



Review

Gastrointestinal complications associated with catheter ablation for atrial fibrillation



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ABSTRACT

Atrial fibrillation is the most common arrhythmia in the United States. With the ageing population, the incidence and prevalence of atrial fibrillation are on the rise. Catheter ablation of atrial fibrillation is a widely accepted treatment modality in patients with drug refractory symptomatic paroxysmal or persistent atrial fibrillation. The close proximity to the left atrium posterior wall makes the thermosensitive esophagus a potential site of injury during catheter ablation of AF leading to various gastrointestinal complications. The major gastrointestinal complications associated with catheter ablation include atri-esophageal fistula, gastroparesis, esophageal thermal lesions and esophageal ulcers. Multiple studies, case reports and series have described these complications with various catheter ablation techniques such as radiofrequency, cryoenergy and high frequency focused ultrasound energy ablation. This review addresses the gastrointestinal complications after AF ablation procedures and aims to provide the clinicians with an overview of clinical presentation, etiology, pathogenesis, prevention and management of these conditions.

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1. Introduction

Atrial fibrillation is the most common arrhythmia in the United States. With the ageing population, the incidence and prevalence of atrial fibrillation are on the rise. Catheter ablation of atrial fibrillation (AF) is a widely accepted treatment modality in patients with drug refractory symptomatic paroxysmal or persistent atrial fibrillation [1]. Since Haïssaguerre and coworkers' description of the role of pulmonary veins in atrial fibrillation [2], catheter ablation strategies have gradually advanced to incorporate newer techniques. Pulmonary vein isolation is the most common strategy practiced worldwide with variable success [3–5]. Numerous studies have demonstrated the safety, feasibility and short to medium-term effectiveness of catheter ablation for AF [6–8].

The close proximity to the left atrial posterior wall makes the thermosensitive esophagus a potential site of injury during catheter ablation of AF leading to various gastrointestinal complications [9–13]. The

major gastrointestinal complications associated with catheter ablation include atri-esophageal fistula (AEF), gastroparesis, esophageal thermal lesions and esophageal ulcers. Multiple studies, case reports and series have described these complications with various catheter ablation techniques such as radiofrequency, cryoenergy and high frequency focused ultrasound energy ablation. This review addresses the gastrointestinal complications after AF ablation procedures and aims to provide the clinicians with an overview of clinical presentation, etiology, pathogenesis, prevention and management of these conditions.

2. Esophageal lesions and ulcers (Table 1)

The distance between the esophagus and left atrium posterior wall endocardium is less than 5 mm in 40% of population [13]. During AF ablation, high temperatures are achieved to electrically isolate the arrhythmogenic foci [14]. This may potential cause damage to the esophagus; ranging from mild erythema to ulceration and in rare instances can lead to esophageal perforation and AEF formation.

Studies have revealed varying incidence of esophageal injury after catheter ablation ranging from 2%–47% [15–24,44]. Contreras-Valdes et al. reported that 37.4% patients had luminal esophageal temperatures (LET) > 39 °C, of which 10% patients had esophageal injury [15]. Halm

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Table 1
Summary of studies involving thermal esophageal lesions/injuries.

Study name	Number of patients	Ablation strategy protocol	Diagnosis modality	Results
Contreas-Valdes et al.	219	Power \leq 25 W energy interrupted if LET $>$ 38 °C	EGD only if LET $>$ 39 °C within 24 h of ablation	37.4% patients (n = 82) underwent EGD, 22 patients had esophageal injuries
Halm et al.	185	Power = 30–50 W (30 W if closer to esophagus)	EGD in all patients within 1–4 days after ablation.	14.6% patients (n = 27) had asymptomatic esophageal lesions.
Di Biase et al.	88	Power: 30–45 W (30 W closer to esophagus) Energy interrupted if LET $>$ 39 °C.	Capsule endoscopy within 24–48 h of ablation.	No endoscopic evidence in patients with LET \leq 41 °C. 17% (n = 15) patients had esophageal lesions. Use of general anesthesia was associated with more esophageal lesions than conscious sedation (48 versus 4%, $p < 0.001$).
Martinek et al.	175	Power: 15–30 W	EGD within 24 h of procedure.	2.9% patients (n = 5) developed esophageal lesions. Use of general anesthesia was not associated with increased risk as compared to conscious sedation (8.6% versus 1.45%, $p = 0.055$)
Yamasaki et al.	104	Power \leq 25 W	EGD within 16–48 h of ablation procedure	9.6% patients (n = 10) had esophageal thermal injuries (2 = esophageal erythema, 2 = esophageal ulcerations and 6 = gastric hypomotility). All patients had BMI $<$ 24.9.
Kiuchi et al.	20	Power: 20–25 W energy interrupted if LET $>$ 39 °C	EGD within 1–5 days after ablation	None of the patients had esophageal mucosal injuries.

