Atrioesophageal Fistula Following Radiofrequency Catheter Ablation of Atrial Fibrillation

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Atrioesophageal fistula (AEF) is a rare but catastrophic complication of catheter ablation of atrial fibrillation (AF), with an incidence of 0.03% to 1.5% per year. We report two cases and review the epidemiology, clinical features, pathogenesis, and management of AEF after AF ablation. The principal clinical features of AEF include fever, hematemesis, and neurologic deficits within 2 months after ablation. The close proximity of the esophagus to the posterior left atrial wall is considered responsible for esophageal injury during ablation and the eventual development of AEF. Prophylactic proton pump inhibitors, esophageal temperature monitoring, visualization of the esophagus during catheter ablation, esophageal protection devices, esophageal cooling, and avoidance of energy delivery in close proximity to the esophagus are some techniques to prevent esophageal injury. Eliminating esophageal injury during AF ablation is of utmost importance in preventing AEF. A high index of suspicion and early intervention are necessary to prevent fatal outcomes. Early surgical repair is the mainstay of treatment.

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KEY WORDS

Atrial fibrillation • Atrioesophageal fistula • Catheter ablation

Atrial fibrillation (AF) is a growing epidemic worldwide, affecting over 6 million people in the United States.[1] Catheter ablation for AF is a widely accepted rhythm control strategy for patients refractory to antiarrhythmic medications; pulmonary vein isolation is the most common strategy practiced.[2] During catheter ablation of AF, radiofrequency or cryoenergy is delivered to the posterior wall of the left atrium, which is located in close vicinity to the esophagus. Although there is anatomic variation of the esophagus, lesions placed near its course can dramatically increase the luminal temperature of the esophagus from its baseline. Thermal injury can occur, leading to inflammation and ulceration with resulting fistula formation.[3]

Atrioesophageal fistula (AEF) is a rare but life-threatening gastrointestinal complication of catheter ablation, with an incidence varying from 0.03% to 1.5% per year but with a mortality rate as high as 71%.[4-6] Although not routinely performed, esophageal evaluation with an esophagogastroduodenoscopy (EGD) after AF ablation finds injury in 7.5% to 30% of patients.[7] As many as 6.3% of all procedure-related deaths after AF ablation are due to AEF formation.[8] We report two cases of AEF formation following catheter ablation and review the clinical presentation, pathogenesis, prevention, and management of AEF.

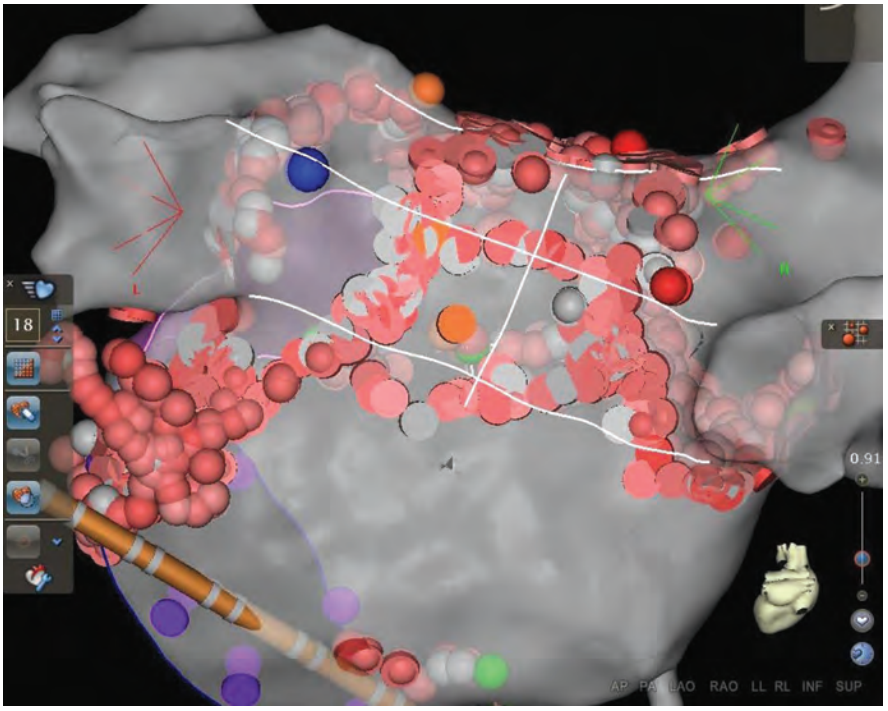

Figure 1. Mapping image of lesions delivered in Case 1.

