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FROM FLUTTERING HEARTS TO RESTLESS STOMACH: NAVIGATING ATRIAL FIBRILLATION, ABLATION AND GASTROPARESIS

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ABSTRACT

Background: We present a case of severe gastroparesis with pyloric spasm secondary to radiofrequency catheter ablation (RFCA) with a focus on procedural risk factors, prevention, and treatment modalities.

Case report: A 39-year-old man with paroxysmal atrial fibrillation underwent RFCA after failed pharmaceutical treatment. The procedure involved three-dimensional mapping and controlled RFCA near the oesophagus. Post-procedure, he developed severe abdominal pain, distention, and vomiting. Imaging revealed stomach distention and pyloric spasm. An upper endoscopy confirmed gastroparesis due to ablation. Metoclopramide was administered, improving food tolerance. However, persistent symptoms led to a gastric emptying study showing delayed emptying, consistent with gastroparesis. Despite treatment with metoclopramide, the patient's symptoms persisted, prompting consideration of esophageal botulinum toxin and medication changes for symptom management.

Conclusions: In a patient with no prior history of abdominal symptoms or chronic gastric disorder post ablation presentation of new-onset abdominal distention, pain, and vomiting ablation-induced gastroparesis should be high on the differential and different treatment modalities should be tried if severe symptoms such as postprandial regurgitation persist. Moreover, better oesophageal protection strategies should be implemented, and guidelines be added to procedure performance limiting excessive force, energy, manipulation, and introduction of oesophageal temperature control holding parameters.

KEYWORDS

Ablation, gastroparesis, atrial fibrillation

LEARNING POINTS

- Gastroparesis can be an under-recognized complication of left atrial ablation.
- Posterior wall ablation near the oesophagus is high-risk for gastrointestinal complications.
- Symptoms usually persist for 3-6 months but may last long term.

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INTRODUCTION/BACKGROUND

Atrial fibrillation is the most common arrhythmia worldwide radiofrequency ablation is the treatment of choice for drug-resistant atrial fibrillation however like most invasive interventions post-procedure complications are a risk. In this case, we present a case of severe gastroparesis with pyloric spasm secondary to radiofrequency catheter ablation (RFCA) with a focus on procedural risk factors, prevention, and treatment modalities.

CASE DESCRIPTION

A 39-year-old male with a past medical history of hyperlipidaemia, obstructive sleep apnoea, non-occlusive coronary artery disease, and paroxysmal atrial fibrillation (PAF) underwent RFCA for his paroxysmal atrial fibrillation after the failure of pharmaceutical treatment. The patient was diagnosed with PAF in 2017 with a CHA^2DS^2-VASc score of 0. He was treated with diltiazem and dronedarone and underwent cardioversion once in 2022. Due to his uncontrolled rate, RFCA was performed.

Eight-French sheaths at the right femoral veins were used for access, and transseptal punctures were performed under fluoroscopic and echocardiographic guidance. Three-dimensional mapping of the left atrium, and pulmonary veins was carried out. Wide area circumferential ablation was performed. RFCA was delivered to left atrium using 3.5 mm tip catheter with an average contact force of 10 grams over the period of 30 seconds at every position. RF was delivered in parts of the left atrium near the oesophagus. Delivery was terminated to allow for oesophageal cooling whenever oesophageal temperature increased by 0.5°C. Total number of energy applications was 38.

Eighteen hours later the patient reported to the emergency department (ED) with complaints of abdominal pain, distention, and vomiting. Per the patient approximately 6 hours after the procedure he could not tolerate any oral intake and gradually the abdominal pain increased. In the ED, the patient was afebrile, in sinus tachycardia with abdominal distention and epigastric tenderness on exam. A computed tomography (CT) scan of the abdomen and pelvis revealed moderate severe distention of the stomach with gas and fluid, the patient was admitted. CT scan with contrast revealed marked distention of the stomach with a transition point at the pylorus. Surgery was consulted and esophagogastroduodenoscopy (EGD) was performed revealing severe pyloric spasm and food retention *(Fig. 1)*. The patient was diagnosed with gastroparesis secondary to ablation and was started on metoclopramide, eventually was able to tolerate oral food and was to be followed, outpatient. The patient was treated for gastric spasm as an outpatient however he continued to experience epigastric pain, postprandial regurgitation, and heartburn. Cardiac causes were ruled out and a gastric emptying study (GES) was performed. The patient did not have any history of such abdominal pain prior to the procedure. The GES *(Fig. 2)* revealed retention of 60 minutes-93% (normal 37-90%),

