Incidence of oesophageal wall injury post-pulmonary vein antrum isolation for treatment of patients with atrial fibrillation

Martin Schmidt1*, Georg Nölker1, Harald Marschang1, Klaus-Jürgen Gutleben1, Volker Schibgilla1, Harald Rittger1, Anil-Martin Sinha1, Guido Ritscher1, Dirk Mayer2, Johannes Brachmann1, and Nassir F. Marrouche3

1Division of Cardiology, Klinikum Coburg, Coburg, Germany; 2Division of Gastroenterology, Klinikum Coburg, Coburg, Germany; and 3Division of Cardiology, Department of Internal Medicine, University of Utah, Salt Lake City, UT, USA

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Aims Oesophageal injury has been reported with delivery of radio-frequency lesions at the left atrium posterior wall in catheter ablation procedures for atrial fibrillation (AF). In this observational study we prospectively assessed endoscopical oesophageal wall changes after pulmonary vein antrum isolation (PVAI) in patients presenting for treatment of AF.

Methods and results Twenty eight patients (18 men; mean age 55 ± 11 years) were ablated using either a cooled-tip or an 8 mm tip ablation catheter. Endoscopy of the oesophagus was performed 24 h after PVAI. If oesophageal wall changes were detected post ablation, a proton-pump inhibitor (PPI) was started and repeat endoscopy was considered. Within 24 h post ablation oesophageal wall changes were confirmed in 47% of our study patients. Erythema was identified in 29% and necrotic or ulcer-like changes in 18% of patients. None of study patients experienced left atrial-oesophageal fistula. A significant correlation between Reflux-like symptoms and oesophageal wall changes was demonstrated. Complete recovery of oesophageal lesions was shown in all study patients 2–4 weeks post ablation.

Conclusion A significant number of patients experienced oesophageal wall injury post PVAI. Initiating PPIs in this group of patients might facilitate recovery of oesophageal wall injuries caused by radio-frequency energy delivery.

KEYWORDS Oesophageal injury; Radio-frequency catheter ablation; Pulmonary vein antrum isolation; Proton-pump inhibitor

Introduction

Since the introduction of left atrial ablation and specifically the pulmonary vein antrum isolation (PVAI) procedure for treatment of atrial fibrillation (AF), a tremendous improvement in success rates and complications has been reported.1–4 A recently defined and introduced fatal complication was oesophageal injury leading to left atrial oesophageal fistula.5–7 Despite the low incidence of the left-atrial oesophageal fistula (0.1%), it became the most feared impasse of the PVAI procedure.7–11 Other serious oesophageal injuries as acute pyloric spasms and gastric hypomotility have been also reported.12 To avoid those serious complications several efforts have been made including reduction of the amount of radiofrequency (RF) energy delivered when ablating within the left atrial posterior wall region or the pulmonary vein (PV) antra. Still, little is known about the type of injuries occurring in the anterior wall of the oesophagus.

Nevertheless to date there is no data about the incidence of endoscopic oesophageal wall changes initiated by RF energy delivery during the AF ablation procedures. In this observational study we report the incidence and characterize the occurrence of oesophageal wall changes immediately post PVAI.

Methods

Study patients

Twenty eight consecutive patients (18 men; mean age 55 ± 11 years) with symptomatic AF refractory to antiarrhythmic therapy were included in the study. Patients’ characteristics concerning type of AF,13 number of antiarrhythmic drugs used, underlying heart disease, functional NYHA class and size of left atrium are given in Table 1. Eleven patients with ischemic heart disease were

* Corresponding author. Tel: +49 9561 2233223.
E-mail address: mschmidtco@arcor.de

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Pulmonary vein antrum isolation protocol

PVAI is described elsewhere.14 Briefly, all antiarrhythmic drugs were discontinued >5 half-lives before the procedure. All 28 patients underwent a procedure that involves a technique isolating all of the pulmonary veins at their antrum. RF ablation was performed under conscious sedation with a continuous intravenous administration of small doses of midazolam (2-5 mg/h) and if needed an intravenous injection of morphine (5-10 mg). Intracardiac echocardiography guided mapping was performed using a 10-Fr 64 element phased-array ultrasound imaging catheter (AcuNav, Siemens Medical Solution). The catheter was introduced through an 11-Fr sheath via the left femoral vein and positioned fluoroscopically guided in the right atrium. The electrophysiologist optimized the ICE images. All PV ostia were defined before transseptal puncture; the transseptal puncture was performed under ICE guidance to visualize the intra-atrial septum. Then, a circular mapping catheter (Lasso, Biosense, Diamond Bar) and an ablation catheter were inserted into the left atrium. On-line information obtained by ICE was used to monitor catheter navigation in the left atrium and to evaluate the position of the mapping catheter in relation to the ostia of the pulmonary veins. Mapping and ablation of the left atrium was performed using either a cooled-tip (14 patients) or an 8 mm tip ablation catheter (14 patients). Using an open irrigation technology (0.9% saline) RF was delivered with a 3.5 mm tip catheter (Thermo-cool, or Navistar-Thermo-cool, Biosense Webster, Diamond Bar; Biosonik Inc., Berlin). The temperature and power were set at 50° and 50 W, respectively, similar to previously reported protocols.13,14 RF delivery was interrupted in the case of impedance rise or if a sudden increase in microbubble density was observed, energy delivery was terminated immediately15).

