

INNOVATIVE COLLECTIONS

COMPLEX CASE STUDY

# Successful Treatment of Esophageal Perforation Following Atrial Fibrillation Ablation with a Fully Covered Esophageal Stent: Prevention of Atrial-Esophageal Fistula

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## Introduction

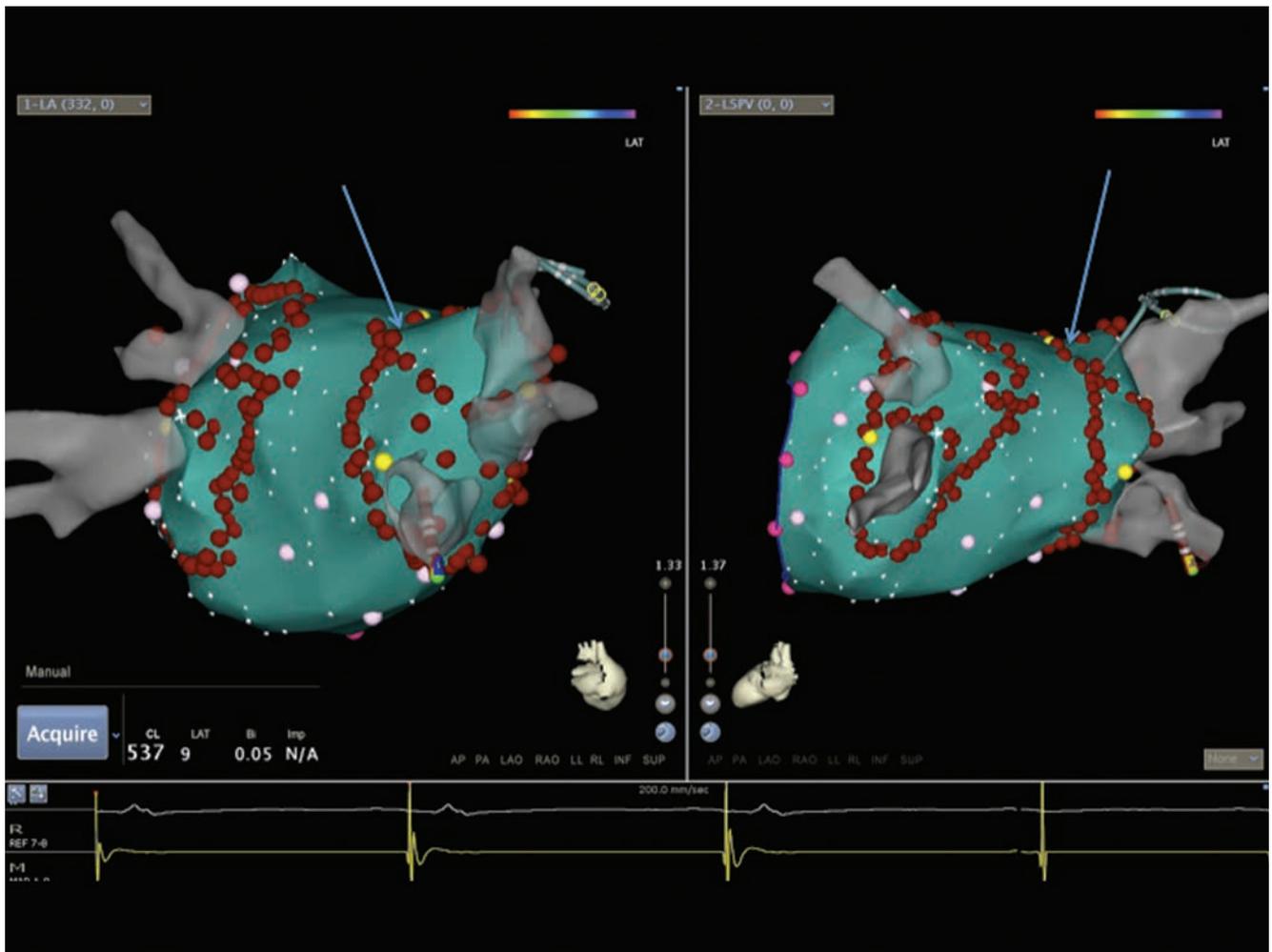
Atrioesophageal fistula (AEF) is an uncommon, under-reported, and typically fatal complication of left atrial (LA) catheter ablation for atrial fibrillation (AF). The prevalence of AEF in the literature has been reported as 0.03%, and is highly dependent upon physician reporting. AEF is associated with a mortality rate of over 80% and major cerebrovascular morbidity from septic thrombi and embolic stroke.<sup>1</sup> We present a case of esophageal perforation following persistent AF catheter ablation, with successful avoidance of AE fistula formation, as a result of early diagnosis and treatment, including endoscopic evaluation of the covered stent 2 weeks following insertion.

