Asymptomatic esophageal perforation following high-power short-duration pulmonary vein isolation—A case of conservative cure

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Introduction

In patients undergoing pulmonary vein isolation (PVI), neither reduction of power, contact force and ablation time at the left atrial (LA) posterior wall (LAPW), nor monitoring of the luminal esophageal temperature (LET) have reduced the incidence of esophageal lesions and atrioesophageal fistula.[1–3] Whether high-power short-duration (HPSD) radiofrequency (RF) protocols, thought to result in shallower lesions,[4] are able to prevent esophageal injury is still unresolved.[5–8]

Case report

We report a 69-year-old female patient (body mass index 33.1 kg/m^2) with esophageal perforation following HPSD-PVI that resolved with endoscopic therapy and did not progress to fistula.

To study the pathophysiology of esophageal injury, endoscopy is performed routinely following PVI in our center, and with that, we had the opportunity to detect and to observe the progression to asymptomatic esophageal perforation. Conventional methods (eg, RF parameters and LET monitoring) all did not predict this complication.

KEY TEACHING POINTS

- High-power short-duration (HPSD) ablation may also lead to severe esophageal damage that can be asymptomatic and may progress to perforation.
- Current lesion assessment tools and luminal esophageal temperature (LET) monitoring are insufficient.
- Improvement in both radiofrequency lesion assessment parameters (also suitable for HPSD protocols) and LET monitoring, modification of ablation strategy (overall avoiding lesions in proximity of the entire longitudinal course of the esophagus, eg, single-ring pulmonary vein / posterior wall isolation), and new energy sources may all help to prevent esophageal injury.

Ablation

Preprocedural computed tomography (CT) showed a severely dilated LA (volume index 51.3 mL/m^2), and the esophagus was positioned along the medial border of the left common pulmonary vein (PV) orifice. PVI was performed with conscious sedation using an HPSD protocol (50 watts for a maximum of 13 seconds [TactiCath; Abbott Inc, Chicago, IL]). Very wide antral PVI left a narrow corridor of atrial tissue at the LAPW, potentially facilitating regular atrial tachycardia. Therefore, a short connecting line

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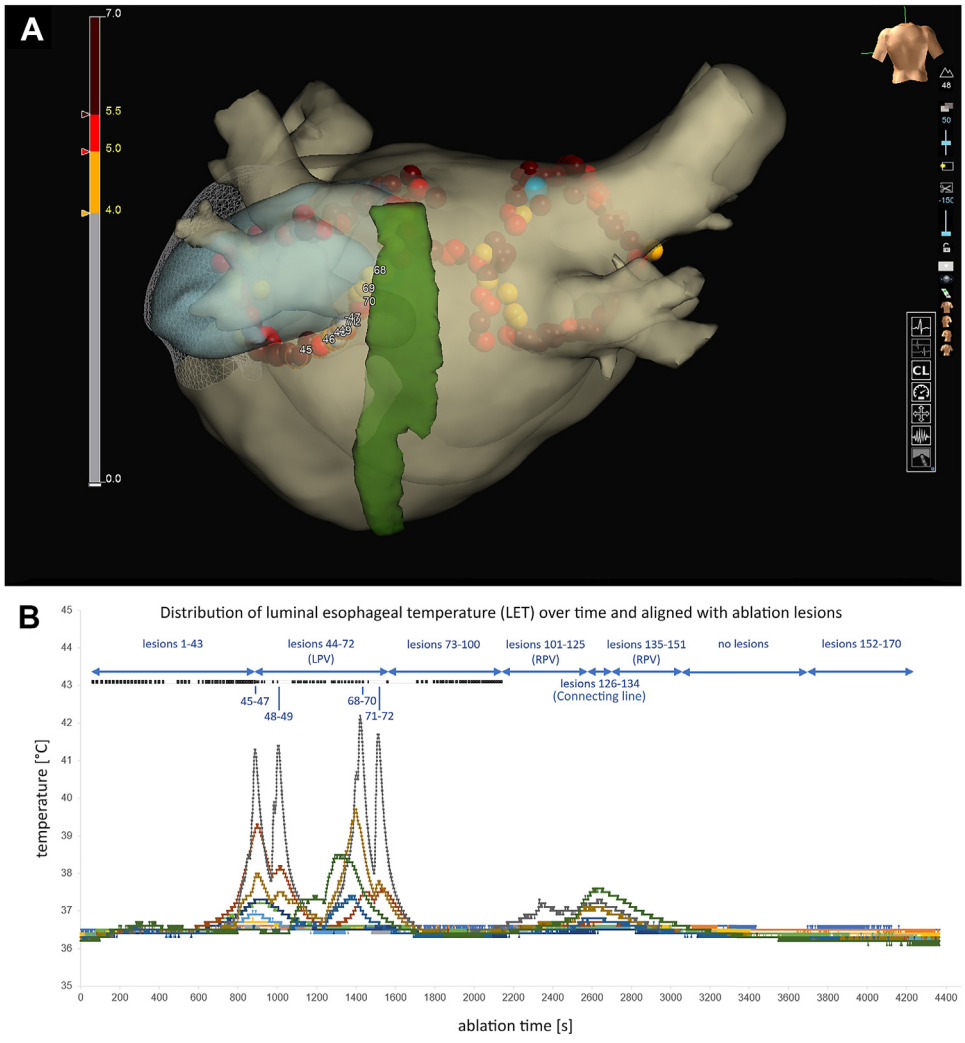

