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Image Report

Acute and subacute neurovascular impact of cryptogenic air emboli

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ABSTRACT

Background: Cerebral air embolism is a rare cause of acute ischemic stroke that is becoming increasingly well-described in the literature. However, the mechanism and severity of this type of injury can vary, with ischemia typically emerging early in the course of care. To the best of our knowledge, delayed ischemia in this setting has not yet been described.

Case Description: A stroke code was called for an unresponsive, hospitalized, 75-year-old man. A computerized tomography (CT) scan of the head revealed air within the right greater than left hemispheric cortical veins with loss of sulcation, concerning for developing ischemia, and CT angiography revealed absent opacification of the distal cortical vessels in the right anterior cerebral artery and middle cerebral artery territories. Magnetic resonance imaging (MRI) of the brain was obtained 5.75 h after the patient's last known well-showed small areas of subtle cortical diffusion restriction. Follow-up CT head within 24 h showed near-complete resolution of the air emboli after treatment with 100% fraction of inspired oxygen on mechanical ventilation. Subsequent MRI, performed 4 days after the initial event, showed extensive cortical diffusion restriction and cerebral edema crossing vascular territories.

Conclusion: This case highlights that cerebral air emboli can cause delayed ischemia that may not be appreciated on initial imaging. As such, affected patients may require intensive neurocritical care management, close neurologic monitoring, and repeat imaging irrespective of initial radiographic findings.

Keywords: Air embolism, Embolism, Neurocritical care, Neuroimaging, Stroke

CASE REPORT

A 75-year-old man with a history of hypertension, atrial fibrillation on apixaban, stage III chronic kidney disease, peripheral vascular disease, and prior *Group B Streptococcal* infective endocarditis with prior bioprosthetic valve replacement was admitted to the hospital with recurrent endocarditis and *Group B Streptococcus* bacteremia. His hospital course was further complicated by right-sided empyema requiring the placement of a chest tube. On hospital day 13, a stroke code was called for unresponsiveness with the last known well 20 min before stroke code activation. On evaluation, the patient was obtunded with forced left gaze deviation concerning

for possible nonconvulsive status epilepticus (NCSE). He was intubated for airway protection and treated acutely with intravenous lorazepam and levetiracetam for possible NCSE. Noncontrast computerized tomography (CT) of the head revealed air within the right greater than left hemispheric cortical vessels with loss of sulcation, suggestive of ischemia due to cerebral air embolism (CAE) [Figure 1a]. CT angiography (CTA) of the head showed absent opacification of the distal cortical vessels in the right anterior cerebral artery and middle cerebral artery territories [Figure 1b]. The distal end of the patient's peripherally inserted central catheter (PICC) line, previously placed for antibiotic administration, was in the appropriate position on the scout CTA imaging [Figure 1c]. The patient was then transferred to the neurosciences intensive care unit for further care, placed in the Trendelenburg position, and continued on 100% fraction of inspired oxygen (FiO_2) through mechanical ventilation. The PICC line was preemptively removed.

Subsequent CT head at 24 hours (h) after the last known well-showed near-complete resolution of the air emboli after continued treatment with 100% FiO_2, though there was the evident loss of sulcation in the right hemisphere [Figure 1d]. Magnetic resonance imaging (MRI) of the brain obtained 5.75 h from the last known well showed areas of subtle cortical diffusion restriction, but there was no evidence of definitive infarction [Figures 2a and b]. Electroencephalogram revealed mild-to-moderate generalized slowing and right hemispheric slowing with attenuation of faster frequencies; no seizures were noted. Repeat MRI obtained 4 days after the stroke code activation (hospital day 17) demonstrated cortical diffusion restriction in a gyriform pattern throughout multiple vascular territories without associated cerebral edema [Figures 2c and d]. On hospital day 18, the patient's neurological examination was notable for improved wakefulness, ability to follow commands, right gaze preference, left homonymous hemianopsia, left upper motor neuron facial droop, and left hemiplegia. His hospital course was further complicated by ventilator-associated pneumonia and prolonged intubation requiring placement of a tracheostomy and a percutaneous gastrostomy tube. He was ultimately discharged to a long-term acute care hospital on hospital day 89.

Retrograde movement of air into the cortical veins resulting in venous infarction was the hypothesized mechanism of this patient's CAE. Although the PICC line was noted to be ipsilateral to the more affected hemisphere, this was confirmed to be in the correct position on CTA scout imaging during the patient's emergent evaluation and, thus, was not felt to be the source of air entry into the venous circulation [Figure 1c]. Beyond chest tube placement and removal, no surgical interventions had occurred before the CAE. A transthoracic echocardiogram was negative for a patent foramen ovale to suggest paradoxical air embolism through the arterial circulation as a possible mechanism. As such, the etiology of CAE in this patient remains cryptogenic.

