Unusual esophageal injury after atrial fibrillation ablation: Early diagnosis and treatment to optimize outcomes

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Introduction

Atrial fibrillation (AF) is the most common clinically relevant arrhythmia globally, with more than 5 million new cases per year.[1] In the United States, about 50,000 ablation procedures for AF are performed annually. Cardiac ablation has a complication rate of less than 5%, and serious, life-threatening complications including cardiac tamponade, pericardial effusion, transient ischemic attack, stroke, pulmonary vein stenosis, and atrioesophageal fistula (AEF) occur in less than 3% of procedures.[2,3] Although AEF is rare, patients with AEF suffer a high mortality rate. Early recognition and prompt treatment of esophageal injury are vital to maximizing the probability of a positive outcome. This case study illustrates the variability in presenting symptoms and the importance of prompt recognition and treatment of postablation esophageal injury.

Case report

A 61-year-old man with a history of myocardial infarction with drug-eluting stent to the right coronary artery, remote ischemic stroke without residual neurological deficits, gastroesophageal reflux disease, and hiatal hernia underwent radiofrequency catheter ablation of persistent AF.

The index ablation procedure included antral pulmonary vein isolation, left atrial roof line, cavotricuspid isthmus line, and focal ablation of a premature atrial contraction originating from the left atrial inferior wall (Figures 1 and 2). Ablation was performed in a power-controlled mode at 50 W with a temperature cutoff of 40°C with a ThermoCool SmartTouch SF irrigated, force-sensing ablation catheter and the CARTO3 mapping system (Biosense Webster, Diamond Bar, CA). An esophageal temperature probe was used, and any heating of the esophagus prompted termination of ablation until return to baseline. Maximum temperature

KEY TEACHING POINTS

- Atrioesophageal fistula (AEF) remains a rare but grave complication of atrial fibrillation (AF) ablation. As the number of AF ablations performed increases, it is expected that even rare complications may become more prevalent. Early recognition of esophageal injury before the development of AEF may decrease the incidence of progression to AEF and improve outcomes.
- Symptoms of AEF are nonspecific and occur weeks after ablation. Patients often do not report pain, and often present outside the electrophysiology department, to clinicians who may not consider AEF on their differential. Esophageal injury and AEF should be on the differential for any patient presenting with fever, neurological changes, and gastrointestinal complaints within 2 months of AF ablation. It is incumbent on electrophysiologists to educate their emergency department and internal medicine colleagues about AEF.
- When AEF is strongly considered clinically, it is important to aggressively treat the patient for presumed AEF, as no imaging study or test can definitively rule out esophageal injury. It is also important to avoid instrumentation and insufflation of the esophagus. These patients must be closely monitored for signs of AEF. Prompt and aggressive treatment may prevent progression to life-threatening and frequently fatal outcomes.

KEYWORDS Atrial fibrillation; Catheter ablation; Esophageal injury; Atrioesophageal fistula; Ablation complication; Septic stroke; Air emboli
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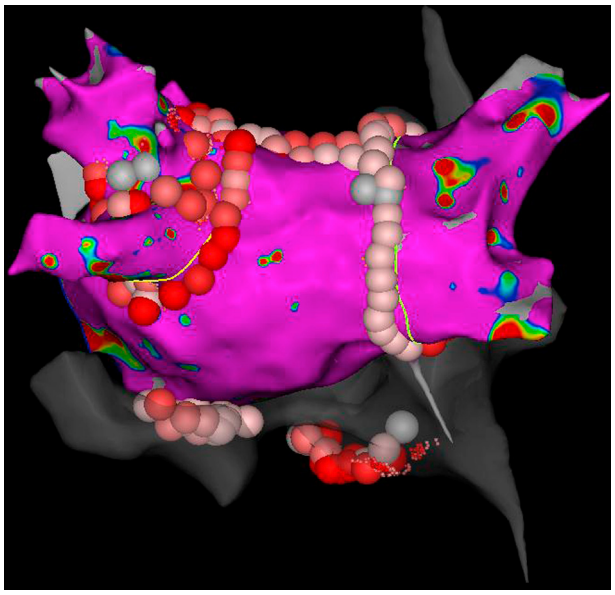