Figure 1 **A:** Three-dimensional reconstruction of the left atrium, esophagus, and pulmonary veins (Abbott EnSite Verismo; Abbott Inc, Chicago, IL). Ablation lesion set (*yellow and red dots*) with 2 wide antral lines encircling the pulmonary veins and an interconnecting posterior line. The left atrial appendage is displayed in blue color. Computed tomography–segmented esophageal course (*green*). The ablation lesions associated with the esophageal perforation site are displayed with the corresponding lesion numbers (45–49 and 68–72), the analysis of the lesion parameters is given in Supplemental Table 1. **B:** Luminal esophageal temperature (LET) curves of all 12 thermocouples showing 2 double peaks (maximum LET 42.2°C; Supplemental Table 1) corresponding to the ablation lesions 45–49 and 68–72. The connecting line (lesions 126–134) should have been performed with a larger distance to the esophageal course (at the left atrial roof rather than at the closest point between the 2 circles). However, analyzing the temperature data, this line was not associated with a suspicious LET rise (max. 37.6°C). The timing of the ablation lesions was integrated in the figure.

was added at the LAPW (at the closest distance between the 2 circles) (Figure 1A). LET monitoring (S-Cath; Circa Scientific, LLC, Englewood, CO) was used during energy application at the LAPW (Figure 1B). RF energy was stopped when LET reached 41.0°C and ablation continued after LET had normalized. Lower temperature limits from other studies have not been shown to prevent lesions.

Esophageal lesion and perforation

Following an unsuspicious clinical course, esophagogastroscopy on day 3 (the next working day) showed an esophageal ulceration close to the LA, associated with periesophageal edema (endoscopic ultrasound) (Figure 2A and 2B). The dosage of proton pump inhibitor therapy was increased (pantoprazole 40 mg twice daily), and sucralfate as well as food and drinking restriction were added.

The patient was kept in hospital and remained asymptomatic. Repeat endoscopy, endoscopic ultrasound, and CT on day 10 showed significant progression to perforation (Figure 3A and 3B); there were no signs of mediastinitis, air trapping, or fistula formation. Careful analysis of all CT slices located the perforation site to the inferoposterior border of the left common PV (Figure 3C and 3D).

