Iatrogenic cerebral venous air embolism

A 62-year-old male patient was brought to our radiological department due to sudden loss of consciousness, visual deviation towards the left and left sided Babinski present.

Imaging Findings:

A 62-years-old Caucasian white male patient had undergone a heart transplant 4 weeks prior to this incidence. Shortly after arrival, an anaesthesiologist inserted a 3-lumen central venous catheter (CVK) into the right subclavian vein, where air leakage was noticed on the day of the event. This same day the patient underwent a routine heart biopsy through the right jugular vein. Later that evening the patient syncopated. An emergency non-enhanced cerebral CT scan and cerebral CT angiography was performed. The studies revealed intracranial air, mainly located in the cortical veins, but also scattered in the cerebral parenchyma. There was a predominance of intracranial air on the right side. No certain signs of intraarterial air embolism were detected, neither were areas of infarction nor bleeding diagnosed. The patient was immediately sent for treatment with hyperbaric oxygen (HBO) and treated for 3 consecutive days. Follow up cerebral CT showed complete regression of intracranial air already after the first treatment. Unfortunately, ischemia and oedema of the brain became apparent, and the patient was declared dead 10 days following the syncopical episode.

Discussion:

With insertion, removal or manipulation of a CVK, passive air suction through the catheter may occur. This is most likely to happen in the superior vena cava of a patient sitting or standing when the intravenous pressure is lower than the atmospheric pressure. Air may also enter a catheter that is not properly closed, due to intrathoracic pressure being below atmospheric pressure during inspiration. In the erect position, air bubbles with their low weight may travel retrograde in veins when the air has higher velocity than blood. With the patient in question, we assume that retrograde migration of air in cranial direction must have occurred. Arterial air embolism is a rare complication during invasive medical or surgical procedures. However, air embolism secondary to insertion, removal or use of a CVK is more common than previously believed. Health professionals must be aware of this complication. It is important to perform the best medical practice possible during these procedures, including keeping the patient in Trendelenburg’s position.

Typically, a relatively large amount of venous gas is well tolerated. Air in the veins may cause arterial embolism through a patent foramen ovale. From there arterial gas has direct access to the systemic circulation.

In general, the symptoms from air embolism are related to organ involved, such as chest pain, dyspnoea or cardiac arrest referring to involvement of lungs and heart. Haemoptysis may occur with pulmonary barotrauma. Clinical signs of cerebral air embolism may be unconsciousness, and generalised or focal seizures. Other symptoms reported are confusion, headache, dizziness, hemiplegia, sensory loss and visual disturbances, to name a few.
Besides myocardial ischemia, which is more commonly seen in arterial gas embolism, there is no major difference in clinical presentation between arterial and venous air embolism.

Air embolism is an emergency, and treatment includes attempts to remove the intravascular air, exit the current procedure, O2 therapy and perform external cardiac massage when cardiac abnormalities are seen. Despite the lack of randomised controlled clinical studies, it is recommended to initiate hyperbaric oxygen treatment immediately. This treatment can be provided in a pressure chamber. With high ambient pressure HBO will reduce the bubble size followed by a decreased obstruction of the vessel and thereby initiating reperfusion. The gas will then be forced into the systemic circulation and through the lungs for filtration. Air emboli can result in significant morbidity, sometimes death. The outcome is less dependent on the type and source of the air embolism, on the contrary, the presence of Babinski signs at the time of admission to the hospital is associated with poor long-term outcome. As already stated, it is essential to start treatment with HBO immediately, whereby treatment within 7 hours or less is associated with better outcome.

In order to confirm or disprove the diagnosis, the patient should be examined with CT, preferably with CT angigraphy, alternatively MRI.

**Differential Diagnosis List:** Iatrogenic cerebral venous air embolism

**Final Diagnosis:** Iatrogenic cerebral venous air embolism

**References:**


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**Figure 2**

Description: Axial unenhanced CT images show complete remission of intravenous air after the first treatment with HBO. Unfortunately, right hemispheric swelling is evident. Origin:
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Figure 4

Description: Coronal image. Air in the sagittal sinus and no signs of arterial air embolism. Origin:
**Description:** Sagittal image. Air in the sagittal sinus and no signs of arterial air embolism. **Origin:**

**Description:** Axial image. Reduced perfusion in the right hemisphere which is thought to be due to venous air obstruction. There are also signs of cerebral edema in the hypoperfused area. **Origin:**
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Description: CTA demonstrating air in the cerebral veins, preferably in the sagittal sinus. Origin:
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