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

Running title: Kumar et al., Esophageal hematoma post AF ablation

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Abstract:

Background - Esophageal hematoma has recently 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 trans-septal puncture. The procedural incidence of esophageal hematoma was 0.27% (3/1110 procedures, mortality 0%). Odonyphagia, regurgitation and hoarseness were the predominant symptoms with an onset within 12 hours. There was absence of fever and neurological symptoms. CT chest 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.

Key words: ablation; atrial fibrillation ablation; atrio-esophageal fistula; esophageal hematoma; transesophageal echocardiography

Abbreviations: AF-atrial fibrillation; GA-general anesthesia; INR-international normalized ratio; LA-left atrium; LMWH-low molecular weight heparin; PVI-pulmonary vein isolation; RF-radiofrequency; TEE-transesophageal echocardiography; 3-D- three dimensional.
Introduction

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. Trans-esophageal echo (TEE) is commonly used at the time of AF ablation procedures to exclude left atrial thrombus, define cardiac function and in some centers to guide trans-septal puncture. Recently, Nguyen et al 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. Whilst the presentation and risk factors for RF-related esophageal injury are well described, only isolated cases of esophageal hematoma have been reported. 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 routinely used in the setting of AF ablation under general anesthesia (GA).

Methods

Study population

This series included 1110 consecutive catheter ablation procedures for AF performed in 817 patients under GA between March 2002 to October 2011 at the Royal Melbourne Hospital, Melbourne Private Hospital and The Alfred Hospital, Melbourne, Australia. AF was paroxysmal in 922 procedures and persistent in 188 procedures.

Pre-procedural Anticoagulation

Patients on warfarin pre-procedure were asked to cease it 5 days before the procedure and low molecular weight heparin (LMWH) commenced at a dose of 1 mg/kg twice daily when
international normalized ratio (INR) fell below 2.0. The last dose of LMWH was given the night prior to the procedure. Aspirin was continued to the night before the procedure.

Transesophageal echocardiography

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 trans-septal puncture.7 After trans-septal puncture, the probe was retracted till 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 PV flow. Imaging was also employed during the procedure if there was hemodynamic instability suggestive of cardiac tamponade.

Ablation procedure

Standard catheters were positioned as previously described. Trans-septal puncture was guided by fluoroscopy and TEE. The point of maximal inter-atrial septal tenting was always defined anatomically by TEE prior to septal crossing.7

Patients received a bolus of 100 IU/kg of intravenous heparin after the first trans-septal puncture and further heparin to achieve an activated clotting time of 300-350 after the second puncture.4 In all patients an irrigated ablation catheter and a circular mapping catheter were deployed in the left atrium (LA).

LA Mapping and Ablation

LA geometry was created using a three-dimensional (3D) non-fluoroscopic mapping system
(CARTO-XP, Biosense-Webster, Diamond Bar, CA, USA or NavX, St. Jude Medical, St. Paul, MN, USA). The esophageal course was segmented from the 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 previously described. Power was limited to 30–35 W at anterior sites with temperature limited to 48 degrees and RF time ≤ 30–60 seconds for each lesion. At posterior sites, power was limited to 20–25W with 10-30 seconds for each lesion. Adjunctive LA ablation (linear ablation or ablation of fractionated electrograms) was only performed in those with persistent AF at the discretion of the operator.

**Post-ablation 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 post procedure and continued until the INR ≥ 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, Chicago, USA) was used for analysis. To test for associations between categorical variables, $X^2$ tests or Fisher’s 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 two-tailed P-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 to those without hematoma (Table 1). There was trend toward a higher incidence of injury in females (0.9%) compared to males (0.1%, $P = 0.11$, Fisher’s exact test, Table 1). 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 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 a pantoprazole infusion. A gastrograffin 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 gastrograffin 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 paroxysmal AF underwent PVI (LA size 41mm). Intubation was performed with ease (Mallampati score II). The TEE probe was inserted without difficulty. PVI was successfully performed and the patient underwent cardioversion to sinus rhythm. The patient complained of dysphagia, odynophagia and regurgitation of food remnants with frothy secretions 12 hours post procedure. CT chest with contrast showed a large intramural esophageal hematoma extending from the posterior esophageal wall at the level of the superior aspect of left atrium for 9 cm inferiorly. There was a large pointed right-sided osteophyte at T8/9 vertebrae, 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 made 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 1 year underwent PVI and a roofline (LA 49 mm). Intubation was performed with ease (Mallampati score I). After induction of general anesthesia, the TEE probe was inserted without difficulty. The patient complained of throat pain, odynophagia and dysphagia starting 8 hours post-procedure and progressing over the next 48 hours. On examination she had a hoarse voice, early stridor and tenderness over the right para-laryngeal 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. Fibreoptic 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 paraglottic 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

RF-related esophageal injury, specifically atrio-esophageal fistula formation is a rare and devastating complication of AF ablation. 1 2 3 Recently, Nguyen et al reported a case of esophageal hematoma, attributed to the use of TEE, as a complication after an AF ablation. 8 Whilst 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, 9 blunt trauma, 10 thrombolysis, 11 or was of idiopathic origin. 12 McCall et al also reported one case of TEE-induced esophageal hematoma post AF ablation, however this was attributed to post-anesthetic emesis. 13

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 to 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 3-12 days post ablation 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 post-procedural thromboembolic risk. CT scan of the chest was the first critical step in excluding atrio-esophageal fistula and in diagnosis of hematoma, which was then 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.