Case 1

A 46-year-old man with a history of persistent AF and multiple cardioversions underwent catheter ablation (Table 1), which included ablation in the left atrium (left and right antrums of the pulmonary veins, left and right carina, atrial roof, posterior line, coronary sinus, mitral isthmus, and complex fractionated atrial electrograms), and in the right atrium (complex fractionated atrial electrograms) (Figure 1). Postablation

TABLE 1

Procedural Characteristics for Both Cases

Procedural Characteristics	Case 1	Case 2
Mapping software	CARTO®[a]	EnSite precision™[b]
Type of catheter	SmartTouch®[a] (3.5 mm, open irrigated tip, force sensing catheter)	CELSIUS®[a] (3.5 mm, open irrigated-tip catheter
Average power setting of catheter, W	30	30
Esophageal temperature monitoring	Yes	Yes
Temperature change to move catheter, °C	+ 0.5	+ 0.5

[a]Biosense Webster, Irvine, CA.
[b]St. Jude Medical, St. Paul, MN.

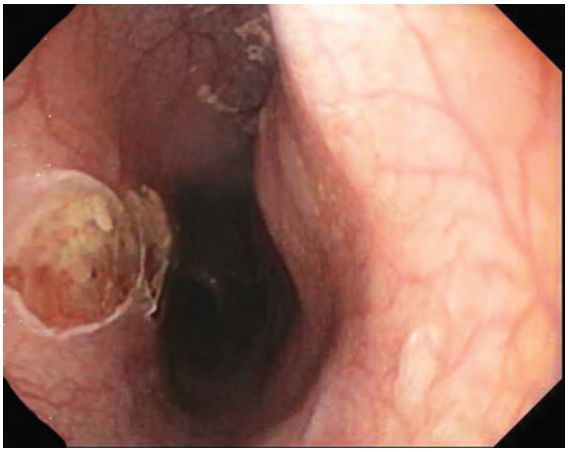

Figure 2. Postablation esophagogastroduodenoscopy showed a 1.3-cm, superficial, cratered esophageal ulcer.

EGD, as part of the clinical protocol at our institution, revealed a 1.3-cm superficial, cratered, and blackened esophageal ulcer with no stigmata of recent bleeding found 33 cm from the incisors (Figure 2). He was subsequently started on omeprazole, 20 mg twice daily, and sucralfate, 1 g four times daily; 21 days later he presented with neurologic symptoms including loss of feeling in his arms and legs bilaterally. Initial computed tomography (CT) scan of the head and brain, and CT angiography of the neck with and without contrast, showed no acute intracranial processes or dissection, but a repeat study 4 hours later, after the patient had a seizure, showed pneumocephalus, with arterial air emboli. The patient remained unresponsive with a Glasgow Coma Scale score of 9. CT chest angiography showed extraluminal air between the left atrium and esophagus as well as right upper abdominal wall and the left lobe of the liver (Figure 3). No air was noted in the cardiac chambers or the aorta. An EGD with minimal insufflation confirmed the presence of AEF, with findings of an esophageal lesion with a constant, slow trickle of bleeding into the esophagus.