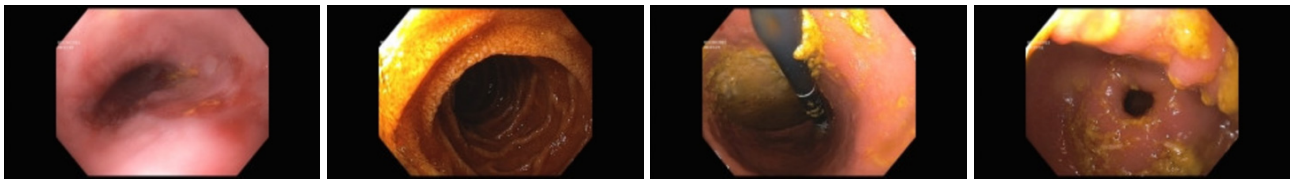


Figure 1. Esophagogastroduodenoscopy showing retention.

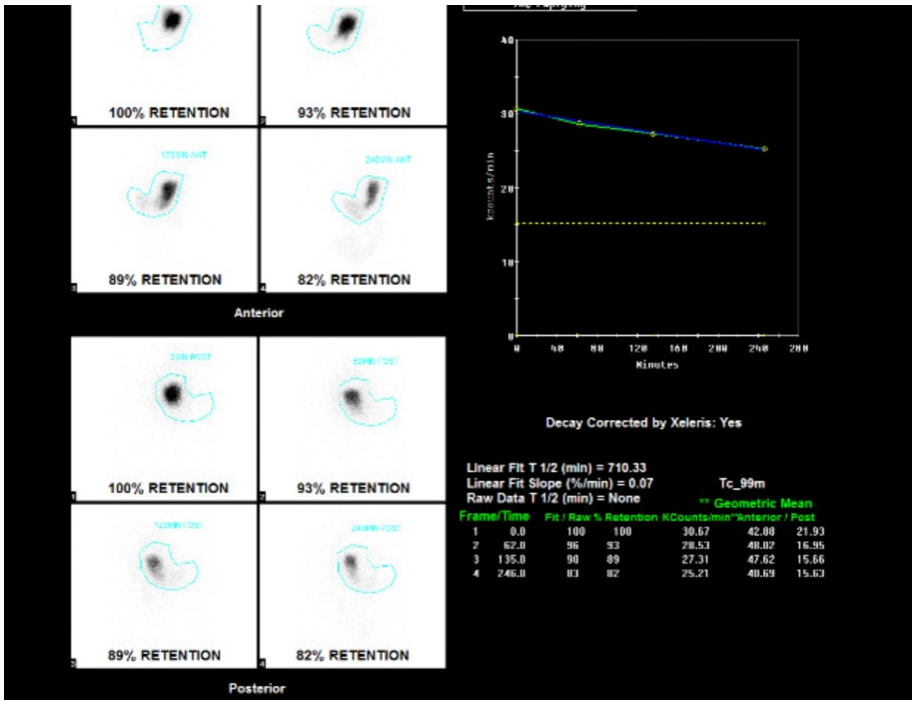


Figure 2. Gastric emptying study showing paresis.

120 minutes-89% (normal 30-60%), and 240 minutes-82% (normal 0-10%) consistent with gastroparesis.

The patient continued to experience post-prandial regurgitation, not well controlled with metoclopramide alone. The gastroenterologist considered oesophageal botox and changing the prokinetic however the patient refused the continued metoclopramide and pantoprazole and now his symptoms only occur occasionally.

