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]

References

1. Go AS, Hylek EM, Phillips KA, et al. Prevalence of diagnosed atrial fibrillation in adults: national implications for rhythm management and stroke prevention: the Anticoagulation and Risk Factors in Atrial Fibrillation (ATRIA) Study. *JAMA*. 2001;285:2370-2375.
2. Cox JL. The surgical treatment of atrial fibrillation. IV. Surgical technique. *J Thorac Cardiovasc Surg*. 1991;101:584-592.
3. Cox JL, Schuessler RB, Lappas DG, Boineau JP. An 8 1/2-year clinical experience with surgery for atrial fibrillation. *Ann Surg*. 1996;224:267-273; discussion 273-275.
4. Raman JS, Seevanayagam S, Storer M, Power JM. Combined endocardial and epicardial radiofrequency ablation of right and left atria in the treatment of atrial fibrillation. *Ann Thorac Surg*. 2001;72:S1096-S1099.
5. Haïssaguerre M, Jaïs P, Shah DC, et al. Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. *N Engl J Med*. 1998;339:659-666.
6. Marrouche NF, Dresing T, Cole C, et al. Circular mapping and ablation of the pulmonary vein for treatment of atrial fibrillation: impact of different catheter technologies. *J Am Coll Cardiol*. 2002;40:464-474.
7. Pappone C, Oreto G, Rosanio S, et al. Atrial electroanatomic remodeling after circumferential radiofrequency pulmonary vein ablation: efficacy of an anatomic approach in a large cohort of patients with atrial fibrillation. *Circulation*. 2001;104:2539-2544.
8. Lin WS, Tai CT, Hsieh MH, et al. Catheter ablation of paroxysmal atrial fibrillation initiated by non-pulmonary vein ectopy. *Circulation*. 2003;107:3176-3183.
9. Oral H, Scharf C, Chugh A, et al. Catheter ablation for paroxysmal atrial fibrillation: segmental pulmonary vein ostial ablation versus left atrial ablation. *Circulation*. 2003;108:2355-2360.
10. Pappone C, Manguso F, Vicedomini G, et al. Prevention of iatrogenic atrial tachycardia after ablation of atrial fibrillation: a prospective randomized study comparing circumferential pulmonary vein ablation with a modified approach. *Circulation*. 2004;110:3036-3042.
11. Cummings JE, Schweikert RA, Saliba WI, et al. Brief communication: atrial-esophageal fistulas after radiofrequency ablation. *Ann Intern Med*. 2006;144:572-574.
12. Doll N, Borger MA, Fabricius A, et al. Esophageal perforation during left atrial radiofrequency ablation: is the risk too high? *J Thorac Cardiovasc Surg*. 2003;125:836-842.
13. Gillinov AM, Pettersson G, Rice TW. Esophageal injury during radiofrequency ablation for atrial fibrillation. *J Thorac Cardiovasc Surg*. 2001;122:1239-1240.
14. Pappone C, Oral H, Santinelli V, et al. Atrio-esophageal fistula as a complication of percutaneous transcatheter ablation of atrial fibrillation. *Circulation*. 2004;109:2724-2726.
15. Scanavacca MI, D'Avila A, Parga J, Sosa E. Left atrial-esophageal fistula following radiofrequency catheter ablation of atrial fibrillation. *J Cardiovasc Electrophysiol*. 2004;15:960-962.
16. Cummings JE, Schweikert RA, Saliba WI, et al. Assessment of temperature, proximity, and course of the esophagus during radiofrequency ablation within the left atrium. *Circulation*. 2005;112:459-464.
17. Marrouche NF, Guenther J, Segerson NM, et al. Randomized comparison between open irrigation technology and intracardiac-echo-guided energy delivery for pulmonary vein antrum isolation: procedural parameters, outcomes, and the effect on esophageal injury. *J Cardiovasc Electrophysiol*. 2007;18:583-588.
18. Martinek M, Bencsik G, Aichinger J, et al. Esophageal damage during radiofrequency ablation of atrial fibrillation: impact of energy settings, lesion sets, and esophageal visualization. *J Cardiovasc Electrophysiol*. 2009;20:726-733.
19. Schmidt M, Nölker G, Marschang H, et al. Incidence of oesophageal wall injury post-pulmonary vein antrum isolation for treatment of patients with atrial fibrillation. *Europace*. 2008;10:205-209.
20. Sánchez-Quintana D, Cabrera JA, Climent V, Farré J, Mendonça MC, Ho SY. Anatomic relations between the esophagus and left atrium and relevance for ablation of atrial fibrillation. *Circulation*. 2005;112:1400-1405.
21. Tsao HM, Wu MH, Higa S, et al. Anatomic relationship of the esophagus and left atrium: implication for catheter ablation of atrial fibrillation. *Chest*. 2005;128:2581-2587.
22. Dagres N, Kottkamp H, Piorkowski C, et al. Rapid detection and successful treatment of esophageal perforation after radiofrequency ablation of atrial fibrillation: lessons from five cases. *J Cardiovasc Electrophysiol*. 2006;17:1213-1215.
23. Halm U, Gaspar T, Zachäus M, et al. Thermal esophageal lesions after radiofrequency catheter ablation of left atrial arrhythmias. *Am J Gastroenterol*. 2010;105:551-556.
24. Zellerhoff S, Ullerich H, Lenze F, et al. Damage to the esophagus after atrial fibrillation ablation: just the tip of the iceberg? High prevalence of mediastinal changes diagnosed by endosonography. *Circ Arrhythm Electrophysiol*. 2010;3:155-159.