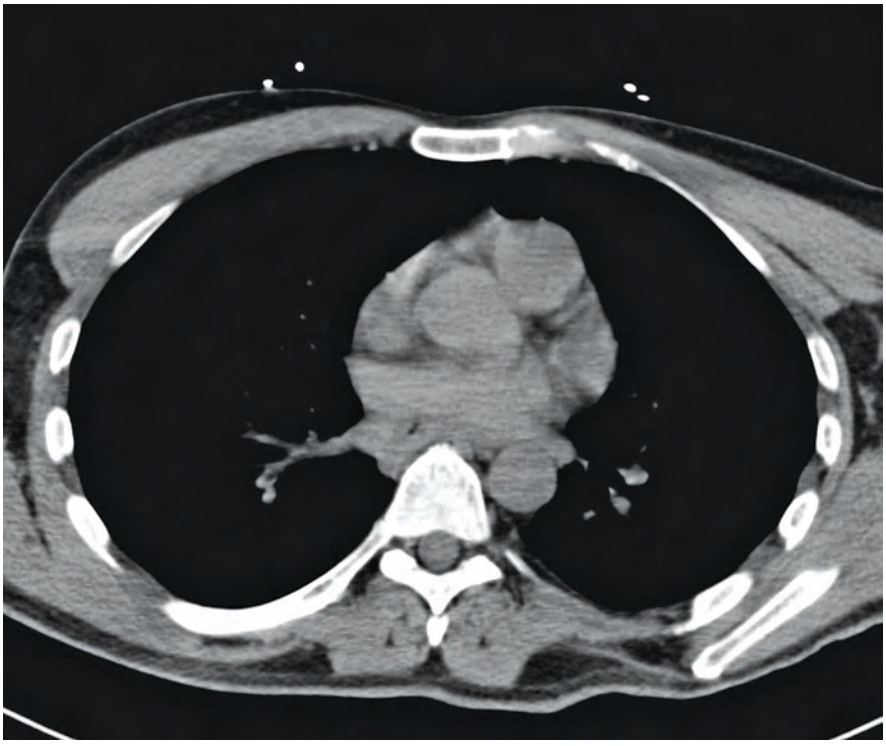

Figure 3. Computed tomography chest angiography showed extraluminal air between the left atrium and esophagus, as well as the right upper abdominal wall and the left lobe of the liver.

During surgery, a fistulous connection between the esophagus and the right inferior pulmonary vein posterior to its insertion into the left atrium was found. Surgical repair included takedown of the AEF, left atrial defect closure with sutures, esophageal defect closure with sutures, and placement of an intercostal muscle flap to separate the pericardium from the esophagus. A postoperative repeat CT showed resolution of intracranial air. The postoperative course was complicated by sepsis with mediastinitis. The patient awoke with right-sided hemiparesis and expressive aphasia. Two months after discharge, the patient remained in normal sinus rhythm with residual right-sided upper and lower extremity weakness, with slow and steady recovery.

Case 2

A 63-year-old woman with a history of paroxysmal AF underwent AF ablation (Table 1) with pulmonary vein isolation and linear and coronary sinus ablations (Figure 4). At a follow-up appointment 2 weeks postprocedure, the patient complained of pleuritic chest pain, bloating, and generalized chest discomfort. She was prescribed colchicine for concern of pericarditis and was restarted on sotalol for postprocedure palpitations consistent with AF. Because of concern about possible esophageal injury, CT angiography of the heart and chest was performed. This showed minimal atelectasis, normal pulmonary veins, and no evidence of AEF. Two weeks later (1 month postprocedure), the patient presented to an outside hospital after a syncopal episode with fever, melena, and hematemesis. She was found to be in AF with a rapid ventricular rate and was transferred for formal cardiac evaluation. On admission, the

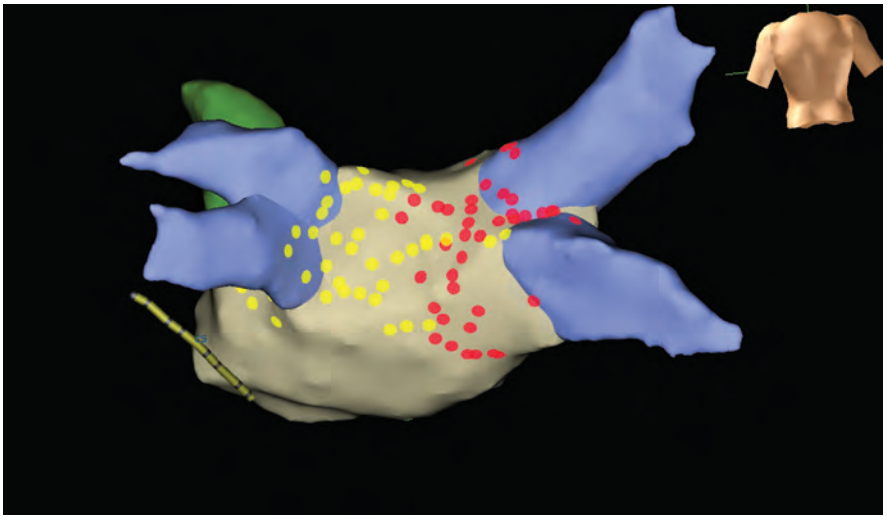

Figure 4. Mapping image of lesions delivered in Case 2.

