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Atrial-oesophageal fistula following percutaneous radiofrequency catheter ablation of atrial fibrillation: the risk still persists

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Aims Atrial-oesophageal fistula is a serious complication related to ablation of atrial fibrillation. As its occurrence is rare, there is a great lack of information about their mechanisms, incidence, presentations, and treatment. The objective of this manuscript is to present a series of cases of atrial-oesophageal fistula in Brazil, focusing on incidence, clinical presentation, and follow-up.

Methods and results This is a retrospective multicentre registry of atrial-oesophageal fistula cases that occurred in eight Brazilian centres from 2003 to 2015. Ten cases (0.113%) of atrial-oesophageal fistula were reported in 8863 ablation procedures in the period. Most of the subjects were male (70%) with age 59.6 ± 9.3 years. Eight centres were reference units in atrial fibrillation ablation with an experience over than 200 procedures at the time of fistula occurrence. Oesophageal temperature monitoring was performed in eight cases using coated sensors in six. The first atrial-oesophageal fistula clinical manifestation was typically fever (in six patients), with a median onset time of 16.5 (12–43) days after ablation. There was a delay of 7.8 ± 3.3 days between the first manifestation and the diagnosis in five patients. The treatment was surgical in six cases, clinical in three and stenting in one. Seven patients died (70%) and two developed permanent neurological sequelae.

Conclusion Atrial-oesophageal fistula remains a serious complication following AF ablation despite the incorporation of protective measures and increased technical experience of the groups. The high morbidity and mortality despite the treatment indicates the need to develop adequate preventive strategies.

Keywords Atrial-oesophageal fistula • Atrial fibrillation • Radiofrequency catheter ablation

Introduction

Percutaneous catheter ablation of atrial fibrillation (AF) treatment has emerged as a promising non-pharmacological treatment of this arrhythmia, becoming applied worldwide. Complications, however, related to the procedure are not negligible and atrial-oesophageal fistula (AEF) is one of the most serious of these, due to high morbidity and mortality.[1] Despite the incorporation of different technological resources and strategies over time, aimed to improve oesophageal protection, the incidence of AEF remains apparently stable, the gravity of manifestation remains unchanged, and treatment measures remain controversial.[2–4] Its rare occurrence, approximately one case in 1000–3000 treated patients,[1–4] makes it difficult to study the understanding of AEF physiopathology, identification of the risk factors, the adoption of effective preventive measures, as well as the development of appropriate therapeutic

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What's new?

- In this study, 10 (incidence of 0.113%) cases of atrial-oesophageal fistula (AEF) were identified in 8 Brazilian centres that performed 8863 procedures from 2003 to 2015.
- Two cases (incidence of 1% in 2 years) occurred in 2003 and 2004 when wide PV isolation was introduced and AEF was not recognized as a complication of AF catheter ablation.
- Other eight (incidence of 0.1% from 2005 to 2015) occurred during the subsequent years, after introduction of preventive measures such as reduced power on posterior wall, oesophageal temperature monitoring, PPI and sucralfate use and endoscopy post-ablation.
- Four (incidence of 0.23% in 2014 and 2015) AEF cases occurred in the last 2 years when new technology to perform deeper lesions were introduced.
- There was a significant delay to recognize AEF; six patients underwent surgical repair, one received oesophageal stent, however, just one patient survived without significant neurological sequelae.
- Patients and physicians must be aware to recognize oesophageal lesions early, in order to introduce therapeutic measures that could avoid its progression.

strategies. Given the scarcity of information on this complication, additional data will undoubtedly contribute to a better understanding of this serious problem.

Methods

This is a retrospective descriptive registry, which consisted of documentation and compilation of AEF cases that occurred in eight Brazilian centres in the period between 2003 and 2015. Electronic forms were distributed to the centres that reported this kind of complication, whose diagnosis had been consistently characterized by imaging method.

Electronic forms were distributed, and contained information about demographics, clinic, preoperative preparation, intra-operative and post-operative care. Additionally, we collected specific information on time from clinical manifestations to diagnosis, adopted treatment and final outcome. The patient information was de-identified by each centre after data collection. The first author of this study evaluated the forms; their information was organized, compiled, described and subsequently re-evaluated and validated by the other authors.