The patient was transferred to the intensive care unit. A surgical intervention (esophageal repair) was discussed but was rejected in the absence of fistula and mediastinitis. Similar to treatment of instrumental esophageal perforation, endoscopic clips were placed at the perforation site to reduce any inflow of saliva and gastric secretions (Figure 3E and

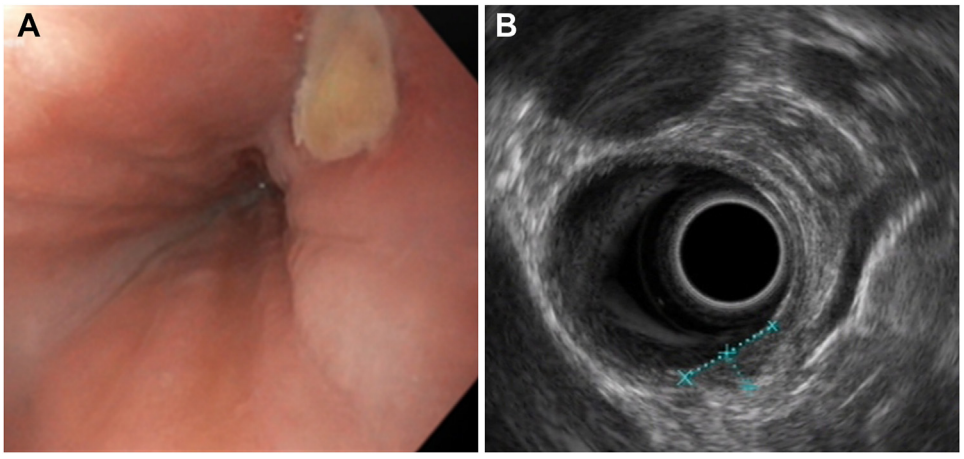

Figure 2 Day 3 following pulmonary vein isolation. Thermally induced esophageal ulcer (**A**), associated with edema (10 × 4 mm, blue-dotted lines) of the esophageal wall (**B**), visualized in endoscopic ultrasound.

3F). A nasointestinal tube was inserted and fixed by a clip in the distal portion of the duodenum to allow oral drug therapy.

Absolute food and drinking restrictions were continued, and pantoprazole 40 mg was administered 3 times daily intravenously. The patient was put on systemic antimicrobial therapy (Meropenem and caspofungin) in analogy to esophageal perforation of other etiologies. Oral anticoagulation (rivaroxaban 20 mg/d) was replaced by intravenous heparin.

Online LET monitoring and offline analysis of PVI data

To understand the pathophysiology of thermally induced perforation, ablation lesion data and LET recordings were extracted and analyzed offline. The presumed lesions associated with the perforation at the inferoposterior border of the left common PV (numbers 45–49 and 68–72) showed lesion size index (LSI) of 4.4–5.5, contact forces of 8–20 grams, and impedance drops of 8–17 ohms, respectively. Owing to the observed temperature increase with lesions 45–49, lesions 68–72 were performed after completion of ablation at the superoposterior border of the left PV (Figure 1A, Supplemental Table 1). There were 4 LET peaks >41.0°C (maximum 42.2°C) with those lesions; the temperature rise from 39.0°C to 41.0°C took 6 seconds, and took another 5 seconds to 42°C. The connecting line showed a maximum of 37.6°C (Figure 1B).

In-hospital follow-up

Throughout the entire hospital stay, the patient remained asymptomatic. Blood chemistry showed a rise of C-reactive protein (maximum 15.2 µmol/L) without leukocytosis and normal procalcitonin levels.

After serial CT scans performed every 3–5 days (with intravenous and oral [water-soluble] contrast agents) excluded mediastinitis, air trapping, or fistula formation, repeat endoscopy and endoscopic ultrasound demonstrated initial healing with granulation at the bottom of the ulcer on day 20, and marked improvement on day 31. The patient was transferred to the regular ward, the nasoduodenal tube removed, and liquid oral nutrition started.

Complete healing and the absence of any esophageal extravasate in the CT scan were seen on day 38. C-reactive protein had normalized. The patient was discharged on day 40 with dietary restrictions (nutrition with liquid or pulpy food, avoidance of hot and cold drinks).