LET = luminal esophageal temperature, EGD = esophagogastroduodenoscopy.

and colleagues found that 15% patients had asymptomatic esophageal injuries (lesion size 2–16 mm) with no endoscopic evidence of esophageal injury in patients with LET less than 41 °C and increased odds of mucosal injury for every 1 °C increase in LET over 41 °C [16].

Di Biase and colleagues demonstrated that 17% (15/88) patients had evidence of esophageal mucosal injury on capsule endoscopy, of which 6 patients were symptomatic [17]. The incidence of esophageal injury was higher in patients undergoing AF ablation under general anesthesia (GA) versus conscious sedation (48% versus 4%, respectively; $p < 0.001$). The authors suggested that reduced esophageal peristaltic movement and lack of swallowing along with fixation of esophagus due to nasogastric tube were responsible for increased incidence of esophageal injury in GA group [19]. Martinek et al. demonstrated that higher radiofrequency energy ablation strategy (25 W compared to 15 W) was associated with increased risk of esophageal injury [18]. However, in contrast to Di Biase et al. findings, Martinek et al. demonstrated no significant difference in the incidence of esophageal injury with GA arm versus conscious sedation arm (2.7% versus 2.2%, respectively; $p = 0.86$) [20].

Yamasaki et al. reported that 9.6% patients (10/104) had esophageal injury or periesophageal nerve injury after AF ablation under conscious sedation with maximum energy of 20–25 W. The authors suggested that thinner the patients (all patients who had esophageal injury had body mass index $<$ 24.9), less esophageal and left atrial connective tissue, higher the risk of mucosal injury [23]. On the contrary, Kiuchi et al. demonstrated that, of 20 patients enrolled in their trial with BMI $<$ 24.9 who underwent AF ablation with a 20 W energy (target LET $<$ 39 °C), none had any evidence of esophageal mucosal injury, thereby suggesting that low power delivery (target LET $<$ 39 °C) as a preventive strategy in patients with low BMI [24].

3. Upper gastrointestinal dysmotility (Table 2)

Upper gastrointestinal (UGI) motility complications resulting from AF ablation include gastroesophageal reflux, pyloric spasm, and gastric hypomotility [19,21,25,26]. The exact mechanism of these injuries is not known, however injury to vagus nerve and its branches is the proposed mechanism. The vagus nerve because of its close proximity to esophagus and LA wall is susceptible to direct injury from AF catheter ablation. There have been numerous case reports and observational studies to understand the effect of catheter ablation on upper GI motility [25–30]. Interestingly, most of the UGI hypomotility seems to be sub-clinical and often diagnosed incidentally on further testing. Shah et al. reported an earliest case of acute pyloric spasm and gastric hypomotility after radiofrequency ablation of AF [25]. Bunch et al. reported two cases of gastroparesis and pyloric spasm 12 h after AF ablation suggesting vagal nerve injury and emphasizing the need for early diagnosis to minimize excessive weight loss [28]. Guiot and colleagues reported that 9% patients who underwent cryoablation developed asymptomatic gastroparesis [29]. Aksu et al. reported that 10% (6/58) patients who underwent cryoballoon ablation developed symptomatic gastroparesis as compared to 2% (1/46) patients who underwent radiofrequency ablation [30].

Lakkireddy and colleagues performed the only prospective, observational study assessing the effect of AF ablation on UGI motility. They enrolled 27 patients who underwent esophageal manometry, gastric emptying study, and sham feeding study before ablation and subsequently at 24 h, 3 months and 6 months. They reported that 74% developed at least one new abnormality on UGI functional testing 24 h post ablation, that eventually normalized in all patients at 6 months follow-up [27].

Table 2
Summary of studies involving upper gastrointestinal hypomotility.

Study name	Number of patients	Ablation strategy protocol	Diagnosis modality	Results
Guiot et al.	66	Cryoablation	24 Holtor, echocardiography and EGD before discharge	6 patients: gastroparesis 5 patients: Transient phrenic nerve palsy.
Aksu et al.	58: cryoablation arm (group 1) 46: radiofrequency ablation (group 2)	Cryoenergy: 240 s for 2 cycles. Radiofrequency ablation: 20–25 W	Gastric emptying scintigraphy in symptomatic patients after ablation	Group 1: 6 patients gastroparesis Group 2: 1 patient gastroparesis.
Lakkireddy et al.	27	Radiofrequency ablation Power: 30 W on posterior wall and 40 W on anterior wall Energy interrupted if LET $>$ 39 °C	Esophageal manometry, gastric emptying study and sham feeding test	20 patients had at least 1 abnormality on UGI function test.

LET = luminal esophageal temperature, EGD = esophagogastroduodenoscopy, UGI = upper gastrointestinal.

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