Esophageal Hematoma After Atrial Fibrillation Ablation 
Incidence, Clinical Features, and Sequelae of Esophageal Injury of a Different Sort

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Background—Esophageal hematoma recently has been reported as a form of esophageal injury after atrial fibrillation (AF) ablation, attributed to the use of transesophageal echocardiography (TEE). We sought to determine the incidence, clinical features, and sequelae of this form of esophageal injury.

Methods and Results—This was a prospective series of 1110 AF ablation procedures performed under general anesthesia (GA) over 9 years. TEE was inserted after induction of GA to exclude left atrial appendage thrombus, define cardiac function, and guide transseptal puncture. The procedural incidence of esophageal hematoma was 0.27% (3/1110 procedures, mortality 0%). Odynophagia, regurgitation, and hoarseness were the predominant symptoms, with an onset within 12 hours. There was absence of fever and neurological symptoms. Chest computed tomography excluded atrio-esophageal fistula and was diagnostic of esophageal hematoma localized to either the upper esophagus or extending the length of the mid and lower esophagus; endoscopy confirmed the diagnosis. Management was conservative in all cases comprising of ceasing oral intake and anticoagulation. Long term sequelae included esophageal stricture formation requiring dilatation, persistent esophageal dysmotility (mid esophageal hematoma), and vocal cord paralysis, resulting in hoarse voice (upper esophageal hematoma).

Conclusions—Esophageal hematoma is a rare but important differential diagnosis for esophageal injury after TEE-guided AF ablation under GA, and can result in significant patient morbidity. Key clinical features differentiate presentation of esophageal hematoma from that of an atrio-esophageal fistula. (Circ Arrhythm Electrophysiol. 2012;5:701-705.)

Key Words: ablation ■ atrial fibrillation ablation ■ atrio-esophageal fistula ■ esophageal hematoma ■ transesophageal echocardiography

Radiofrequency (RF) ablation of atrial fibrillation (AF) is associated with a risk of collateral esophageal injury, of which an atrio-esophageal fistula is the most devastating complication.1–3 Transesophageal echo (TEE) is used commonly at the time of AF ablation procedures to exclude left atrial thrombus, define cardiac function, and in some centers to guide transseptal puncture.4–7 Recently, Nguyen et al8 reported a case of an esophageal hematoma as a complication after an AF ablation. This was attributed to the use of TEE during the procedure. While the presentation and risk factors for RF-related esophageal injury are well described, only isolated cases of esophageal hematoma have been reported.9–13 As such, our understanding of this condition remains limited and the differentiating clinical features from an atrio-esophageal fistula remained undefined. The aim of this study was to describe the incidence, characteristics, and sequelae of esophageal hematoma in a large center, where TEE is used routinely in the setting of AF ablation under general anesthesia (GA).

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Transesophageal Echocardiography
General anesthesia (GA) was used in all reported procedures. After induction of GA and neuromuscular blockade, an experienced cardiac anesthetist or an echocardiologist inserted a well-lubricated TEE probe. All cardiac anesthetists and echocardiologists had extensive experience in TEE insertion and manipulation. TEE was performed to exclude left atrial (LA) thrombus, define atrial and septal anatomy, and to guide the transseptal puncture. After transseptal puncture, the probe was retracted until it was outside the cardiac silhouette and left in situ in a neutral, unlocked position, with probe function either on “freeze” or probe disconnected from the machine. Imaging was repeated at the completion of the procedure to detect pericardial fluid and to image pulmonary vein (PV) flow. Imaging also was employed during the procedure if there was hemodynamic instability suggestive of cardiac tamponade.

Ablation Procedure
Standard catheters were positioned as previously described. Transseptal puncture was guided by fluoroscopy and TEE. The point of maximal interatrial septal tenting was defined anatomically by TEE prior to septal crossing. Patients received a bolus of 100 IU/kg of intravenous heparin after the first transseptal puncture and further heparin to achieve an activated clotting time of 300 to 350 after the second puncture. In all patients an irrigated ablation catheter and a circular mapping catheter were deployed in the LA.

Left Atrium Mapping and Ablation
LA geometry was created using a 3-dimensional nonfluoroscopic mapping system (CARTO-XP, Biosense-Webster, or Ensite NavX, St. Jude Medical). The esophageal course was segmented from the computed tomography (CT) scan and marked on the mapping system during the ablation. The ablation strategy in all patients was to perform circumferential point-by-point ablation to achieve pulmonary vein isolation (PVI) at the antrum, as described previously. Power was limited to 30 to 35 W at anterior sites, with temperature limited to 48 degrees and RF time ≤30 to 60 seconds for each lesion. At posterior sites, power was limited to 20 to 25 W, with 10 to 30 seconds for each lesion. Adjunctive LA ablation (linear ablation or ablation of fractionated electrograms) was performed only in those with persistent AF, at the discretion of the operator.

