Texas Society of Neuroradiology (TSNR)
Excerpta Abstract
2026 Annual Meeting – Dallas, TX
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Radiologic Diagnosis of Cerebral Air Embolism: A Rare but Critical Entity

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Clinical History

A 70 year-old-male with a history of AML s/p remote bone marrow transplant, graft vs host disease and pulmonary fibrosis initially presented to an outside hospital after a mechanical ground level fall wherein he sustained a scalp laceration without loss of consciousness. Initial imaging workup showed pneumomediastinum and no acute intracranial findings.

Early the following morning, the patient reported bilateral visual loss and was found to have punctate acute ischemic infarcts in the bilateral occipital lobes. Shortly afterwards, mental status declined and he became encephalopathic with a GCS of 3, tachycardic with respiratory distress, hypoxia and fever. A repeat CT head in the early afternoon showed right frontal intravascular air.

The patient was transferred to our institution the next day in order to potentially undergo hyperbaric oxygen therapy. A repeat CT the following day showed resolution of air, and MRI brain with and without contrast showed findings consistent with extensive sequela of recent cerebral air embolism. By then, the patient was not deemed a candidate for hyperbaric treatment and was subsequently treated with FiO2 at 100%.

Imaging Findings

Non-contrast CT showed extra-axial intravascular air along sulci in superior right frontal lobe extending to the paracentral lobule. Repeat CT head showed resolution of air with new right frontoparietal parenchymal hypoattenuation in the region of prior air, consistent with acute ischemic infarcts from cerebral air embolism.

MRI brain with and without contrast showed findings consistent with extensive sequela of recent cerebral air embolism with extensive bihemispheric (right more than left) mixed cytotoxic and vasogenic edema with a predominant distal vascular bed/watershed distribution, with associated avid perivascular and leptomeningeal enhancement throughout these regions.

Discussion

Cerebral air embolic ischemic infarcts are rare, yet potentially catastrophic events. The air may enter via the arterial or venous route, including iatrogenic (most often) causes such during invasive procedures (i.e. central venous catheter manipulation, intravascular injection, endovascular procedures, surgeries) or non-iatrogenic causes such as trauma or decompression sickness.

The pathophysiology of injury with cerebral air embolic infarcts is comprised of three main mechanisms which account for the MR imaging manifestations, including direct ischemia from end artery occlusion by the gas emboli (cytotoxic edema), inflammation and blood-brain-barrier breakdown resulting from a gas-bubble related foreign body reaction (enhancement), and elevated venous pressure promoting vasogenic edema and potentially venous infarctions.

While CT may directly show the extra axial air, it is not a reliable/sensitive finding given the rapidity at which it may be resorbed (as shown in this case with the resolution of air on CT the following day).

Clinically, patients often present with nonspecific encephalopathic symptoms. Treatment includes hyperbaric oxygen therapy (if appropriate) and 100% FiO2.

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Teaching Point

The MR imaging patterns of cerebral air embolic infarcts is characterized by a distal border-zone predominant pattern of mixed cytotoxic and vasogenic edema along with leptomeningeal and perivascular enhancement. Of note, the finding of enhancement in the acute phase is a relatively underrecognized/underreported feature, only recently brought to attention in a case series published in AJNR in August 2024.

References

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Figures