Statistical analysis

Shapiro–Wilk's *W* test was used to demonstrate normality of variables. Variables with normal distribution are presented as mean ± SD and variables with non-normal distribution are presented as median and range. Nominal variables were presented as absolute number (*n*).

Results

Demographic, clinical and echocardiographic characteristics

Clinical characteristics of the patients are presented in *Table 1*. This series consisted of 10 patients that were diagnosed with AEF after AF ablation in 8 Brazilian centres between the years 2003 and 2015. In this period, 8863 AF ablations were performed in the centres, and the incidence of AEF was 0.113% in this population. The cases occurred in the years 2003, 2004, 2008, 2011 (two cases), 2012, 2014 (two cases), and 2015 (two cases). The incidence of AEF has not changed over the years (*Figure 1*). All patients who developed AEF were white, seven were men, with a mean age of 59.6 ± 9.3. There was no evidence of structural heart disease in any case and the mean left atria diameter was 38 ± 5 mm. Atrial fibrillation was paroxysmal in six and persistent in four cases, with the chronicity of AF ranging from 12 to 216 months. Two patients had prior AF ablation (11 months and 4 years before).

Operative characteristics of the cases

All the procedures were performed with the patient under general anaesthesia. *Table 2* shows data from the ablation of each patient. Only one patient received proton pump inhibitor prior to the ablation. The ablation was performed using 8 mm tip catheter in three patients, and the remaining procedures were performed with irrigated tip catheters (including two with a contact force sensor). Electroanatomic mapping was used in seven cases and intracardiac echocardiography in four cases. In all patients, pulmonary vein ostia were targeted. Complementary lines of ablation in the left atrium were made in three patients, two with roof and posterior wall lines and another one exclusively on the roof. Empirical reduction of radiofrequency (RF) power during the posterior wall ablation was used in three patients with upper limit set to 25 W. Radiofrequency power delivery was based on the oesophageal temperature monitoring in the other seven cases.

Postoperative characteristics

Postoperative oesophageal protection was performed in all cases with proton pump inhibitor with the addition of sucralfate in three cases (*Table 3*). Early clinical manifestations suggestive of oesophageal damage occurred in two patients within the first 48 h following the ablation procedure. The symptoms reported were odynophagia, feeling of fullness, and chest pain. No oesophageal diagnostic evaluation was performed before symptom onset in nine patients during this period, and in one patient endoscopy was performed 8 and 14 days after ablation (*Figure 2*) due to oesophageal temperature increase during ablation (*Table 4*).

The first clinical manifestation of AEF was fever in six patients (60%), associated with chest pain in two cases. In the four remaining patients, the initial manifestation was isolated chest pain, haematemesis, dysphagia, and chest pain, respectively. The median time between the ablation and the first clinical manifestation of AEF was 16.5 days (range 12–43). Immediate investigation and concomitant diagnostic imaging was performed in five patients. In the remaining five, there was a delay of 7.8 ± 3.3 days between the first clinical manifestation and the definitive diagnosis of AEF. Neurological manifestations occurred in nine patients. Specific manifestations were variable, including seizures, syncope, paresis, hemiplegia, and coma.

Imaging methods used to diagnose AEF were computed tomography (CT) scan in six patients and magnetic resonance imaging (MRI) in two. The appearance of air in the mediastinum and/or inside the cardiac chambers confirmed the diagnosis in seven patients (*Figure 3*). In one case, the MRI performed with oesophageal contrast allowed to view the extravasation of contrast into the

