Doxycycline-induced pill esophagitis

A 23-year-old white woman with a past history of chronic urticaria presented to our department following 2 weeks of progressive dysphagia for solids and liquids. She also complained of chest pain, odynophagia, and a 3-kg weight loss. She denied having prior episodes of food impaction, heartburn, or acid regurgitation. She had suffered 3 months earlier from a subacute onset of polyarthritis involving the small and large joints of the lower limbs, and a low-grade fever. At the time, viral serology and immunology tests were negative, and a presumptive diagnosis of serum sickness was made. Her oral contraceptive pill (OCP) was discontinued and her symptoms subsided. She did not receive any further medications, except for a short course of antibiotics for facial acne 2 weeks prior to her current presentation. Her physical examination was unremarkable.

Upper gastrointestinal endoscopy revealed a corrugated esophagus with circumferential pseudomembrane formation and discrete areas of ulceration (Fig. 1). The findings were limited to the mid-esophagus. Histopathology demonstrated inflammatory infiltration of the esophageal papillae, with necrosis and detachment of the superficial epithelium. The subepithelium contained fibrinoid nodules, abundant lymphocytes, and eosinophils (10/high-power field [HPF]) (Fig. 2). These findings were consistent with doxycycline-induced esophagitis.

Doxycycline is a common yet under-reported cause of pill esophagitis. Mucosal injury is the result of prolonged contact of the acidic drug with the esophageal mucosa. Therefore, patients who ingest the pill with insufficient water and then remain recumbent have the highest risk. Curiously, patients with pre-existing esophageal dysmotility are not at increased risk, perhaps because they are likely to be more careful with their swallowing. Pills tend to lodge at areas of esophageal narrowing, such as the levels of the aortic arch, the left main bronchus, and the gastroesophageal junction. The use of doxycycline for gynecological infections and for acne vulgaris is thought to account for the slight female preponderance of this condition; however, reliable epidemiological data are lacking.

Despite her history of atopy, endoscopic and histological findings in this patient were not compatible with a diagnosis of eosinophilic esophagitis. Similarly, the current episode is unlikely to be related to an undiagnosed rheumatological disease, such as Sjögren’s syndrome or scleroderma, given her negative serology and her response to discontinuation of the OCP.

Most cases of pill esophagitis heal without intervention over 3–10 days. Our patient had discontinued doxycycline a few days before her presentation. She was managed with anti-secretory medication, although this treatment is unsubstantiated. Her symptoms abated, and a follow-up endoscopy after 2 weeks was completely normal.

References

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Competing interests: None

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Bibliography
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