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Characteristic MR Imaging Findings of Cerebral Air Embolism Infarcts: A Case Series

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ABSTRACT

SUMMARY: Infarcts from cerebral air embolism are rare events with potentially catastrophic clinical consequences. The imaging features of cerebral air embolism are not well-defined in the literature. We report a novel constellation of MR imaging findings of cerebral arterial air emboli–induced infarcts in a series of 6 patients. Awareness of the more distinguishing MR imaging patterns of cerebral air embolism may help establish this diagnosis and facilitate implementation of timely treatment.

ABBREVIATIONS: ACA = anterior cerebral artery; CAE = cerebral air embolism

Cerebral air embolism (CAE) is a rare cause of stroke associated with high morbidity and mortality.[1,2] Strokes from air emboli occur when intravascular air migrates distally into the arterial circulation disrupting physiologic CBF and gas exchange, most often at smaller distal branches along territorial borderzones. The source of intravascular air can be arterial or venous, with the latter resulting in infarct when gas subsequently enters the arterial circulation typically through a pulmonary or cardiac right-to-left shunt such as a patent foramen ovale. It has also been suggested that venous air could result in a CAE venous infarct via retrograde flow intracranially.[2,3] While the true incidence of CAE is unknown, in 2020 the US FDA Manufacturer and User Facility Device Experience Database found 328 reports of systemic air embolism, with other reports estimating upward of 1 in 772 interventional procedures complicated by a clinical systemic air embolism.[4,5]

The etiology for air embolic events is most often iatrogenic when intravascular air may inadvertently be introduced during an invasive procedure and, less likely, due to noniatrogenic trauma or decompression illness seen rarely in scuba diving accidents.[6] Procedures known to be associated with cerebral air emboli include central venous catheter manipulation, endovascular procedures, laparoscopic surgeries, cardiothoracic surgeries, and certain neurosurgical procedures performed with the patient in a seated position such as posterior fossa surgery.[1] While clinically, an air embolism may be difficult to diagnose because patients often present with nonspecific encephalopathic symptoms of altered consciousness, seizures, and various strokelike episodes, the diagnosis should be considered in cases in which there is a temporal relationship between a postoperative/postinterventional procedural state and the onset of symptoms.

There are several proposed pathophysiologic mechanisms of brain injury in CAE, all of which may be contributory. First, an embolized intra-arterial air bubble may occlude vascular flow, depriving the brain parenchyma of oxygen, ultimately causing neuronal metabolic processes to fail, allowing water and sodium to enter the cell, and resulting in cytotoxic edema. Second, the surface of the air bubble lodged in the vascular lumen may trigger a foreign body response through cellular and humoral immune mechanisms, resulting in vasogenic edema.[1,7] Third, the air bubble may irritate the vascular endothelium leading to a further increase in surrounding vasogenic edema.[1,7,8] Accumulation of vasogenic edema and inflammation-induced in situ thrombus formation may also result in further compromise of perfusion to neuronal tissue.[1,7,9]

Imaging can play a critical role in establishing a diagnosis of air embolism. In some cases, the diagnosis can be made on CT, where gas-attenuation foci may be seen peripherally in vascular beds along borderzones with surrounding vasogenic and cytotoxic edema; however, these changes are not present in all cases, and CT has been described as having limited sensitivity for CAE.[4,10] On MR imaging, the gas emboli may appear as susceptibility foci, a finding that, similar to CT, is not always seen and can make ischemic lesions of air emboli challenging to distinguish from other causes of stroke. The proposed inflammatory component of CAE may manifest as vasogenic edema,[2,11] or if

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Patient demographics, etiology of air embolism, and imaging findings

