INTRODUCTION

Brunner’s gland hyperplasia is a benign tumor of the duodenum and it is rarely associated with clinical symptoms. We report on a 64-yr-old man with Brunner’s gland hyperplasia who had undergone a duodenocephalo-pancreatectomy. The reason is that he presented upper gastrointestinal obstructive symptoms and the esophagogastroduodenoscopic finding revealed the lesion to be an infiltrating type mass on the second portion of the duodenum with luminal narrowing. An abdominal computed tomography showed a 2.5 cm-sized mass in the duodenal second portion with a suspicious pancreatic invasion and 7 mm-sized lymph node around the duodenum. Duodenocephalo-pancreatectomy was successfully performed. Histological examination revealed a Brunner’s gland hyperplasia. The final diagnosis was the coexistence of Brunner’s gland hyperplasia and pancreatic heterotopia with a pancreatic head invasion. The literature on Brunner’s gland hyperplasia is reviewed.

CASE REPORT

A 64-yr-old man visited a private clinic with episodes of epigastric discomfort, dyspepsia, nausea, and vomiting for several days. A duodenal mass was found on esophagogastroduodenoscopy. He was then referred to our hospital. On admission, he had no significant past medical history, but had a weight loss of 7 kg. Laboratory values were as follows: white blood cells 10,950/μL, hemoglobin 12.9 g/dL, platelets 295 K/μL, aspartate aminotransferases (AST) 25 IU/mL, alanine aminotransferases (ALT) 10 IU/mL, alkaline phosphatase (ALP) 72 IU/L, gamma glutamyl transpeptidase (gamma-GT) 102 IU/L, and total bilirubin 0.7 mg/dL. Other laboratory values were within normal limits. An esophagogastroduodenoscopy showed an infiltrating type mass on the second portion of the duodenum with luminal narrowing. So, the scope could not pass through the narrowed lumen (Fig. 1). A 3-D abdominal computer tomography (CT) was then performed, which showed a 2.5 cm-sized mass in the second portion of duodenum and a loss of fat plane between the duodenal mass and pancreas, which gave suspicion of a pancreatic invasion and a 7 mm-sized lymph node around the duodenum. The stage based on the CT finding was stage III (T3, N1, Mx) (Fig. 2). The tissue from the endoscopic biopsy showed moderate and chronically active duodenitis. The literature on Brunner’s gland hyperplasia is reviewed.
denocephalopancreatectomy, because we could not completely exclude malignancy due to the CT finding and suspected that the lesion leading to duodenal obstruction in the second portion of duodenum included the ampulla.

The histological examination of the second portion of the duodenum showed a diffuse nodular type of Brunner’s glands hyperplasia with inflammation of the ampulla, and there was reactive lymphadenopathy of regional lymph nodes. Pancreatic heterotopia was found in the subserosa of the duodenum (Fig. 3). Finally, the patient was diagnosed with a diffuse nodular type of Brunner’s gland hyperplasia with duodenal obstruction. The patient was discharged 25 days after the operation without any complications.

**DISCUSSION**

Patients who have Brunner’s gland hyperplasia of the duodenum are frequently affected with chronic pancreatitis (4), duodenal ulcers, and chronic erosive gastritis. The cause of Brunner’s gland hyperplasia is thought to be chronic inflammation, chronic stimulus by excessive secretion of gastric acids, or the decrease of the pancreatic exocrine function. However, it is recently thought that the hyperactivity of the exocrine modulating factor (hormone, vagus nerve, and intestinal mucous membrane factor) is the major cause of Brunner’s gland hyperplasia (5).

The clinical manifestations of Brunner’s gland hyperplasia are nonspecific, such as epigastric discomfort, abdominal distension, or dyspepsia etc. Occasionally, it can lead to hemorrhages, acceleration of peristalsis, diarrhea, transient and partial bowel obstruction and duodenal intussusception due to the mass (3).

