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Dabigatran-induced esophagitis

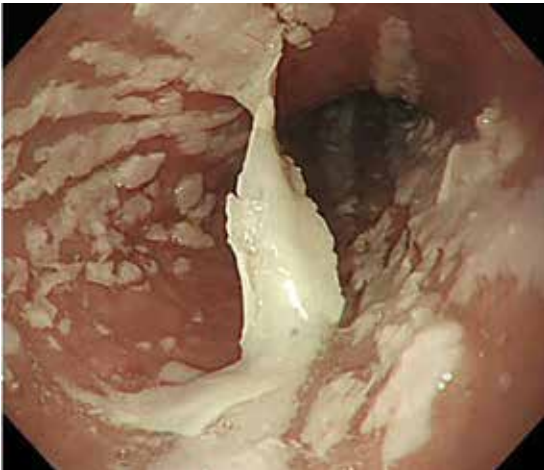


Figure 1. Endoscopy shows longitudinal sloughing mucosal casts in the middle and lower esophagus.

A 74-YEAR-OLD MAN presented to the gastroenterology clinic with a 2-day history of retrosternal discomfort. His vital signs were normal, and laboratory testing showed a normal leukocyte count.

Esophagogastroduodenoscopy (EGD) revealed longitudinal sloughing mucosal casts in the middle and lower esophagus (**Figure 1**).

Esophageal candidiasis would be suspected on the basis of endoscopic findings of white or yellowish plaque-like lesions that could not be washed away.¹ However, in this patient, the lesions were exfoliative, a picture compatible with chemical or “pill esophagitis.” The patient had no risk factors for esophageal candidiasis such as human immunodeficiency virus infection, alcohol abuse, or diabetes mellitus. Further, no *Candida* species were detected in EGD samples sent for microscopic study.

TABLE 1
Risk factors and medications associated with pill esophagitis
Risk factors
Poor pill-taking habits—inadequate liquid with the pill, lying down immediately after taking a pill
Elderly patient
Esophageal strictures
Esophageal motility disorders
Drug formulation (capsules, slow-release tablets)
Medications
Antibiotics, eg, doxycycline and tetracycline
Nonsteroidal anti-inflammatory drugs
Bisphosphonates
Quinidine
Potassium chloride
Ferrous sulfate
Dabigatran

The tablets contain tartaric acid, and if they get stuck in the esophagus, the acid leaching out can damage the mucosa

The patient had been taking dabigatran 110 mg twice daily for 2 years because of non-valvular atrial fibrillation. He was also taking amlodipine 2.5 mg/day for hypertension.

Risk factors and medications commonly associated with pill esophagitis are shown in **Table 1**. Further questioning of the patient revealed that he took his medications with very little water. Apixaban was prescribed as an alternative to dabigatran, in addition to the proton-pump inhibitor rabeprazole 10 mg/day. The symptoms disappeared within a few days.

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Figure 2. Complete healing of the esophageal mucosa 1 month later.

At follow-up 1 month later, EGD confirmed that the esophageal mucosa had completely healed (Figure 2).

DABIGATRAN-INDUCED ESOPHAGITIS

Dabigatran, an oral direct thrombin inhibitor, is widely used for thromboprophylaxis. Unlike other direct oral anticoagulants, it contains

tartaric acid as an excipient, which reduces variability in the drug's absorption. However, if the tablet gets stuck in the esophagus, the acid leaching out can damage the esophageal mucosa.

Although no study has investigated the overall prevalence of dabigatran-induced esophagitis, a retrospective database review of 91 patients taking dabigatran and undergoing upper-gastrointestinal endoscopy reported that 19 (20.9%) had endoscopic signs of dabigatran-induced esophagitis.²

Typical symptoms are the acute onset of chest pain, epigastralgia, odynophagia, and dysphagia. But patients can also have no symptoms or only mild symptoms.^{2,3}

Despite dabigatran's anticoagulant activity, there have been few reports of bleeding, perhaps because the lesions tend to be superficial on the surface of the esophageal mucosa.

Symptoms usually resolve within 1 week after stopping dabigatran and starting a proton pump inhibitor. To prevent mucosal injury, patients should be instructed to take dabigatran with sufficient water and to remain in an upright position for at least 30 minutes afterward.⁴

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