Sex	Age (yr)	Source	MRI Brain Findings	Extracranial Findings
M	68	Left atrial-esophageal fistula status post cardiac ablation procedure	Distal borderzone–predominant distribution; vasogenic edema + cytotoxic edema; leptomeningeal enhancement	Atrial-esophageal fistula
M	68	Left atrial-esophageal fistula status post cardiac ablation procedure	Distal borderzone–predominant distribution; vasogenic edema + cytotoxic edema; leptomeningeal enhancement	Atrial-esophageal fistula, pneumomediastinum
F	47	Laparoscopic right renal mass surgery	Distal borderzone–predominant distribution; cytotoxic edema; leptomeningeal enhancement	Pneumomediastinum
M	64	Open chest wound	Distal borderzone–predominant distribution; cytotoxic edema; vasogenic edema.	Right anterior chest wall wound
F	86	Aortic valve replacement	Distal borderzone–predominant distribution; vasogenic edema + cytotoxic edema; leptomeningeal enhancement	NA
F	53	Meningioma resection	Mixed cytotoxic-vasogenic edema; leptomeningeal enhancement	NA

Note:—M indicates male; F, female; NA, not applicable.

inflammation results in sufficient breakdown of the BBB, CAE could theoretically result in enhancement of the perivascular leptomeninges, similar to other pathologic processes such as meningitis.[12] These changes can be captured on both noncontrast MR imaging and contrast-enhanced MR imaging of the brain, with the latter potentially offering increased opportunities for detection of this inflammatory pathology.[1,12,13]

The findings on MR imaging reflecting the inflammatory pathophysiology of CAE are not well-described in the literature, and to our knowledge, a link between leptomeningeal enhancement and CAE has not been previously reported. In this case series, we present 6 patients with clinically suspected air embolism who underwent immediate MR imaging of the brain. We report characteristic acute MR imaging findings of a mixed cytotoxic-vasogenic edema pattern, peripheral borderzone distribution, and leptomeningeal enhancement that supports the proposed dual mechanism of ischemia and inflammation contributing to the pathophysiology of CAE. Recognition of these imaging features can help confirm a presumptive CAE diagnosis and facilitate prompt initiation of a CAE-specific treatment strategy.

Case Series

Institutional review board (Mayo Clinic and University of Colorado Anschutz Medical Campus) approval was obtained for this retrospective case series. A retrospective search of the electronic health records of Mayo Clinic and the University of Colorado health systems was performed to identify neuroimaging examinations performed between January 1, 2004, and December 31, 2023. Cases were identified by searching for key words such as "air embolism" and/or "air emboli" on any neuroimaging examination performed during the study period. This search yielded 7 cases: Six had MR imaging within 3 days of the suspected air embolism event and were included in this series, while 1 study contained only noncontrast CT brain imaging and was excluded from this series. Imaging data, along with associated clinical notes and procedural reports contained in the electronic medical record of each patient, were reviewed jointly by 2 board-certified neuroradiologists with 11 and 5 years of neuroradiology experience, respectively. Imaging findings were reported by consensus interpretation. Demographic, clinical, and imaging results from this case series are summarized in the Table.

Patient 1

A 68-year-old man with a history of atrial fibrillation presented with acute alteration of mental status 2 weeks following a cardiac ablation procedure. CTA of the brain obtained approximately 12 hours from symptom onset did not reveal evidence of large-vessel occlusion. CTA of the neck demonstrated a fistulous communication between the left atrium and esophagus, with evidence of intra-atrial gas and trace contrast extravasation into the esophageal lumen (Online Supplemental Data). A contrast-enhanced MR brain imaging obtained immediately after the patient's CTA on hospital day 1 demonstrated a mixed pattern of cytotoxic and vasogenic edema in a distal vascular bed distribution, with associated leptomeningeal enhancement adjacent to regions of edema (Fig 1). The findings were consistent with acute air emboli from a post cardiac ablation atrial-esophageal fistula. The patient was treated with supportive care but the clinical condition rapidly deteriorated and he died on hospital day 3.

