completely from his complaints despite the persistence of the eosinophilic esophagitis. Thus, the fluctuation of the esophageal symptoms in patients with eosinophilic esophagitis could be caused by precipitating factors, such as ingested substances or to the local action of microorganisms. These factors may activate the local eosinophils, *i.e.*, they may potentiate the release of neurotransmitters or neurotoxins, as pointed out by Attwood *et al.* (3). As is known, there is much evidence to suggest an important role for eosinophils in the pathophysiology of allergic diseases. In these conditions, such as asthma, uncontrolled eosinophil activation may lead to the release of lipid mediators, basic proteins, cytokines, VIP, and substance P (4, 5). In asthma, these eosinophil products may cause bronchoconstriction and contribute to airway hyperresponsiveness (6). The lack of clearance of esophageal contents in these patients may facilitate the overgrowth of *Candida albicans* or other types of microorganisms that may consequently precipitate the activation of eosinophils and the disturbances of esophageal motility.

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Helicobacter pylori and Gastric Outlet Obstruction

To the Editor: We strongly support de Boer's (1) and Borody *et al.*'s (2) views that eradication of *Helicobacter pylori* (*H. pylori*) markedly reduces the complications of duodenal ulcer (DU), especially the pyloric strictures. Before the discovery of the *H. pylori* organism, the complications of DU were more frequent, but with its eradication such complications are rare nowadays. We have a similar experience in the Kashmir valley, which is a highly endemic area for peptic ulcer disease having a point prevalence of 4.72% (3) and having a significant association with *H. pylori* (4). In the past 4 yr, we had six patients with DU-related gastric outlet obstruction who had clinical and endoscopic evidence of significant obstruction and were positive for *H. pylori* (rapid urease test plus Giemsa staining). We treated these patients with triple therapy, and repeated endoscopic follow-ups revealed a significant dilation and clinical improvement in all. None of the patients required surgery. Similar experience to that of de Boer *et al.* (5) has been reported by others (6). Although reversal of stenosis appears impossible theoretically, clinical experience has demonstrated the same. We support Tursi *et al.*'s view (6) that the resolution of associated luminal edema following *H. pylori* eradication allows early improvement in the gastric emptying and symptoms. Thus, we suggest that *H. pylori*-positive DUs with gastric outlet obstruction should receive adequate eradication therapy (triple or quadruple), whereas dilation or surgery should be reserved for patients who do not respond to such medical therapy.

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