

Atrioesophageal Fistula: A Rare Complication of Radiofrequency Ablation

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ABSTRACT

A 75-year-old woman was admitted with fever, chills, altered mentation, and right-sided weakness. A month earlier, she had undergone catheter radiofrequency ablation for treatment of chronic atrial fibrillation. A magnetic resonance imaging scan of her brain revealed septic emboli with multiple bilateral cerebral and cerebellar infarcts, as well as extensive bilateral leptomeningeal enhancement. Blood cultures were positive for *Streptococcus mitis*, *Rothia mucilaginosa*, *Streptococcus pneumoniae*, and *Candida albicans*, which suggested a connection between gastrointestinal and cardiovascular systems. A chest computed tomography scan with contrast showed a curvilinear low attenuation structure communicating between the esophagus and the left pulmonary vein—an atrioesophageal fistula. Ten days after admission, the patient died from multiple cerebral septic emboli secondary to atrioesophageal fistula following radiofrequency ablation.

INTRODUCTION

A rare complication of radiofrequency ablation is atrioesophageal fistula,¹ which typically manifests as a new onset of neurological symptoms or as systemic polymicrobial infection and/or gastrointestinal bleeding. Fatal events usually are caused by polymicrobial sepsis and cerebral air embolism.² We report the case of a 75-year-old woman who died from multiple cerebral septic emboli secondary to atrioesophageal fistula following radiofrequency ablation for chronic symptomatic atrial fibrillation.

CASE REPORT

A 75-year-old woman came to our emergency department for evaluation of altered mentation and possible stroke with right-sided weakness. She had a 10-year history of symptomatic paroxysmal atrial fibrillation despite medical therapy with sotalol, amiodarone, dronedarone, and dofetilide. Five weeks earlier, while wintering in Arizona, she had seen her electrophysiologist, who recommended

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radiofrequency ablation of the left atria and pulmonary veins. She underwent the procedure, during which the electrophysiologist noted difficulty locating the right inferior pulmonary veins due to her anatomy. The procedure was further complicated by recurrent episodes of ventricular tachycardia and atrial fibrillation that required multiple cardioversions. Following the procedure, she was believed to be stable.

Ten days later she returned with episodic dizziness associated with blurred vision. A computed tomography (CT) scan of the

brain was negative for any acute pathology. She was subsequently seen in neurology, where she was started on steroids and referred for vestibular therapy, owing to symptoms consistent with vestibular neuritis.

Approximately 3 weeks later she returned to the emergency department for evaluation of altered mentation and possible stroke with right-sided weakness. She had a fever of 38.3°C, chills, and leukocytosis (white blood cell count of 16 440/ μ L [16.44×10^9 /L] with 20% bands).

An initial head CT scan was performed and indicated no acute hemorrhage or mass. Subsequently, a magnetic resonance imaging (MRI) scan of her brain was obtained (Figure 1), the results of which were concerning for septic emboli with multiple bilateral cerebral and cerebellar infarcts, as well as extensive bilateral leptomeningeal enhancement. Blood samples were collected for culture, and the patient was started on broad spectrum antibiotics.

Within a few hours of admission, the patient developed a new left-sided hemiparesis. Results of a repeat head CT scan were negative for acute hemorrhage. A transthoracic echocardiogram was performed to investigate for possible infective endocarditis, but no vegetations were apparent on the valves. Further evaluation for infective endocarditis with transesophageal echocardiogram indicated no gross thrombus or vegetation.

The patient's neurological status continued to fluctuate. Lumbar puncture was performed after several days of withholding rivaroxaban. Cerebrospinal fluid analysis indicated inflammatory changes

with no evidence of bacterial infection.

Initial blood cultures were positive for *Streptococcus mitis*, *Rothia mucilaginosa*, *Streptococcus pneumonia*, and *Candida albicans*. The polymicrobial nature of her infection suggested a connection between gastrointestinal and cardiovascular systems, but a CT scan of the abdomen and pelvis showed no obvious abdominal pathology to account for the positive blood cultures. A chest CT scan with contrast was obtained (Figure 2), which indicated a curvilinear low attenuation structure communicating between the esophagus and the left pulmonary vein. Our patient's clinical condition and imaging studies led to an atri-esophageal fistula diagnosis, a complication of her cardiac radiofrequency ablation. Approaches to therapy were discussed with the family, who chose comfort care in the setting of her rapidly declining neurological status. The patient died on the 10th day of her hospital admission.