Figure 1 Posteroanterior projection of an electroanatomic map of the left atrium (purple) and coronary sinus/right atrium (gray) with superimposed ablation tags for antral pulmonary vein isolation, left atrial roof line, focal atrial tachycardia originating from inferoposterior left atrium, and typical tricuspid flutter.

during the case remained below 38°C. Ablation on the posterior wall was limited to 8-second maximum duration with goal force 10 g or greater, with a goal total force-time integral 400 gram-seconds. The patient recovered after the procedure and went home the same day. He was seen in follow-up 10 days postablation and was in sinus rhythm at the time, with no complaints.

Twenty-one days after ablation, he presented to the emergency room with a 3-hour history of sudden-onset, nonradiating chest pain. He denied bleeding, dysphagia, or neurological symptoms. He had a blood pressure of 168/85 mm Hg but otherwise had normal vital signs. He appeared uncomfortable, but well; his lungs were clear, his abdomen was soft and nontender, and no focal neurological findings were observed. He was in sinus rhythm on electrocardiogram; laboratory studies were significant for mild leukocytosis. Echocardiogram showed normal left ventricular size and function and a trace pericardial effusion. He was diagnosed with acute pericarditis and discharged home on ibuprofen 800 mg every 8 hours. He returned 7 hours later with worsening pleuritic chest discomfort that prevented him from lying supine and a reported fever of 38.7°C. Rhythm on electrocardiogram had changed to AF with a ventricular rate of 106 beats per minute with no ischemic changes. Laboratory studies showed a persistent, mild leukocytosis.

He was admitted with a diagnosis of pericarditis and fever. His chest pain initially resolved with nonsteroidal anti-inflammatory drugs, but when it returned the following morning, an AEF was suspected. Computed tomography (CT) with intravenous contrast showed a 5 mm × 6 mm gas collection posterior to the left atrium; however, no gas was seen in the left atrium and no contrast was seen in the esophagus (Figure 3). This study was concerning for an esophageal perforation but showed no evidence of fistula to the left atrium. An esophagram with gastrografin contrast showed persistent, nonspecific staining of the mid esophagus without frank rupture or fistula.

Once esophageal injury was suspected, antibiotics, antifungals, bowel rest, and proton pump inhibitor (PPI) therapies were initiated. Repeat esophagram with iohexol contrast 4 days after admission showed no extravasation of contrast or dysmotility. Repeat CT of the chest with intravenous and oral contrast 1 week after initial presentation showed "mild inflammation surrounding the distal esophagus with a possible single bleb of pneumomediastinum versus small diverticulum." No extraluminal oral contrast was observed to suggest esophageal rupture or leak, and no contrast was seen in the esophagus following administration of intravenous contrast. The patient was discharged after a 9-day hospitalization on a pureed diet, amoxicillin and clavulanic acid 875 mg/125 mg every 8 hours, and fluconazole 400 mg daily. His symptoms improved, and his esophageal injury never progressed to a fistula.

When the patient followed up 96 days postablation, he was in sinus rhythm, and his symptoms had resolved aside from some mild dysphagia and heartburn. CT of the chest 14 months postablation no longer showed air or inflammation in the space between the left atrium and the esophagus.

Discussion

AF ablation provides improved rhythm control and quality of life compared to antiarrhythmic drugs, is associated with