Out-of-hospital follow-up

The patient remained asymptomatic and in sinus rhythm. During repeat CT scan and endoscopic and endosonographic evaluation on day 53 and on day 172, complete healing of the esophageal lesions without any visible alterations of the mucosa was confirmed.

Discussion

The presented case with esophageal perforation (albeit not fistula) following HPSD-PVI is unusual but offers several teaching points regarding the time course and pathophysiology of lesion progression.[9,10]

In the absence of any symptoms and detected by routine postinterventional endoscopy/ultrasound, dramatic progression of an esophageal ulcer to perforation occurred within a brief time period. The combined LET information (Figure 1) and the CT-derived anatomic localization of the perforation site (Figure 3) at the inferoposterior border of the left common vein orifice allowed identification and detailed analysis of the culprit RF lesions (matching the LET maximum).

Similar to a recently reported fatal case,[11] neither RF lesion parameters (energy, LSI, impedance drop, wall contact force) nor LET monitoring suggested a higher risk for esophageal damage (Supplemental Tables 1 and 2). The distance of the ablation lesions to the esophagus has also not shown to be a reliable indicator of safety.[12]

Only routine endoscopic surveillance following PVI detected this major complication; and as reported in another case,[13] closure of the perforation site by endoscopic clips, in

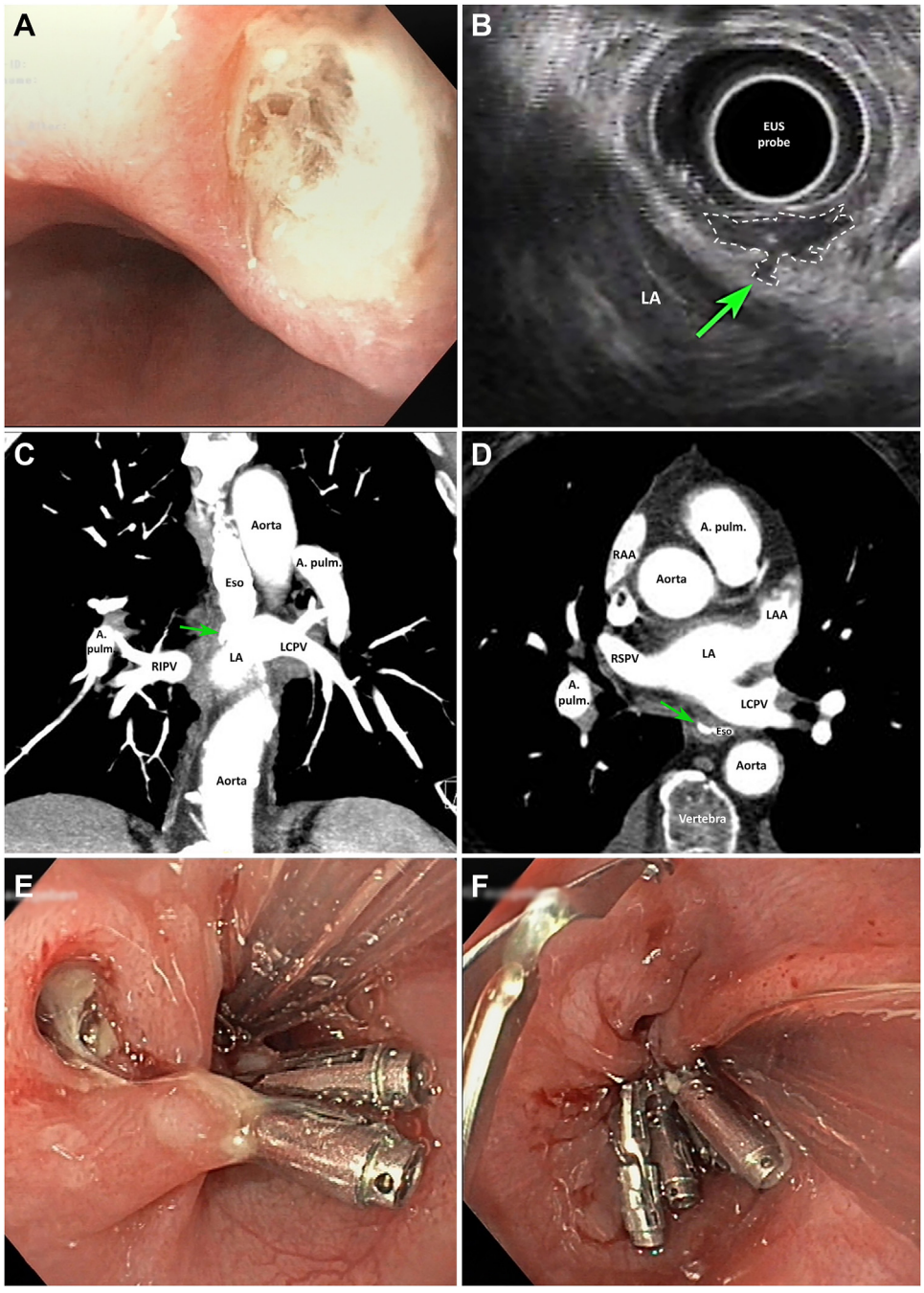

Figure 3 Day 10 following pulmonary vein isolation. Progression of esophageal ulcer to perforation: **A:** perforation site in endoscopy; **B:** edema of the esophageal wall (*dotted line*) and perforation (*green arrow*) visualized by endoscopic ultrasound; **C, D:** computed tomography scan with oral dye extravasate (**C**, horizontal view; **D**, transversal view). **E, F:** Endoscopic clip therapy (after exclusion of fistula and mediastinitis) for endoluminal closure of the perforation site. A. pulm = pulmonary artery; Eso = esophagus; LA = left atrium; LAA = left atrial appendage; LCPV = left common pulmonary vein; RAA = right atrial appendage; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein.

analogy to the albeit different pathophysiological mechanism of perforation by esophageal instrumentation, was sufficient for the healing.

Future directions