DISCUSSION

Cardiac radiofrequency ablation is associated with serious adverse effects, such as pericardial effusion/tamponade, hemo/pneumothorax, diaphragmatic paralysis, valve damage, sepsis, abscess or endocarditis, atrial flutter/tachycardia, stroke, pulmonary vein stenosis, arteriovenous fistula, atri-esophageal fistula, and, sometimes, death.^{3,4} Although the rate of atri-esophageal fistula after recent radiofrequency ablation is less than 1%, its fatality rate is greater than 70%, either secondary to neurological causes, such as air embolism, or to polymicrobial sepsis.¹ Early signs and symptoms typically include neurological symptoms, such as altered mental status, seizures, hemiparesis, and stroke. Other symptoms may include fever, lethargy, weakness, chest pain, dysphagia, hematemesis, or melena.⁵ Symptoms can manifest anywhere from 2 to 6 weeks after radiofrequency ablation.

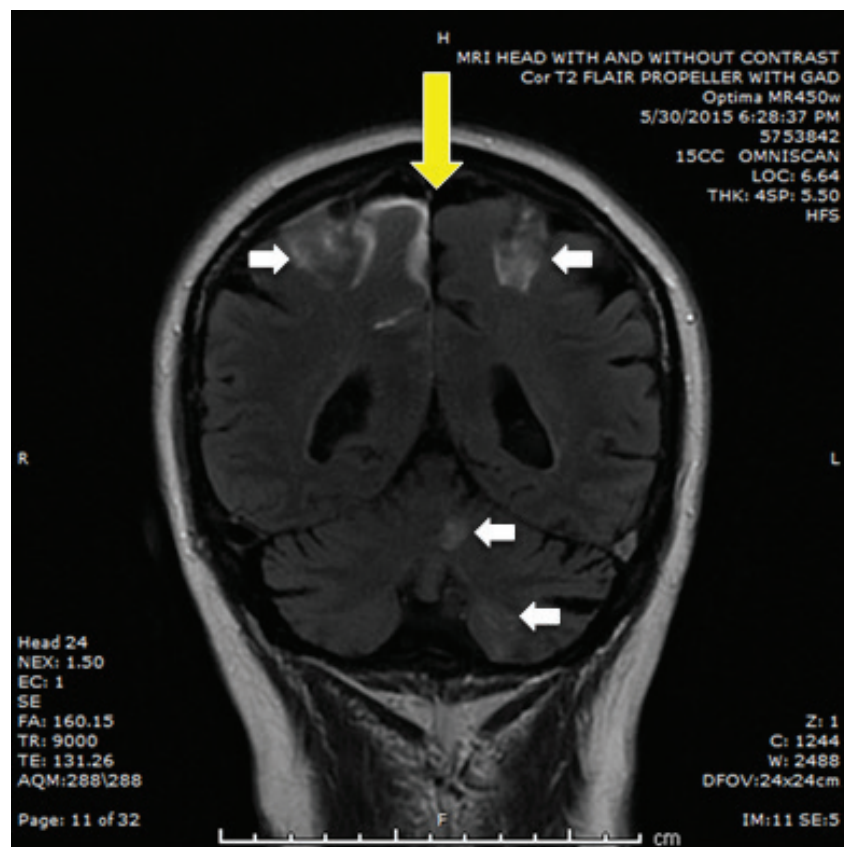
Both anatomical and procedural factors have been suggested as causes of fistula development after radiofrequency ablation. The proximity of the esophagus to the left atrium is the most important factor responsible for the pathogenesis of esophageal mucosal injury during catheter ablation. Patients with left atrial dilatation have thinner fat pads and a larger contact area between the esophagus and the left atrium.⁶ Extremely small patients also have been posited to be at greater risk.⁷ Of the possible procedural factors, thermal injury is believed to be the most likely, and the risk is thought to increase as temperature and duration increase.⁸ General anesthesia, too, has been suggested as a possible cause of esophageal injury

because it limits the usual motility of the esophagus.⁹

The delayed presentation of atri-esophageal fistula after ablation suggests that mechanical perforation of the atrial wall during ablation is unlikely to be responsible for its development. Thermal injury is thought to affect the microvasculature of esophageal tissue, leading to ischemic necrosis of the mucosal layers. The progression of esophageal ulceration to atri-esophageal fistula formation has been associated with gastric hypomotility, esophagitis, and resultant acid reflux from vagal plexus injury.^{6,10}

