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Case Report

Atrioesophageal Fistula After Atrial Fibrillation Ablation: A Case Report

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ABSTRACT

Atrioesophageal fistula is a rare, potentially fatal complication of atrial fibrillation ablation that is often missed by clinicians. We report the case of a patient who presented with infectious symptoms 4 weeks after undergoing atrial fibrillation ablation. Our case emphasizes that prompt diagnosis and surgical intervention are crucial to reduce the high morbidity and mortality rates associated with this highly concerning complication.

Atrial fibrillation (AF) ablation is a treatment option for symptomatic refractory AF. Atrioesophageal fistula (AEF) formation is an extremely rare complication, with an incidence of 0.03% and mortality of up to 93% without surgical treatment.¹ Even with surgical intervention, mortality can be as high as 20%.² Patients with AEF present with a broad array of symptoms but typically develop neurologic symptoms, fevers, and hematemesis 1-6 weeks after ablation, with rapid deterioration. Therefore, prompt diagnosis and referral for surgery is of utmost importance to reduce mortality stemming from this serious complication.

Case

A 57-year-old man with history of AF and obstructive sleep apnea presented to the emergency department with chest pain and palpitations with a duration of 1 day. Three weeks prior to presentation, he had undergone radiofrequency ablation for his AF. His initial vitals were notable for tachycardia to 140 beats per minute and tachypnia to 25 breaths per minute. On exam, the patient was alert and oriented. He had coarse

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RÉSUMÉ

La fistule atrio-œsophagienne est une complication rare, mais potentiellement fatale de l'ablation de la fibrillation auriculaire que les cliniciens négligent souvent. Nous rapportons le cas d'un patient qui présentait des symptômes d'infection quatre semaines après avoir subi l'ablation de la fibrillation auriculaire. Notre cas démontre que le diagnostic précoce et l'intervention chirurgicale sont cruciaux pour réduire les taux élevés de morbidité et de mortalité associées à cette complication très préoccupante.

bibasilar breath sounds, and cardiac exam was notable for irregularly irregular tachycardia. Initial labs revealed a serum creatinine level of 1.6 mg/dL, a normal complete blood count, normal lactic acid level, and negative serial troponin levels. An electrocardiogram showed atrial flutter with variable atrioventricular block. Chest X-ray showed a patchy consolidation in the left lung base. A computed tomography (CT) angiogram of the chest showed no pulmonary emboli. The patient received antibiotics for presumed pneumonia and was started on diltiazem drip.

On day 2 of hospitalization, the patient became confused and lethargic, and developed new left-sided hemiparesis. He was febrile to 40°C and tachycardic to 180 beats per minute. A repeat electrocardiogram showed persistent atrial flutter. Repeat lab tests showed leukocytosis of 15×10^9 /L, and a lactic acid level of 4 mmol/L. Given his clinical deterioration despite sepsis treatment, and history of recent ablation, AEF was suspected. A chest CT with oral and intravenous contrast showed contrast extravasation from the esophagus into the left atrium, confirming a diagnosis of AEF (Fig. 1). An initial CT scan of the brain showed no acute parenchymal disease, but a repeat scan showed diffuse right cerebral infarcts. The patient subsequently became hypotensive, requiring vasopressors and intubation for airway protection. Antibiotic coverage was broadened, and an amiodarone drip was started. The patient was emergently transferred to a cardiothoracic surgery center where he underwent a 2-stage surgery for left atrial (LA) wall repair with pericardial pledgets and esophageal wall repair with a vascularized serratus anterior muscle flap. The patient was

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Ethics Statement: This report has adhered to the relevant ethical guidelines.

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²⁵⁸⁹⁻⁷⁹⁰X/© 2022 The Authors. Published by Elsevier Inc. on behalf of the Canadian Cardiovascular Society. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

- AEF is a rare but serious complication that usually manifests 1-6 weeks following AF ablation.
- Clinicians should maintain a high index of suspicion for AEF in patients who have undergone recent AF ablation and present with infectious and/or neurologic symptoms.
- Diagnosis of AEF is confirmed by chest CT with oral contrast, which can show contrast extravasation into the left atrium.
- Once AEF is diagnosed, immediate surgical repair is crucial to reduce morbidity and mortality stemming from this highly concerning complication.

extubated on postoperative day 1 and remained in stable condition. He was discharged 3 weeks later to a rehabilitation facility, with persistent left-sided weakness.