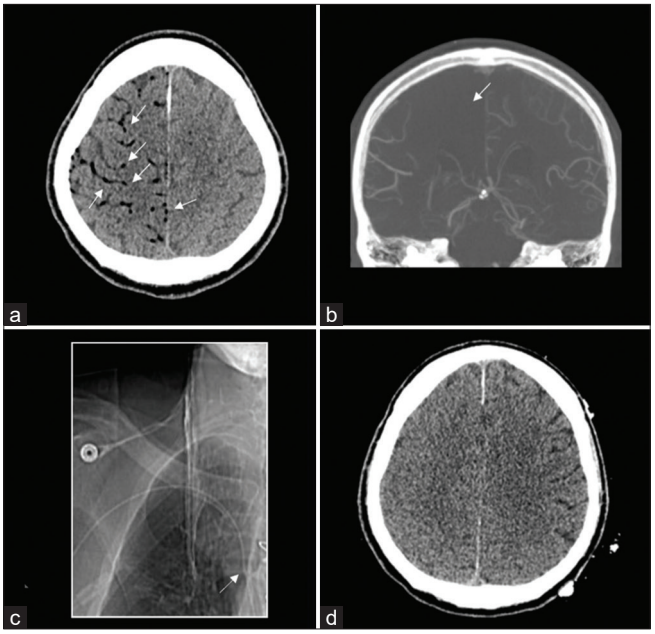

Figure 1: Initial computerized tomography (CT) demonstrating air emboli (a, white arrows). Loss of blood flow noted in the affected area on CT angiography (CTA) (b, white arrow). CTA scout image showing R-sided peripherally inserted central catheter with correct placement (c, white arrow). Repeat CT within 24 h after initial CT shows improvement in air after 100% fraction of inspired oxygen (d).

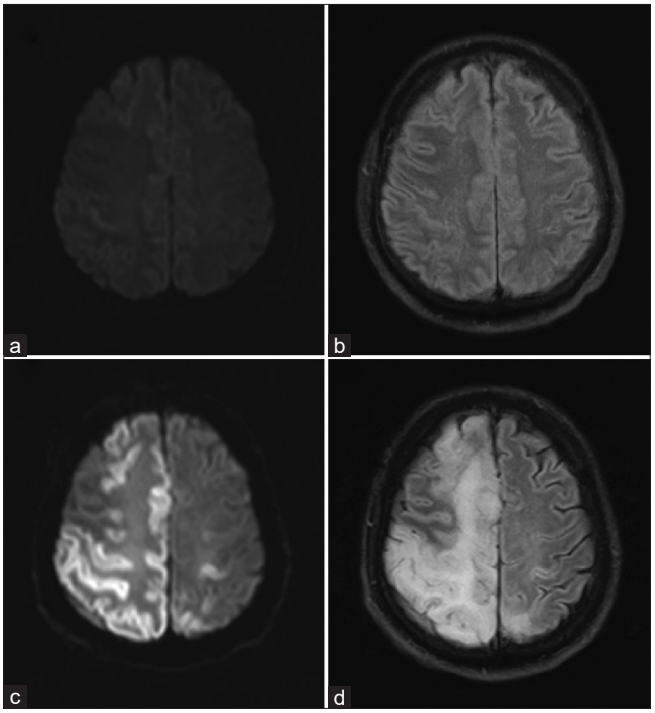

Figure 2: Axial diffusion-weighted imaging (DWI) (a) and fluid-attenuated inversion recovery (FLAIR) (b) images from magnetic resonance imaging (MRI) brain obtained 5.75 h after last known normal and repeat MRI from day 5 (c: DWI; d: FLAIR), demonstrating delayed evolution of cortical ischemia with adjacent white matter edema.

CAE is a rare cause of acute ischemic stroke that is becoming increasingly well-described in the literature.[1,3] The cause of CAE is often iatrogenic and typically occurs during medical procedures or in association with medical devices, such as intravascular catheters.[3] The presentation of CAE includes reduced or altered level of consciousness, focal neurological deficits, increased muscle tone, and/or seizures.[1] Noncontrast CT of the head can reveal the presence of air in the sulci if obtained early. However, even in the absence of air on the CT head, a high degree of clinical suspicion is required for diagnosis, as intracerebral air is rapidly absorbed through arterioles;[1-3] existing literature suggests that CAE can cause injury through two possible mechanisms: (1) local obstruction of blood flow by air emboli resulting in ischemic infarction or (2) or direct endothelial injury with resultant blood–brain barrier breakdown and *in situ* thrombus formation leading to ischemic infarction.[3,4] With respect to imaging characteristics, multiple areas of restricted diffusion along the cortical gray matter in a gyriform pattern involving both cerebral hemispheres with significant cerebral edema are common MRI findings associated with CAE (venous more than arterial).[3]

As there are no trials evaluating the treatment of CAE, management strategies are based on case reports and case series. Initial management of CAE involves identification and removal of potential sources of air entry, positioning the patient using the Durant's maneuver (left lateral decubitus and Trendelenburg position), high-flow oxygenation, and, if available, use of hyperbaric oxygen therapy.[3,5] Although air embolism carries a high mortality rate of 21%, a recent retrospective study assessing CAE suggests that functional outcomes may vary, and patients tend to improve over time.[3,5] Larger cohort studies are needed to assess functional outcomes, morbidity and mortality, and the effectiveness of interventions in patients with CAE.

A unique feature of our case is the radiographic presentation of delayed cerebral ischemia with cerebral edema on MRI associated with CAE. Based on available radiographic data, the cerebral edema and ischemia appear to have developed sometime between the 5.75 h MRI and the 24 h CT head, though the size of the edema appears to have continued to increase between the 24 h imaging and the MRI completed 4 days later. The MRI findings of cortical diffusion restriction with cerebral edema that is out of proportion to the infarct burden suggest that the air emboli were likely in the venous circulation. The mechanism and severity of injury associated with CAE can vary, with ischemia typically emerging early. To the best of our knowledge, delayed cerebral ischemia secondary to CAE has not yet been described. This has important implications for patient care, as affected patients may require more intensive neurocritical care and closer neuromonitoring than initially anticipated to optimize treatment after CAE. Although the cause of this patient's air emboli remains cryptogenic at this time, this case highlights that CAE can cause delayed ischemia that may not be appreciated on initial brain imaging.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

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