In all patients ICE imaging was able to visualize the longitudinal extent of contact between the left atrial, posterior wall and the oesophagus as demonstrated in Figure 1. All lesions at that site were tagged using the CARTO system.

Further detailed information concerning the technique for PVAI is given in Ref.14.

Endoscopy protocol

In all patients endoscopy of the oesophagus was performed 24 h after PVAI. Eso wall changes were defined as no change, erythema, necrosis or atrio-oesophageal fistula (characterized by the gross appearance of epithelial sloughing). If eso wall changes/injuries were detected endoscopically post ablation, a proton-pump inhibitor (PPI) was started (omeprazole 40 mg once a day for 2 weeks) and a repeat endoscopy was considered 2 weeks later. All endoscopy reports were reviewed by two independent gastroenterologists to assess for eso wall changes that could have been initiated during the PVAI.

Follow-up

All patients were monitored overnight on a telemetry unit after the procedure. Warfarin (INR 2-3) was restarted in all patients on the day of PVAI. If eso wall changes/injuries were observed endoscopically a repeat endoscopy was considered 2 weeks later. If oesophageal injury was still noted 2 weeks post PVAI, control endoscopy was performed 4 weeks later or until complete recovery of lesions was documented.

Statistical analysis

Continuous variables are expressed as the mean ± SD. Data distributions were first assessed for normality and for aberrancies. The Student’s t-test was used for comparisons of continuous variables. Pearson χ² test was used for categorical data comparisons. Values of P ≤ 0.05 were considered statistically significant.

Results

Intracardiac echocardiogram (Figure 1) was used to define the number (9 ± 4 lesions) and duration (3.0 ± 2.5 min) of left atrial lesions delivered within proximity to the oesophagus.

In all study patients an endoscopy of the eso was performed 24 h after PVAI. Symptoms of reflux were present in 50% of patients within 24 h post PVAI. A correlation was observed between reflux-like symptoms and oesophageal wall injury (r = 0.9).

Table 1  List of the types of atrial fibrillation (AF), number of antiarrhythmic drugs used, underlying heart disease, functional NYHA class and size of left atrium

<table>
<thead>
<tr>
<th>Patients</th>
<th>28</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male/female</td>
<td>18/10</td>
</tr>
<tr>
<td>Age (years)</td>
<td>53 ± 8</td>
</tr>
<tr>
<td>Left atrial size (cm)</td>
<td>4.4 ± 0.8</td>
</tr>
<tr>
<td>Duration of AF (years)</td>
<td>5.1 ± 3.4</td>
</tr>
<tr>
<td>Antiarrhythmic drugs</td>
<td>2 ± 3</td>
</tr>
<tr>
<td>Paroxysmal AF</td>
<td>17</td>
</tr>
<tr>
<td>Persistent/permanent AF</td>
<td>11</td>
</tr>
<tr>
<td>NYHA class</td>
<td>2.2 ± 1.3</td>
</tr>
<tr>
<td>Underlying heart disease</td>
<td></td>
</tr>
<tr>
<td>Ischemic</td>
<td>11</td>
</tr>
<tr>
<td>Hypertensive</td>
<td>8</td>
</tr>
<tr>
<td>None</td>
<td>9</td>
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on daily aspirin 100 mg. There were no non-steroid antiinflammatory agents used 4 weeks prior to PVAI. Moreover, there were no reflux-like symptoms in any patient 4 weeks prior to PVAI. Each patient was interviewed concerning reflux-like symptoms or other abdominal problems after admission prior to PVAI procedure. None of the 28 patients had any symptoms of reflux or any other gastric complaints within 4 weeks prior to ablation.

All patients signed a written consent approved by the institutional ethics committee.

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| Persistent/permanent AF | 11 |
| NYHA class | 2.2 ± 1.3 |
| Underlying heart disease | |
| Ischemic | 11 |
| Hypertensive | 8 |
| None | 9 |

Figure 1  Intracardiac echocardiogram demonstrating the left atrium with left atrial posterior wall contiguous to the oesophagus.

was titrated downwards by 5 W decrements until microbubbles generation subsided and RF delivery was continued. If a brisk shower of microbubbles was observed, energy delivery was terminated immediately15).