patient developed acute ST elevation on electrocardiogram. Shortly thereafter, she became unresponsive, with left gaze deviation and posturing. A head CT showed edema of the occipital and temporal regions but no cerebral arterial occlusion. The patient vomited 400 mL of bright red blood and underwent an EGD, which showed an esophageal ulcer behind the left atrium. Pulsating cardiac tissue was seen with persistent pooling of blood in the middle third of the esophagus (Figure 5). The patient was taken emergently to surgery for repair via right posterolateral thoracotomy. Findings were adherence of the right inferior pulmonary vein to the esophagus with maceration and edema of the surrounding tissue. The atrial defect was closed with a pericardial patch. However, it was not possible to primarily repair the macerated segment of the esophagus; therefore, the chest was packed and the surgery was terminated. The patient had very labile blood pressure and severe neurologic deficits. The patient's family requested withdrawal of care, and the patient expired 25 hours after presentation.

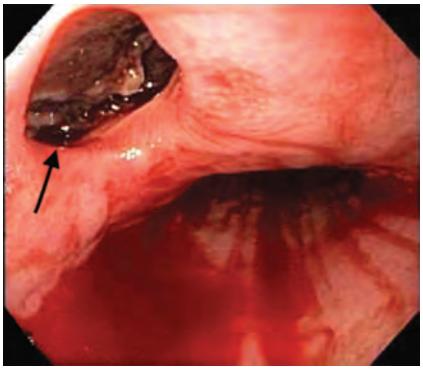

Figure 5. Esophagogastroduodenoscopy showed an esophageal ulcer behind the left atrium with pulsating cardiac tissue (*arrow*).

Discussion

Presentation

A high index of clinical suspicion is required in patients after AF catheter ablation in order to accurately diagnose and promptly treat AEF.[9] The most common symptoms on presentation include fever, hematemesis, and neurologic deficits within 2 months after the ablation.[6] The neurologic deficits stem predominantly from air or gastroesophageal contents that traverse the esophagus to the left atrium and onward to the systemic circulation. Embolization of air, gastroesophageal contents, or thrombi into the cerebral vasculature presents as transient ischemic attack, embolic stroke, air embolism, abscesses, meningitis, seizures, or sudden loss of consciousness.[10] Additional symptoms include altered mental status and seizure. Chavez and colleagues[6] reported no association between timing of symptoms and mortality.

The preferred diagnostic test is CT of the chest with intravenous contrast. Common radiologic findings include multiorgan infarcts consistent with air embolism, pneumomediastinum, pericardial effusion, intra-arterial air, inflammation of atrial and esophageal tissue, and frank communication between the atrium and pericardium or esophagus with extensive systemic septic or food emboli.[6,11] EGD may be considered in patients with equivocal work-up, but cases of cerebral air embolization following air insufflation during endoscopy in patients with AEF have been reported.[11] Caution must be exercised by using minimal insufflation with the patient placed in a Trendelenburg position.

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Pathogenesis

The exact pathogenesis of AEF formation is unknown. Radiofrequency ablation causes thermal injury to the collagen, elastin, and proteins thereby decreasing tensile strength of esophageal tissue.[12] Lemola and associates[13] performed a CT analysis, which

found the mean thickness of the posterior left atrium wall to be 2.2 ± 0.9 mm and the mean thickness of the esophageal wall adjacent to the posterior left atrium to be 3.6 ± 1.7 mm. Typically, a fat pad, with thickness of 0.9 ± 0.2 mm, is located between the atrium and esophagus, which aids in preventing conductive thermal injury.[14] However, Lemola and associates[13] found that in 96% (48/50) of patients, the fat pad was discontinuous. The gap was primarily located at the level of the mid posterior left atrium, between the superior and inferior pulmonary veins with a mean diameter of 18 ± 10 mm.[13] One hypothesis for AEF formation is that, when radiofrequency heats the posterior left atrial wall, conductive heating of the anterior esophagus occurs.[4] Thermal injury to the anterior esophagus injures intraluminal arteries, resulting in focal ischemia and necrosis that form esophageal ulcerations.[15]