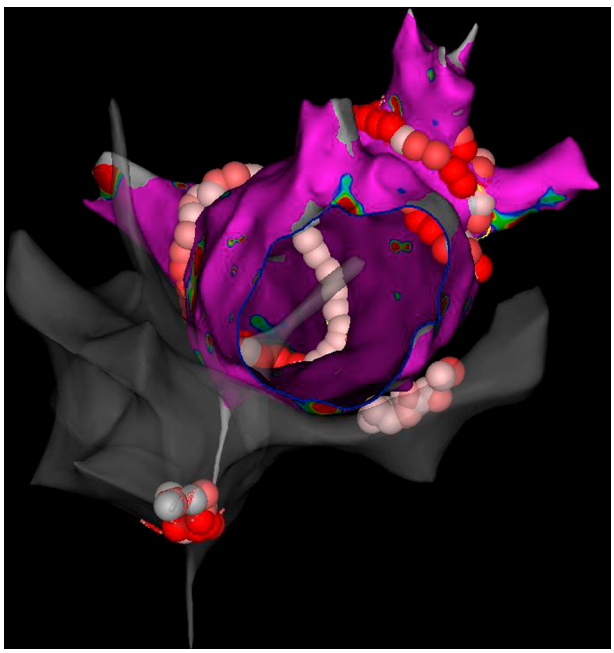

Figure 2 Left anterior oblique projection of posteroanterior electroanatomic map of the left atrium (purple) and coronary sinus/right atrium (gray) with superimposed ablation tags for antral pulmonary vein isolation, left atrial roof line, focal atrial tachycardia originating from inferoposterior left atrium, and typical tricuspid flutter.

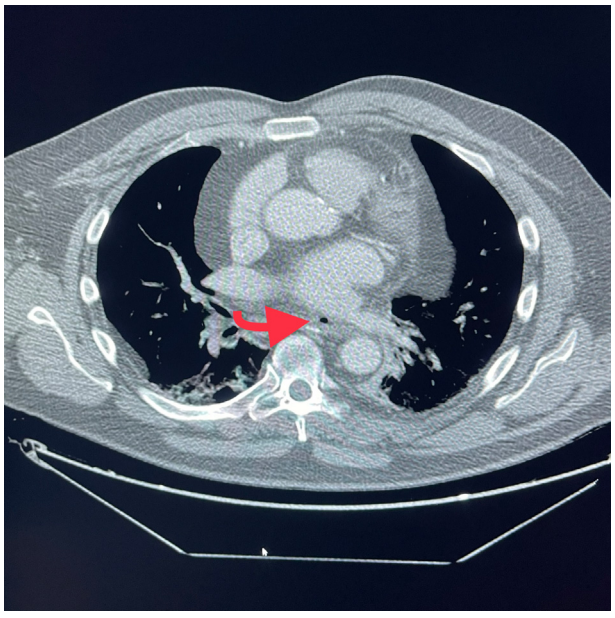

Figure 3 Axial computed tomography scan after the patient presented with fever and pleuritic chest pain showing small air collection (*red arrow*) located adjacent and posterior to the left atrium.

improved overall survival in patients with advanced heart failure, and is the standard of care for symptomatic AF refractory to antiarrhythmic drugs.[3–5] The number of AF ablation procedures is growing exponentially, as it is increasingly effective and safe. The mortality rate of AF ablation is at only 0.01%, with the cause of death most commonly related to cardiac tamponade, stroke, or AEF.[1,4] Serious complications after AF ablation include pericardial effusion and tamponade, hemothorax and pneumothorax, transient ischemic attack and stroke, pulmonary vein stenosis, and AEF.[4,5]

The prevalence of esophageal injury after AF ablation and its progression to AEF is unknown.[3,6] Esophageal injury tends to be a slow, progressive process ranging from shallow mucosal inflammation to deep esophageal ulceration that progresses to fistula formation invading the mediastinum, pericardium, and left atrium.[7] Symptoms of esophageal injury include pleuritic chest pain, nausea, fever, throat pain, and abdominal pain. Endoscopic sonography has demonstrated that mucosal changes of the esophagus after ablation can be reversible with bowel rest, antimicrobials, and PPIs but may progress to esophageal perforation and AEF.[1,7] AEF is a rare (0.015%–0.04%) but dreaded complication of AF ablation in which a fistula develops between the esophagus and the heart's left atrium. AEF is universally fatal without intervention.[1,3,8]

Symptoms of esophageal injury include pleuritic chest pain, nausea, fever, throat pain, and abdominal pain. It is unclear how often esophageal injury occurs postablation or how often injury progresses to esophageal ulceration and AEF. Screening for esophageal lesions postablation may not predict the risk of subsequent AEF.[6,9] Injury may progress to AEF days to weeks after ablation, with a peak presentation time of 21 days postablation.[1,3,8] As esophageal injury progresses to AEF, recurrent strokes, sepsis, and gastrointestinal (GI) bleeding develop.[1,3,5,8] The late presentation, nonspecific symptoms, and failure to report a history of AF ablation frequently results in the misdiagnosis of esophageal injury and AEF.