Patient 2

A 68-year-old man with a history of atrial fibrillation presented 11 days status post cardiac ablation procedure with acute altered mental status, left lower-extremity weakness, a right visual field deficit, and melena. CTA of the head and neck obtained on hospital day 1 was notable for pneumomediastinum with gas interposed between the esophagus and left atrium, suspicious for an atrial-esophageal fistula (not shown). A follow-up same-day contrast-enhanced MR brain imaging revealed a mixed pattern of cytotoxic and vasogenic edema in a distal vascular bed distribution, with associated leptomeningeal enhancement adjacent to regions of edema (Fig 2). The findings were consistent with acute air emboli from a post cardiac ablation atrial-esophageal fistula. The patient was treated with supportive care and died on hospital day 1.

Patient 3

A 47-year-old woman presented with acute alteration in mental status during the immediate postprocedural recovery period

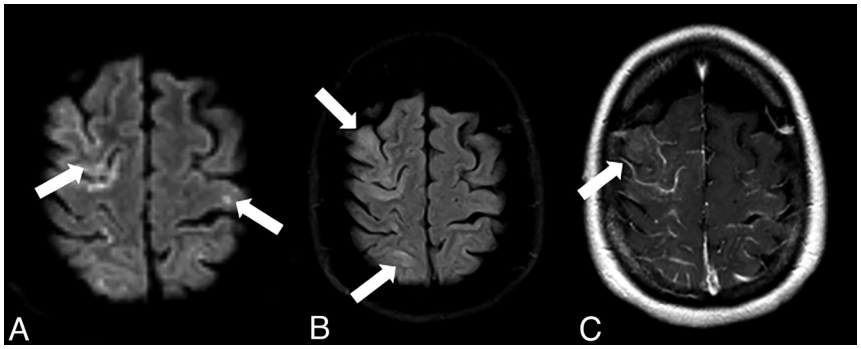

FIG 1. Contrast-enhanced brain MRI in a 68-year-old man with suspected CAE revealing a mixed pattern of cytotoxic (*white arrows*, DWI sequence, *A*) and vasogenic edema (*white arrows,* T2-FLAIR sequence, *B*) in a distal vascular bed distribution, with associated leptomeningeal enhancement adjacent to regions of edema (*white arrow*, postcontrast [MultiHance; Bracco Diagnostics] 3D gradient-echo T1 sequence, *C*).

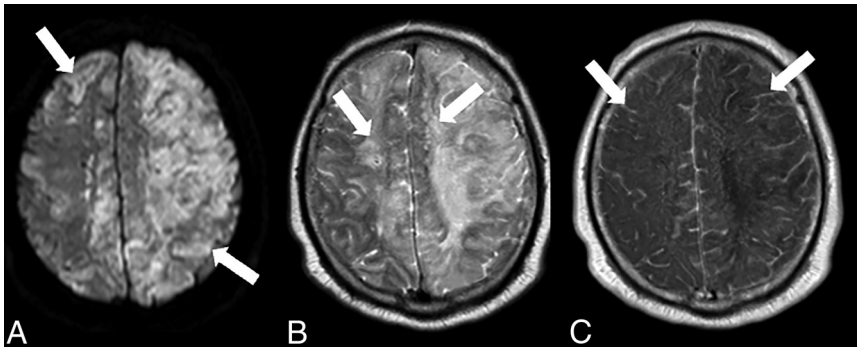

FIG 2. A 68-year-old man with symptoms suspicious for CAE. Contrast-enhanced brain MRI demonstrates a mixed pattern of cytotoxic (*white arrows,* DWI sequence, *A*) and vasogenic edema (*white arrows*, T2-FLAIR sequence, *B*) in a distal vascular bed distribution, with associated leptomeningeal enhancement adjacent to regions of edema (*white arrows*, postcontrast [MultiHance; Bracco Diagnostic] 2D spin-echo T1 sequence, *C*). CTA of the head and neck was notable for pneumomediastinum with gas interposed between the esophagus and left atrium (not shown). The findings were consistent with acute air emboli from a post cardiac ablation atrial-esophageal fistula.