## Case presentation

A 56-year-old man with symptomatic, drug refractory persistent AF, underwent wide area LA circumferential

pulmonary vein isolation by radiofrequency (RF) ablation including cavotricuspid isthmus ablation. He was anticoagulated with dabigatran 150 mg bid (Pradaxa, Boehringer-Ingelheim, Germany) prior to and immediately following ablation. Dabigatran was discontinued 24 h prior to the ablation. Cardiac magnetic resonance imaging (MRI) and transesophageal echocardiogram were performed preprocedure, confirming no LA thrombus, despite persistent AF. Under general anesthesia, a 3.5-mm-tip saline irrigated bidirectional D/F curve Thermo-cool catheter (Biosense Webster, Diamond Bar, CA) with 17 mL/min saline flow was used for RF ablation. Transseptal access sheaths included an SL-0 8.5-Fr (St. Jude Medical, St. Paul, MN) and Preface 8-Fr sheath (Biosense Webster, Diamond Bar, CA) with the Thermo-cool catheter placed in either one, allowing for optimal tissue contact based on anatomy. A 15–25 mm variable Nav-Lasso catheter (Biosense Webster, Diamond Bar, CA) was used to target pulmonary vein potentials and confirm entrance and exit block. Unfractionated heparin (UFH) infusion was administered to a target ACT of 300–350 throughout the case using a nurse-managed protocol. Using CARTO-III (Biosense Webster, Diamond Bar, CA), a three-dimensional electroanatomic map was created of the left atrium and all pulmonary veins (**Figure 1**). Ablation was performed at 25–30 W on

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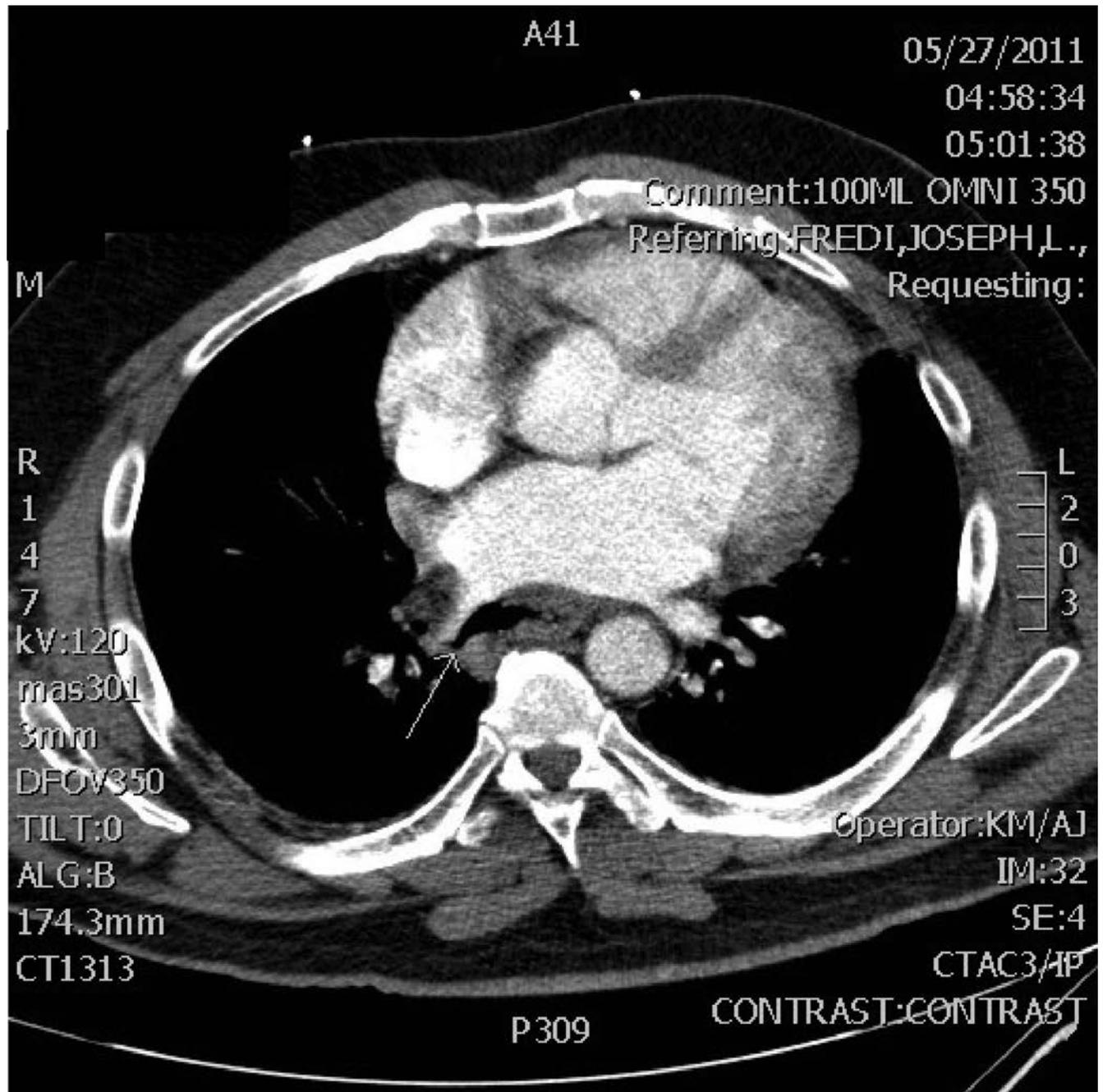