Table 1 Clinical characteristics of patients with AEF

Case	Year	Age (years)	Gender	BMI	Heart disease	Comorbidities	AF classification	AF evolution time (months)	Previous ablations	LA diameter (mm)	LA volume (mL/m^2)	LVEF (%)
1	2003	72	M	28.3	Absent	Absent	Paroxysmal	24	No	35	–	Normal
2	2004	71	F	22.2	Absent	Hypertension	Persistent	216	No	41	–	62
3	2008	66	F	20.3	Absent	Pulmonary emphysema, Sjogren's syndrome	Paroxysmal	24	No	31	–	73
4	2011	60	M	–	Absent	Hypothyroidism	Paroxysmal	N/A	No	32	–	69
5	2011	61	M	–	Absent	Sleep apnoea	Paroxysmal	N/A	No	40	–	74
6	2012	50	F	22.5	Absent	Absent	Persistent	12	No	44	35	70
7	2014	41	M	26.5	Absent	Absent	Persistent	24	No	31	35	57
8	2014	61	M	26	Absent	Absent	Paroxysmal	15	Yes	42	–	63
9	2015	58	M	–	Absent	Hypertension	Paroxysmal	36	No	41	–	–
10	2015	56	M	37.2	Absent	Hypertension, obesity	Persistent	96	Yes	45	–	62

BMI, body mass index; LA, left atrium; LVEF, left ventricular ejection fraction.

pericardium (case 3: pericardio-oesophageal fistula). The diagnosis was made by upper gastrointestinal endoscopy for haematemesis investigation in the two remaining patients. The first patient (case 1) in the series, held in 2003,[4] the AEF was diagnosed prior to the first AEF report. The other case (case 7) corresponded to a patient admitted 28 days after ablation due to extensive stroke with development of haematemesis. Endoscopy without gas inflation was performed; the patient underwent open-chest surgery allowing the visualization of a large lesion on the left atrium posterior wall. Interestingly these two patients had undergone MRI (case 1) and CT (case 7) on the 10th and 2nd days after ablation, respectively, due to respiratory distress; retrospective review of the images showed no evidence of oesophageal damage in those moments. The type of fistula was atrio-oesophageal in nine cases and esophago-pericardium in one patient (case 3), with no radiological or surgical evidence of left atrial involvement.

The treatment was surgical in six cases, non-surgical in three and oesophageal stenting in one (*Figure 4*). The surgical treatment involved a suture of left atrium in two patients, plus the oesophagus with additional interposition of intercostal muscle or pericardial membrane patch between both structures in four (*Figure 4A*). One patient (*Figure 4B*) had postoperative re-fistula treated with an oesophageal stent. Non-surgical, clinical treatment corresponded to general measures such as hydration, antibiotic therapy, intravenous proton pump inhibitors administration, chock treatment, and ventilatory support. One recent patient (case 9, 2015) was treated primarily by oesophageal stenting. Interesting, endoscopy performed 6 weeks after stent implantation showed complete sealing of the oesophageal lesion. Unfortunately, this patient developed severe neurologic sequelae and ultimately died due to pulmonary infection. Time to hospital discharge or death ranged from 1 to 76 days, median of 16 days. Seven patients (70%) died in a median time of 4 days (range: 1–76 days) after ablation. From the three patients that survived, two developed permanent neurological sequelae, but were able to be discharged in a median time of 30 days (range: 30–60).

Discussion

Since the first reports in the middle of last decade,[5,6] AEF was established as the most dramatic and feared complication of AF ablation, despite the rarity of its occurrence, because of its unpredictability, severity, and difficult management. The information presented in the existing literature has been confined to case reports, international multicentre registries and studies that compiled data from published or unpublished case reports, seeking to identify the best therapeutic strategy.[1–4,7,8] Due to the low incidence of AEF, several studies have attempted to identify risk factors not for the occurrence of AEF, but also for the occurrence of oesophageal injury, on the assumption that this functions as a precursor to future occurrence of AEF.