Postablation Care and Anticoagulation
Heparin reversal was not performed at the completion of the procedure. Either full or half dose (at the discretion of the electrophysiologist) LMWH was commenced approximately 6 hours postprocedure and continued until the international normalized ratio was ≥2.0. Concurrently, warfarin was started on the first night after the procedure.

Statistical Analysis
The Statistical Package for the Social Sciences for Windows (SPSS, release 15.0) was used for analysis. To test for associations between categorical variables, \( \chi^2 \) tests or Fisher exact test were used. Mean values were compared using the Student t test. Mann-Whitney U- or Kruskal-Wallis tests were used for continuous variables, where normal distribution was not present. A 2-tailed probability value of <0.05 was considered statistically significant.

Results
During the period of the study, 3 patients experienced esophageal hematoma (procedural incidence 0.27%). No patient died as a consequence. Length of stay in patients with esophageal injury was a median of 6 days (range, 4–12 days). There were no significant differences in clinical or procedural features between patients with esophageal hematoma compared with those without hematoma (Table). There was trend toward a higher incidence of injury in females (0.9% of all females) compared with males (0.1% of all males; \( P=0.11, \) Fisher’s exact test; Table 1).

<table>
<thead>
<tr>
<th>Table. Baseline Characteristics Comparing Those Patients With Esophageal Hematoma With Those Without Hematoma</th>
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<tr>
<td><strong>Esophageal Hematoma</strong></td>
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<tr>
<td>Age, mean (SD), y</td>
</tr>
<tr>
<td>Female gender, n, %</td>
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<tr>
<td>Persistent AF</td>
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<tr>
<td>LA size, mean (SD), mm</td>
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<tr>
<td>Fluoroscopy time</td>
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<td>Ablation time</td>
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<tr>
<td>Body mass index</td>
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<td>Body surface area*</td>
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*Calculated with the Mosteller formula body surface area=Sqrt[height in cm x weight in kg]/3600).

Individual cases are described below. In this series of patients, there were no cases of atrio-esophageal fistula.

Patient 1
A 57-year-old female with hypertension and a 1-year history of paroxysmal atrial fibrillation (PAF) underwent PVI (LA size 40 mm). Intubation was performed with ease (Mallampati score I). The TEE probe was inserted without difficulty. The patient developed progressive dysphagia and odynophagia within 12 hours of the procedure, culminating in inability to swallow saliva, with no neurological symptoms or fever. Thoracic CT scan on day 1 showed a hematoma arising from the posterior wall of the esophagus, with narrowing of the lumen and deviation to the left. It extended from the level of the carina down the esophageal hiatus at the level of the diaphragm. There was no free gas or contrast extravasation in the mediastinum, with a clear fat plane visible between the esophagus and the PVs and LA. Anticoagulation was ceased; the patient was made nil orally, started on intravenous ceftriaxone and metronidazole, and received a pantoprazole infusion. A gastrografin swallow on day 5 confirmed marked esophageal thickening but no acute stricture (Figure 1A). Gastroscopy performed 2 months later for persistent mild dysphagia showed minor narrowing at 3 cm above the gastrointestinal junction, which was successfully dilated. She experienced persistent dysphagia 12 months later; repeat gastrografin swallow showed no strictures (Figure 1B) and a gastroscopy was normal. She was diagnosed with mild residual esophageal dysmotility.

Patient 2
A 48-year-old man with an 8-year history of PAF underwent PVI (LA size 41 mm). Intubation was performed with ease (Mallampati score I). The TEE probe was inserted without difficulty. The patient underwent cardioversion to sinus rhythm. The patient complained of dyspnea. PVI was performed successfully and the patient underwent videofluoroscopy to sinus rhythm. The patient complained of dysphagia, odynophagia, and regurgitation of food remnants with frothy secretions 12 hours postprocedure. Chest CT with contrast showed a large intramural esophageal hematoma extending from the posterior esophageal wall at the level of the superior aspect of the left atrium extending for 9 cm inferiorly. There was a large pointed right-sided osteophyte at T8/9 vertebræ, which projected anteriorly to focally indent the mural hematoma (Figure 2A). Gastroscopy showed a large hematoma 10 cm long that obstructed...
the lumen at 29 cm from the mouth. The gastroscope was able to pass behind it (Figure 2B). The patient was nil by mouth, anticoagulation ceased, and intravenous pantoprazole was commenced for 72 hours. The patient made a rapid recovery and was discharged 4 days later on thin fluids. There was no residual odynophagia or dysphagia at 3 months follow-up.