Several options to prevent esophageal lesions may be discussed. LET is a poor surrogate of tissue temperatures (in deeper esophageal wall layers), and a precise LET limit has not yet been defined. Whereas excessive LET (eg, >41°C) is associated with esophageal lesions,[3] fistulas have been reported without suspicious LET rise.[14] Restriction of LET to 39°C is an option but is probably not enough to rely on. LETs of 38°C–39°C are very common with RF lesions at the LAPW; thus stopping RF might challenge the long-term efficacy of PVI.

Ablation lesion indices (eg, LSI) have significant limitations, too. Both combining and splitting closely coupled lesions (done automatically by the 3D system) will result in

misleading information (the former with false high values suggesting effective transmural lesions, the latter with false low values suggesting safety and low risk for collateral damage). In addition, LSI becomes visible after 6 seconds and is, therefore, often not helpful when HPSD protocols are used. At first notification, LSI is often already >4.5 (in many electrophysiological centers the target at the LAPW),[15] and shorter lesions will not show any LSI value at all (in the presented case 4 of 10 RF lesions [Supplemental Table 1]).

Esophageal perforation and atrioesophageal fistula are extremely rare. The impact of endoscopy to prevent mucosal lesion progression to perforation still has to be defined. Endoscopy will have to be reserved for individual cases. Unfortunately, indicators for esophageal injury are not yet defined. Perhaps our recently reported tool of the calculated area under the curve of LET[12] (considering the temporal and spatial components in addition to temperature maximum) may be an initial step to limit the need for endoscopic workup.

In a previous study, periesophageal edema but no esophageal lesions were seen with HPSD ablation.[12] In contrast, the same energy protocol resulted in esophageal perforation in the reported case. Whether periesophageal edema plays a role in progression to perforation/fistula or is just an unsuspicious thermic side effect not reaching the mucosa is not well understood. However, tissue edema may well be a surrogate for vascular injury leading to necrosis and may facilitate bacterial invasion and inflammation. Currently, vessel disruption and inflammation are not detectable, and therefore edema should be considered in future studies on lesion progression.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2022.07.005.

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