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Once the AEF has formed, hematemesis is a common symptom, whereas exsanguination is relatively uncommon. Patients who present with no gastrointestinal bleeding are thought to have an AEF functioning as a one-way valve, allowing esophageal contents into the heart without blood entry into the esophagus.[10] Alternatively, because intraesophageal pressures can range from 30 to 180 mm Hg during peristalsis and swallowing, respectively, for roughly 7 seconds at a time, this may preferentially force a one-way migration of esophageal contents into the less pressurized LA via the AEF.[16]

Prevention

There are no specific techniques or ablation strategies that have been proven to lessen the risk of AEF.[5] However, there are several proposed strategies to minimize the incidence. These include the following:

1. Assessment of the esophageal position in preprocedural CT/magnetic resonance imaging.[11]

2. Tagging of the esophagus in real-time visualization of its course during the ablation procedure via three-dimensional electroanatomic mapping system, intracardiac echocardiography, or fluoroscopy after swallowing barium paste, or insertion of a luminal marker such as nasogastric tube, catheter, or temperature probe.[15]

3. Continuous monitoring of the luminal esophageal temperature (LET).

4. Reduction of ablation power applied to the left atrial posterior wall.[17]

5. Frequent movement of ablation catheter when radiofrequency energy is applied to the left atrial posterior wall near the esophagus.

6. Mechanical movement of the esophagus away from the direct effects of catheter ablation with an endoscopic stylet or probe.[18]

7. Placing a barrier between the esophagus and the posterior left atrium during catheter ablation.[19]

A prior study looked at contrast-enhanced spiral CT scans with barium swallow versus multiplanar and three-dimensional reconstructions to assess the anatomic relationship of the esophagus and left atrium.[20] Accurate correlation was found between the two imaging modalities in reference to the anatomic relationship of the esophagus and left atrium.[20] However, there can be changes of up to 15 mm between the preprocedural esophageal positioning based on imaging before or on the day of the procedure and the periprocedural positioning of the esophagus, which may limit the usefulness of this approach.[11]

Another study used periprocedural contrast-enhanced rotational angiography to obtain volumetric three-dimensional images of the left atrium and pulmonary veins.[15] Real-time imaging would then detect changes in esophageal positioning, allowing the operator to adjust ablation sites to avoid ablation within 3 to 5 mm of the esophagus.

In a retrospective study, a comparison of esophageal mucosal injury in patients with and without LET monitoring during AF catheter ablation was observed.[17] A single-thermocouple esophageal probe was used to monitor the LET, and ablation applications were stopped once the LET was ≥38.5°C. They found a significantly lower incidence of esophageal injury in the group who received LET monitoring versus the control group (6% vs 36%). Limitations to LET monitoring include positioning of the temperature probe and temperature differences between the luminal and mural temperatures.[17] Additionally, it has been proposed that the esophageal temperature probe could act as a heat sink, and lead to significant tissue heating and subsequent damage.[21]

There has also been extensive literature on the role of esophageal cooling. Sohara and colleagues[22] suggested that cooling the esophagus with hypotonic saline infusion mixed with iopamidol when LET exceeds 39°C during radiofrequency hot balloon ablation might decrease the incidence and severity of esophageal thermal injury.

The use of robotic navigation (RN)-assisted power settings versus manual catheter ablation has also been studied.[23] Subjects in the manual group limited to 30W showed no esophageal lesions, minimal lesions (erythema with intact mucosa), or shallow ulcerations. However, every patient in the RN group limited to 30W had ulceration, and one subject developed an esophageal perforation. In the second RN group, power was limited to 20W, and only one minimal lesion was found. This study suggested that decreasing the power delivered during ablation could prevent esophageal injury and thus the risk of AEF.

Other studies have looked at the role of esophageal displacement to prevent esophageal injury in patients undergoing radiofrequency or laser balloon AF ablation.[18] An endotracheal stylet placed into a thoracic chest tube was inserted into the esophagus to mechanically deviate it away from the ablation site. The esophagus was moved an average of 2.8 cm at the pulmonary vein level. LET monitoring showed no esophageal temperatures >40°C during the procedure. No esophageal injury was found due to thermal injury in 95% of patients; 12 patients (63%) showed signs of esophageal trauma related to deviating the esophagus, but no clinical sequelae were found.

