Esophageal Injury Following Radiofrequency Ablation for Atrial Fibrillation: Injury Classification

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Atrial fibrillation is a major cause of stroke and the most common arrhythmia that is clinically significant, with prevalence rates of 3.8% in individuals 60 years of age or older and 9.0% in individuals over 80 years of age.[1] In 2001, the prevalence of atrial fibrillation was projected to increase 2.5-fold by 2050 due to the rapidly growing elderly population.[1] As a result, there has been an increase in radiofrequency ablation (RFA) techniques to treat paroxysmal and persistent atrial fibrillation. In the maze procedure, which was developed in 1991 by Cox, incisions are made within the left atrium (LA) to interrupt the reentrant circuits responsible for atrial fibrillation.[2,3] However, due to the procedure's long operative time and morbidity rate, most clinicians have adopted a variation of the procedure which uses intraoperative and percutaneous RFA to create transmural lines of electrically inactive scar tissue within the LA endocardially and within the right atrium epicardially.[4] The approach to RFA changed dramatically in 1998 with the discovery by Haïssaguerre and associates that the majority of ectopic atrial beats originated somewhere within 1 or more of the 4 pulmonary veins (PVs) due to the extension of muscular bands from the LA into the PVs.[5] Following this discovery, mapping and ablation of arrhythmogenic foci of both the PVs and the LA have been performed, with today's procedures showing success rates of 60–90%.[6-10]

Although RFA has been effective at treating atrial fibrillation, complications have been reported in the literature, the most serious of which is a left atrial-esophageal fistula that forms secondary to thermal esophageal injury.[11-15] Despite the possibility of RFA inducing esophageal injury, few endoscopic studies have examined the esophagus following RFA, and no studies have attempted to classify lesions in order to stratify patients for postoperative follow-up.[16-19] This case series consists of 3 patients who underwent percutaneous RFA and experienced acute onset of esophageal symptoms. As these cases illustrate the spectrum of injury that can develop from thermal injury to the esophagus, these findings were used to propose a classification system for determining patient follow-up.

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Case Reports

Patient #1

A 53-year-old white woman presented with acute retrosternal chest pain, dysphagia, and odynophagia 1 day after undergoing RFA of the PVs. The patient was afebrile with stable vital signs, and her laboratory studies had no significant abnormalities. An electrocardiogram showed normal sinus rhythm with no ST-T wave abnormalities. The patient subsequently underwent an esophagogastroduodenoscopy (EGD), which revealed a small ulcer with adherent exudate at 25 cm, a finding that was consistent with thermal injury (Figure 1). The patient was instructed to take sucralfate 1 g three times daily and to adhere to a clear liquid diet until her symptoms resolved. One week later, she had complete resolution of her symptoms and had resumed a regular diet without any further complications.

Patient #2

A 69-year-old white man presented with complaints of dysphagia 1 day after undergoing RFA of the PVs and tricuspid isthmus. He was afebrile with stable vital signs, and his laboratory studies had unremarkable findings. The patient was referred to our service for further evaluation of possible thermal injury. An EGD showed a 6-mm linear ulcer on the anterior wall of the esophagus at 35 cm, as well as an adjacent 2-mm ulcer; both lesions

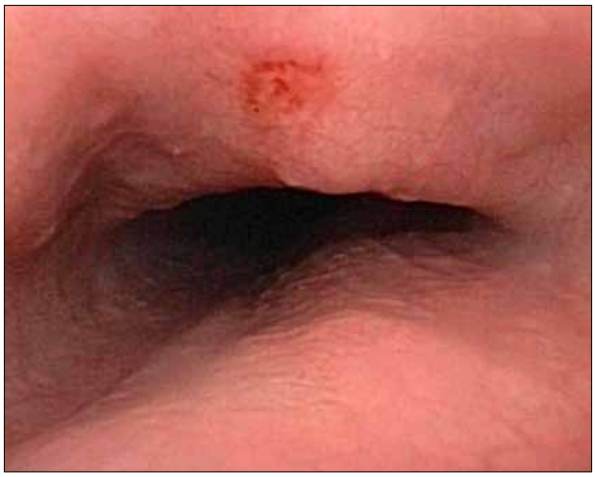

Figure 1. Mucosal erythema with adherent exudate.

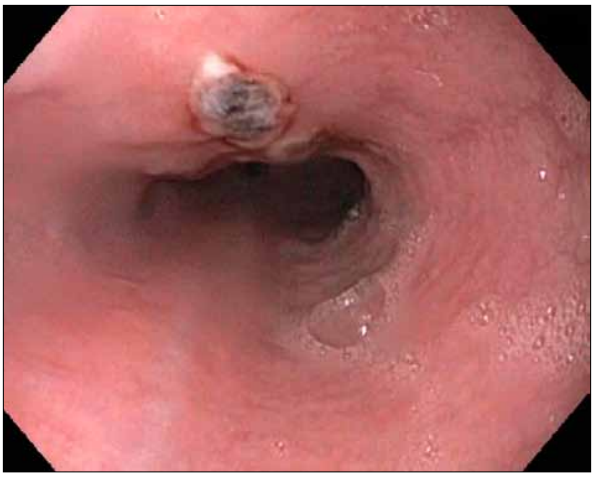

Figure 2. Shallow ulceration covered with black coagulum.