RF-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 can also 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 haemodynamic instability,11 12 airway compression resulting in respiratory distress13 and catastrophic gastrointestinal bleeding and hypovolemic shock if the intramural hematoma extravasates into the esophageal lumen.9 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.\textsuperscript{1,2,3} Although specific recommendations on short, intermediate and long term follow up are lacking, a number of important points can be learnt from on review of cases from around the world where esophageal hematoma from varying causes has been diagnosed and treated.\textsuperscript{9} Generally, treatment should be conservative with analgesia, cessation of anticoagulation and cessation of oral intake. Re-introduction 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 \textasciitilde 2 weeks.\textsuperscript{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.\textsuperscript{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 significantly improved. Low molecular weight heparin should be avoided due to its limited reversibility. In our series, we recommended 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. 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 closely followed with no resulting
adverse sequelae from esophageal hematoma.

Intermediate follow up should constitute repeat endoscopy and gastrograffin 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/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 intra-operative cardiac echocardiography.

The described procedural incidence of TEE-related esophageal injury was low; this risk must be balanced against the risks posed by trans-septal puncture without imaging and the costs of intra-cardiac 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 a 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.

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**References:**


Table 1: Baseline characteristics comparing those patients with esophageal hematoma to those without hematoma.

<table>
<thead>
<tr>
<th></th>
<th>Esophageal hematoma (n=3)</th>
<th>No hematoma (n=1007)</th>
<th>p value</th>
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<tbody>
<tr>
<td>Age, mean (SD), yrs</td>
<td>50 ± 2.7</td>
<td>57 ± 8.9</td>
<td>0.17</td>
</tr>
<tr>
<td>Female gender, n, %</td>
<td>2 (66.7)</td>
<td>228 (22.6)</td>
<td>0.11</td>
</tr>
<tr>
<td>Persistent AF</td>
<td>1 (33.3)</td>
<td>153 (15.2)</td>
<td>0.36</td>
</tr>
<tr>
<td>LA size, mean (SD), mm</td>
<td>43 ± 5</td>
<td>41 ± 7</td>
<td>0.62</td>
</tr>
<tr>
<td>Fluoroscopy time</td>
<td>36.7 ± 8.6</td>
<td>39.4 ± 22.4</td>
<td>0.81</td>
</tr>
<tr>
<td>Ablation time</td>
<td>46.8 ± 10.1</td>
<td>41.5 ± 23.2</td>
<td>0.60</td>
</tr>
<tr>
<td>Body mass index</td>
<td>29.8 ± 3.5</td>
<td>28.4 ± 4.8</td>
<td>0.61</td>
</tr>
<tr>
<td>Body surface area*</td>
<td>2.1 ± 0.08</td>
<td>2.1 ± 0.26</td>
<td>1</td>
</tr>
</tbody>
</table>

*Calculated with the Mosteller formula BSA=Sqrt[(height in cm x weight in kg)/3600]

AF-atrial fibrillation, kg-kilograms, LA-left atrial, m-meters, mm-millimeters, mins-minutes, PVI-pulmonary vein isolation, SD- standard deviation.
Figure Legends:

Figure 1: Barium swallow on Patient 1 (A) day 5-post ablation (B) and 1-year post ablation: (A) Barium swallow performed day 5 post ablation showing marked esophageal wall thickening (white arrows) consistent with esophageal hematoma; the esophago-gastric junction is marked with asterix (B) Barium swallow performed 1 year post ablation showing no residual wall thickening or strictures.

Figure 2: (A) Contrast CT (oral and intravenous) of patient 2, day 1 post ablation (B) Gastroscopy performed day 2-post ablation. (A) A large intramural filling defect (28 mm in anteroposterior diameter and 42 mm in transverse diameter), consistent with haematoma (*), extends within the posterior wall of the oesophagus from the level of the T7/8 intervertebral disc and superior aspect of left atrium for 9 cm inferiorly, at least to the level of T11. Only a very thin layer of intraluminal contrast medium (bold arrow) is able to extend past the haematoma. It is confined to the most anterior aspect of the oesophageal lumen. A moderately large, pointed right sided osteophyte at the T8/9 disc level (dotted arrow) projects directly anteriorly to focally indent the mural haematoma. Note no free gas or contrast is present in the mediastinum and a clear fat plane is present between the oesophagus and the pulmonary veins/left atrium/aorta, excluding an atrio-esophageal fistula. (B) Gastroscopy showed a large hematoma (*) 10 cm long that obstructed the lumen at 29 cm from the mouth (i and ii). The gastroscope was able to pass behind it (iii).
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