**Patient 3**

A 49-year-old woman with a 4-year history of PAF progressing to persistent AF in the last year underwent PVI and a LA roofline (49 mm). Intubation was performed with ease (Mallampati score I). After induction of GA, the TEE probe was inserted without difficulty. The patient complained of throat pain, odynophagia, and dysphagia starting 8 hours postprocedure and progressing over the next 48 hours. On examination she had a hoarse voice, early stridor, and tenderness over the right paralaryngeal gutter region. CT neck and chest showed a 2-cm hematoma of the upper esophageal wall obstructing the esophageal lumen and displacing the trachea. There was no free gas in the surrounding structures, suggestive of esophageal perforation. Fiberoptic nasopharyngolaryngoscopy showed a hematoma in the pharynx/pyriform muscle, and the posterior lobe of the right thyroid. The right vocal cord was paralyzed from blood in the parapharyngeal space. She was commenced on intravenous dexamethasone, and anticoagulation was ceased. A repeat CT scan 1 week later showed no enlargement of the hematoma. There was resolution of odynophagia over the following week. The hoarse voice resolved over 6 months.

**Discussion**

Radiofrequency-related esophageal injury, specifically atrio-esophageal fistula formation, is a rare and devastating complication of AF ablation.1–3 Recently, Nguyen et al8 reported a case of esophageal hematoma, attributed to the use of TEE, as a complication after an AF ablation. While the presentation and risk factors for RF-related esophageal injury are well defined, our understanding of esophageal hematoma is based on isolated case reports mostly in the non-AF ablation setting, where the hematoma was attributed to endoscopic intervention,6 blunt trauma,10 thrombolysis,11 or was of idiopathic origin.12 McCall et al13 also reported 1 case of TEE-induced esophageal hematoma post-AF ablation; however, this was attributed to postanesthetic emesis.

The present study describes the incidence and clinical presentation of TEE-induced esophageal hematoma, specifically in the AF ablation setting where over 1000 consecutive procedures were performed with TEE-guidance under GA and high dose anticoagulation. We observed that TEE-induced esophageal hematoma is rare, with a procedural incidence of 0.27%. There was a trend toward higher risk in females compared with males. Importantly, the distinctive clinical features that may differentiate this type of injury from an atrio-esophageal fistula are the early presentation (within 12 hours) and the absence of neurological symptoms and fever. In contrast, atrio-esophageal fistula usually presents 3 to 12 days postablation with a range of symptoms from profound sepsis, endocarditis, mediastinitis, or gastrointestinal exsanguination.1–3

Esophageal hematoma carries substantial morbidity with prolonged hospital stay, potential for residual esophageal strictures, and dysmotility in the long-term. In addition, anticoagulation must be ceased at a time of significant postprocedural thromboembolic risk. CT scan of the chest was the first critical step in excluding atrio-esophageal fistula and in diagnosis of hematoma, which then was confirmed by endoscopic means. It is critical to note that endoscopy should only be employed if atrio-esophageal fistula is truly excluded, as gas insufflation during endoscopy in a patient with atrio-esophageal fistula can be devastating. If possible, endoscopy without gas insufflation is preferred.

Radiofrequency-related esophageal injury is attributed to direct thermal damage to the left atrium or its blood supply, or damage to vagus nerve branches that lie in close proximity to the left atrium. The latter also can cause autonomic disturbances, such as gastric hypomotility.14 In contrast, TEE-induced esophageal hematoma is likely to result from a small mucosal tear followed by “aggressive” heparin anticoagulation.

The presentation of esophageal hematoma can be catastrophic; if large enough, compression of adjacent chambers, such as the left atrium, can cause hemodynamic instability,11–12 airway compression resulting in respiratory distress,13 and catastrophic gastrointestinal bleeding and hypovolemic shock if the intramural hematoma extravasates into the esophageal lumen.7 Although none of these consequences were observed in this setting, recognition of TEE-induced esophageal complication as distinct from RF-related injury
appears critical for management. The former is best treated conservatively with cessation of oral intake and anticoagulation and has a good prognosis, whereas the latter often requires aggressive surgical intervention and carries a high mortality.1–3 Although specific recommendations on short, intermediate, and long-term follow-up are lacking, a number of important points can be learnt from a review of cases from around the world, where esophageal hematoma from varying causes has been diagnosed and treated.9 Generally, treatment should be conservative with analgesia, cessation of anticoagulation, and cessation of oral intake. Reintroduction of oral intake should be with fluid, followed by soft diet, and progressing to normal diet based on the extent of clinical recovery. In most cases of esophageal hematoma from other causes, resolution of symptoms is noted by ~2 weeks.9 Parenteral nutrition may be considered for patients with delayed recovery who start to exhibit malnourishment, although this would be expected to be extremely rare.

