

Esophageal T-tube: A novel approach to atrioesophageal fistula repair



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Introduction

It is estimated that major complications occur in 4% of patients undergoing catheter ablation of atrial fibrillation (AF).¹ One of the most dreaded complications of AF ablation is atrioesophageal fistula (AEF), which results from collateral thermal injury to the esophagus owing to its close relation to the posterior wall of the left atrium. Patients present with fever, sepsis, esophageal symptoms, and/or devastating stroke weeks after the procedure.² Although rare, AEF has a high mortality rate even when treated aggressively, suggesting the optimal management approach remains ill-defined.³ We report the first case of successful surgical outcome of an AEF with direct left atrial repair followed by an esophageal T-tube.

Case report

A 52-year-old man traveled to Australia to compete in a triathlon and presented to our hospital with 5 days of fever, epigastric pain, and vomiting 29 days after undergoing radiofrequency ablation for AF in his home country.

Operative records from the hospital where he had undergone catheter ablation described circumferential pulmonary vein isolation performed using the ESI NAV-X mapping system and Tactiath Quartz 75 contact-force irrigated radiofrequency ablation catheter (St Jude Medical, St Paul, MN). Ablation time, power settings, temperature, impedance, and contact-force values were not reported. Esophageal temperature monitoring was not used. Medical comorbidities included ankylosing spondylitis treated with ibuprofen. Post-ablation proton pump inhibitor was not administered.

He was febrile to 38.8°C with rigors and diaphoresis. White blood cell count was $8.0 \times 10^9/L$ and C-reactive protein 76 mg/L. He was admitted under the Infectious Diseases Service and empirically treated with intravenous (IV) flu-

cloxacillin and gentamicin. On the first day of admission he was found unresponsive, pale, and diaphoretic and received cardiopulmonary resuscitation for 2 minutes. After regaining consciousness, he had fixed rightward gaze, dense right hemiparesis, and dysphasia, which improved over 24 hours. Brain computed tomography (CT) scan revealed no acute abnormality. Blood cultures grew multiple streptococcal species and antibiotics were changed to IV benzylpenicillin, vancomycin, and gentamicin. Transthoracic echocardiography was normal. Brain magnetic resonance imaging (MRI) demonstrated multifocal areas of diffusion restriction throughout both cerebral hemispheres, consistent with cardioembolism, in addition to a more diffuse signal abnormality within the hippocampi, suggestive of concurrent hypoxic-ischemic injury. An urgent electrocardiographic-gated cardiac CT scan was then performed with IV and oral contrast. This demonstrated a mildly hyperdense pericardial collection with gas locules adjacent to the left pulmonary veins, with beaking of the posterior wall of the left atrium toward the esophagus, consistent with AEF (Figure 1). There was no active extravasation of contrast from the left atrium to the mediastinum or esophagus or intracardiac air.

The patient was taken urgently to the operating theatre for repair of the AEF under the care of both cardiothoracic and upper gastrointestinal surgeons. A right anterior thoracotomy was performed through the fourth intercostal space. Cardiopulmonary bypass was established. Upon opening of the pericardium, the pericardial fluid was purulent and there was an infective rind overlying the heart. There was a necrotic 2 × 2-cm area at the posterior left atrium. The left atrium was opened via an incision in the Sondergaard groove. The breach in the posterior left atrial wall was identified close to the ostium of the left inferior pulmonary vein, with clearly defined margins. The defect was repaired using a bovine pericardial patch and the left atrium closed (Figure 2A).

The esophageal perforation was then identified via intraoperative endoscopy at 35 cm from the incisors and a T-tube was placed into the esophagus via an incision just posterior to the hilum of the right lung. This was sutured in place and brought to the skin through the right lateral chest wall (Figure 2B and C). After the patient was weaned from

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KEY TEACHING POINTS

- Fever, sepsis, esophageal symptoms, upper gastrointestinal tract bleeding, and/or neurologic symptoms occurring days to weeks after atrial fibrillation ablation should prompt evaluation for atrioesophageal fistula (AEF).
- Contrast-enhanced thoracic computed tomography or magnetic resonance imaging is the appropriate diagnostic test. Transesophageal echocardiography and esophageal endoscopy are contraindicated owing to the risk of fatal massive air embolism.
- Urgent surgical intervention is indicated to mitigate the high morbidity and mortality of AEF. After direct repair of the left atrial defect, the esophageal defect may be repaired directly or using a T-tube. Esophageal stenting should not be used owing to very poor outcomes.

cardiopulmonary bypass, a pericardial flap was generated to lie abutting the posterior wall of the left atrium.