One study placed an inflated intrapericardial balloon in the oblique sinus of porcine models via percutaneous epicardial access to displace the esophagus.[19] Esophageal temperature was monitored during radiofrequency ablation to the left atrial posterior wall. The interposition of the balloon between the esophagus and the left atrium allowed for an increased distance of 12.3 ± 4.0 mm, which consistently prevented LET increase.[19] Although there are associated complications with the required pericardial access, it can be considered for those patients in whom pulmonary vein isolation is difficult to achieve secondary to LET increase.[11]

Management

A high index of clinical suspicion should be exercised to appropriately diagnose and treat AEF. Early surgical intervention is the treatment of choice to prevent serious complications and fatalities.[6] Reported surgical interventions include both direct intracardiac and transthoracic extracardiac repair with and without cardiopulmonary bypass.[24] Pledgeted sutures for primary repair of the esophagus with pericardial patches and/or muscle flaps for reinforcement are often used and pericardial patches are often used for primary repair of the esophagus.[25] Reinforcement creates a physical barrier, which may help to prevent refistulization.[25] Some success has also been shown with cervical esophagus diversion and gastric drainage, as reported by St Julien and associates.[24] The authors performed an incision in the left neck, anterior to the sternocleidomastoid, and tied two sutures around the esophagus, which were tagged by metal clips. The esophagus was decompressed with an 18-French Malecot drain via a stab incision. A Stamm gastrostomy and feeding jejunostomy were then performed to reduce the contamination from the fistula. This resulted in complete healing of the fistula with no evidence of a remnant tract or fistula during the follow-up.[25] Other modalities including hyperbaric oxygen have been shown to increase the neurologic recovery of patients.[26]

Although esophageal stenting for AEF has been proposed as a treatment option, it is associated with poor outcomes and, therefore, is no longer recommended.[27] Stenting, however, may have a temporary role as a bridge to definitive surgical treatment.[28]

We recommend a multidisciplinary approach with close communication between the gastroenterologist, cardiologist, and surgeon that is critical for successful and appropriate management of AEF. We suggest that a neuroradiologist review imaging studies in suspected cases, as it can be easily overlooked, as demonstrated by our case. We also recommend adjunctive treatment with antibiotics and nutritional support in the postoperative intensive care setting.

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Conclusions

AEF is a rare complication of catheter ablation for AF but is often fatal. Performing EGD immediately after ablation to identify patients with high-grade esophageal lesions is extremely helpful to ensure close following of such patients. Unfortunately, at this time, there are no proven measures to prevent progression to fistula once a high-grade esophageal lesion is found. However, early suspicion with symptoms of fever, leukocytosis, and neurologic symptoms manifesting within 1 to 3 weeks of left atrial posterior wall ablation must be acted upon aggressively. Diagnosing this entity early and receiving definitive treatment with surgical intervention is key for reducing mortality. There are no specific techniques or ablation strategies that have been proven to lessen the risk of AEF. However, reduction of ablation power, less force of catheter contact, and less time of ablating along the posterior wall are plausible strategies to employ. Prophylactic proton pump inhibitors, esophageal temperature monitoring, visualization of the esophagus during catheter ablation, esophageal protection devices, esophageal cooling, and avoidance of energy delivery in close proximity to the esophagus are techniques that have been proposed to prevent esophageal injury. ■

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MAIN POINTS

- Atrial fibrillation (AF) is a growing epidemic worldwide, affecting over 6 million people in the United States. Catheter ablation for AF is a widely accepted rhythm control strategy for patients refractory to antiarrhythmic medications.
- Atrioesophageal fistula (AEF) is a rare but life-threatening gastrointestinal complication of catheter ablation, with an incidence varying from 0.03% to 1.5% per year but with a mortality rate as high as 71%. As many as 6.3% of all procedure-related deaths after AF ablation are due to AEF formation.
- Prophylactic proton pump inhibitors, esophageal temperature monitoring, visualization of the esophagus during catheter ablation, esophageal protection devices, esophageal cooling, and avoidance of energy delivery in close proximity to the esophagus are techniques that have been proposed to prevent esophageal injury.
- Currently, there are no proven measures to prevent progression to fistula once a high-grade esophageal lesion is found. A high index of clinical suspicion should be exercised to appropriately diagnose and treat AEF. Early surgical intervention is the treatment of choice to prevent serious complications and fatalities.

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