Nonceliac diaphragm disease of the duodenum

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In 1988, Bjarnason et al (1) and Lang et al (2) described the clinical and pathological features associated with small intestinal strictures in patients who had received nonsteroidal anti-inflammatory drugs. These small intestinal strictures were very distinctive and unique to the known forms of small intestinal pathology associated with nonsteroidal anti-inflammatory drugs. Investigators used the term 'diaphragm disease' to describe the pathological changes (2). The observations were later confirmed by others detailing solitary or multiple diaphragm-like strictures, particularly in the distal small intestine and colon (3,4). In a subsequent report (5), a duodenal diaphragm-like stricture was reported in a Belgian man after consumption of an over-the-counter preparation that contained acetylsalicylic acid (ASA) and sodium bicarbonate.

Many other conditions have been associated with duodenal stricture formation and include congenital forms of duodenal stenosis (6-8), other disease-induced strictures including those related to intrinsic small bowel disorders (9-11), medication-induced strictures (ie, potassium chloride) (9), abdominal trauma (12,13) and infections such as tuberculosis (14). The present report documents a middle-aged adult with obstructive symptoms and endoscopically detected membranous stenosis of the descending duodenum. Although there was no history of medication use, the changes were typical of duodenal diaphragm disease.

CASE PRESENTATION

A 58-year-old, Indo-Canadian male was evaluated for recurrent bouts of upper epigastric pain and distension that was exacerbated by meals. There was no nausea, vomiting, change in appetite or weight loss. A barium study of the stomach and duodenum showed a dilated duodenum without ulceration (Figure 1). Radiographic studies of the jejunum revealed diaphragm-like strictures in the descending duodenum. Other reported causes such as celiac disease and drug-induced small bowel diaphragms were excluded. Possibly, the changes seen in this patient were related to ethnic food-induced, mucosal injury to the upper gastrointestinal tract. Further studies are needed to evaluate potential toxicity or protective effects of different ethnic diets and their relationship with the development of different intestinal diseases.

Key Words: Celiac disease; Drug-induced small bowel strictures; Duodenal membranous stenosis; Duodenal stricture; Duodenal ulcers


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num and ileum were normal. Retained food debris was also reported, possibly from delayed emptying of the duodenum. A chest radiograph was normal. Serological studies for *Helicobacter pylori* were negative. There was no history of potassium chloride, ASA or other nonsteroidal anti-inflammatory drug use. There was no history of abdominal trauma, rheumatological disorder or familial history of gastrointestinal diseases such as Crohn's disease or celiac disease. An initial endoscopic examination revealed a normal esophagus and stomach, but there was retained food debris. Endoscopic examination was repeated after a 24 h fluid diet. The mucosa was normal except for two distinct membranous strictures or webs of the descending duodenum (Figure 2). Mucosal biopsies from mucosa of the descending duodenum beyond the second stricture and between strictures, and from the duodenal bulb were normal. Biopsies from the margin of the web-like membranous strictures revealed reactive epithelial cellular changes with mucin depletion. No erosive change was evident. Fibrosis in the submucosal region was present, but there were no iron-laden macrophages or granulomas (2). Gastric biopsies were normal, while silver stains for *H pylori* were negative. Clinical improvement and resolution of his abdominal pain followed endoscopic balloon dilation and empirical omeprazole therapy.

**DISCUSSION**

The differential diagnosis of small intestinal ulceration and stricture formation is extensive (8). This is generally classified into broad groups such as congenital, mechanical, inflammatory, vascular, neoplastic and miscellaneous. Most formations are self-evident from clinical history and are rare. Granulomatous disease, especially with broad-based strictures, raises the possibility of Crohn’s disease (9,10) or an infection such as tuberculosis (14). Congenital duodenal stenoses, including webs and rings, may occur and are evident soon after birth (6,7). Occasionally, they may be associated with Down’s syndrome (7,8). Post-traumatic strictures that simulate Crohn’s disease rarely have been described in the ileum (12), but stricture of the duodenum and jejunum has been recorded in association with physical child abuse (13). Ischemic strictures may develop in the more distal small intestine, are usually broad-based and typically have iron-laden macrophages (2). Ulceration in the small intestine may be related to celiac disease, sometimes with intestinal lymphoma (15,16). Strictures have also been associated with celiac disease, particularly in the duodenum (17,18). Drug-induced strictures and diaphragm-like changes have been reported with a number of medications, including potassium chloride, ASA and other nonsteroidal anti-inflammatory medications. Usually, these occur in the distal small intestine and colon (1-4) rather than the duodenum (5). In spite of careful, clinical and pathological evaluation, the cause of some small intestinal strictures remain unexplained. Some of these differ from the changes seen in the present case because the strictures are broad-based and often
require surgical resection to resolve symptoms. In contrast, the diaphragm-like strictures seen in the present patient were quite distinctive and multiple; they usually involve the distal small intestine or colon (19). Pathological features of diaphragm disease, as previously detailed in this journal (19), also seem quite specific and, in some instances, appear to be directly related to medication use (2). In the present patient, no specific cause could be defined.

Little information is available on the effects of different dietary factors in the pathogenesis of gastroduodenal disease. Nevertheless, prior epidemiological and experimental animal studies from India have suggested that environmental, particularly dietary factors, may be important in the pathogenesis of duodenal ulcer disease (20,21). Moreover, studies from Bombay have demonstrated pathologic effects of spices, including chili powder, on the mucosa of the upper gastrointestinal tract (22). The prevalence of most duodenal diseases in India, however, is not well defined; for example, most duodenal strictures have been attributed to prior ulcer disease or tuberculosis (14). Even in these patients, however, it may be speculated that some caustic dietary factor could be important. It is known, for example, that salicylates, including ASA and other salicylate-like compounds, may be found in a variety of foods (23,24). Whether these would be present in sufficient amounts in the Indian diet to cause any toxic effects in the upper gastrointestinal tract is not known. Further studies are needed to determine whether dietary factors, including those associated with ethnic diets, play a role in the pathogenesis of duodenal disease, including ulcers or strictures, in South Asians.

REFERENCES
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