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# The Fatal Link: Atrial-Esophageal Fistula after Atrial Fibrillation Catheter Ablation

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## Abstract

Catheter ablation, a cornerstone in the management of symptomatic atrial fibrillation (AF), has been shown to be successful in rhythm control in most patients. It is, however, not without the risk of serious complications, including atrial-esophageal fistula (AEF). This article reports the case of a 64-year-old man whose AF catheter ablation course was complicated by AEF, leading to a fatal outcome. This unfortunate outcome serves to highlight the importance of early diagnosis and treatment of AEF. Clinicians should also be cognizant of AEF risk factors. They should be capable of recognizing the clinical presentation of AEF signs and symptoms so that diagnosis and treatment can be made in a timely manner. After diagnosis, surgery is the preferred treatment, which has been shown to improve outcomes and reduce mortality.

**Keywords:** Atrio-esophageal fistula; Catheter ablation; Atrial fibrillation; Hemodynamic instability

## Introduction

Catheter ablation has become a key element in the management of AF, the most prevalent persistent cardiac arrhythmia. Although the procedure is considered safe and effective, it does pose risks, such as the potentially fatal AEF. It is a feared complication that can occur due to thermal esophageal trauma during the procedure, creating a pathological conduit between the left atrium and esophagus. This complication, if undiagnosed and untreated in its early phase, can lead to hemodynamic deterioration, systemic embolization, and sepsis, with a high rate of mortality [1]. We present an unfortunate case of a fatal outcome following AEF after catheter ablation for AF in a 64-year-old man.



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## History of Presentation

The patient is a 64-year-old male with a medical history that includes abdominal aortic aneurysm, coronary artery disease, hypertension, hyperlipidemia, type 2 diabetes, a renal transplant from a deceased donor due to IgA nephropathy, a pulmonary vein isolation procedure performed four years before, and a successful catheter ablation (posterior left atrial wall isolation) executed five weeks before his emergency department visit. He presented with a 24-hour history of fever, chills, headache, and intermittent periods of disorientation. Upon examination, he had a supple neck without any focal neurologic abnormalities. He appeared lethargic but was alert and well-oriented. His vital signs revealed a temperature of 102 F and no hemodynamic instability. The remainder of the systemic examination was unremarkable.

The initial lab findings indicated an eGFR (estimated glomerular filtration rate) of 62 ml/min/1.73 sqm, normal lactate levels, and an elevated creatinine level of 1.4 mg/dl, increased from a baseline of 1.1 mg/dl. He had a white blood cell (WBC) count of 8.2 TH/uL with a normal differential, and the urinalysis did not show any abnormal findings. The electrocardiogram (ECG) indicated normal sinus rhythm, and the chest X-ray did not reveal any focal consolidation. The contrast-free CT scan of the head showed no evidence of acute territorial infarction, mass effect, or cerebral hemorrhage. The distal esophagus was thickened on a contrast-free CT scan of the abdomen and pelvis, raising concerns for esophagitis. Based on these findings, the possible diagnoses were pneumonia, meningitis/encephalitis, esophagitis, and infective endocarditis.

The patient was initially admitted to the medicine floor. Blood and urine cultures were obtained, and he was empirically started on vancomycin and cefepime. He also received intravenous fluid boluses as part of the sepsis protocol. The patient experienced tachycardia, tachypnea, and another fever spike of 104°F on the second day of his hospital stay. During the medical examination, he was confused, and a new left-sided weakness was discovered. Subsequently, he was moved to the intensive care unit (ICU) due to severe respiratory distress and an altered mental state. He needed to be intubated for airway protection as his mental state progressively deteriorated. The arterial blood gas revealed metabolic acidosis and significant lactic acidosis. Due to hypotension, which was not responsive to fluid boluses, norepinephrine and vasopressin were started to stabilize blood pressure, along with stress dose steroids. His antibiotic coverage was broadened to include meropenem and nystatin. The right frontoparietal lobe had small subacute cortical infarcts, whereas the right medial cerebellar lobe showed subacute intraparenchymal hemorrhage on the head MRI. No obvious sign of intracardiac thrombus or infective endocarditis was seen on transthoracic echocardiography (TTE), but it showed mild regurgitation along with mitral annular calcification. The ejection fraction (EF) was 42%, down from 60% a year earlier. When cerebrospinal fluid from a lumbar puncture was analyzed, it showed increased protein, neutrophilic pleocytosis, and xanthochromia, all indicative of a CSF infection. Gram-positive rods, Lactobacillus rhamnosus, were grown in blood cultures. Later, the patient experienced hemodynamic instability and ventricular tachycardia, necessitating electric cardioversion and initiation of an amiodarone infusion. Considering the recent history of catheter ablation and concerns for potential post-procedural complications, a CT chest was ordered. It revealed many air bubbles in the esophagus adjacent to the posterior wall in the left atrium, prompting concerns for an AEF (Figure). Because of persistent encephalopathy, a CT head was done, which showed intracranial hemorrhage with new embolic cortical infarcts.

After multidisciplinary discussions, surgical intervention was deferred due to the patient's poor prognosis, given multisystem dysfunction and neurological deficits. With the help of the palliative medicine team, a family meeting was held to discuss the goals of care. Palliative care was initiated, and the patient expired within 24 hours.


