Pearls & Oy-sters:
Cerebral venous air embolism after central catheter removal
Too much air can kill

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PEARLS

1. Cerebral air embolism is a rare but potentially catastrophic consequence of central venous catheter removal.
2. Treatment is often supportive; hyperbaric therapy should be considered but is not readily available in most hospitals and its efficacy for cerebral venous air embolism is unknown.

OY-STERS

1. Place the patient in the Trendelenburg position before removing the catheter to minimize the risk of air emboli going to the brain.
2. Clinical suspicion of this diagnosis is essential, as signs and symptoms are not specific and brain imaging may not show the presence of air in the cerebral vasculature.
3. Even patients without patent foramen ovale are at risk for cerebral air embolism.

CASE REPORT A 95-year-old woman with a history of hypertension presented from a long-term care facility for altered mental status in the context of dehydration and hypoglycemia. Multiple attempts to obtain a peripheral IV failed, and a triple lumen catheter was placed into the right internal jugular vein using the Seldinger technique under ultrasound guidance. No complications followed the procedure. The patient was then rehydrated and given multiple ampules of D50, leading to prompt resolution of the hypoglycemia and improvement in her mental status. After resolution of symptoms, the central line was removed, with the head of the bed at 30°. Within minutes of removal of the central line, the patient developed severe respiratory distress and hypoxia with desaturation to 80% on a non-rebreather mask with 100% oxygen. On examination, the patient was unconscious and gasping for air with severe suprasternal retractions; she was tachypneic and tachycardic, and her blood pressure was 210/100 mm Hg. All limbs were flaccid. She was placed in the left lateral decubitus position as resuscitation efforts were activated. She was then intubated and transferred to the intensive care unit.
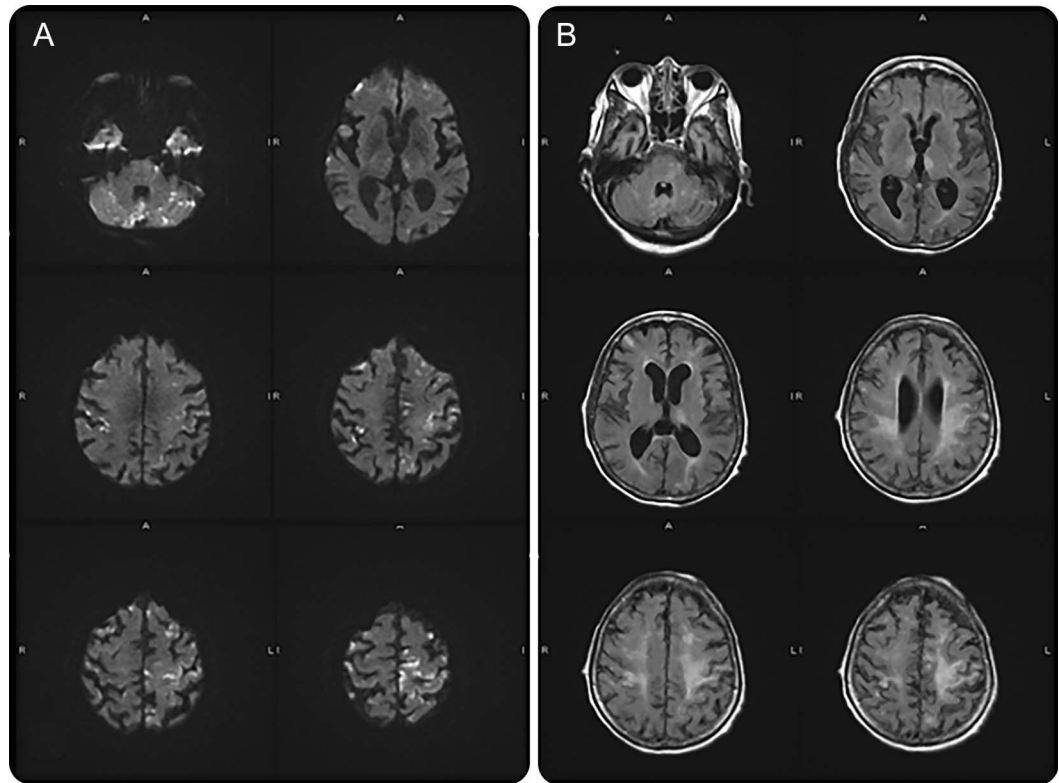
A stat CT angiogram of the head and neck did not show signs of stroke, bleeding, or vascular occlusion. Chest CT with contrast showed dilation of the distal esophagus with fluid and debris within the distal trachea and left mainstem bronchus, without evidence of pulmonary embolism, and consistent with aspiration. An echocardiogram revealed an ejection fraction of 79%, normal left ventricular function, and mildly dilated left atrium, but no thrombi, vegetations, or evidence of patent foramen ovale (PFO) with agitated saline contrast. Troponin I peaked about 8 hours after the event (0.468, normal range 0–0.034), simultaneous creatine kinase MB was elevated (4.92, normal range 0–2.30), and multiple EKGs were consistent with non-ST elevation myocardial infarction. Prolonged cardiac telemetry failed to show atrial fibrillation.