were covered with a black coagulum (Figure 2). The patient was instructed to remain nil per os with total parenteral nutrition and was started on pantoprazole 40 mg twice daily and sucralfate 1 g four times daily. A repeat EGD obtained 1 week later revealed 2 healing ulcers on the anterior esophageal wall at 36 cm with minimal erythema. The patient was advanced to a soft mechanical diet and continued on pantoprazole and sucralfate therapy, with another EGD scheduled for the following week. The third EGD demonstrated minimal erythema on the anterior esophageal wall at 35 cm. The patient was advanced to a regular diet, and no further endoscopies were required. The patient remained afebrile, with resolution of his symptoms following the final EGD.

Patient #3

A 63-year-old white woman was referred to our service with complaints of nausea and worsening, severe (10/10), epigastric abdominal pain that radiated to her back 5 days following RFA of the PVs. These symptoms had predated the procedure by 2 months and were thought to be unrelated to it. Her laboratory studies were remarkable only for anemia (hemoglobin level, 10.0 g/dL). An electrocardiogram revealed normal sinus rhythm with nonspecific T-wave changes. Due to the worsening nature of her epigastric pain, presence of anemia, and history of RFA, an EGD was performed. This procedure revealed a large adherent clot on the anterior esophageal wall at 30 cm, which was overlying a visible vessel (Figure 3). No active bleeding was noted at that time. After cardiothoracic surgery consultation, the patient underwent a computed tomography (CT) scan of the chest. The scan did not show any signs of esophageal thickening or pneumomediastinum. A barium swallow study did not show any signs of perforations or fistulous tracts. A repeat EGD obtained 3 days after the initial study revealed a large healing ulcer at 30 cm with a new bluish circumferential discoloration of the esophageal mucosa at 17–24 cm. As there were no signs of perforation on any of these studies and the ulcer appeared to be healing, the patient was discharged home with instructions to take pantoprazole 40 mg twice daily and sucralfate liquid 1 g four times daily. A repeat EGD was scheduled for 1 month later to confirm healing of the esophageal ulcer.

The patient presented on Postoperative Day 17 with acute-onset chest pain and fever. A CT scan of the chest revealed pneumomediastinum and pneumopericardium with a moderate pericardial effusion, suggestive of an esophagopericardial fistula and esophageal perforation. During an emergent thoracotomy, a 1-cm esophageal perforation was discovered and repaired just posterior to the LA; in addition, a pericardial window was created for drainage of a pericardial empyema. Extensive irrigation of the pleural and pericardial cavities was performed, and 2 chest tubes were placed bilaterally. Gastrografin studies continued to show persistent leaks with high chest tube outputs on 2 separate occasions; therefore, after 1 month of conservative therapy, the patient underwent an esophagectomy with gastric pull-up. During the procedure, a large esophageal perforation (0.9 cm in diameter) was noted.

Discussion

Esophageal injury is a known complication of RFA of the PVs and LA for treatment of atrial fibrillation. The etiology of the injury is multifactorial. As stated by Gillinov and associates, the goal of RFA is to create a series of transmural lesions in the LA; however, only the temperature and duration of the energy delivered can be controlled.[13] There is no way to control the depth of the lesion created; thus, it is possible to create a nontransmural lesion, resulting in an unsuccessful operation, or a lesion that is too deep, consequently causing injury to adjacent structures. Additionally, it has been shown that intestinal tissue is far more

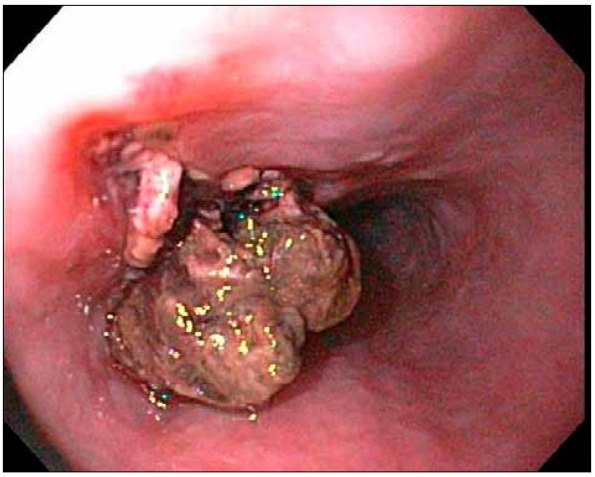

Figure 3. Deep ulceration with an adherent clot.

susceptible to radiofrequency-induced thermal injury than muscle tissue and that the convection of heat generated within the LA by the ablation probe can result in esophageal injury without the probe coming into direct contact with the esophagus.[12]

In order to establish a better understanding of the anatomic relationship between the esophagus and the posterior LA wall, Sánchez-Quintana and colleagues conducted a study to examine the course of the esophagus by gross dissection.[20] In 40% of the cadaveric specimens, there was less than 5 mm between the esophagus and the LA endocardium.[20] The thinnest layer of the fat pad has been consistently seen at the level of the inferior PVs, explaining the close proximity of the esophagus and the inferior PVs.[21] Furthermore, anterior to the esophageal adventitia are the esophageal arteries and vagus nerve plexus, which can be easily injured with transmural ablation of the LA or PVs. Damage to these structures could cause neurovascular compromise of the esophagus, leading to necrosis and perforation.