Review

Esophageal Injury Following Left Atrial Ablation

Bashar J. Qumseya, MD[1]
Fred Kusumoto, MD[2,3]
Herbert Wolfsen, MD[1]

[1]Division of Gastroenterology and Hepatology, [2]Division of Cardiovascular Diseases, [3]Electrophysiology and Pacing Laboratory, Mayo Clinic, Jacksonville, Florida

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

Address correspondence to:
Dr. Herbert Wolfsen, Mayo Clinic, Division of Gastroenterology and Hepatology, 4500 San Pablo Road, Jacksonville, FL 32224; Tel: 904-953-6319; Fax: 904-953-7366; E-mail: wolfsen.herbert@mayo.edu

have AF. It has been projected that, because of changing demographics, this number will increase to 10 million in 20 years.[1] A rate-control strategy is used to treat many patients with AF, although some patients require antiarrhythmic medications to maintain sinus rhythm. Unfortunately, antiarrhythmic medications are effective in only 40–70% of patients.

For patients who fail an antiarrhythmic drug trial due to inefficacy or drug intolerance, percutaneous catheter ablation has emerged as an important treatment option.[2] Generally, ablation for AF is performed by placing a series of lesions around the pulmonary vein ostia in the posterior left atrium (LA). Initial single-center reports found success rates of 90% with AF ablation, but recent randomized multicenter studies using rigorous definitions for AF recurrence have found success rates ranging from 60% to 80%, depending on patient selection.[3-5] Catheters used for AF ablation vary, and lesions can be produced with either cryoenergy or radiofrequency energy. However, most AF ablations are performed using radiofrequency energy in a monopolar configuration with current travelling from a small tip electrode within the heart (producing thermal injury to the cardiac tissue) to a larger return electrode placed on the skin. To produce a deeper lesion, the most commonly used catheter has a 4-mm tip electrode that is continuously irrigated with saline to produce higher tissue temperatures below the surface. Many techniques have been developed to control the size of the lesion created (eg, by varying the duration of energy application, changing the flow of irrigation, or modifying the amount of power used), but it is extremely difficult to predict the actual depth of lesion formation in real time while performing AF ablation.