DISCUSSION

Severe gastroparesis following ablation is a relatively uncommon complication. Some authors have called it atrial fibrillation gut syndrome. This case report identifies potential risk factors for onset and treatment methodologies for patients with unresolved symptoms.

Gastroparesis is a condition characterized by delayed gastric emptying into the small intestine presenting as bloating, nausea, vomiting, early satiety, and abdominal pain. While mostly caused by diabetes, iatrogenic injuries are the second most common cause of gastroparesis in the United States[1].

The mechanism of radiofrequency and cryoablation-induced gastroparesis is not clearly defined; however it is thought to be caused by collateral thermal injury to periesophageal vagus nerve fibres during ablation especially when ablating the posterior left atrial wall[2]. The anatomy of the oesophagus itself has been a contributing factor with one report stating that a left-sided oesophagus was more prone to injury to the vagal trunk and fibres supplying the pylorus[3].

The incidence of ablation-induced gastroparesis was not well documented until recently. A multicentre Japanese study followed 23,322 patients from 2014-2023 post-cryoablation and reported an incidence of 0.23%[4] much like the results of a 2017 study of 5,380 patients followed post-RFCA over 17 years that reported an incidence of 0.28% of gastroparesis[5].

Gastric anatomy and posterior wall ablation show a significant correlation considering the proximity of the vagal trunk to the posterior left atrial wall. One study also reported the relation between the ablation index (AI) and the incidence of upper gastrointestinal (UGI) complications. It was found that a higher AI was associated with a significant increase in UGI complications, and gastroparesis was found in 13.5% of patients with UGI complications[6]. According to Aksu et al of the 104 patients post ablation 7 developed gastroparesis of whom 6 were post-cryoablation (CBA) and 1 post-RFCA[7]. Moreover, symptoms were typically reported after 48 hours. This could be either due to different tolerance thresholds of patients or the level of nerve irritation/injury. Our patient however reported the onset of symptoms 6 hours post procedure. Luminal oesophageal temperature control and low energy, low force targeted ablation frequency have been recommended by the American College of Cardiology[8] but in the case of our patient and in the case reported by Akhtar et al[9] this did not prevent gastroparesis. It was also advised to mechanically move the oesophagus during the procedure[10] and while that reduces the risk of thermal injury it increases the risk of mechanical injury. The latest randomized control trial utilizing an enoSTEM device for controlled active thermal protection showed a significantly reduced risk for combined endpoints (i.e., abnormalities, mucosal lesions, and gastroparesis) and is the first method to show any effective reduction in UGI complications[11]. Little to no data exists regarding the number of RF applications, length of procedure, experience, and skill of the physician, and the total circumference of area needed to be ablated.

The diagnostic algorithm followed at almost all places has been a CT abdomen-pelvis followed by EGD and confirmation by GES. The treatment of choice in most cases has been prokinetic agents. It is important to note that most of these medications cause QT prolongation and, in our case, domperidone was avoided due to its proarrhythmic effect. Mirtazapine and dietary modification of a low-fat low-fibre diet are also recommended. In some studies symptom resolution was observed within 3-6 months, however in our patient who continues to experience postprandial regurgitation, and in the case reported by Akhtar et al[9] symptoms continued despite metoclopramide treatment > 6 months.

Gastric electric stimulation may be considered in patients with refractory paresis.

CONCLUSION

In a patient with no prior history of abdominal symptoms or chronic gastric disorder post ablation presentation of new-onset abdominal distention, pain, and vomiting ablation-induced gastroparesis should be high on the differential. Moreover, better oesophageal protection strategies should be implemented, and guidelines added to procedure performance limiting excessive force, energy, manipulation, and introduction of oesophageal temperature control holding parameters. Furthermore, research regarding gastroparesis risk associated with RFCA vs. cry balloon ablation, Ablation index, and wide area circumferential ablation technique needs to be investigated.

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