Several markers of oesophageal damage have been previously suggested, including endoluminal temperature levels, a low body mass index, use of general anaesthesia in interventions, ablation technique, power employed on the posterior wall, functional gastroesophageal changes induced by ablation, and others.[9–13] The identification of such markers has implicated in adoption of

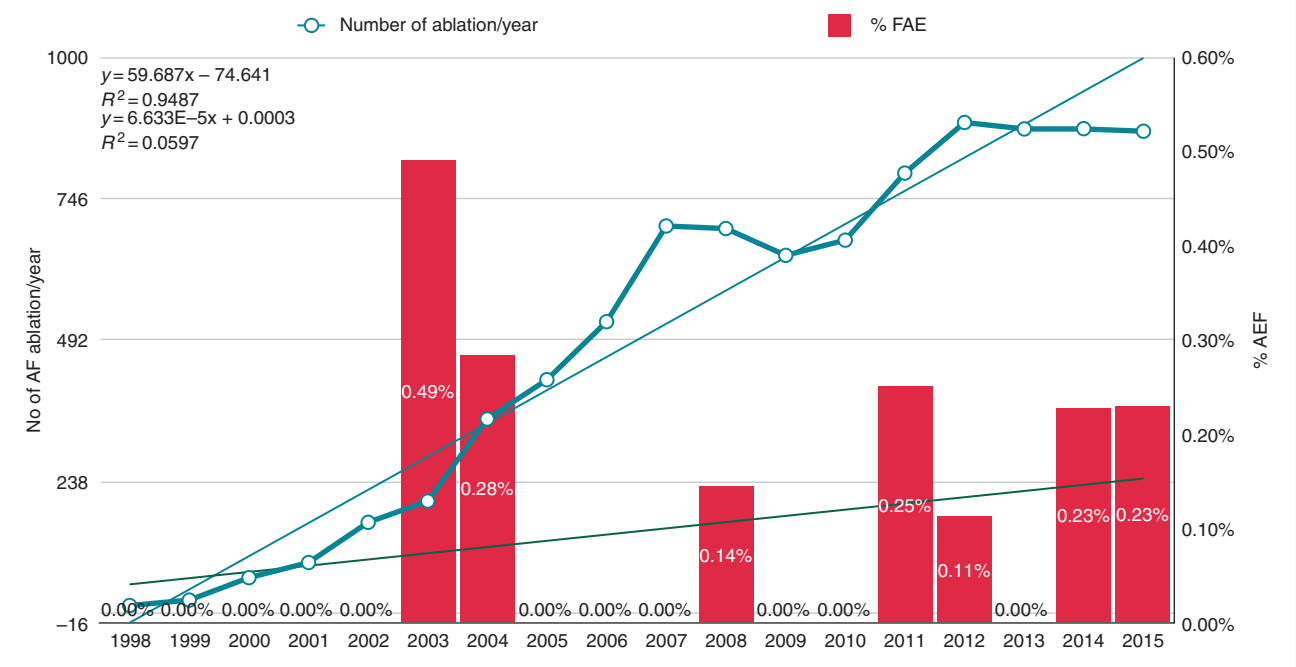

Figure 1 Incidence of atrioesophageal fistula in the different years of this series. Blue line represents the number of AF ablation procedures performed in the eight centres. Blue (R^2: 0.94) and yellow (R^2: 0.06) lines represent trendline of number of procedures and incidence of AEF, respectively.

oesophageal protective measures, such as preventive use of proton pump inhibitors in pre and postoperative period, use of oesophageal cooling during ablation, oesophageal temperature monitoring, power reduction in the ablation of the posterior wall and use of simple sedation during the procedures[9,11,13–16] It is important to emphasize that the decision for taking each of these measures is empirical, as no evidence of benefit has been demonstrated in any prospective study, and controversial. For example, while monitoring of oesophageal temperature is expected to predict thermal injury to the oesophagus, several recent studies suggest that some temperature probes used for monitoring are related to an increased risk of oesophageal injury.[17,18]

The incidence of AEF has been variably reported as 0.04% in a global survey including 16 309 patients,[19] 0.03% in a cohort of 20 425 patients,[20] and 0.011% in a recent survey including 191 215 AF ablation procedures.[4] The incidence of AEF in our population was 0.113%, which was higher than previously reported surveys. One possible explanation is that only centres that report complications such as AEF were selected, and that questionnaire-based surveys may under-report AEF incidence. Another is that all groups involved in this survey used the same strategy approaching the antra of PVs. This is an important point since no AEF cases had been described when PVI was performed into the PV ostia (segmental isolation).