Despite the relative safety of the procedure, 2–6% of patients may experience major complications, including stroke, cardiac tamponade, pulmonary vein stenosis, and extracardiac injury.[6,7] The esophagus is separated from the posterior LA by a thin (0.9 ± 0.2 mm) layer of fat. Therefore, the esophagus is particularly prone to thermal injury during AF ablation.[8] Reported esophageal injuries include perforation, atrio-esophageal fistula formation, and peri-esophageal nerve injury.[9] In particular, development of an atrio-esophageal fistula can be difficult to detect and is almost uniformly fatal if not treated promptly. The incidence of this complication is not well established but appears to be approximately 0.05%; the associated mortality rate reported in the literature is very high (93%), with 13 deaths occurring in 14 reported cases.[10] Symptoms of esophageal injury are subtle but often include chest pain, hematemesis, and dysphagia. Development of atrio-esophageal fistula is often associated with neurologic abnormalities. These symptoms have been reported within 3 days of the initial procedure and up to 5 weeks afterward. Pappone and associates reported that 50% of patients with an atrio-esophageal fistula were not correctly diagnosed until autopsy.[10]

Esophageal changes have been reported after AF ablation in up to 47% of patients who underwent the procedure.[11] Schmidt and colleagues performed endoscopic evaluations of 28 patients within 24 hours of AF ablation.[11] They found erythema on the posterior wall of the esophagus in 29% of the patients and ulcer-like changes in 18% of the patients. Interestingly, the researchers reported that all of these patients complained of a reflux-like sensation.

Keshishian and associates reported on 3 patients with esophageal injury following AF ablation.[12] The first patient presented with dysphagia and odynophagia the day after the procedure, and an endoscopy revealed a small ulcer. The authors suggested that this type of injury could be classified as a class I thermal injury. However, the presence of transesophageal injury resulting in mucosal ulceration is clearly evidence of more advanced injury, compared to the patients in the study by Schmidt and associates, who had only mucosal erythema and no major symptoms.[11] Perhaps patients with erythema, no ulcers, and no major symptoms are better classified as having a class I thermal injury.

This class of patients appears to do well, with no subsequent major complications. Although treatment with gastric acid blockers such as proton pump inhibitors (PPIs) is frequently recommended, it should be remembered that the injury is coming from deeper tissues. Thus, the use of PPIs in those patients may not be helpful. However, short-term therapy with PPIs is unlikely to be harmful. Additionally, such therapy may be reasonable, given the relative inexpense of generic PPIs. The critically important concern is that the presence of erythema within the lumen of the esophagus provides little appreciation for the deeper tissue injury. Since the catheter is in the LA, the mucosa of the esophagus is farthest from the heat source. Thus, significant injury has occurred to the outer layer of the esophagus if surface mucosal erythema or ulceration is visible on endoscopy. Serial computed tomography (CT) scans following ablation may be necessary to monitor the condition of these deeper tissues.

The second patient in the case series by Keshishian and coworkers presented with dysphagia 1 day after ablation.[12] The patient had esophageal ulceration in 2 locations with what appeared to be a black spot. The authors suggested that such an injury should be classified as a class II thermal injury. This grade suggests that class II patients may have incurred more damage to the esophagus than class I patients. The authors suggested that such patients be treated with PPIs and sucralfate and undergo repeat endoscopy and close monitoring. In this situation, the use of PPIs is warranted, given the more severe injury to the esophagus. In our opinion, these patients should also be educated and encouraged to have a high level of suspicion for signs and symptoms of worsening esophageal injury; these patients must proceed to the emergency room if they have any signs of chest pain, continued dysphagia, fevers, or intestinal bleeding.

Keshishian and associates recommended keeping patients nil per os if their postablation endoscopy reveals eschar, large clots, or visible vessels.[12] The authors also recommended that these patients start PPI therapy, undergo a CT chest scan, and consult a cardiothoracic surgeon. These steps were used in the management of the third patient in the case series by Keshishian and colleagues, but the patient developed an atrio-esophageal fistula by Day 17 postprocedure.[12] Therefore, the question is: What else could have been done in this patient? Clearly, there have not been any randomized trials looking at such patients and optimal therapies. Keshishian and associates have suggested imaging the deeper tissues between the LA and the esophagus, as well as using endoscopic ultrasonography (EUS) to evaluate the depth of the injury.[12] Based on our experience, it would be rather difficult to use EUS to assess the degree of acute injury following ablation. One possibility may be to insert covered, removable esophageal stents to seal the area prophylactically and prevent fistula formation in patients who are found to have stigmata for severe esophageal injury. However, further patient studies are needed before such interventions can be considered.

