Eosinophilic enteritis presenting as a perforated duodenal ulcer

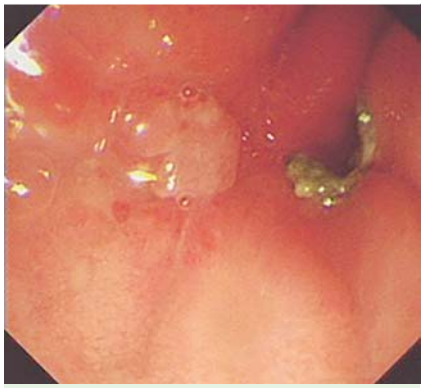

Fig. 1 Esophagogastroduodenoscopy showing gastritis and a duodenal ulcer with stenosis.

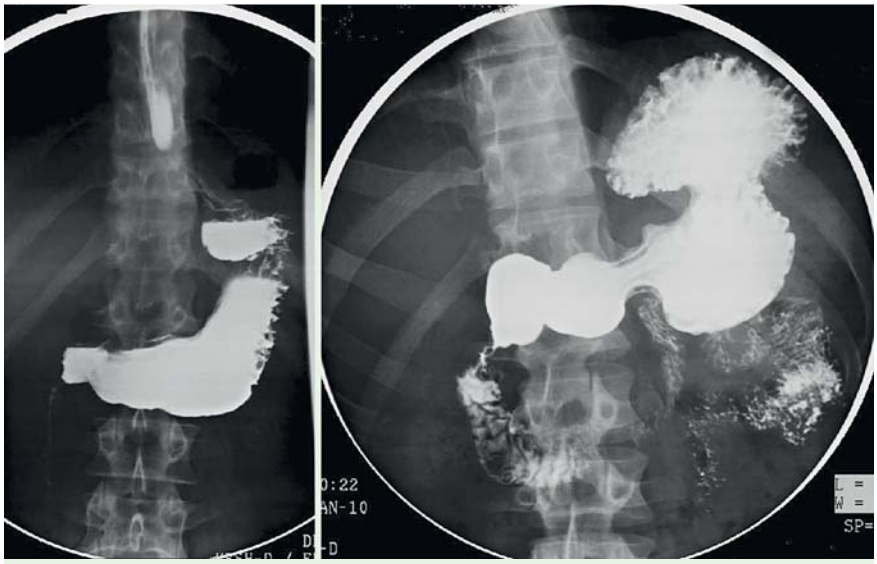

Fig. 2 Barium meal showing a short segment of narrowing in the first part of the duodenum with peripheral ulceration suggesting chronic ulceration with secondary fibrotic changes and subsequent narrowing.

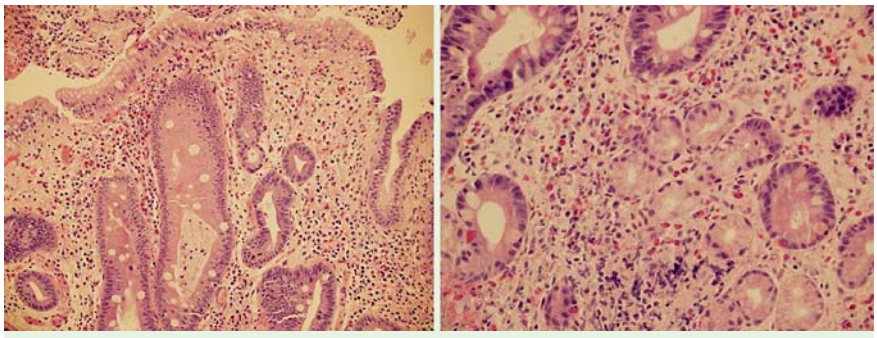

Fig. 3 Biopsies from the duodenal ulcer showing a marked eosinophilic infiltrate, diagnostic of eosinophilic gastroenteritis (EGE).

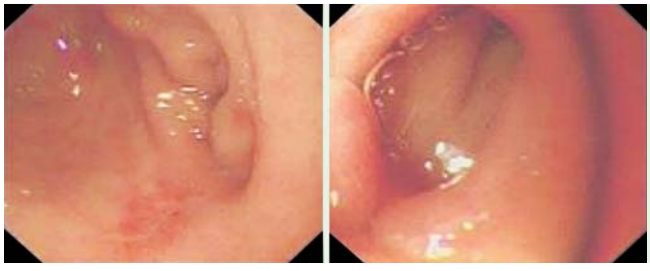

Fig. 4 Esophagogastroduodenoscopy showing a healed duodenal ulcer and a postbulbar stenosis that was subsequently dilated.

Eosinophilic gastroenteritis (EGE) is a rare condition of unknown etiology that is characterized by eosinophilic infiltration in the layers of the gastrointestinal tract [1]. It can affect any part of the gastrointestinal tract but most commonly affects the stomach [1,2]. This report describes a rare case of EGE presenting as a perforated duodenal ulcer with subsequent duodenal stenosis.

A 26-year-old man was referred to us with duodenal obstruction following a laparotomy for a perforated duodenal ulcer. He had undergone repair of the ulcer and 2 weeks later had presented with vomiting and weight loss. Endoscopy showed gastritis and a duodenal ulcer with stenosis (**Fig. 1**). A barium meal showed narrowing at the first part of the duodenum (**Fig. 2**). He was given treatment for *Helicobacter pylori* and pantoprazole. A repeat endoscopy 2 months later showed a deformed pylorus, prepyloric nodular mucosa, and an almost circumferential duodenal ulcer with significant narrowing.

Biopsies from the ulcer showed a marked eosinophilic infiltrate diagnostic of EGE (**Fig. 3**). He received prednisolone as a tapered course over 1 month and pantoprazole, after which, there was a marked improvement in his symptoms. A repeat endoscopy showed a healed duodenal ulcer and a postbulbar stricture (**Fig. 4**). Central radial expansion balloon dilation was performed. He received another course of steroids and 1 month later was well and gaining weight with no vomiting.

Talley et al. suggested three diagnostic criteria for EGE: (i) gastrointestinal symptoms; (ii) demonstration of eosinophilic infiltration in the gastrointestinal tract, or presence of high eosinophil count in fluid; (iii) no evidence of parasitic or extraintestinal disease [3]. EGE has been classified, depending on the extent of bowel wall involvement, into mucosal, muscular, and serosal [1,3]. In the past, peptic ulceration accounted for most cases of gastric outlet obstruction. Nowadays, other causes such as malignancy and EGE must be excluded.

EGE may also present acutely [4,5] as was the case for our patient who presented with peritonitis secondary to a perforated peptic ulcer, with a subsequent stricture

that was presumed to be postsurgical but proved to be due to EGE. To the best of our knowledge, this is the first report of EGE presenting as a perforated duodenal ulcer.

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Competing interests: None

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Bibliography

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