In the evaluation of our results it is clear that despite the technological progress in AF ablation over these 12 years, incorporating sophisticated features to ablation procedures for the treatment of AF, such as electroanatomic mapping, intracardiac echocardiography, catheters with contact force sensors and the adoption of oesophageal protective measures, such as luminal temperature monitoring, RF power reduction on the posterior wall, use of proton pump inhibitors with or without sucralfate, AEF is still occurs, with high morbidity and mortality rate. Interestingly, 40% of the cases occurred in the recent 2 years, probably related to an increase in the number of ablation procedures (*Figure 1*).

In this series of 10 patients, only one survived without serious sequelae. This finding is in agreement with previous reports that describe a high mortality and morbidity related to AEF.[1–4,7,8,19,20] Ideally, factors associated with poor outcomes with AEF would be identified. Two of the 10 patients (20%) had symptoms potentially associated to the oesophageal damage in the first 48 h after ablation. It is possible that a more aggressive investigation of these cases at this stage, for example through endoscopy could identify oesophageal injury, resulting in treatment and follow-up measures to intervene on the natural history of the condition. However, we must bear in mind two aspects: (i) symptoms occurring the first hours after recovery from an ablation for AF have several alternate potential aetiologies; (ii) it is unknown if early oesophageal injury is related to the risk of fistula. Noteworthy is the fact that the case 7 underwent chest tomography (CT) in the second postoperative day for unrelated reasons, which did not identify peri-oesophageal or mediastinal changes suspicious for oesophageal injury. This indicates a slow and insidious pathophysiological mechanism. It is possible that endoscopic ultrasound, as a sensitive, simple and low cost diagnostic test, could be used to visualize not only the oesophageal integrity but also the surrounding tissues.[21]

An important aspect in this series is that in five patients there was a delay of 7.8 $\pm$ 3.3 days between the first clinical manifestation and the confirmation of the diagnosis. Perhaps, earlier identification of

Table 2 AF ablation characteristics

Case	Pharmacological oesophageal protection prior to ablation	EAM	ICE	Ablation technique	Roof line	Posterior line	Catheter	Power reduction in posterior wall	Oesophagus temperature monitoring probe	Irrigation rate during ablation (mL/min)	PPI use following ablation	Sucralfate use following ablation
1	No	No	No	Circumferential	No	No	8 mm	No	Not employed	–	Yes	No
2	No	Yes	No	Circumferential	Yes	Yes	8 mm	No	Not employed	–	Yes	No
3	Yes	No	No	Circumferential	No	No	Irrigated	Yes 25 W	Single Sensor Coated	17	Yes	No
4	No	Yes	No	Circumferential	No	No	Irrigated	No	Single Sensor Coated	17	Yes	No
5	No	Yes	No	Circumferential	No	No	Irrigated	No	Single Sensor Coated	17	Yes	No
6	No	Yes	Yes	Circumferential	Yes	No	Irrigated	Yes 25 W	Multiple Sensors Coated	30	Yes	No
7	No	Yes	Yes	Circumferential	No	No	Irrigated	No	Pre-shaped Multiple Sensors Uncoated	30	Yes	No
8	No	Yes	Yes	Circumferential	No	No	Irrigated tip with contact force sensor	No	Single Sensor Uncoated	17	Yes	Yes
9	No	Yes	Yes	Circumferential	No	No	Irrigated tip with contact force sensor	No	Single Sensor Coated	17	Yes	Yes
10	Yes	No	No	Circumferential + Box	Yes	Yes	8 mm	Yes 25 W	Single Sensor Coated	–	Yes	Yes

EAM, electroanatomical mapping; ICE, intracardiac echocardiography; PPI, proton pump inhibitor.

