LETTER TO THE EDITOR

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An Unusual Cause of Altered Mental Status: Recurrent Cerebral Air Embolism

Atrial fibrillation remains the most common cardiac arrhythmia affecting 2.7 to 6.1 million adults in the United States and contributes to significant morbidity and mortality with more than 467,000 annual admissions and more than 99,000 annual deaths.^{1,2} Current medical therapy focuses on controlling the rate or restoring sinus rhythm and concurrent anticoagulation with vitamin K antagonist based on risk stratification to decrease the risk of thromboembolic events. Medically refractory cases commonly resort to cardiac ablation to restore sinus rhythm and alleviate symptoms. But cardiac ablation is not a benign procedure and is associated with a 4.5% incidence of major complications and a 0.1% mortality rate.^{3,4}

Our case demonstrates an unusual but established complication of atrial fibrillation involving atrial-esophageal fistula (AEF) formation and subsequent cerebral air emboli. In a 2010 international survey of radiofrequency ablation procedures, there was a 0.04% rate of AEF formation,³ and an incidence of 0.1% to 0.9% in patients presenting with sudden neurological symptoms after cardiac ablation.⁴ Currently, atrial fibrillation uses the application of radiofrequency energy by transvenous electrodes to cardiac tissue, which leads to irreversible coagulative necrosis and formation of transmural, nonconducting myocardial tissue.⁵ Although the high power delivery ensures formation of sufficient lesions, the combination of high power and the anatomical proximity of the esophagus posterior to the left atrial wall increases the risk of complications such as cardiac perforation or AEF formation.

Complications from air emboli arise because air in the arterial circulation occludes microcirculation and results in end-organ ischemia. Additionally, oxygen free radicals contribute to more inflammatory changes and edema, leading to worsening damage and neurological deficits not limited to the occluded vascular supply.⁵ The AEF can also provide bacterial introduction into the bloodstream with risk for subsequent sepsis.

A 69-year-old male with medically refractory atrial fibrillation underwent cardiac ablation for atrial fibrillation with rapid ventricular response. During ablation, temperature did not exceed 28°C, and no events were noted by the cardiologist. The patient tolerated the procedure well and was discharged 2 days following ablation. Nine days after discharge, severe chest pain led to readmission with discovery of an AEF by computed tomography (CT) angiography and subsequent Gastrografin swallow study following surgery recommendations. The AEF was surgically repaired with intercostal muscle flap buttress, and closure confirmed with Gastrografin swallow study.

Sixteen days after surgical repair and discharge, the patient developed neurological deficits, including progressively worsening left-sided paresis, hemisensory deficits, and dysarthria. Blood cultures and complete blood count obtained on admission were unconcerning for sepsis. CT and magnetic resonance imaging (MRI)/magnetic resonance angiography revealed air in the right frontal lobe, in the left atrial appendage, and left ventricular apex, with no evidence of air in the pericardium or mediastinum. The findings were concerning for a persistent AEF despite surgical repair. The patient urgently received hyperbaric oxygen with resolution of cerebral air emboli confirmed on repeat MRI of the brain and rapid improvement in speech and mental status. With concern for repeat cerebral air emboli from a persistent AEF, the surgery department was consulted. Before repeat thoracotomy, surgery requested further imaging, including a repeat Gastrografin swallow study to confirm the possibility that a persistent AEF was the source of the air emboli. Repeat Gastrografin swallow study did reveal persistent AEF despite prior surgical closure. Unexpectedly and before surgical closure, the patient developed rapid decline in mental status leading to death. Repeat imaging revealed worsening cerebral air emboli.

The incidence of stroke secondary to air emboli is rare, but associated with high mortality, with untreated air emboli mortality rates exceeding 90%. Specific to cerebral air emboli secondary to AEF, previous case studies have documented diagnostic difficulty and delay as likely contributors to the high mortality rate.⁶ Appropriate clinical suspicion remains the best diagnostic and treatment tool followed by prompt imaging modalities such as MRI or CT of the head to confirm the presence of cerebral air emboli and the presence of an AEF.⁵

Treatment of cerebral air emboli encompasses the acute management of cerebral air emboli, closure of the AEF, and preventive strategies. Any patient with proven cerebral air emboli presenting with neurological deficits should receive hyperbaric oxygen therapy acutely. High-flow supplemental oxygen (i.e. a high fraction of inspired oxygen) increases the rate embolized air absorbed by increasing the partial pressure of oxygen and decreasing the partial pressure of nitrogen in blood, producing a positive pressure gradient for the diffusion of nitrogen from the air to the blood and vice versa.⁷ After medical stabilization, patients should also receive surgical closure of the fistula to prevent recurrent cerebral air emboli, but closure is still associated with a 71% mortality if diagnosis is delayed.⁸

Preventive strategies include modification of catheter ablation energy settings, esophageal temperature monitoring, detection of microbubbles during ablation, and esophageal cooling devices.⁴ Commonly, electrophysiologist will modify the catheter ablation energy when specifically ablating lesions on the posterior left atrial wall. Although the decrease in energy may decrease damage to surrounding tissue, the efficacy of successful ablation of atrial fibrillation is diminished.³

Recent smaller studies indicate nondrug-attained sinus rhythm compared with standard medical therapy may lead to improved mortality and morbidity. Additionally, ongoing studies such as Catheter Ablation Versus Antiarrhythmic Drug Therapy for Atrial Fibrillation and the Early therapy for Atrial Fibrillation for Stroke Prevention Trial aim specifically at collecting more evidence on the efficacy of cardiac ablation. Ultimately, radiofrequency will likely continue to be used. Though preventive strategies and improved technology will potentially decrease the rate of complications, clinical suspicion and knowledge of side effects remains the first step in diagnosis and treatment. Specific to our case, no other case studies have involved recurrent air embolism

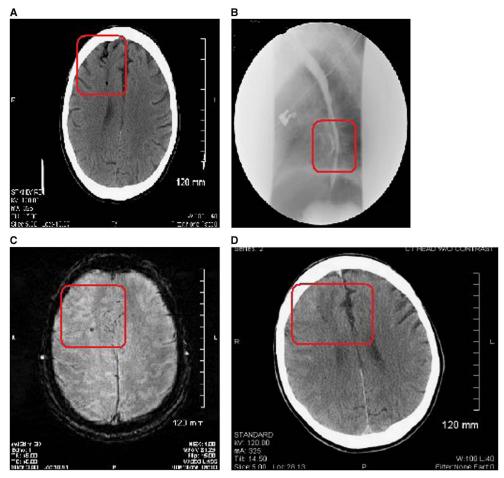


Figure 1: A. CT head upon admission with evidence of air emboli in the right front lobe. B. Gastrografin confirming atrialesophageal fistula. C. MRI Brain confirming air emboli. D. CT Head with evidence of recurrent air emboli after prior improvement.

following successful surgical repair.⁶ Our case and the high mortality associated with air emboli demonstrate the necessity of including cerebral air emboli in the differential of patients presenting to the hospital after cardiac ablation, despite prior treatment.

DISCLOSURES

The authors have no disclosures.

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