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In all study patients an endoscopy of the eso was performed 24 h after PVAI. Symptoms of reflux were present in 50% of patients within 24 h post PVAI. A correlation was observed between reflux-like symptoms and oesophageal wall injury (r = 0.9).
Within 24 h post ablation, erythema (Figure 2) was found in 8 of the 28 patients (29%), necrotic or ulcer-like changes (Figure 3) were seen in 5 of 28 patients (18%). No left atrial-oesophageal fistula was seen in our study group. In all 13 patients oesophageal wall changes were seen at the anterior aspect of the oesophagus within vicinity to the left atrial posterior wall. There was no correlation between oesophageal wall changes and underlying heart disease or patients’ drug regimen. All patients with documented oesophageal wall changes (47% of patients) were started on omeprazole, 40 mg per day for 2 weeks duration. Repeat endoscopy 2 weeks after the ablation procedure revealed recovery of all erythematous changes.

Complete recovery of eso lesions was detected latest 4 weeks after PVAI in all of the five patients in whom diagnosis of necrotic wall changes was made 24 h after PVAI. Table 2 lists the findings related to the acute and chronic oesophageal wall changes after PVAI and in Table 3 findings related to ablation with open-irrigated 3.5 mm tip catheter and 8 mm tip non-irrigated catheter are listed.

**Discussion**

In this prospective study endoscopic analysis of oesophageal wall changes showed a high incidence of oesophageal injury during PVAI procedures utilizing open irrigated or 8 mm tip ablation catheters. We demonstrated that around half of our study population experienced oesophageal wall injury after PVAI. This is an important finding and supports the ongoing debate on how to best minimize oesophageal injury during the AF ablation procedure.

**Previous studies**

The detection of the oesophageal–left atrial anatomical relationship and monitoring of appropriate transmural lesion formation during RF energy delivery would help to avoid injury within the oesophageal wall and hence left atrial oesophageal fistulas.

Lemola et al. highlighted the importance of real-time imaging using barium swallow during the AF ablation procedure to track the frequent mobility of the oesophagus. Although useful, but this might lead to incomplete isolation of the PV antra or targeting complex fractionated electrograms, because significant part of the PV antrum or fractionated potentials are located within the posterior wall area.

Monitoring of the left atrial lesion formation during ablation would be an ideal strategy to avoid oesophageal injury. Unfortunately today’s technology would allow us to only indirectly monitor or suggest oesophageal injury during energy delivery. Oesophageal temperature monitoring during RF ablation is an approach recently introduced. Nevertheless, the potency of this approach avoiding oesophageal wall injury before it takes place is questionable. As demonstrated by Cummings et al. no correlation between delivered energy and oesophageal temperature was detected. A recent comparison of open irrigation tip catheter set at 50 and 50 W with the phased array ICE-guided energy delivery proved open irrigation ablation technology to be superior in terms of efficacy and safety in PVAI. Another study demonstrated in an in-vivo canine model that microbubble formation detected using ICE was occasionally absent during RF delivery around the PV ostia with tissue temperatures > 80°C, and it was concluded that microbubble formation could not be a consistent marker of tissue overheating. The inaccuracy of this method was supported by our data, in which about one-fifth of patients (18%) undergoing ICE-guided energy delivery experienced necrotic changes within the oesophageal wall.
In our study no atrio-oesophageal fistula was seen after PVAI, but the most common pathologic finding was focal erythema of the anterior oesophageal wall in a region remote from the gastrooesophageal sphincter. Such changes would be highly unusual in a control population not undergoing an RF ablation procedure. Oesophageal injury post transoesophageal echocardiography is a very rare complication.27,28 Only study patients with persistent or permanent AF (11 of 28 patients) underwent TEE prior to the ablation procedure. Also this would decrease the possibility of correlation between oesophageal wall changes and TEE-related injury in our patient population. Moreover, wall injuries were not observed more frequently in patients who underwent TEE prior to PVAI.

**Conclusions**

In summary we showed that erythema of the oesophagus seems to be a common finding in patients undergoing PVAI procedures, with important clinical relevance. Further efforts are needed to improve the current available real-time imaging technology to further avoid complications and improve outcomes of AF ablation procedures.

Since there was a correlation between reflux-like symptoms and oesophageal lesions we would recommend every patient with symptoms of reflux post PVAI to be followed closely using endoscopy. Repeat endoscopy should be done to track changes and recovery. A prophylactic therapy with a proton-pump inhibitor as a routine therapy or in case of reflux-like symptoms and oesophageal lesions would be recommended.

**Conflict of interest:** none declared.

**References**