BRIEF COMMUNICATION

Celiac disease: Diagnosis at laparoscopy

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A 27-year-old woman was found to have an abnormal small bowel at the time of an elective laparoscopic tubal ligation. Subsequent investigation revealed celiac disease to be the underlying cause. The changes identified are believed to reflect an increased intestinal blood flow, which has been identified in the diagnostic imaging literature to be associated with active celiac disease. Further study of this feature of celiac disease may provide additional insights into its pathophysiology.

Key Words: Celiac disease; Laparotomy; Mesenteric circulation

The clinical presentation of celiac disease includes several common and a number of unusual presentations. The present case falls into the latter category, and the authors discuss the features that highlight aspects of the pathophysiology of this disease that have received little attention in the literature.

CASE PRESENTATION

A 27-year-old woman with a 10-year history of uncomplicated type 1 diabetes was referred for a tubal ligation. Evaluation in the preoperative assessment clinic revealed gastroesophageal reflux for which she took Tums (GlaxoSmithKline, USA); no other gastrointestinal symptoms were noted. Abdominal examination was normal.

At laparoscopy the small bowel was found to be grossly abnormal with circumferential erythema, dilated blood vessels and lacteals (Figure 1). Interoperative consultation with general surgery was obtained and she was subsequently referred for gastroenterology consultation.

Further functional inquiry confirmed the use of Tums for vague abdominal discomfort. She had a tendency to constipation and reported occasional episodes of abdominal bloating and borborygmi. Her weight had fallen by 6.5 kg over the previous year despite a normal food intake.

Small bowel radiography was normal and at endoscopy ‘scalloping’ was seen in the proximal small bowel. The immunoglobulin G antiendomysial antibody titre was positive to 1:1280 (normal less than 1:2.5) and the lactulose/mannitol ratio was increased to 0.092 (normal less than 0.025). Severe villous atrophy, crypt hyperplasia and inflammatory infiltrate consistent with celiac disease was seen in the histopathology of the duodenum (Figure 2). Bone densitometry revealed osteopenia.

At follow-up one month following initiation of a gluten-free diet, she felt well. Management of her diabetes had become somewhat more problematic because of a rise in her blood glucose levels and her abdominal discomfort had improved. She did not return for follow-up evaluation after six to nine months, as recommended, and was subsequently interviewed by telephone. This revealed that, initially, on gluten restriction she had considerable difficulty controlling her diabetes and experienced a gain in weight from 44.5 kg to 50 kg. She continues to be free of gastrointestinal symptoms 18 months following initiation of a gluten-free diet.

DISCUSSION

We tend to think of gluten-sensitive enteropathy as a disease of the small bowel mucosa, and we focus our attention on the underlying immunological events, the histopathology and the functional consequences of the changes to the intestinal mucosa (1,2). However, as confirmed by this case, there is more extensive involvement of small bowel that include substantial changes to intestinal blood flow. Several studies using angiography and/or Doppler ultrasound have shown an increase in mesenteric blood flow in celiac disease in both adults and children (3-8). This increase varies directly with disease activity and normalizes with treatment. It has been speculated that the release of inflammatory mediators in the intestinal mucosa, local hormonal and nutrient factors as well...
as changes in the microcirculation that result in mucosal shunting, contribute to a hyperdynamic mesenteric circulation (3,4). The systemic hypotension that is commonly observed in active celiac disease (9) may occur as a consequence of mesenteric redistribution of blood flow, or perhaps common factors underlie both cardiovascular changes.

The diagnosis of unsuspected celiac disease at abdominal surgery is likely to remain an interesting and remarkable occurrence. Further investigation of the mechanism and effect of these vascular changes may yield useful insights into the pathophysiology of this disorder.

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REFERENCES