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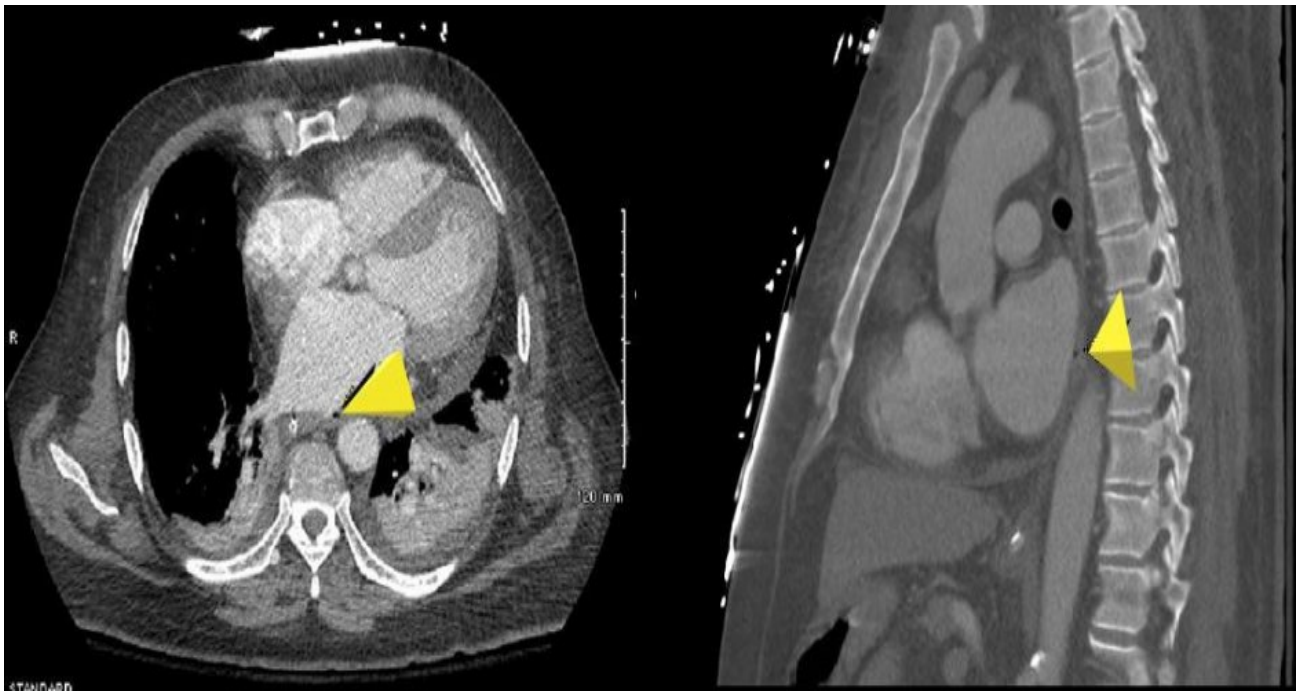

**Figure:** Multiple air bubbles located between the esophagus and the posterior aspect of the left atrium, which is concerning for atrial-esophageal fistula. This is associated with a small pseudoaneurysm along the left atrium's posterior wall.

## Discussion

Ablation is among the most widely used treatments for AF. However, complications like AEF, although thought to happen in less than 0.1% - 0.25% of AF ablation treatments, are a cause for concern [2]. The esophagus's anatomical position posterior to the left atrium raises the risk of injury during the procedure. Mechanisms implicated in esophageal injury include ischemia, infection, direct thermal injury, and aggravation of acid reflux [3]. Research has sought to establish a connection between the occurrence of AEF and the type of ablation performed. Percutaneous radiofrequency ablation has been linked to higher incidences of esophageal injury. However, additional energy sources, like surgical ablation [6], high-intensity focused ultrasound [5], and cryoablation [4], have also been associated with AEF. When using similar radiofrequency ablation parameters, robotic guidance AF ablation has been discovered to have a greater rate of esophageal damage than manual ablation [7].

The diagnostic challenge in identifying AEF is distinguishing it from other complications following ablation, as initial signs and symptoms are often nonspecific. Initial imaging, such as a chest CT scan with contrast, may also be inconclusive. AEF usually presents 1 - 6 weeks after this procedure, manifesting nonspecific symptoms like fever, malaise, chest discomfort, nausea, dysphagia, and odynophagia. These symptoms should alert physicians to investigate and rule out AEF promptly. Early diagnosis is crucial as it may reduce morbidity and mortality, improving the probabilities of recovery, particularly if the esophageal perforation is identified before it communicates with the atrium [8].

The gold standard diagnostic techniques for diagnosing AEF are chest CT and MR imaging. Endoscopy should be avoided as it carries dangers, including risks of further perforation, air embolism leading to strokes and multi-organ damage, sepsis, and septic shock. Given that the death rate is close to 100% if prompt intervention is not received, early surgical repair deserves to be seriously considered upon diagnosis [9].


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The optimal surgical approach to AEF involves a combined repair of the left atrium and esophagus. This procedure is typically performed via sternotomy with cardiopulmonary bypass, which allows for thorough debridement and secure closure of the defect in the atrial wall [10]. Depending on the extent of injury, esophageal repair may involve resection of necrotic tissue and using tissue flaps, such as omental or muscle flaps, to create a barrier between the esophagus and the atrium, thereby reducing the risk of recurrence. Postoperative management includes the administration of broad-spectrum antibiotics, providing enteral nutritional support, and vigilant monitoring for signs of infection or leakage. Timely surgical intervention has been consistently associated with significantly improved survival rates compared to conservative or endoscopic treatments.

This example starkly illustrates the fatal results of a delayed diagnosis in AEF. Early surgical consultation, timely imaging, and a high index of suspicion are essential to improving survival in affected patients. Raising awareness among electrophysiologists, intensivists, and emergency providers is essential to recognizing and managing this complication before it becomes irreversible.

## Conclusion

AEF is a rare yet potentially lethal side effect of AF ablation. Establishing standards for the early diagnosis of this potentially deadly consequence and recommendations on the safety of ablation treatments are essential. After the procedure, patients should be closely monitored for any warning signs or symptoms of AEF, allowing for early detection and interventions that improve outcomes.

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