He was extubated on the first postoperative day. Initially, feeding was achieved with total parenteral nutrition. Serial thoracic CT scans with IV and oral contrast demonstrated a small leak of oral contrast into the gas-filled space between the esophagus and atrium, anterior to the tip of the esophageal T-tube. This resolved over the course of 5 weeks. On the 36th postoperative day he returned to the operating theatre for removal of the esophageal T-tube. Gastroscopy demonstrated that the T-tube was intact. A fistulogram via the distal T-tube demonstrated no extravasation of contrast, with contrast seen to track through to the esophagus. The T-tube was transected at the skin and pulled out through the esophagus via a snare under endoscopic visualization. Nasogastric feeding was commenced 7 days after removal of the esophageal T-tube following a CT scan that demonstrated no evidence of esophageal leak, and oral feeding was started after 10 days. The patient made a good neurologic recovery. On the 57th day of admission he was retrieved back to hospital in his home country.

Discussion

Together with cardiac tamponade and stroke, AEF is a potential cause of death following catheter ablation of AF.⁴ The incidence approximates 0.03%–0.08%,⁵ but the true incidence may be higher owing to underreporting. The extreme rarity of AEF accounts for the limited understanding of its pathogenesis and treatment. Esophageal mucosal changes consistent with thermal injury were detected by endoscopy in up to 47% of patients soon after AF ablation, and esophageal ulcerations were observed in 14%–18%.^{6,7} The mechanism of progression of early mucosal lesions to AEF remains unknown. AEF formation has been reported with multiple ablation energy

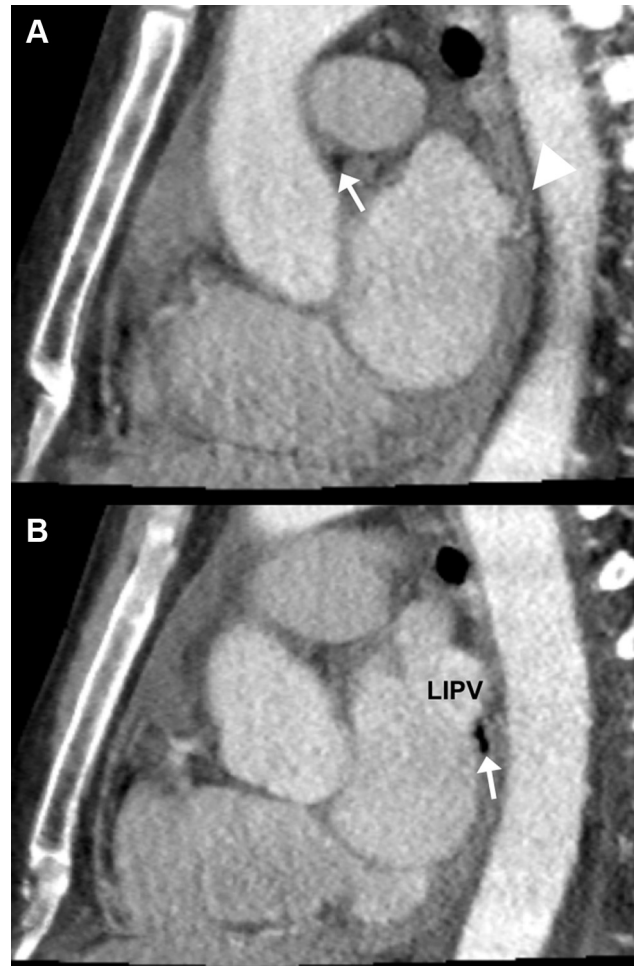


Figure 1 Contrast thoracic computed tomography scan shows **A**: beaking of the posterior wall of the left atrium toward the esophagus (*arrowhead*) and **B**: gas locules (*arrow*) adjacent to the left inferior pulmonary vein (LIPV), consistent with atrioesophageal fistula.

sources, including radiofrequency, cryoablation, high-intensity focused ultrasound, and surgical ablation procedures.² Ablation strategies incorporating more extensive lesion sets at the posterior left atrium increase the magnitude and duration of local tissue heating and the risk of esophageal injury. Prolonged transesophageal echocardiogram probe dwell time, the use of general anesthesia, and gastric acid reflux are other potential factors that have been implicated.⁸ It is possible that use of nonsteroidal anti-inflammatory drugs in our patient contributed to impaired mucosal defenses by disrupting the normal cytoprotective prostaglandin barrier in the esophagus, exacerbating an initial mucosal injury.

Common strategies that have been employed to prevent AEF formation include identification of esophageal position in relation to the left atrial posterior wall with preprocedural CT or MRI and avoiding ablation in the vicinity of the esophagus, limiting energy output and duration at posterior sites, and esophageal temperature monitoring.⁸ Cases of AEF have occurred despite these preventative measures. Consequently, a high index of suspicion must remain if a patient presents with suggestive symptoms, including fever, upper



Figure 2 Atrioesophageal fistula repair. **A:** Endocardial view of posterior left atrial wall showing bovine pericardial patch repair of the atrial perforation. **B:** Esophageal T-tube prior to insertion. **C:** Course of the T-tube from the esophagus to the chest wall posterior to the right lung.

gastrointestinal symptoms, and/or neurologic events, in the days to weeks following an AF ablation. The mean time from ablation to presentation is 20 ± 12 days (range 2–60 days).²

