Fundic gland polyps and duodenal gastric heterotopia – is this an unusual association?

Fig. 1 Esophagogastroduodenoscopy showing multiple small, sessile polyps in the fundus and upper body of the stomach in a 72-year-old woman on proton pump inhibitor therapy for more than 10 years.

Fig. 2 Polypoid sessile lesion in the duodenal bulb.

Fig. 3 Histology of the gastric polyps: cystically dilated glands lined by normal cell types of the oxyntic mucosa (hematoxylin and eosin, $\times 10$).

Fig. 4 Histology of the duodenal lesion: intestinal mucosa (upper right), foveolar mucosa (upper left), and crypts of the fundic type (below) (hematoxylin and eosin, $\times 10$).

A 72-year-old woman with a past history of heartburn and acid regurgitation had been on proton pump inhibitor (PPI) therapy for more than 10 years with effective symptom control. Her past medical history was unremarkable, and there was no familial history of colorectal cancer or polyposis.

The patient now presented with the new onset of early satiety without weight loss, dysphagia, or gastrointestinal bleeding. Laboratory data were unremarkable. Esophagogastroduodenoscopy revealed multiple small (4–10 mm), sessile polyps in the fundus and upper body of the stomach (● Fig. 1), and the larger polyps were removed. In addition, a 2.5-cm polypoid sessile lesion suspicious for adenoma was identified in the duodenal bulb (● Fig. 2) and biopsied.

Histopathologic examination of the resected gastric polyps revealed fundic gland polyps (FGPs) without epithelial abnormalities or dysplasia (● Fig. 3). Biopsy of the duodenal polyp revealed foveolar epithelia and gastric fundic glands (● Fig. 4). Duodenal gastric heterotopia (DGH) in a patient with sporadic FGPs was diagnosed.

FGPs, the most common type of gastric polyps, have been associated with familial adenomatous polyposis syndromes and the long-term use of PPIs [1]. Endoscopic series have reported an incidence of DGH of 1.9% [2]. A subdivision of this entity into congenital and reactive subtypes has been proposed, the latter being part of a spectrum related to active duodenitis [2, 3]. Recently, however, some studies have described an association between FGPs and DGH, suggesting that hypergastrinemia induced by the use of PPIs may cause hyperplasia within congenital DGH, leading to the formation of macroscopic lesions visible on endoscopy [2, 3].

This case illustrates an exuberant presentation of DGH in an association that, if confirmed in further studies, one might expect to be increasing with the increasingly widespread use of PPIs in recent years.

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Bibliography

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