The most common presenting symptoms of AEF are fever (73%) and neurological symptoms (72%), which include confusion, seizures, meningitis, postprandial transient ischemic attack, hemiplegia, and stroke.[1,8,10] In published case studies, GI symptoms were present in only 41% of patients with AEF.[1,8] Systemic emboli and septicemia are more common than GI bleeding because pressures in the esophagus are greater than intra-atrial pressure allowing esophageal contents to enter the left atrium while preventing blood from entering the esophagus.[10] Blood cultures positive for bacteria from the oral cavity, fever associated with neurological symptoms, and postprandial neurological symptoms are concerning for AEF.

The causes of AEF are not fully understood but are likely due to thermal injury to the esophagus that leads to inflammation, coagulation necrosis, ischemia, and esophageal ulceration.[3] The anterior esophageal nerve plexus and esophageal arteries pass millimeters from the pulmonary veins and posterior atrial wall, predisposing the esophagus to thermal injury during catheter ablation.[3,7,8]

There are no validated methods to prevent AEF.[5] Common preventative methods include esophageal temperature monitoring, esophageal cooling, preprocedure localization of the esophagus, active displacement of the esophagus during ablation, and prophylactic PPI use. Despite these measures, AEF persists.[1,4,8]

When AEF is suspected, no test can definitively rule it out. CT of the chest with oral, water-soluble contrast is the most useful diagnostic test.[4] Signs of AEF imaging studies include chest CT with contrast in the esophagus, air in the left atrium, pleural or pericardial effusions, and esophageal thickening. A contrast esophagram may show extravasation of contrast material.[1,3,10] Brain imaging that shows air emboli or transthoracic echocardiography that shows an "aquarium sign," or air bubbles in the left atrium confirm the diagnosis of AEF.[1,8] Laboratory studies often show leukocytosis, elevated serum C-reactive protein level, thrombocytopenia, and elevated erythrocyte sedimentation rate. More than 70% of patients with AEF develop symptoms of infection, and blood cultures may be positive for polymicrobial infection with oral or GI organisms.[1]

Once AEF is suspected, transesophageal echocardiography and esophagogastroduodenoscopy should be avoided because instrumentation of the esophagus can cause air to enter the left atrium, leading to massive cerebral embolism and enlargement of the fistula tract.[1,3–5] In our patient, there were no definitive imaging studies of AEF, but esophageal injury with subsequent pneumomediastinum vs esophageal bleb was observed. Although it is possible such injury occurred as a result of esophageal temperature probe placement and movement, we felt it necessary to treat for

potential AEF owing to its clinical likelihood following ablation and high mortality if left untreated.

Conservative treatment of esophageal injury includes broad-spectrum antibiotics, supporting nutritional status, and PPIs. Studies suggest that early detection and aggressive medical treatment can reverse early esophageal injury. Studies also show that about 10% of esophageal ulcers progress to esophageal perforation and subsequent AEF.[7] Esophageal stenting to treat AEF carries a high mortality rate, and stent insertion complications include pneumopericardium, stent dislocation, and hematemesis.[1,7] Medical management and esophageal stenting alone after the development of AEF has a mortality rate of 97%.[1]

Aggressive and timely surgery is the cornerstone of AEF management and offers the highest survival rate, but the mortality rate remains between 33% and 41% even with surgical intervention.[1,5,8] There is no agreed-upon surgical approach, but successful surgery generally involves resecting the necrotic parts of the esophagus and closure of the left atrium with a muscle flap or pericardial patch to separate the esophagus and the left atrium.[3,5,10]

Conclusion

With the increase in AF ablation procedures, clinicians can expect to see rare complications more frequently. The variability in presentation and nonspecific symptoms lead to a broad differential, so several medical specialties may initially see these patients. Increased awareness will ensure prompt diagnosis, minimize esophageal manipulation, and ensure emergent, aggressive surgical intervention and early medical therapy with the goal of improved clinical outcomes of AEF.[1,8,10]

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