonary artery catheter (PAC). Hemostasis valve is an integral part of the

introducer sheath and is designed to prevent fluid from the inside lumen of the catheter from leaking when there is positive intraluminal pressure and to prevent air from entering the catheter when there is negative intraluminal pressure. Introducer sheath self-sealing valves maintain competence with pressure changes under simulated *in vivo* pressure conditions of up to –30 cm water pressure (MacGregor et al., 1998). It is recommended that an obturator cap be used over the valve to prevent air embolism when the catheter is not in use. This cap seals the external surface of the valve apparatus using an O-ring located on the external surface of the side port-valve assembly. The obturator also penetrates through the valve itself to provide the mechanical seal. Introducer valves that are penetrated with a PAC or other catheter must be observed closely for leakage of fluid around the PAC. This implies a faulty valve that may entrain air at low pressures. Data demonstrates that in the absence of visible leakage, the least amount of pressure required to entrain air was –125 mm Hg (MacGregor et al., 1998). The maximum negative intrathoracic pressure that can be generated in humans remains unclear however studies using maximal inspiratory pressures at the mouth have ranged from approximately –50 to –150 mm Hg, and esophageal transducer experiments during a sniff test have shown maximal transdiaphragmatic pressures in healthy adults to range from –82 to –204 cm H_2O (–61 to –152 mm Hg) (Black and Hyatt, 1969; Leech et al., 1983; Wilson et al., 1984).

Diagnosis requires a high degree of suspicion, particularly in case of neurologic symptoms around the insertion, use or removal of a CVC. Computed tomography of the head or magnetic resonance imaging done immediately after reveals the presence of air in the cerebral vasculature, but may be false negative if there is a delay in imaging (Caulfield et al., 2006). Bedside echocardiogram with agitated saline injection is helpful to differentiate between intracardiac shunt or intrapulmonary shunt.

Treatment is supportive. The patient is placed in the Trendelenburg position on the left, known as Durant's maneuver (Heckmann et al., 2000). This keeps air trapped in the heart away from the right ventricular outflow tract and may help in reducing the blockage of the vasculature. Oxygen therapy is recommended but it remains unclear whether hyperbaric oxygen therapy has any role (Heckmann et al., 2000).

Aspiration of air during venous air embolism may be required in case of an "air lock" in the right ventricular outflow tract. Multilumen or Swan Ganz catheters have been shown to be ineffective in aspirating air, with success rates between 6 and 16% (Bedford et al., 1981; Colley and Artru, 1987, 1989; Bowdle and Artru, 1988; Hanna et al., 1991; Artru, 1992). This may be related to the narrow luminal diameter, but offers the highest chance of success when there is already a catheter near the right atrium or ventricle. The best available device that has been studied is the Bunegin-Albin multiorifice catheter with success rates ranging from 30–60% (Colley and Artru, 1987, 1989; Bowdle and Artru, 1988). It has been reported that in a case

of venous air embolism, the withdrawal of 15 ml of air percutaneously from the right heart resulted in prompt hemodynamic improvement (Stallworth et al., 1950). Typically, 15–20 ml of air may be aspirated using this technique. Currently no data is available to support emergent catheter insertion for air aspiration during acute hemodynamic compromise in the setting of venous air embolism.

Prevention is the best treatment. It is imperative to correct dehydration before the procedure to increase CVP for decreasing the gradient that is necessary for air embolism to occur. During CVC placement, ensuring occlusion of needle hub and catheter (Pronovost et al., 2004), keeping connections secure when not in use prevents air embolism from the catheter (Opeskin et al., 1998). CVC removal must always be done in supine or Trendelenburg position with Valsalva maneuver to increase CVP. An impermeable dressing consisting of petrolatum gauze, sterile gauze and transparent dressing should be applied after removal (Hsiung and Swanson, 2000) and pressure should be held to the site for 1 to 5 minutes (Thielen and Nyquist, 1991), preferable longer in order to close off any patent catheter tracts.

In our patient, cerebral air embolism occurred likely due to two mechanisms. A large volume of air entrainment into the venous circulation through a dysfunctional one-way valve in the sheath introducer for pulmonary artery catheter led to a paradoxical air embolus through a functional right-to-left intracardiac shunt due to severe pulmonary hypertension (Eisenmenger physiology). There was also retrograde flow of air through the internal jugular vein to the cerebral venous vasculature as demonstrated in cranial imaging studies.

Conclusion

Cerebral air embolism is an exceptionally rare complication of central venous catheterization and can be associated with high mortality. A high clinical suspicion must be present in any patients exhibiting neurologic symptoms surrounding CVC insertion, manipulation or removal. Prevention and prompt diagnosis may decrease morbidity and mortality.

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