Brain MRI showed areas of restricted diffusion bilaterally in the cerebellum, temporal lobes, frontal lobes, left occipital lobe, and thalami, consistent with different arterial vascular territories, including middle, anterior, and posterior cerebral arteries. It also showed restricted diffusion in the left more than right parasagittal regions along the cortical gray matter in a gyriform pattern. Additional abnormalities were seen much more on fluid-attenuated inversion recovery sequence than on diffusion-weighted imaging (including bilateral thalamus and confluent white matter changes in both hemispheres), suggestive not only of multiple embolic infarctions, but also of extensive vasogenic edema, with edema out of proportion to the ischemic lesions (figure). No source of stroke was identified and a clinical diagnosis of air embolism was made.

A 4-hour video EEG did not show epileptiform activity. The patient was extubated 5 days after the event. Her neurologic examination continued to be poor: she was alert but had no verbal output and could not follow commands; pupils were symmetric and reactive to light. She had blink to threat bilaterally and minimal movements of all extremities except for the right arm, which moved off the bed to noxious stimuli. This was a significant change, as the patient could talk and had no major neurologic deficits at baseline.

Figure   Brain MRI reveals multiple infarcts throughout the supratentorial and infratentorial parenchyma

(A) Diffusion-weighted imaging sequence shows areas of restricted diffusion bilaterally in the cerebellum, temporal lobes, frontal lobes, left occipital lobe, and thalami, consistent with different arterial vascular territories, including middle, anterior, and posterior cerebral arteries. Restricted diffusion is also observed in the left more than right parasagittal regions along the cortical gray matter in a gyriform pattern. (B) Fluid-attenuated inversion recovery sequence shows additional abnormalities including the bilateral thalami and confluent white matter changes in both hemispheres, suggestive not only of multiple embolic infarctions but also of extensive vasogenic edema.

DISCUSSION Cerebral air embolism is a rare cause of stroke and the diagnosis can be difficult because signs and symptoms are often nonspecific. One of the procedures with a high risk for air embolism is the removal of central venous catheters, which is a fairly common procedure performed by all residents.

Presentation is varied and can be characterized by acute onset of various degrees of altered mental status, headache, dizziness, chest pain, paresthesias, seizures, hemiparesis, aphasia, akinetic mutism, and homonymous hemianopia. Up to 50% of patients will be unresponsive at some time.[1,2] Following air embolism, severe cardiovascular and pulmonary dysfunction can be seen, depending on the amount of air and the location within the body where the bubbles are entrapped.[3]

There are different etiologies for arterial gas embolism. The first mechanism is by direct infusion of air bubbles into the pulmonary vein. Pulmonary vessels act as a filter for air bubbles that originate in the venous circulation and provide protection for the coronary and systemic circulation. If the filter is overburdened, the air emboli can penetrate the filter. The second mechanism is passage of venous bubbles to the arterial circulation via any right to left shunt, including a PFO.[4] Interestingly, a review of the literature showed that a saturated physiologic arteriovenous pulmonary shunt was the reason for right-left shunting in 60% of such patients.[1]