Table 3 Post ablation characteristics

Case	Early symptoms of oesophagus damage	AEF clinical manifestations	First AEF clinical manifestation	Time to initial clinical manifestation of AEF (days)	Time to definitive diagnosis of AEF (days)	Delay in diagnosis (Days)	AEF diagnosis confirmation method	Fistula type	Treatment	Clinical conditions at the time of treatment choice	Treatment description
1	No	Fever, haematemesis, neurological	Fever	14	22	8	UGE	Atrio-oesophageal	Clinical	Critical (Shock)	Clinical and hemodynamic support
2	No	Fever, neurological	Fever	14	14		MRI	Atrio-oesophageal	Surgical	Unstable	Oesophagus and left atrium repair, intercostal muscle interposition
3	Yes	Fever, chest pain, shoulder pain	Chest pain	21	21		MRI with oesophageal contrast	Oesophagus-Pericardium	Surgical	Unstable	Oesophagus repair with bovine pericardial patch, left atrium repair
4	No	Fever, chest pain, neurological	Fever	18	18		CT	Atrio-oesophageal	Surgical	Critical (Coma and shock)	Left atrium repair
5	No	Haematemesis, Neurological	Haematemesis	43	43		CT	Atrio-oesophageal	Clinical	Stable	Clinical and hemodynamic support, enteral nutrition, antibiotic therapy
6	No	Fever, chest pain, neurological	Fever Chest pain	14	21	7	CT	Atrio-oesophageal	Surgical	Unstable	Oesophagus repair with bovine pericardial patch, left atrium repair
7	Yes	Fever, haematemesis, chest pain, neurological	Fever Chest pain	12	16	4	UGE	Atrio-oesophageal	Surgical	Critical (Shock)	Left atrium repair
8	No	Dysphagia, fever, neurological	Dysphagia	15	28	13	CT	Atrio-oesophageal	Clinical	Critical (Coma and shock)	Clinical and hemodynamic support
9	No	Fever, chest pain, neurological	Chest pain	21	28	7	CT	Atrio-oesophageal	Stenting	Unstable	Oesophagus stent implantation
10	No	Fever, neurological, septic shock	Fever	24	24		CT	Atrio-oesophageal	Surgical	Critical (coma and shock)	Oesophagus repair with bovine pericardial patch, left atrium repair

UGE, upper gastrointestinal endoscopy; MRI, magnetic resonance imaging; CT, computed tomography.

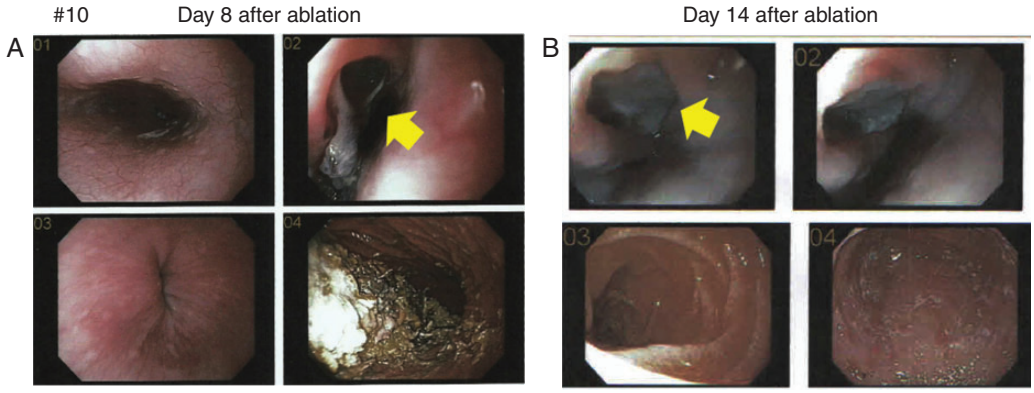

Figure 2 Endoscopy performed in patient 10. In (*A*), on endoscopy performed 8 days after ablation, there is a large dark lesion on oesophageal anterior wall (yellow arrow), that is also present in the endoscopy performed 14 days after ablation (*B*).