Proton pump inhibitors may help in attenuating acid-reflux mediated exacerbation of esophageal injury and may help improve rate of recovery of the hematoma through the ulcerative phase. These should be administered intravenously until oral intake is re-established, after which time oral treatment will suffice. However, the evidence for this is lacking but the approach seems rational.9 Antibiotics are likely to be of limited benefit. Anticoagulation should be recommenced only when there are signs of active bleeding or hematoma enlargement and swallowing has improved significantly. Low molecular weight heparin should be avoided due to its limited reversibility. In our series, we recommended recommencing warfarin at a low dose in the absence of low molecular weight heparin or unfractionated heparin and allowed the international normalized ratio to reach therapeutic range slowly with frequent monitoring. This was initiated after clinical stability was established. In rare cases where active arterial bleeding, esophageal perforation with mediastinitis, or rapid enlargement of the hematoma with respiratory distress is evident, an operative approach may be necessary. As reported in the present study, these recommendations were followed closely with no resulting adverse sequelae from esophageal hematoma.

Intermediate follow-up should constitute repeat endoscopy and gastrografin swallow to exclude persistent structural or functional abnormalities of the esophagus. Both intermediate and long-term follow-up should include the expertise of a gastroenterologist or endoscopist. There may be a need for
endoscopic dilatation. If repeat catheter ablation is planned for recurrence of AF, the operator should strongly consider the use of other imaging modalities, such as intraoperative cardiac echocardiography.

The described procedural occurrence of TEE-related esophageal injury was low; this risk must be balanced against the risks posed by transseptal puncture without imaging and the costs of intracardiac echo, with the associated risk of groin complications from a large sheath insertion. An examination of asymptomatic injury related to TEE would be of interest in a future study employing routine endoscopic surveillance. However, the present study focused purely on the incidence and sequelae of severe, symptomatic, clinically relevant esophageal complications related to TEE.

**Study Limitations**

The study has limited statistical power for identifying correlates of hematoma that results from having observed only 3 events. Larger multicenter data may shed light on predictors of esophageal hematoma.

**Conclusions**

Esophageal hematoma, although rare (0.27%), should be recognized as an important differential diagnosis in the investigation of suspected esophageal injury after an AF ablation. The condition carries significant patient morbidity and residual clinical sequelae. Key clinical features differentiate the presentation of esophageal hematoma from that of an atrio-esophageal fistula, and prognosis is favorable with conservative management.

**Disclosures**

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**CLINICAL PERSPECTIVE**

Atrial-esophageal fistula is the most feared and devastating complication of atrial fibrillation (AF) ablation. Although rare (0.05%–1.2%), it is associated with a high mortality (~50%), often with catastrophic presentation at ~12 days postablation with profound sepsis, endocarditis, mediastinitis, gastrointestinal exsanguination, or neurological demise. The present study describes the incidence, clinical features, and sequelae of esophageal hematoma as a “different sort” of esophageal injury in a prospective study of 1110 consecutive AF ablation procedures, guided by transesophageal echo (TEE). TEE commonly is used at the time of AF ablation procedures to exclude left atrial thrombus, define cardiac function, and guide transseptal puncture. Esophageal hematoma has been described in isolated case reports; however, our understanding of the condition remains limited. We found that the incidence of esophageal hematoma was low (0.27%), with a more benign presentation to that of fistula. It typically presented early postablation (within 12 hours), with absence of fever and neurological symptoms. Symptoms were attributed to intraluminal (odynophagia, regurgitation) or extraluminal esophageal compression (hoarseness from recurrent laryngeal nerve palsy). Computed tomography scan was the most critical and absolutely the first step in excluding atrio-esophageal fistula. Only then was endoscopy performed to confirm the diagnosis, as air insufflation during endoscopy otherwise may have had catastrophic consequences for a undiagnosed fistula. Management was conservative, comprising of ceasing oral intake and anticoagulation and commencement of proton pump inhibitors. The prognosis was benign with no deaths; however, long term sequelae that required attention included esophageal stricture formation requiring dilatation, persistent esophageal dysmotility, and vocal cord paralysis.
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