

Esophageal Injury Due to Long-term Use of Dabigatran

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To the Editor

A 79-year-old man visited the emergency department complaining of a 3-day history of dysphagia. The patient had been taking dabigatran (220 mg/day) for his permanent atrial fibrillation for the past eight years. His physical examination revealed no pathology. A chest computed tomography (CT) revealed esophageal wall thickening (Figure 1a), and an esophagogastroduodenoscopy revealed vertically oriented white strips of sloughing mucosal casts in the middle thoracic esophagus (Figures 1b and 1c). A histopathological examination of a biopsy specimen of sloughing casts revealed necrosis of the superficial squamous epithelium without evidence of malignancy (Figure 1d). We diagnosed him with dabigatran-induced esophagitis (DIE). We diagnosed him with dabigatran-induced esophagitis (DIE).

A thorough history-taking revealed that he took dabigatran with little water and lay down right away afterward. Thus, we replaced dabigatran with edoxaban and advised the patient to drink sufficient water and maintain an upright position after drug ingestion. This resulted in a remarkable improvement in his symptoms within a few days. After several months, esophageal lesions were undetectable via chest CT and gastrointestinal endoscopy (Figures 1e and 1f). The patient showed no recurrence of dysphagia.

Dabigatran is a direct oral anticoagulant (DOAC) commonly used for thromboprophylaxis. Dabigatran capsules are unique; unlike other DOACs, they contain tartaric acid as an excipient. Prolonged exposure of the esophageal mucosa to the drug can lead to DIE.¹ The prevalence of DIE was 21% in patients who had gastrointestinal endoscopy and were on dabigatran therapy.² DIE may be asymptomatic or present along with one or more of the following symptoms: chest pain, dysphagia, epigastralgia, and heartburn.³ DIE presents with the characteristic endoscopic finding of longitudinal sloughing of the epithelium in the middle to the distal esophagus, which is the key to its diagnosis.² Treatment options include instructing patients how to take dabigatran to avoid lodging

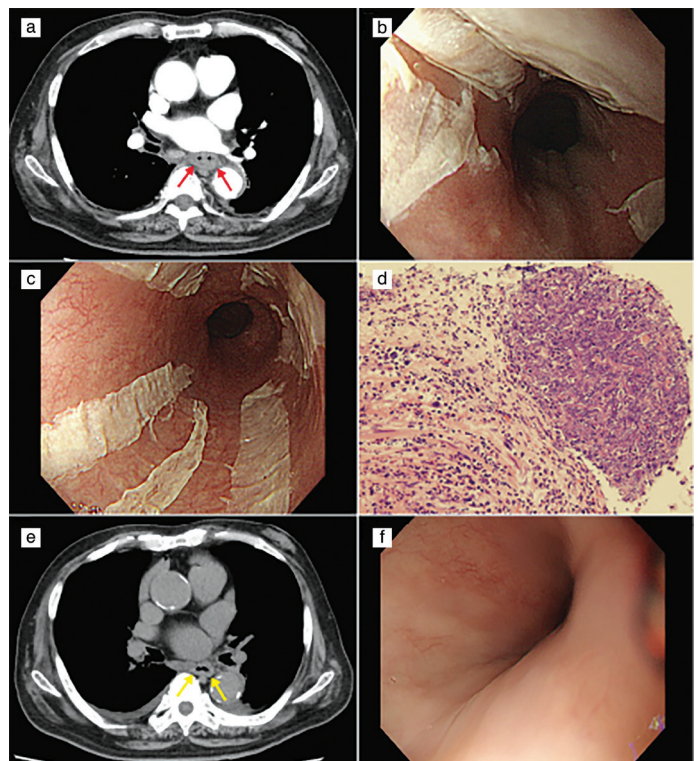


FIG 1. (a) Computed tomography (CT) of the chest showing esophageal wall thickness in the middle segment of the thoracic esophagus (red arrows). (b, c) Esophagogastroduodenoscopy reveals vertically oriented white strips of sloughing epithelium in the middle thoracic esophagus. (d) Hematoxylin and eosin staining show necrosis of the superficial squamous epithelium (x200). (e) Chest CT showing the disappearance of the wall thickening of the esophagus (yellow arrows). (f) Esophagogastroduodenoscopy shows that the esophageal mucosa had healed completely



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in the esophagus (i.e., taking dabigatran with plenty of water and maintaining an upright position for 30 minutes or more after taking it) and switching to another DOAC.^{4,5} Taking dabigatran after a meal or concomitant therapy with a proton pump inhibitor may also be effective.⁶ Previous reports showed that DIE usually develops shortly or approximately one year after dabigatran initiation.^{2,3} This is an extremely rare case in which DIE became apparent eight years after its commencement. If a patient taking dabigatran presents with chest or abdominal symptoms, clinicians should consider that DIE may be the cause regardless of its start date.

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