# **Esophageal perforation during atrial ablation** Gabrielle Aiello MD<sup>1</sup>, Minh Chung DO<sup>1</sup>, Xiang You MD<sup>1</sup>, Kaitlyn Vu MS1<sup>2</sup>, Amethyst Wilder MD<sup>1</sup> 1. Memorial Health University Medical Center, 2. Mercer University School of Medicine

### Introduction

Despite the safety profile of atrial radiofrequency (RF) ablation, esophageal perforation is a rare, life-threatening complication with an incidence rate between 0.1% to 0.25% (1). Esophageal perforation is often diagnosed late when a patient develops atrial-esophageal fistula (AEF) leading to poor prognosis.

- AEF results from severe thermal or ischemic injury and exhibits high overall mortality rate of 63.16% (1).
- Esophageal ulcers typically occur hours to days post-ablation preceding AEF formation.
- Most common clinical presentations of AEF include fever, neurological symptoms, and chest pain/discomfort with mean onset of  $30 \pm 12$  days post-ablation (2).

Most patients with esophageal perforation developed AEF, 71%, while others had pericardial-esophageal fistula, 14%, and minority had no fistula formation, 14% (3).

CT usually shows distinctive free air in mediastinum whereas MRI can visualize cerebral air embolism. Diagnostic strategies for prompt identification and reduction of AEF remain limited and challenging due to the infrequent complication and delayed onset.

# **Case Presentation**

A 56-year-old male presented to the hospital for chest discomfort after several days of tachycardia with a heart rate of 145 bpm. He underwent radiofrequency ablation for atrial fibrillation (AFib) 10 days prior. Upon admission, his past medical history included paroxysmal AFib, esophageal reflux, essential hypertension (HTN), and mixed hyperlipidemia. He was diagnosed with Atrial Flutter with Rapid Ventricular Response (RVR) with suspected post-ablative pericarditis, treated successfully with urgent cardioversion. Initial imaging showed pleural effusion and tension pneumothorax, but no signs of AEF or esophageal perforation. Three days since admission, he had recurrent Atrial Flutter with RVR and restored to normal sinus rhythm with cardioversion. The next day, an esophagram revealed esophageal perforation of right lower thoracic esophagus, which was repaired with stent placement via EGD. Four days later, his symptoms worsened, and he developed cough and leftsided hemiparesis. Stroke workup was negative. CT chest showed evidence of a new AEF. An air embolism was suspected. He was subsequently transferred to an outside hospital (OSH) for further AEF evaluation and repair. At OSH, he had atrial repair followed by thoracotomy with stent removal and additional esophageal procedures. His postoperative course was complicated by heparininduced thrombocytopenia. In the setting of a modified anticoagulation course, he developed a medial cerebral artery (MCA) stroke. Complications from this ultimately led to the patient's death.

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## **Diagnostic Imaging**



Image 1: Esophageal Perforation on Initial Esophagram.



Image 3: Computed Tomography (CT) Chest: Locule of gas anterior to the esophageal stent suggestive of an atrio-esophageal fistula.

### Timeline: Initial procedure $\rightarrow$ AEF diagnosis.

Timeline:		
Day:		Event:
0	<u> </u>	Atrial fibrillation ablation
10		Readmission: SOB, palpitations, weakne
11		Echocardiogram: normal. X-ray: small ri
13		X-ray: large right hydropneumothorax.
14		Esophageal perforation diagnosed (imag
15		Repeat Esophagram: no leak
16		Increased leukocytosis. CT head ordered
		CT/MRI BATT negative. CT chest showed
		The patient was transferred to OSH for



The defect measured 10-15mm in diameter (6).

ess  $\rightarrow$  Paroxysmal A. flutter  $\rightarrow$  Direct cardioversion ight pleural effusion.

ages 1 & 2). Stent placed via EGD.

ed  $\rightarrow$  acute left-sided hemiparesis after coughing  $\rightarrow$ d an atrio-esophageal fistula (image 3) for repair.

- higher body mass index (3).

In this case, the patient developed sudden chest discomfort 10 days postablation. Although initial CT with IV contrast showed no esophageal findings, clinical suspicion was high given chest pain out of proportion to other findings (required a PCA pump ultimately), unexplained pleural effusion and pneumothorax. Thus, esophagram with PO contract was performed revealing right lower thoracic esophagus perforation, which was repaired with stenting via EGD. Four days later, patient developed neurological symptoms and subsequent CT chest revealed new AEF. He was transferred to OSH for fistula repair via thoracotomy.

Given the high mortality rate of AEF, the prevention of esophageal injury is crucial. Studies suggest esophageal cooling via direct water injection, catheter contact force limitation, proton pump inhibitors, and non-thermal ablation techniques to improve esophageal protection (4). Since all ablation methods may cause AEF formation, there may be a potential increase in incidence as increasingly more ablations are performed (4). Surgical repair is highly suggested; a case series described open-heart (internal) LA repair of AEF with better outcomes compared to endoscopic and external LA repair (5).

Physicians should keep a high index of suspicion for AEF in any patients with severe chest pain after ablation for  $30 \pm 12$  days post-ablation. While studies showed CT with oral or IV contrast is a safe and sensitive way to diagnose AEF, other diagnostic tests, such as esophagram, are recommended for early interventions. EGD with stenting should be avoided with AEF or potentially with all esophageal perforation post-ablation due to possible neurological injury. Further research is required to compare outcomes of endoscopic repair via EGD stent placement versus surgical repair via thoracotomy in esophageal perforation without fistula formation. In observational studies, primary surgical fistula repair remains the optimal treatment for AEF. Further research will enhance early detection and improve the prognosis of AEF formation.

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### Discussion

The mechanism of AEF development is not fully understood, however, it is related to the anatomical proximity of left atrium (LA) and esophagus.

Primary esophageal wall injury most likely involves 1-way valve from esophageal lumen to LA secondary to ulcer penetration (1).

• Risk factors for esophageal perforation resulting in death or severe neurologic deficits include decreased left ventricular ejection fraction and

### Conclusion

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