Table 4 Outcome after AEF diagnosis and treatment

Case	Hospital stay time (days)	Time to death	Hospital stay time (days)	Final outcome	Neurological sequelae
1	3	3		Death	
2	60		60	Discharge	Yes
3	28	28		Death	
4	4	4		Death	
5	30		30	Discharge	Yes
6	60		60	Discharge	No
7	1	1		Death	
8	4	4		Death	
9	76	76	76	Death	
10	3	3		Death	

AEF after symptom onset could have changed the prognosis of these patients. It is important to consider the underestimation of symptoms by the patient could contribute to a delayed diagnosis. In this sense, it is possible that a system of active monitoring for symptoms in the first 6 weeks after ablation may assist in the early diagnosis of AEF. Fever was the most consistent clinical feature, appearing as the first manifestation in six patients and a component of the clinical syndrome in nine patients. One of the cases of this series had extremely late clinical occurrence of symptoms (43 days), highlighting the importance of careful observation for those symptoms for a period greater than 1 month.

Despite the lack of consistent information in the literature about the best type of treatment, some observational studies suggest that early surgical treatment of AEF directed towards the left atrium and to oesophageal repair with separation of these structures (either by a muscle flap or by pericardial patch) can lower morbidity and mortality.[7,8] In our series, surgical treatment was adopted in six patients; two underwent left atrial repair, and four underwent left atrial and oesophageal suture repair with interposition of an intercostal muscle or pericardial patch between the structures. Just two patients in the surgical group survived. Also, only one patient survived when a non-surgical approach was used (out of four patients in this group). However, we must keep in mind one important aspect: most of the patients in this series presented with neurological and hemodynamic compromise (including shock or coma), and treatment assignment was at the discretion of the treating physician after clinical evaluation. Thus, it is possible that the high mortality rate with or without intervention is not related to treatment modality, but reflects poor patient prognosis at the time of diagnosis and therapeutic decision-making.

Limitations

This is a retrospective compilation of cases of AEF and some important information regarding clinical history and procedures may be missing or inaccurate. Additionally, other patients could have presented this complication without the knowledge of the authors. However, AEF often has a fatal outcome and is frequently reported by the family. Thus, we do not expect to have missing cases of AEF in this population. As a compilation of cases, it is not possible to identify predictors of the occurrence of atrial-oesophageal fistula. Not all Brazilian centres that perform AF catheter ablation were

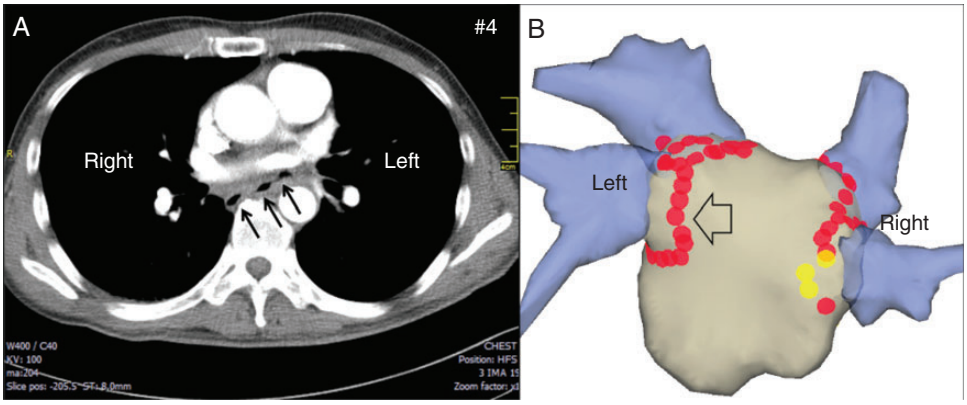

Figure 3 Images from patient 4 of the series. (*A*) Chest CT image demonstrating the presence of AEF outlined by air propagating from oesophagus towards left inferior pulmonary vein antrum (arrows). Note that fistula trajectory from the left atrium is orthogonal to the oesophagus position. (*B*) Electroanatomic mapping image (posterior view) obtained during ablation. Spherical markers identify isolation lines around pulmonary veins antra. In this case, the region in which oesophageal temperature increased (yellow markers) does not correspond to the left atrium region where AEF formed (open arrow).