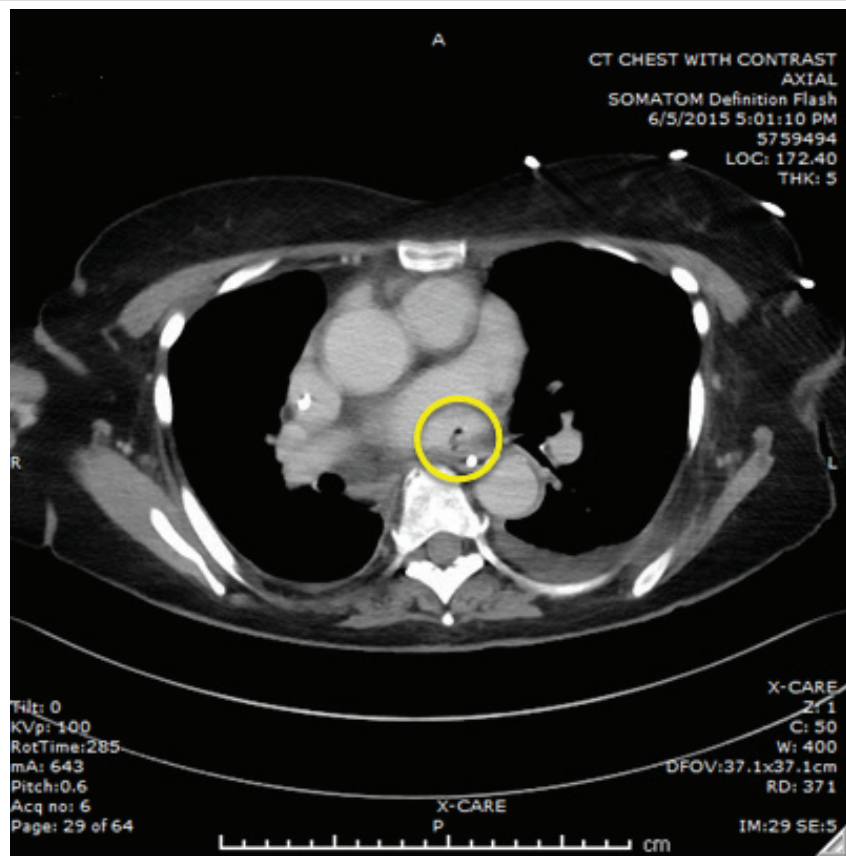
Progressive enlargement of atri-esophageal fistula can be promoted in two ways. One mechanism involves the relative higher intra-atrial pressure compared to esophageal pressure, which could cause a significant amount of blood to pass through the fistula, leading to gastrointestinal bleeding. The other mechanism involves increased esophageal pressure. Esophageal peristalsis can increase esophageal pressure to 10 times greater than intra-atrial pressure, leading to introduction of air in the cardiovascular system causing air emboli and polymicrobial sepsis.^{1,11} Performing invasive diagnostic procedure such as upper endoscopy and transesophageal echocardiogram, or even placing a nasogastric feeding tube, can promote esophageal peristalsis and, hence, enlargement of atri-esophageal fistula.

Figure 1. Magnetic Resonance Imaging (MRI) Scan of the Brain.



MRI of the brain (T2 Flair images with gadolinium enhancement) demonstrates multiple cerebral infarcts (white arrows) and leptomeningeal enhancement (yellow arrow).

Figure 2. Computed Tomography (CT) Scan of the Chest With Contrast



CT scan of the chest with contrast shows curvilinear low attenuation communication between the esophagus and the left pulmonary vein (center of yellow circle).

A CT scan of the chest seems to safely provide necessary clinical and anatomical information. Surgical repair is the definitive treatment and improves chance of survival. Muscle flap or pericardial patch is used to separate the esophagus and the atrium. However, the general and neurological condition of the patient guides treatment because surgical repair involves many risks.³

A few successful cases of nonsurgical treatment with esophageal stenting and pericardiocentesis have been reported. In these cases, the thermal injuries were small, and a scar was formed.¹² However, stenting is known to have an increased risk for air embolus.^{2,3} Regardless of surgical intervention, early administration of antibiotics and supporting the patient's nutritional status via total parenteral nutrition were paramount in all cases.¹³ Aggressive prophylactic treatment with a proton pump inhibitor is theorized to reduce the risk for atrioesophageal fistula formation, but evidence is lacking. For the neurological sequelae, early use of hyperbaric oxygen has been described, but no specific data validate its effectiveness.¹⁴

CONCLUSION

Having high suspicion is probably the most important factor in early recognition of atrioesophageal fistula and avoiding interventions that have the potential to worsen the fistula and, in turn,

increase mortality. Early administration of antibiotics and nutritional support improves outcomes. In patients with favorable general and neurological status, however, surgical intervention is the definitive and most commonly used approach to correct atrioesophageal fistula.

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