Diffuse circumferential hyperplasia of Brunner's glands causing obstruction in the duodenum in a 12-year-old child

12 yaşındaki çocukta duedonumda obstrüksiyona neden olan yaygın Brunner bez hiperplazisi

To the Editor,

Brunner's glands consist of submucosal mucin-secreting glands, which are located in the duodenum (1). Brunner's gland hyperplasia (BGH) is usually asymptomatic. A few cases causing obstruction, hemorrhage or intussusceptions in adults have been reported in the literature (2). We report a case of a 12-year-old boy with BGH presented with duodenal obstruction.

A 12-year-old boy was admitted to our emergency department with significant vomiting and weight loss over the course of one week. On the present admission, physical examination revealed extensive abdominal tenderness, especially in the epigastric region. An upper endoscopy showed mild esophagitis, completely open pylorus and considerably widened bulb. The mucosa seen in the intersection between the first and second part of the duodenum was edematous and hyperemic, and the instrument did not pass through at that level because of severe narrowing (Figure 1). Barium examination of the upper gastrointestinal tract revealed that the stomach and the first part of the duodenum were distended, passage through the second part of the duodenum was delayed, and retrograde filling of the stomach was observed. Histological examination revealed BGH. Lobules of Brunner's glands were extended into the lamina propria and were separated by delicate fibrous septa (Figure 2). The cells constituting the glands were cytologically bland and showed no mitotic activity. An antral biopsy specimen was negative for *Helicobacter pylori,* and serum gastrin level was within normal limits. An abdominal computed tomography was performed, which showed an enlarged stomach and bulb, but no masses were detected. According to the subsequent assessment made together with pediatric surgery and radiology, the patient un-

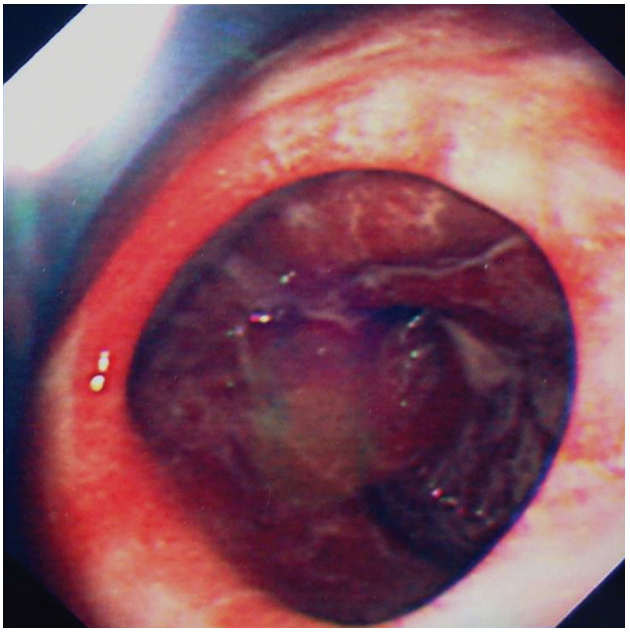

Figure 1. Endoscopic photograph shows the enlarged duodenal bulb with luminal narrowing.

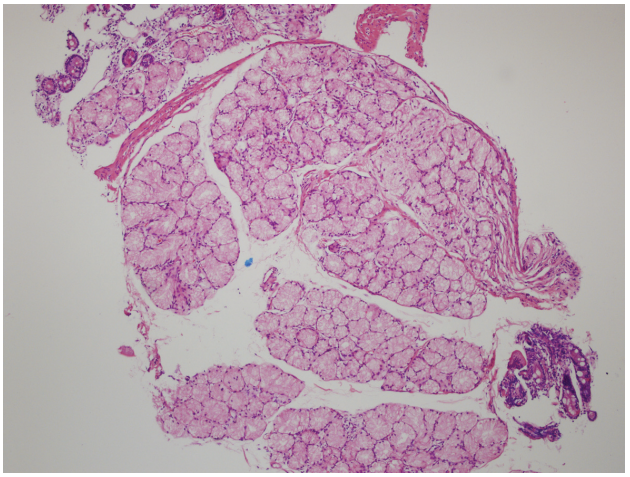

Figure 2. Lobules of Brunner's glands are separated by delicate fibrous septa, HEx100.

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derwent a surgical procedure that included gastroduodenoplasty.

Histopathologic examination of the operation specimen showed marked circumferential BGH. The patient had an uneventful postoperative recovery. He remains in good health without any dyspeptic symptoms 18 months after the operation.

Brunner's glands were first described by Brunner in 1688. These tubuloalveolar glands extend from the pylorus to the second portion of the duodenum and secrete pepsinogen, urogastrone and mucus in response to the acid in the duodenum (1-3). BGH mostly presents during the 5th and 6th decades and is quite rare in the pediatric age group.

The pathogenesis for the development of BGH remains unknown, although gastric hyperacidity may play an important role. In contrast, 20% of cases are reported to have hypoacidity (4,5,7). Other conditions associated with increased incidence of BGH are *H. pylori* infection, uremia and chronic pancreatitis (8-10). In our case, uremia and pancreatitis were not considered, and *H. pylori* was negative in both rapid urease test and histological examination of antral biopsy specimens; serum gastrin level was within the normal range.

The majority of cases are asymptomatic. Nevertheless, the condition may cause epigastric pain, obstruction, diarrhea, gastrointestinal hemorrhage, obstructive jaundice, and pancreatitis depending on the size, type and location of BGH (1,4,8). Duodenal obstruction in BGH is caused mostly by a polyp or hamartoma, while circumferential hyperplasia is rare (2). Our patient presented with severe vomiting and had a rare case of duodenal obstruction caused by circumferential BGH without any mass.

Endoscopic investigations are important in diagnosing BGH. However, the sensitivity of the endoscopy is 72-84% (6). Endoscopic biopsy usually gives a negative result because it is often not deep enough to reach the submucosal tissue. On the other hand, histopathologic examination of the endoscopic biopsy and extracted specimen indicated BGH.

Treatment of BGH is conservative if the patient is asymptomatic. Proton pump inhibitor and antacids can be given. If the patient is symptomatic, endoscopic resection, laparoscopy or surgery may be required. Gastroduodenoplasty may be necessary if safe excision is not possible in the diffuse or circumscribed nodular types presenting as obstructive lesions (9). In our case, duodenal bypass was performed due to obstruction between the first and second portions of the duodenum. Antacid therapy was continued postoperatively and the patient remained free of symptoms 18 months later.

In conclusion, although unusual in childhood, diffuse circumferential BGH should be noted as a potential cause of duodenal obstruction. Although acid suppression therapy alone may be sufficient in mild cases, duodenal bypass should be considered in severe cases such as ours.

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