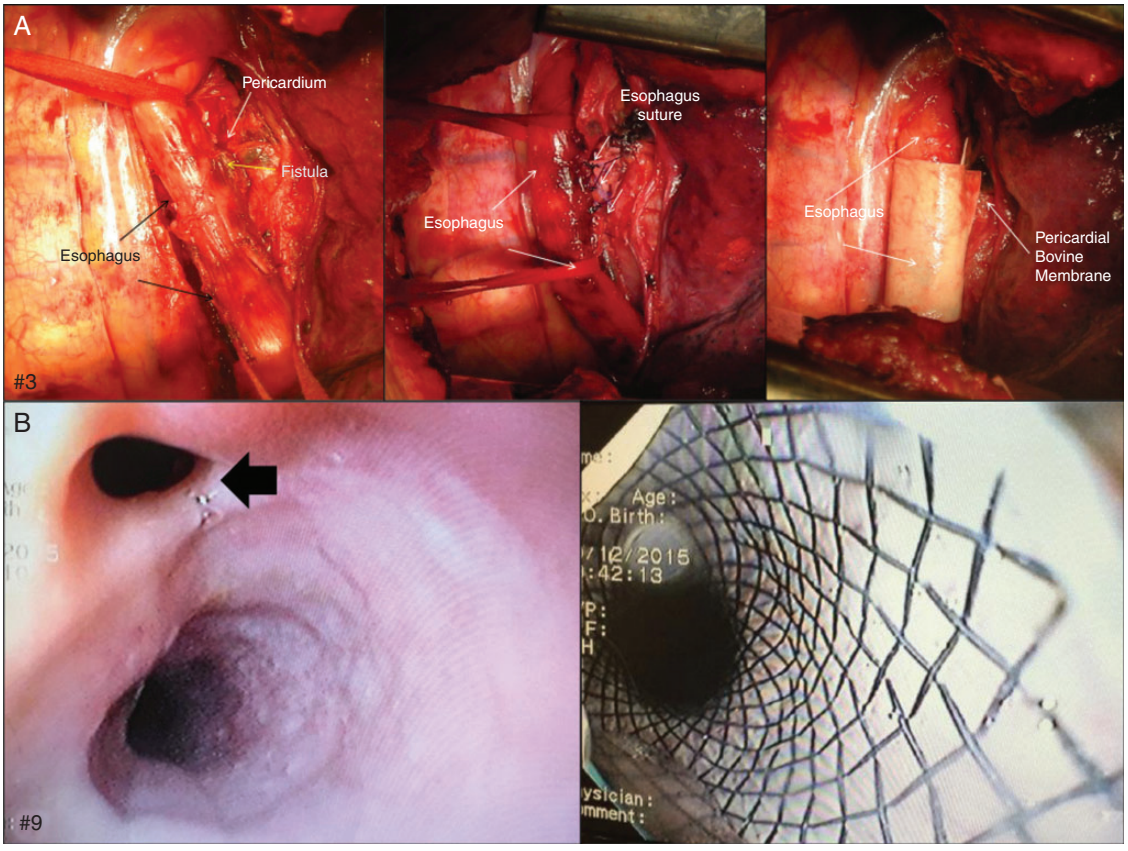

Figure 4 Treatment of oesophageal fistula: (*A*) Surgical procedure to correct the fistula in case 3. On the left: Open chest surgical view of oesophageal pericardial fistula; On the middle: oesophagus and pericardial sutures; On the right: bovine pericardial sheath interposed between oesophagus and pericardial sutures. (*B*) Oesophageal stenting: Upper gastrointestinal endoscopy images obtained from the case 9. On the left: Lower oesophagus shown prior to stent implantation demonstrating the large fistula orifice (black arrow). On the right: Final oesophagus appearance following stent implantation.

contacted, but the experienced centres that recognized such complication.

Conclusion

Despite the incorporation of protective measures and increased technical experience of the groups, AEF still occurs, and presents challenges in terms of prevention, diagnosis, and treatment, with high morbidity and mortality rate. Those aspects indicate the need to develop adequate preventive strategies to avoid oesophageal damage, to make early diagnosis if it occurs and to take preventive measures to avoid its progress to AEF manifestation.

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