following laparoscopic resection of a renal mass. Contrast-enhanced brain MRI (Fig 3) demonstrated distal infarcts in the bilateral MCA and anterior cerebral artery (ACA) territories with associated leptomeningeal enhancement. A contrast-enhanced CT of the chest (Online Supplemental Data) revealed diffuse chest wall and mediastinal emphysema. A transthoracic echocardiogram found a right-to-left atrial shunt. Findings were consistent with CAE related to laparoscopic insufflation, resulting in intravascular introduction of gas, presumably venous with secondary arterial embolization via the patient's interatrial shunt. The patient was treated with hyperbaric oxygen therapy, and, at follow-up, had a 90-day mRS score of 4.

Patient 4

A 64-year-old man presented with a history of diabetes and multiple thoracic surgeries complicated by infections resulting in a chronic nonhealing open right anterior chest wound. The patient reported hearing a rush-of-air sound while undergoing a routine packing procedure of his chest wound, which was immediately followed by a loss of consciousness. A noncontrast brain CT obtained 3 hours following the onset of symptoms (Fig 4) revealed extra-axial gas in the right frontal lobe. Noncontrast brain MRI obtained on hospital day 2 (Fig 4 and Online Supplemental Data) demonstrated a mixed pattern of cytotoxic and vasogenic edema in the bilateral right-greater-than-left MCA-ACA borderzones and susceptibility signal corresponding to some residual extra-axial gas in the right frontal lobe. Chest CT with contrast (Online Supplemental Data) showed the patient's open right anterior chest wall wound. Findings were consistent with CAE stemming from intravascular introduction of gas along the cavity of the patient's chest wall during wound packing. The patient was treated with hyperbaric oxygen therapy and, in follow-up, had a 90-day mRS score of 1.

Patient 5

An 86-year-old woman with a history of aortic stenosis presented for routine aortic valve replacement surgery. Intraventricular air was inadvertently introduced during placement of a left ventricular vent. Postoperatively, the patient demonstrated confusion and bilateral upper-extremity weakness. Contrast-enhanced MR brain imaging was obtained on postoperative day 2 (Fig 5), demonstrating a mixed pattern of cytotoxic-vasogenic edema scattered throughout the cerebral hemispheres in a predominantly distal borderzone distribution, with subtle associated leptomeningeal enhancement along the left anterior ACA-MCA borderzone. The patient was treated with hyperbaric oxygen therapy and, in follow-up, had a 90-day mRS score of 4.

Patient 6

A 53-year-old woman presented with bilateral leg numbness postoperatively immediately following left parietal meningioma resection in the setting of a clinically suspected intraoperative air embolism. Six-hour postoperative contrast-enhanced MR brain imaging was obtained (Online Supplemental Data), demonstrating a gross total resection of the meningioma with a mixed pattern of vasogenic edema and cytotoxic edema in the left frontal and parietal lobes with associated leptomeningeal enhancement in the bilateral frontal-parietal lobes and the right cerebellar hemisphere. Findings were compatible with clinically suspected air emboli. The patient was managed conservatively and follow-up brain MR imaging obtained approximately 2 weeks after discharge (not shown) demonstrated near-complete resolution of