**Figure 1:** CARTO III (Biosense-Webster, Diamond Barr, CA) electroanatomic map of left atrium. Red point tags show ablation sites. The blue arrow points to location of esophageal perforation. Yellow tags show location of pulmonary vein antral isolation.

the posterior wall and 30–35 W on the anterior wall and roof. Antral pulmonary vein isolation was performed for all four veins. Intraluminal esophageal temperature was continuously monitored using a pediatric orogastric tube with temperature probe, and RF terminated for temperatures  $>38^{\circ}\text{C}$ , or temperature rise  $>1.5^{\circ}\text{C}$  over baseline. Luminal temperature remained less than  $38.5^{\circ}\text{C}$  at all times, as recorded with the thermistor probe continuously adjusted on fluoroscopy adjacent to the site of active RF delivery. Maximal recorded esophageal temperature was  $38.2^{\circ}\text{C}$  at the site of esophageal perforation. RF lesions were considered complete after 30 s with a 10-ohm impedance drop using saline-irrigated ablation with power titration. Impedance was closely monitored during RF ablation with no steam pops or abrupt impedance rise observed. The patient converted to sinus rhythm during RF ablation and was discharged the following day on amiodarone 200 mg daily, prednisone 30 mg daily for 3 days, omeprazole 20 mg twice daily for 2 weeks, and dabigatran 150 mg twice daily.

He presented to the emergency department 2 weeks post ablation with progressive inspiratory substernal

chest pain, which had become abruptly worse 12 h prior to admission, and fever up to  $101^{\circ}\text{F}$ . He had leukocytosis, with white blood cells of 17,000. Computed tomography (CT) angiography of the chest showed focal collection of air adjacent to the right aspect of the esophagus, at the antrum of the right superior pulmonary vein (RSPV)–LA junction, indicating esophageal perforation (**Figure 2**). Broad-spectrum intravenous antibiotics and dabigatran were immediately administered. Six hours after admission, he developed acute neurologic changes of aphasia, right-sided hemiparesis, and respiratory decompensation and was emergently intubated. Cerebral angiography showed hyperemia in the left middle cerebral artery territory consistent with an acute stroke. An esophago-gastroscopy was performed by thoracic surgery, demonstrating linear ulceration and dense inflammatory change in the mid-esophagus at the level of the LA, with placement of a  $155 \times 23$  cm fully covered Wallflex stent (Boston Scientific, St. Paul, MN) occluding the esophageal perforation. The risks and benefits of esophageal endoscopy in the setting of acute perforation were discussed extensively with the family.



**Figure 2:** Computed tomography angiogram of chest; the white arrow shows location of esophageal perforation and mediastinal air.

The patient was an inpatient for 2.5 weeks post esophageal stent placement. He was treated with intravenous ertapenem for mediastinitis for 4 weeks post perforation. A jejunostomy tube was placed with enteral feeding for 30 days post stent placement (Nothing by mouth for 30 days post stent placement (NPO) × 4 weeks). An acute gastrointestinal (GI) bleed occurred 2 weeks following stent placement. Upper GI endoscopy revealed mucosal inflammation and friable, oozing tissue at the distal aspect of his stent, likely attributable to wide open reflux and mechanical irritation (**Figure 3**). Anticoagulation was stopped. Hematocrit stabilized, and

he was discharged home with enteral nutrition, on no anticoagulation, in sinus rhythm. A barium swallow 2 weeks later confirmed healing of his perforation with no residual leakage. Diet was advanced and the J-tube removed. Repeat CT angiography of the chest confirmed resolution of esophageal perforation, without mediastinitis. The Wallflex stent was successfully removed 4 weeks after deployment. Dabigatran was resumed for stroke prevention after stent removal. One year post procedure he remains free of AF and is off antiarrhythmic medications. He has no residual symptoms from his septic embolic stroke.