Discussion

AF ablation is a widely performed procedure for symptomatic, refractory AF. The 2 technologies currently used for ablation are cryoballoon and radiofrequency ablation, both of which have similar efficacy and safety profiles.¹

AEF is a highly concerning complication of ablation that can occur from thermal damage to the esophagus that occurs during ablation of the posterior LA wall. One study reports that the risk of AEF may be associated with the use of general anesthesia, non-brushing ablation technique, and higherenergy settings.³ Additional publications, however, have reported a decreased temperature elevation of the esophagus and less esophageal injury, which may be protective against AEF formation if a high-power short-duration approach is used rather than a low-power long-duration ablation technique.⁴

AEF formation is believed to occur from esophageal ulceration secondary to thermal damage that leads to formation of a 1-way valve fistula from the esophagus to the left atrium.⁵ Methods of esophageal protection, including periprocedural proton-pump inhibitor use and mechanical displacement of the esophagus away from the catheter tip, did not reduce esophageal injury. Intracardiac echocardiography and esophageal temperature monitoring are methods that may reduce the likelihood of esophageal injury, but only limited data support use of one method over the other. The only variable that reliably confers esophageal protection is minimizing catheter energy during ablation, but this approach can reduce efficacy of the procedure. Higher catheter energy but a shorter duration has also been shown to be more protective than a longer duration with lower catheter energy.⁴

Moreover, a novel technique known as pulsed field ablation uses a nonthermal approach for pulmonary vein isolation, and a recent study showed promising outcomes relating to safety and efficacy with this method.⁶

Symptoms of AEF typically manifest 2-4 weeks after ablation, and they are variable, but infectious and neurologic symptoms are common.⁷ Hematemesis may also be present in AEF; however, in these cases, esophagogastroduodenoscopy should be avoided due to the risk of air embolism during

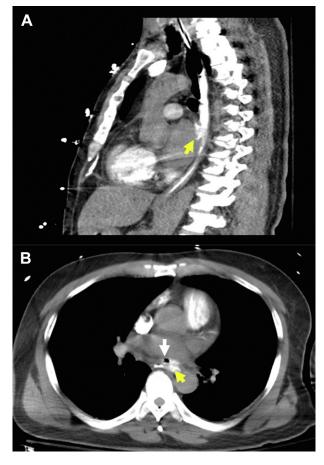


Figure 1. Computed tomography of the chest with intravenous and oral contrast. (A) Sagittal view showing contrast extravasation from the esophagus to the left atrium (arrow). (B) Cross-sectional view showing oral contrast extravasation (yellow arrow) and extraluminal air (white arrow).

insufflation. Any patient who has undergone ablation should in fact avoid all insufflation, including bronchoscopy, gastroscopy, and inadvertent esophageal intubation within the 5-week postablation window, due to the risk of AEF. Our patient presented with infectious symptoms and rapidly developed neurologic symptoms, presumably from embolic phenomena, which prompted further investigation of AEF. In retrospect, reviewing the initial CT angiogram of the chest suggests that a small air pocket is near the left atrium; however, this study had significant motion degradation, and not all radiologists reviewing the imaging can determine whether this finding was an artifact or was diagnostic. Because AEF is rare and initially presents as sepsis, prompt diagnosis can be challenging. Therefore, a crucial need is for clinician to maintain a high index of suspicion for this deadly complication in all patients with recent ablations who present for infectious symptoms, even if fever is absent.⁸

AEF workup should include CT of the chest, with oral and intravenous contrast. In patients who are unable to swallow, administering oral contrast may be challenging. Orogastric and nasogastric tubes ending in the stomach are unsuitable, as they bypass the area with suspected esophageal defect. In our patient, a nasogastric tube was pulled back to a level proximal

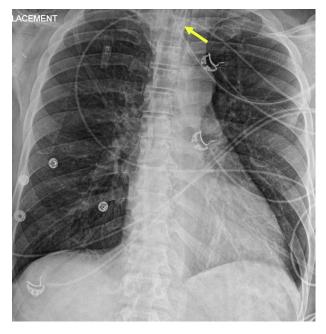


Figure 2. Anteroposterior chest X-ray showing the tip of the nasogastric tube in the proximal esophagus (arrow).

to the suspected defect prior to administering oral contrast, suggesting that the left side of the lesion was the likely culprit (Fig. 2). Also crucial is that oral contrast be administered immediately prior to the scan, due to the short esophageal transit time. Contrast extravasation from the esophagus is diagnostic.

Once AEF is diagnosed, early referral to cardiothoracic surgery is necessary to reduce morbidity and mortality. Surgical repair involves resecting the fistula tract and patching of the esophageal and LA defects. Prior to surgery, broadspectrum antimicrobials are warranted and should cover oral flora as well as fungi, given the esophageal content exposure.

In conclusion, AEF is a serious, potentially fatal complication of AF ablation. We emphasize early imaging to exclude AEF in all patients who have undergone recent AF ablation and present with infectious or neurologic symptoms. Surgery is the only definitive treatment, and every effort should be made to expedite diagnosis and referral for surgical repair.

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Disclosures

The authors have no conflicts of interest to disclose.

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