As always, an ounce of prevention is worth a pound of cure. The goal should be to prevent esophageal injury during AF ablation. Studies have looked at methods of minimizing damage to the esophagus during AF ablation, such as reducing power in the posterior LA wall (closest to the esophagus), monitoring temperature in the esophagus, and irrigating the esophagus with cool water.[9,13,14] Alternatively, EUS could be used before the procedure to assess the actual size of the area between the LA and the esophagus. Such information is easily obtained via EUS and could identify patients at highest risk for injury post–AF ablation. In such patients, esophageal irrigation and reduction of the ablative power near the posterior LA wall are indicated.

References

1. Miyasaka Y, Barnes ME, Gersh BJ, et al. Secular trends in incidence of atrial fibrillation in Olmsted County, Minnesota, 1980 to 2000, and implications on the projections for future prevalence. *Circulation*. 2006;114:119-125.
2. European Heart Rhythm Association (EHRA); European Cardiac Arrhythmia Society (ECAS); American College of Cardiology (ACC); American Heart Association (AHA); Society of Thoracic Surgeons (STS); Calkins H, Brugada J, Packer DL, et al. HRS/EHRA/ECAS expert consensus statement on catheter and surgical ablation of atrial fibrillation: recommendations for personnel, policy, procedures and follow-up. A report of the Heart Rhythm Society (HRS) Task Force on catheter and surgical ablation of atrial fibrillation. *Heart Rhythm*. 2007;4:816-861.
3. Haïssaguerre M, Jaïs P, Shah DC, et al. Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. *N Engl J Med*. 1998;339:659-666.
4. Wilber DJ, Pappone C, Neuzil P, et al; ThermoCool AF Trial Investigators. Comparison of antiarrhythmic drug therapy and radiofrequency catheter ablation in patients with paroxysmal atrial fibrillation: a randomized controlled trial. *JAMA*. 2010;303:333-340.
5. Pappone C, Rosanio S, Oreto G, et al. Circumferential radiofrequency ablation of pulmonary vein ostia: a new anatomic approach for curing atrial fibrillation. *Circulation*. 2000;102:2619-2628.
6. Cappato R, Calkins H, Chen SA, et al. Worldwide survey on the methods, efficacy, and safety of catheter ablation for human atrial fibrillation. *Circulation*. 2005;111:1100-1105.
7. Bertaglia E, Zoppo F, Tondo C, et al. Early complications of pulmonary vein catheter ablation for atrial fibrillation: a multicenter prospective registry on procedural safety. *Heart Rhythm*. 2007;4:1265-1271.
8. Lemola K, Sneider M, Desjardins B, et al. Computed tomographic analysis of the anatomy of the left atrium and the esophagus: implications for left atrial catheter ablation. *Circulation*. 2004;110:3655-3660.
9. Takahashi A, Kuwahara T, Takahashi Y. Complications in the catheter ablation of atrial fibrillation: incidence and management. *Circ J*. 2009;73:221-226.
10. Pappone C, Oral H, Santinelli V, et al. Atrio-esophageal fistula as a complication of percutaneous transcatheter ablation of atrial fibrillation. *Circulation*. 2004;109:2724-2726.
11. Schmidt M, Nölker G, Marschang H, et al. Incidence of oesophageal wall injury post-pulmonary vein antrum isolation for treatment of patients with atrial fibrillation. *Europace*. 2008;10:205-209.
12. Keshishian J, Young J, Hill E, Saloum Y, Brady PG. Esophageal injury following radiofrequency ablation for atrial fibrillation: injury classification. *Gastroenterol Hepatol (N Y)*. 2012;8:411-414.
13. Okaji Y, Tsuno NH, Tanaka M, et al. Pilot study of anti-angiogenic vaccine using fixed whole endothelium in patients with progressive malignancy after failure of conventional therapy. *Eur J Cancer*. 2008;44:383-390.
14. Tsuchiya T, Ashikaga K, Nakagawa S, Hayashida K, Kugimiya H. Atrial fibrillation ablation with esophageal cooling with a cooled water-irrigated intraesophageal balloon: a pilot study. *J Cardiovasc Electrophysiol*. 2007;18:145-150.