**Figure 3:** Endoscopy shows moderate mucosal inflammation with friable tissue and minor oozing of blood at the inferior aspect of covered esophageal stent placed 2 weeks earlier.

## Discussion

The low incidence of AEF poses a challenge in determining effective preventive measures during and immediately following posterior wall LA ablation. Strategies proposed to prevent esophageal injury during AF ablation include reduced power titration while ablating the posterior LA wall (for open saline irrigation catheters), limiting RF delivery time, monitoring intraluminal esophageal temperature, using conscious sedation rather than general anesthesia for better pain perception, and monitoring intraprocedural esophageal position in relation to the posterior LA.<sup>2-5</sup> Endoscopic evaluation for esophageal injury post RF ablation reveals a 36–48% incidence of esophageal erosion in patients undergoing standard LA ablation protocols (pulmonary vein isolation by wide area antral ablation). Predictors of esophageal damage and potential for subsequent development of AEF include persistent AF ablation, power >30 W, esophageal temperature rise to >40°C, intraoperative pain, and anatomic positioning of the esophagus by CT scan (proximity to LA wall).<sup>2,4-6</sup> A critical factor in reducing patient morbidity and mortality following thermal esophageal injury from RF ablation, prior to development of esophageal perforation or full atrial fistulization, is early recognition and aggressive treatment.<sup>7</sup> We present a case of esophageal perforation

and stroke from septic cerebral embolization, 2 weeks following persistent AF ablation, with prevention of AEF formation and excellent patient outcome after esophageal stenting. Esophageal–LA fistula formation was highly likely in this case without intervention, given the overlay of CT findings of mediastinitis, esophageal perforation and LA wall edema with phlegmon directly over the RSPV–LA antral junction.

Esophageal stenting is an increasingly common treatment for malignant or inflammatory esophageal fistula and spontaneous esophageal perforation in the surgical literature.<sup>8,9</sup> Cases of esophageal stenting for esophageal perforation and AEF post AF ablation have been reported with variable outcomes.<sup>10,11</sup> Surgical correction is not feasible in the majority of cases due to inflammatory loss of mediastinal tissue planes, and intense friability of the LA wall. In our case, CT surgery declined consideration for direct repair. The anatomic location of posterior wall LA esophageal fistulae require extensive deep thoracic dissection and may not be safe in the setting of poor tissue plane visibility, and need for ongoing anticoagulation. Neurological complications immediately after stent placement for AEF have been reported and attributed to air emboli resulting from insufflation during endoscopy.<sup>12</sup> In this case, the patient developed neurologic symptoms prior to stent placement due to septic embolization from the LA wall, with no

additional neurologic symptoms during or after stent placement. Though full thickness fistula formation was prevented, the marked inflammatory response and phlegmon on the posterior LA–roof junction near the right superior pulmonary vein likely led to adherent thrombus, which was seeded from bacteremia in the setting of acute mediastinitis. He has since made a full neurologic recovery.

Early recognition and treatment are essential in the reduction of morbidity and mortality associated with esophageal perforation, an under-reported complication of LA ablation.<sup>7</sup> The most common signs and symptoms are fever, dysphagia, sudden and severe chest/epigastric pain, leukocytosis, and neurologic symptoms.<sup>7,10,11</sup> In this case, chest pain, leukocytosis, and fever were present upon initial presentation, and neurologic symptoms occurred within 18 h of symptom onset. The standard practice of performing a transesophageal echocardiogram to exclude LA appendage thrombus, and placement of a small orogastric temperature probe during the ablation procedure could possibly cause esophageal injury, but the delayed presentation by 2 weeks argues the perforation was mediated by thermal injury in this case. In addition, our protocol use of oral prednisone 30 mg daily for 3 days to reduce early post-ablation AF recurrence could have contributed to poor esophageal healing after injury, and its further use will be reconsidered.

Esophageal perforation can occur despite precautions to prevent esophageal damage during AF ablation. In this case, power was limited to 25–30 W on the posterior wall, intraluminal esophageal temperature was monitored, and esophageal position was monitored in relation to the LA by intracardiac echocardiography and a radio-opaque orogastric tube. Despite these precautions, the patient developed an esophageal perforation. Current methods to avoid esophageal injury are inadequate, and additional methods to avoid RF delivery to the esophagus such as mechanical displacement with an esophagogastroduodenoscopy (EGD) probe, active esophageal cooling with intraluminal saline, or epicardial surgical ablation may be considered. The immediate recognition and early treatment of esophageal perforation following AF ablation with a fully covered stent prevented AEF formation in this case, and prevented a fatal outcome. Continued systemic anticoagulation, enteral feeding via

a jejunostomy tube, and broad-spectrum intravenous antibiotics prevented further septic embolization, allowing for a successful recovery.

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