Brunner’s gland hyperplasia, a submucosal tumor, can be diagnosed by endoscopy, biopsy, abdominal CT, and other radiologic techniques. Only about 0.3-4.6% of the benign tumor originating from the duodenum can be found by the endoscope (6). Brunner’s gland hyperplasia is vary rare and
represents 10.6% among this amount (7).

Brunner's gland hyperplasia is distributed mainly on the duodenal bulb (57%), the duodenum's second portion (27%), and its third portion (7%). It is rarely on the pylorus (5%), jejunum (2%), or the terminal ileum (2%) (8).

In 1934, Feyrter classified the abnormal glandular proliferation into the following three types: diffuse nodular hyperplasia (type 1), occupying most of the duodenum; circumscript nodular hyperplasia (type 2), the most common type mainly present in the duodenal bulb; and, adenomatous hyperplasia (type 3), which may be sessile or pedunculated (4, 9-12).

This first-mentioned type corresponds with the Brunner's gland hyperplasia found in our patient. This diffuse nodular hyperplasia (type 1) is often mistaken for a malignancy.

The diagnosis of Brunner's gland hyperplasia is supported by endoscopy and radiologic findings. However, sensitivity of the endoscopy is 72-89% and occasionally, the endoscopy is not useful in making the diagnosis (3, 7, 13, 14). Thus, we found that abdominal CT was useful in this case.

There is no consensus on the therapeutic principle for Brunner's gland hyperplasia because follow-up study is insufficient. The medical treatment is to control gastric hyperacidity, which is one cause of Brunner's gland hyperplasia. However, the regression of Brunner's gland hyperplasia is rare (7). Therefore, excision appears to be the treatment of choice. We should completely remove the lesion by endoscopic resection or surgical resection when Brunner's gland hyperplasia leads to symptoms and complications or when definite diagnosis is necessary (7, 15-17).

According to a recent report, other research opinions state that non-symptomatic Brunner's gland hyperplasia should be resected by all means because it undergoes malignant transformations in some cases (7, 18).

There are various methods for the resection of Brunner's gland hyperplasia. For instance, endoscopic polypectomy should be performed if the lesions are less than 1 cm in diameter, if the size is excessively large or if the tumor is the sessile type. Then, surgical polypectomy, duodenal wedge resection, or partial gastrectomy that is extended to the duodenal bulb should be done (7). However, there is one report of a duodenocaphalopancreatectomy, in a Brunner's gland hyperplasia case involving the duodenal second portion with a pancreatic invasion (7). Domenico Lusco et al. reported the case of a voluminous Brunner's gland hyperplasia induced stenosis of the duodenal second portion and a pancreatic duct dilatation on an abdominal CT finding. Even though the frozen biopsy showed no evidence of malignancy, they decided to carry on with the operation because they could not exclude the possibility of malignancy (7).

Our case is similar to the case by Domenico Lusco et al. The abdominal CT scans of this case showed the findings of duodenal cancer with a pancreatic head invasion and a regional lymph node enlargement. Also, the patient had symptoms of nausea, vomiting, and epigastric pain due to obstruction. This case helped us to decide on the operation. The histological findings of frozen biopsy from the operation had no evidence of malignancy, but showed pancreatic heterotopia. However, sensitivity of frozen biopsy is 83-92% (19-22) and its diagnostic accuracy is 91-97% (19, 23, 24). So, we could not exclude malignancy even after a frozen biopsy. The excision area should be minimal in Brunner's gland hyperplasia because it is a benign tumor. However, if the tumor involves the second portion of duodenum and invades the pancreatic area just as in this case, then duodenocaphalopancreatectomy should be considered.

Another unique aspect of this case is the coexistence of Brunner's gland hyperplasia and pancreatic heterotopia. The relation of ectopic pancreas and Brunner's gland hyperplasia is not clear. Although there is less connection between the cause of Brunner's gland hyperplasia and the pancreatic heterotopia, we need further studies about the relationship of Brunner's gland hyperplasia with pancreatic heterotopia.

In our case, a duodenocaphalopancreatectomy was performed due to symptoms of Brunner's gland hyperplasia with a pancreatic invasion, which induced epigastric discomfort, nausea, and vomiting.

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