Esophageal injury secondary to RFA was first described in 2001; the patient presented 9 days after RFA with an elevated white blood cell count, odynophagia, and a transmural esophageal perforation of the anterior wall that was fatal.[13] Doll and coworkers found that 4 of 387 patients (1%) who underwent intraoperative RFA developed left atrial-esophageal fistulae that were diagnosed secondary to neurologic defects from air emboli, massive hematemesis, or septic shock.[12] Although all of these patients had unremarkable early postoperative courses, they began to develop signs of perforation 6–12 days following RFA.

Following these reports of esophageal injury with intraoperative RFA, the first reports of atrial-esophageal fistula formation following percutaneous RFA came from Scanavacca and associates and Pappone and colleagues in 2004.[10,15] In these reports, 3 patients presented with nonspecific signs and symptoms, including persistent fever, dysphagia, odynophagia, sepsis, endocarditis, and neurologic ischemia.[10,15] These patients presented to their physicians 10–21 days following RFA, but esophageal injury was not initially considered in the differential diagnosis. Cummings and coworkers reported on 9 fatal cases of atrial-esophageal fistula formation in a retrospective case series based on anonymous identification in 2006.[11] These 9 patients presented 10–16 days postoperatively with a constellation of symptoms similar to those previously reported. However, only 4 patients received the correct diagnosis before death; the remaining patients were diagnosed at autopsy. Due to the considerable delay in diagnosis, Dagres and colleagues attempted to identify criteria for rapid detection of esophageal perforation.[22] The researchers found that, although symptoms were generally nonspecific, patients presented with 2 distinct symptoms (fever and severe chest or epigastric pain) that occurred 1–4 weeks following the procedure.

Esophageal perforation following RFA is rare but fatal. Therefore, it is imperative for physicians to consider this complication in the differential diagnosis, as early intervention is essential for avoiding mortality. The 3 patients presented in this case series demonstrate the spectrum of esophageal injury that can occur following RFA. Atrial-esophageal fistula formation appears to usually take at least 1 week, which allows an EGD to be performed within 24–72 hours of RFA with a low likelihood of inducing air embolization. Although endoscopic analyses of the esophagus have been performed for thermal injury, there has been no attempt to classify these injuries with the intention of stratifying patients for further follow-up.[19,23] The following injury classification system is proposed based on endoscopic findings to facilitate rapid detection and management of these patients.

Class I Thermal Injury

Patients with this injury have erythema or discoloration of the anterior esophageal wall with minimal mucosal disruption. These lesions should be shallow erosions without vessel involvement and should be less than 5 mm, as illustrated by Patient #1. These patients appear to have a low risk of frank perforation, and they can be managed with proton pump inhibitors and sucralfate, with clinical follow-up to assess for symptom worsening.

Class II Thermal Injury

As demonstrated by Patient #2, patients with this degree of injury have ulcers of the anterior esophageal wall with or without exudate. These lesions tend to penetrate further into the esophageal wall, without extension into the muscularis externa. In addition, these lesions should not have an overlying clot or vessel involvement. Patients with this injury appear to have an intermediate risk of developing a perforation. In addition to medical management with proton pump inhibitors and sucralfate, these patients require close follow-up and repeat endoscopy to ensure lesion improvement.

Class III Thermal Injury

Patients with this degree of injury have deep ulceration that extends into and beyond the muscularis. These lesions can have evidence of eschar formation, overlying clot, or necrosis. The lesions may also have vascular involvement, as shown by Patient #3. As a result, there is an increased probability of compromising tissue blood flow and worsening injury. Patients with this injury have a high risk of developing an esophageal perforation and atrial-esophageal fistula. They should be considered nil per os and started on medical therapy. Cardiothoracic surgery consultation is warranted as well as a CT scan of the chest. If imaging findings do not require an emergent operation, a repeat endoscopy can be considered to reassess healing of the ulcer.

Conclusion

Larger prospective studies are necessary to evaluate the usefulness of this classification system as well as the proposed follow-up guidelines. In addition, more in-depth evaluation of these lesions should be considered via endoscopic ultrasound to further quantify the extent of injury to the esophagus as well as the surrounding tissue.[24]

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Review

Esophageal Injury Following Left Atrial Ablation

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Atrial fibrillation (AF) is the most common arrhythmia encountered in clinical medicine. Current estimates suggest that 4–5 million individuals in the United States

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