The presentation of AEF may mimic more common conditions such as infective endocarditis and upper gastrointestinal bleeding, which may prompt the need to perform transesophageal echocardiography or endoscopy. However, esophageal instrumentation is strictly contraindicated. If performed, these procedures may have disastrous consequences, as insufflation of the esophagus can lead to massive air embolism, catastrophic cerebrovascular events, and death.² The appropriate diagnostic test is a contrast-enhanced CT or MRI of the chest, which may show pneumomediastinum, hemopericardium, and/or intracardiac air.²

Once a diagnosis of AEF is established, urgent surgical intervention offers the only hope for a good outcome. Although early series described near-universal fatal outcome,⁹ it is possible that greater awareness has led to improved outcomes with early diagnosis and treatment. Overall mortality remains high at approximately 60% in reported cases: 83% in patients managed conservatively and 34% in those who underwent operative management.² The residual high mortality in operated cases suggests that the optimal management approach may not be defined.

The optimal approach to address the esophageal perforation in AEF is not known. In nonablation patients when spontaneous esophageal perforation or iatrogenic perforation owing to esophageal instrumentation presents early (within 24 hours) it is generally accepted that the best treatment is primary surgical repair.¹⁰ Esophageal perforation presenting later than 24 hours is associated with prolonged exposure to local inflammation with tissue-damaging enzymes and bacterial products, resulting in necrotic and edematous esophageal tissues that hold sutures poorly. Primary repair in this setting is difficult and more likely to fail. This is reflected in the high mortality following surgery in late compared to early presentations of esophageal perforation. Primary repair for acute esophageal perforation has a mortality rate of approximately 10%, but this increases to 20%–40% when repair is delayed beyond 24 hours.¹¹ Management options for late esophageal perforation include

direct repair and esophageal stenting and more extensive approaches, such as esophagectomy or esophageal diversion and exclusion.¹² Esophageal stenting has been performed in AEF, with very poor results. There is 1 report of successful outcome following esophageal stenting for AEF, but all other reported cases have resulted in death.¹³

The use of a T-tube to treat delayed esophageal perforation overcomes some of the limitations of primary repair.¹⁴ The rationale is to convert the perforation into a controlled fistula that drains the area surrounding the perforation of inflammatory exudate as well as esophageal secretions and gastric acid. This allows delayed healing of the esophageal perforation and reduces the risk of mediastinal infection. The T-tube repair uses a large-diameter 6- to 10-mm tube with 2 limbs lying inside the esophageal lumen. The tubing is brought through the defect and the esophageal wall closed loosely around the tube with fine interrupted absorbable sutures. The tube is externalized and secured to the chest wall, and it has been recommended that the tube be anchored to the diaphragm to prevent aortic erosion from pressure necrosis (Figure 3). Healing is monitored with serial contrast CT scans and the majority of T-tubes are removed within 3–6 weeks.

Linden and colleagues¹¹ reported a single-center experience of 43 operations for esophageal perforation where a T-tube repair was used in a significant proportion of cases. The majority of perforations in this series were spontaneous (51%), with iatrogenic perforation owing to dilatation,

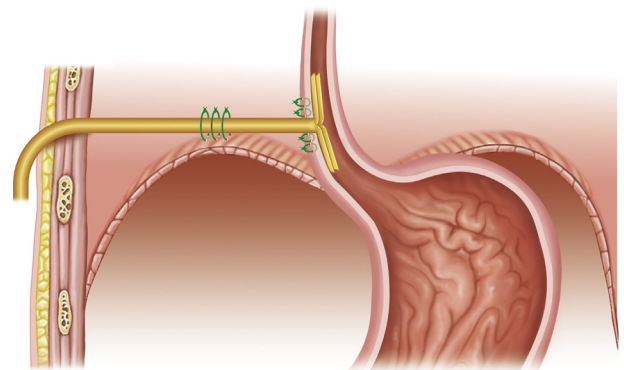


Figure 3 T-tube repair of atrioesophageal fistula. See text for explanation.

endoscopy, and transesophageal echocardiography representing the second most common cause (35%). The remaining 14% were owing to foreign bodies, malignancy, or trauma, but none were related to AF ablation procedures. Most acute (<24 hours) perforations underwent primary repair with a mortality rate of 5%. The majority of patients who presented after 24 hours were treated with an esophageal T-tube, with a mortality rate of only 8%. This is much better than the 20%–40% mortality reported in the literature for late perforation treated with primary repair.^{11,15} It is not clear if the persistent high mortality following operative repair in the setting of AEF relates mainly to failure of the primary esophageal repair or to other factors. However, given the excellent results that have been achieved with T-tube repair of late esophageal perforations in other settings, we believe it should be strongly considered in the management of AEF.

Conclusion

This case demonstrates the first successful use of an esophageal T-tube in the repair of an AEF following catheter ablation of AF. With increasing numbers of these procedures being performed, despite preventative measures, further cases of AEF will occur. Early diagnosis and prompt surgical intervention are critical to improve the chances of survival, and repair of the esophageal perforation using a T-tube should be strongly considered.

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