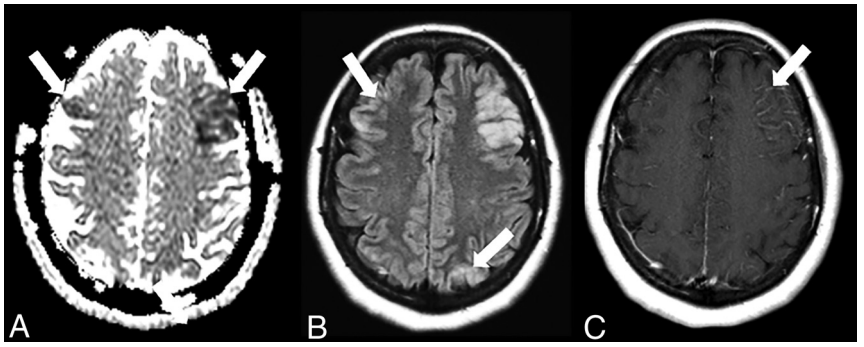

FIG 3. Contrast-enhanced brain MRI in 47-year-old woman suspected of having a CAE event, demonstrating a mixed pattern of cytotoxic (*white arrows,* DWI sequence, *A*) and vasogenic edema (*white arrows,* T2-FLAIR sequence, *B*) scattered throughout the cerebral hemispheres in a predominantly distal borderzone distribution, with subtle, associated leptomeningeal enhancement along the left anterior ACA-MCA borderzone (*white arrow,* postcontrast [MultiHance; Bracco Diagnostics] 2D spin-echo T1 sequence, *C*). Findings were consistent with CAE related to laparoscopic insufflation, resulting in intravascular introduction of gas.

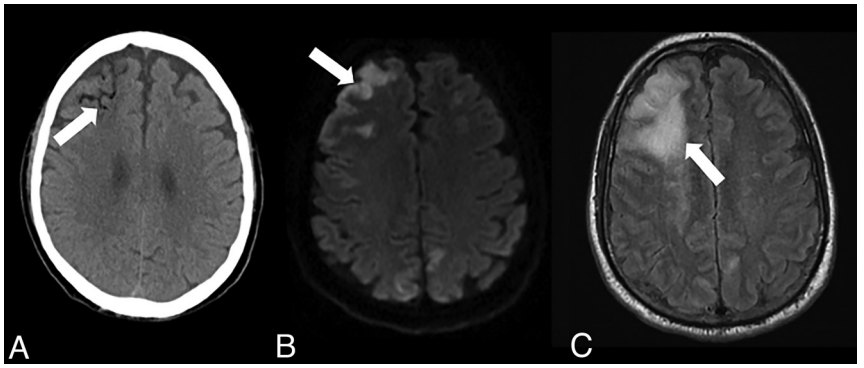

FIG 4. A 64-year-old man with suspected CAE. A noncontrast brain CT reveals extra-axial gas in the right frontal lobe (*white arrow*, *A*). Non-contrast-enhanced brain MRI demonstrates a mixed pattern of cytotoxic (*white arrow*, DWI sequence, *B*) and vasogenic edema (*white arrow*, T2-FLAIR sequence, *C*) in the bilateral right-greater-than-left MCA-ACA borderzones. Findings were consistent with CAE stemming from intravascular introduction of gas along the cavity of patient's chest wall during wound packing.

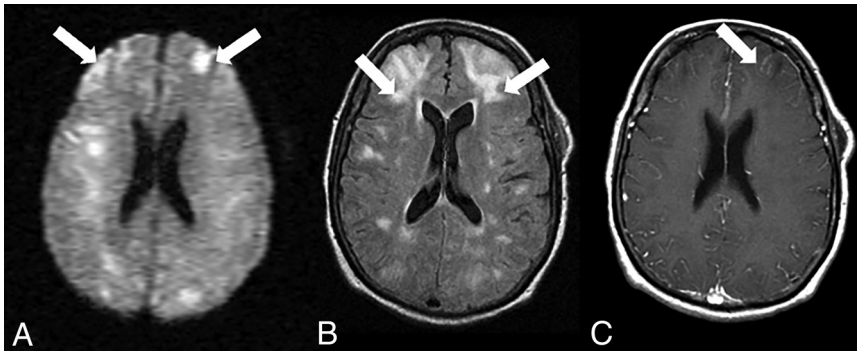

FIG 5. An 86-year-old woman with intraoperative CAE. Contrast-enhanced brain MRI demonstrates a mixed pattern of cytotoxic (*white arrows,* DWI sequence, *A*) and vasogenic edema (*white arrows,* T2-FLAIR sequence, *B*) scattered throughout the cerebral hemispheres, in a predominantly distal borderzone distribution, with subtle, associated leptomeningeal enhancement (*white arrow* postcontrast [ProHance; Bracco] 3D gradient-echo T1 sequence, *C*) along the left anterior ACA-MCA borderzone. The findings represented sequelae of CAE stemming from inadvertent intravascular introduction of air during an aortic valve replacement surgery.

the leptomeningeal enhancement. At 90 days postictus, the patient had a mRS score of 1.

