EJCRIM
European Journal
of Case Reports
in Internal Medicine

ESOPHAGEAL DAMAGE FOLLOWING LEFT ATRIAL ABLATION IN A PATIENT ON DABIGATRAN: ADDING INSULT TO INJURY

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Received: 18/08/2024
Accepted: 26/08/2024
Published: 12/09/2024

Conflicts of Interests: The Authors declare that there are no competing interests.
Patient Consent: The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the case.

How to cite this article: Jalloul S, Jreij C, Karam K, Salem J, Chaptini L. Esophageal damage following left atrial ablation in a patient on dabigatran: adding insult to injury. *EJCRIM* 2024;**11**:doi:10.12890/2024_004832

ABSTRACT

Dabigatran, a commonly prescribed anticoagulant medication, has been associated with esophagitis, referred to as dabigatran-induced esophagitis (DIE). We report a case of DIE occurring in a patient following left atrial ablation for atrial fibrillation. This case emphasizes the importance of recognizing the possible combined detrimental effects of left atrial ablation and dabigatran on the esophageal mucosa and highlights the clinical and endoscopic characteristics associated with DIE.

KEYWORDS

Sloughing esophagitis, left atrial ablation, dabigatran-induced esophagitis

LEARNING POINTS

- Dabigatran-induced esophagitis (DIE) should be considered in patients on dabigatran developing esophageal symptoms after radiofrequency ablation for atrial fibrillation.
- DIE is a condition characterized clinically by symptoms related to esophageal dysfunction and histologically by significant inflammation of the esophageal mucosa.
- Physicians should be aware of the signs and symptoms of DIE and must educate patients on proper medication administration to avoid such risks.

INTRODUCTION

Dabigatran-induced esophagitis (DIE) is a condition characterized clinically by symptoms related to esophageal dysfunction and histologically by significant inflammation of the esophageal mucosa. The pathogenesis of DIE is still largely unclear, but its impact on the esophagus can be severe, leading to mucosal sloughing and causing dysphagia and odynophagia. Similarly, left atrial ablation causes esophageal damage due to the close proximity of the posterior wall of the left atrium to the esophagus.

EFIM
European Federation of Internal Medicine

Publisher
smc media srl

CASE DESCRIPTION

A 66-year-old man presented to the hospital after 2 weeks of progressively worsening symptoms of dysphagia and odynophagia. He underwent left atrial ablation 3 weeks prior to presentation. His prescribed medications included a beta blocker, disopyramide, and dabigatran, which he has been taking for 5 years. He denied epigastric pain, regurgitation, nausea, vomiting or weight loss. Physical examination was unremarkable. Laboratory studies were within normal limits. A computed tomography scan of the chest and abdomen was unremarkable. An esophagogastroduodenoscopy (EGD) revealed longitudinal sloughing mucosal casts with thick white material in the distal 10 cm of the esophagus *(Fig. 1)*. Biopsies revealed significant inflammatory alterations in the esophageal mucosa with squamous epithelium, neutrophilic infiltration, and partial epithelial degeneration but no increase in eosinophilic count, dysplasia, goblet cells, or viral inclusions. Staining for cytomegalovrius yielded a negative result.

The patient was started on sucralfate gel three times daily along with esomeprazole 40 mg twice daily. His symptoms partially improved on this regimen but recurred after decreasing esomeprazole to once daily. His dabigatran was subsequently switched to another anticoagulant agent which led to the complete resolution of the symptoms.

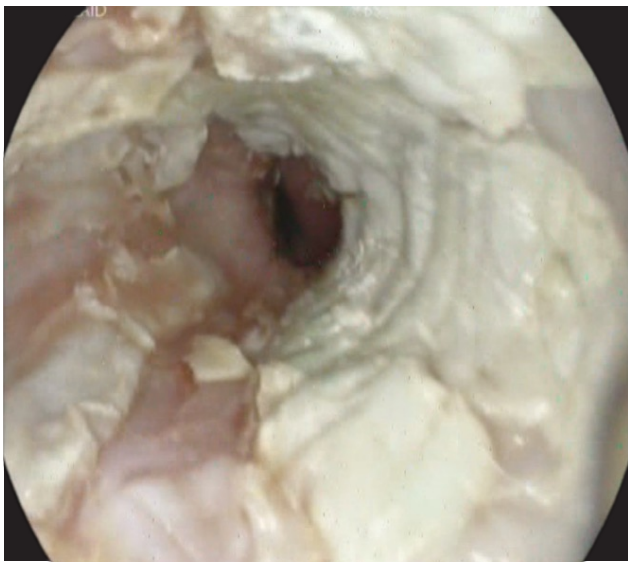

Figure 1. Esophagogastroduodenoscopy showing sloughing mucosal casts in a longitudinal fashion at the level of the distal esophagus.