The passage of bubbles can transiently obstruct cerebral blood flow, but if the bubbles are small, they will rapidly be absorbed without significant damage, and symptoms will resolve. On the other hand, large air emboli can take hours to be absorbed, and blood flow can be significantly impaired. Oxygenation can fall below the levels required for neuronal function and survival, resulting in stroke.[5] Some investigators have hypothesized, based on an experimental model, that if air bubbles are detected by CT, their volume is likely to be larger than 10^{-2} mL. Based on this model, they speculated that these bubbles are reabsorbed very slowly (hours up to days) and this could justify hyperbaric oxygen therapy even if delayed.[5] There are not enough data to justify a precise recommendation based on the bubble size and further studies are needed.

Much of the existing literature with regards to air emboli is focused on arterial strokes, but intravascular air can also result in venous infarcts.[6] In our case, the

MRI revealed multiple infarctions in different arterial territories, and also showed bihemispheric areas of restricted diffusion in a gyriform pattern along the cortical gray matter. As previously observed in other reports, this MRI finding may be seen in cases of cerebral venous air embolism and is thought to be related to the number and size of the air emboli. Multiple small emboli may obstruct the blood flow in end-artery and cortical vein territories and cause infarctions.[6,7] The pathophysiology of retrograde venous air embolism is poorly understood, but it appears from laboratory data that air bubbles have a high probability of rising retrograde against venous blood flow when the patient is in the upright position.[8] Moreover, the contact of bubbles with the endothelium of the blood–brain barrier results in an inflammatory response leading to the breakdown of the barrier and cerebral edema, causing decreased blood flow, as was likely in our patient.

Cerebral air embolism should be suspected in any patient presenting with acute neurologic dysfunction following the insertion or removal of a central venous catheter. The risk of embolism seems to be increased during procedures performed in the sitting position[9]; therefore, patients should always be placed in a Trendelenburg position to minimize the risk. The procedure should be performed at the end of inspiration. Immediately after removing the catheter, an impermeable dressing should be applied to the site and pressure should be held for at least 5 minutes.[10]

The diagnosis of cerebral venous air embolism can be challenging, as no imaging technique alone has shown sufficient diagnostic accuracy,[2] and the diagnosis mainly relies on clinical presentation and temporal correlation with catheter manipulation. Head CT can be diagnostic if done immediately after the onset of symptoms, before air is reabsorbed.[11]

Treatment of cerebral air embolism is mainly supportive and aimed at increasing oxygenation and reducing complications. Administration of oxygen not only reduces hypoxemia, but also decreases air bubble size.[12] Sometimes ventilatory support is necessary to achieve normocapnia. Systemic hypertension as the result of bubble entrapment in the cerebral circulation is expected and it may be considered therapeutic for a brief period of time, because it can help to spread the emboli through different vascular beds, including veins, capillaries, and arterioles. Prolonged hypertension needs to be avoided, and normotension should be achieved.[12] While hyperbaric oxygen treatment is generally considered useful in cases of arterial gas embolism,[12] little is known about its efficacy for venous air embolism. Moreover, not many hospitals have immediate and easy access to a hyperbaric chamber, making this treatment difficult for logistical reasons.[1]

Our case is of particular interest for different reasons. First, it describes a rare but potentially catastrophic complication of central venous catheter removal, a procedure that is commonly performed by residents of all specialties. Second, the distribution of the infarctions is consistent not only with different arterial territories as may be seen in embolic strokes, but the evidence of areas of restricted diffusion along the cortical gray matter raises the possibility of venous air embolism with cortical damage in end-artery and cortical vein territory. In fact, while arterial embolism is a well-recognized entity, only recently has the scientific literature started to focus on the possible pathophysiology, presentation, and treatment of cerebral venous air embolism.

AUTHOR CONTRIBUTIONS

Dr. Luca Bartolini designed and drafted the article. Dr. Kathleen Burger contributed to the design of the article and reviewed the manuscript.

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