DISCUSSION

Infarcts from CAE are uncommon events with potentially catastrophic consequences. The diagnosis relies on a strong clinical suspicion coupled with imaging features confirming air emboli and/or resulting infarcts. While the imaging finding of intracranial intra-arterial air and infarcts is diagnostic, the former finding may not be reliably detected.[4,10] In this study, we report a novel pattern of imaging features on MR imaging in a series of 6 patients with air emboli that may aid in the diagnostic work-up of such patients and enable the timely implementation of treatment.

There is a paucity of literature on the MR imaging features of air embolic infarcts. In a case series, Caulfield et al[14] reported findings of multiple small cortical foci of diffusion restriction in a gyriform pattern highlighting the small size of the embolic air and its propensity to lodge in distal end-artery territories of the brain. Brown et al[4] reported similar findings in their case series of air emboli, reporting scattered regions of diffusion restriction, with a propensity for distal boarder zone distribution and linear susceptibility artifacts corresponding to extra-axial air on CT in 25% of MR cases. A case series by Tsetsou et al[10] reported extra-axial air in only 1 of 5 cases, further highlighting the limited sensitivity of this finding in making a diagnosis of CAE. Brito et al[11] reported 4 cases of CAE, 2 of which had brain MRI demonstrating infarcts without an imaging correlate for extra-axial gas, which had been present on the preceding CT. This observation could be attributed to either the dynamic nature of CAE, in which fast re-absorption of gas emboli could limit detection of this finding at follow-up, or could reflect limited sensitivity of MR over CT for extra-axial gas detection.

Another finding described in some reports is a vasogenic edema pattern of cerebral lesions, suggesting the possibility of venous infarct or an inflammation-

induced mechanism of parenchymal injury.[2,3,11] Last, Oka et al[15] reported a delayed onset of MR findings in CAE appearing 22 hours after an initial brain MRI with normal findings obtained immediately after the embolic event. Notably absent in prior imaging studies of CAE were reports of the contrast-enhanced MR imaging features of this stroke etiology.

Our study affirms previously reported MR imaging features of air embolism of a distal borderzone distribution, vasogenic edema, and extra-axial gas and adds to the existing body of knowledge of CAE by introducing an association between such infarcts and acute MRI findings of leptomeningeal enhancement. The leptomeningeal enhancement observed in this case series occurred in the acute phase in the setting of clinically suspected CAE and in 5/5 of the cases performed with IV contrast. The enhancement was adjacent to the regions of acute infarcts. While gyriform parenchymal enhancement in infarcts is an expected imaging feature in the subacute phase during infarct maturation as the BBB becomes compromised, leptomeningeal enhancement is not an expected imaging feature in acute infarcts, suggesting that leptomeningeal enhancement on MR imaging in the acute phase may be a characteristic imaging feature of air embolism.