DISCUSSION

Atrial fibrillation ablative therapy employs either cryoenergy or radiofrequency energy. The depth of thermal injury inflicted during ablation cannot be predicted, and complications occur in around 2 to 6% of cases[1]. Potential complications include cardiac tamponade, pulmonary vein stenosis and extra-cardiac injuries. From an anatomical perspective, the esophagus and left atrium are in close proximity, with only a thin layer of fat separating the two structures[2]. Therefore, the esophagus is prone to thermal injury during ablation. Esophageal injuries include perforation, atrio-esophageal fistula, and peri-esophageal nerve injury. Chest pain, dysphagia and hematemesis are symptoms of esophageal injury. Symptoms can occur within 3 days and up to 5 weeks post-ablation[1]. Endoscopic esophageal changes include erythema of the posterior wall of the esophagus and ulcer-like formation. Treatment with both proton pump inhibitors (PPIs) and sucralfate has been recommended[1].

Novel oral anticoagulant medications, such as dabigatran, a direct thrombin inhibitor, have gained popularity over the past decade as a treatment modality for thromboembolic disease. DIE has been reported as a potential side effect of dabigatran[3]. A study that analyzed the prevalence of DIE and its endoscopic characteristics in 91 patients on dabigatran undergoing endoscopy found DIE in 19 patients (20.9%). DIE was more prevalent in males (74 %) and elderly patients with a median age of 75. Eighteen of the 19 patients with DIE (94.7%) had longitudinally sloughing epithelial casts in the mid and lower esophagus. This endoscopic finding is considered characteristic of DIE[4]. Patients with DIE present with odynophagia, dysphagia, chest pain and heartburn[3,4]. However, dabigatran-induced asymptomatic esophageal mucosal injury has also been described[5].

While the pathogenesis of DIE remains unclear, most studies suggest that the tartaric acid core present in dabigatran capsules may cause direct local damage to the esophageal mucosa, and subsequent peristalsis may induce exfoliation of the affected epithelium. In addition, tartaric acid plays a role in lowering the gastric pH which can aggravate gastrointestinal mucosal injury and lead to ulceration and even bleeding[3,4,6]. There is no connection between the dose of dabigatran and esophagitis[3].

The differential diagnosis for DIE includes other forms of exfoliative esophagitis, such as esophageal necrosis, hot beverage ingestion, caustic ingestion, autoimmune bullous dermatosis like pemphigus vulgaris, and eosinophilic esophagitis. None of these risk factors were present in our patient.

Discontinuation of the drug and supportive measure, including the use of PPIs, has been shown to hasten mucosal recovery. However, prevention of DIE remains the most effective management option. Drinking a full glass of water and maintaining an upright position for at least 30 minutes after the intake of dabigatran may help in the prevention of DIE[3,4,6]. Despite adequate preventive measures, discontinuing the drug is necessary when serious adverse reactions occur.

In our case, the patient had been taking dabigatran without complications for an extended duration. We propose that the onset of DIE might be linked to damage resulting from the ablation procedure. It is plausible to suggest that the inflammation and luminal narrowing induced by the ablation could have prolonged the exposure of the esophagus to dabigatran pills, potentially precipitating DIE. Findings similar to ours were reported in a study published in an abstract form looking at the prevalence and characteristics

of esophagitis after radiofrequency ablation for atrial fibrillation in 77 patients taking different anticoagulants. Endoscopy was performed on all patients 3 days after ablation and revealed that 5 of the 19 patients on dabigatran developed DIE, characterized by the hallmark longitudinal sloughing epithelial casts. No abnormal endoscopic findings were observed in patients receiving other anticoagulants. After replacing dabigatran with apixaban in half of the DIE-group and applying preventive measures in the other half still receiving dabigatran, clinical and endoscopic follow-up revealed resolution of symptoms and alleviation of endoscopic abnormalities[7].

CONCLUSION

DIE should be considered in patients on dabigatran developing esophageal symptoms after radiofrequency ablation for atrial fibrillation. Physicians should be aware of the signs and symptoms of this entity and must educate patients on proper medication administration to avoid such risks.

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