Three main pathophysiologic mechanisms have been proposed to account for the cerebral lesions of CAE: 1) ischemic lesions due to the interruption of cerebral arterial flow, which is supported by findings of cytotoxic edema; 2) inflammation due to a gas bubble–generating foreign body reaction with endothelial damage, breakdown of the BBB, and activation of an immune cell–mediated response;[1,2,6] and 3) elevated venous pressures in the intracranial dural venous sinuses and cortical veins, promoting vasogenic edema and venous infarcts for cases in which there is retrograde venous air embolism.[2,3] While the precise pathophysiology of brain injury in CAE is unknown, proposed ischemic and inflammatory mechanisms may co-exist. The observed leptomeningeal enhancement and concomitant vasogenic pattern of edema observed in this case series may offer more support for an inflammatory component of CAE pathophysiology and would be concordant with prior studies demonstrating links between air emboli endothelial damage and induction of leukocyte and platelet adhesion.[2,6,8] While the leptomeningeal enhancement could, in part, reflect hyperemia from underlying seizure activity, the observation of multifocal enhancement at sites of infarct would favor inflammation over seizure activity as the underlying etiology because multifocal epileptogenic foci would be uncommon.

Considering the limited sensitivity of CT and MR for detecting extra-axial air, contrast-enhanced MRI may be a useful adjunct to the typical stroke imaging work-up because a leptomeningeal pattern of enhancement coinciding with acute infarcts may help confirm or raise suspicion of a CAE diagnosis. Considering the limited sensitivity of CT and MR for detecting extra-axial air, contrast-enhanced MRI may be a useful add-on to the typical stroke imaging work-up. A suggested CAE imaging work-up would start with a noncontrast brain CT, followed by CTA of the head and neck, and contrast-enhanced MR imaging of the brain. If extra-axial gas were detected on the CT of the brain, the patient could likely forgo the remainder of the imaging in favor of starting immediate CAE-directed therapy. If no extra-axial gas was detected on CT and CTA ruled out other acute vasculopathy, then contrast-enhanced MR imaging could help increase confidence in a presumptive CAE diagnosis if distal borderzone infarcts with a mixed cytotoxic/vasogenic edema pattern and leptomeningeal enhancement were present.

There are important treatment implications to a presumptive CAE diagnosis because this will initiate a treatment regimen focused on removing air from the circulation rather than on thrombolysis and anticoagulation. The recommended treatment for cerebral air embolism includes immediately placing the patient in a head down/Trendelenberg and left lateral decubitus position to prevent cephalic flow of air bubbles and to preferentially trap air in the right atrium and right ventricle.[7] Recommended treatment also includes implementation of high-concentration oxygen therapy and hyperbaric oxygen therapy.[1,7,10,16] While mortality from CAE is estimated at 20% in the acute phase, there are limited data regarding the prognosis and effectiveness of CAE treatment.[2] In one of the larger and more recent case series by Brown et al,[4] 26% of patients had an mRS score of 0 one year after the embolism event, and functional improvement after discharge from the hospital was common, suggesting a cautious approach to early prognostication following air embolic events.

Limitations of this study include its retrospective design and small sample size. We acknowledge that the association between enhancement observed in this series and inflammatory pathophysiology is presumptive and can only truly be confirmed with histologic sampling. We also acknowledge that there is some potential overlap of imaging findings of CAE with other inflammatory stroke etiologies. In the absence of visible air on CT or SWI, the imaging features of suspected CAE should be correlated with a temporal relation to a recent air embolic–inducing event to avoid misdiagnosis with other inflammation-producing infarcts such as septic emboli, fat emboli, or vasculitis. An additional limitation of this case series is the variable MR protocols using 2D, 3D, spin-echo, and gradient techniques as well as a postcontrast T2 FLAIR sequence.

The small sample size of this case series precluded meaningful analysis regarding the accuracy of various postcontrast sequences for detection of CAE, a topic that would warrant future investigation in a larger study cohort.[13]

CONCLUSIONS

We report characteristic MR imaging patterns of cerebral arterial air emboli–induced infarcts in a series of 6 patients. Brain MRI with the addition of contrast-enhanced sequences may be a useful diagnostic adjunct to the standard stroke imaging work-up in cases of suspected CAE. Awareness of the characteristic MR imaging features of CAE to include leptomeningeal enhancement could potentially aid in the detection and timely management of neurologic complications in this patient population.

Disclosure forms provided by the authors are available with the full text and PDF of this article at www.ajnr.org.

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