CASE REPORT

Bali Medical Journal (Bali MedJ) 2023, Volume 12, Number 2: 1608-1611
P-ISSN.2089-1180, E-ISSN: 2302-2914

Bali Medical Journal

INTRODUCTION

Brunner’s glands are acinotubular glands which are located mostly in the deep mucosal or submucosal layers of the proximal duodenum. Hyperplasia of these glands is reported in almost 1 in 50 upper gastrointestinal endoscopies and accounts for approximately 5 to 10 percent of all benign duodenal tumors. Brunner’s gland hyperplasia is usually asymptomatic. It can take form of polypoid or mass lesions and precipitate pyloric obstruction, gastrointestinal hemorrhage, and intussusception in children. Although its benign feature and usually respond to medical treatment, transformation to adenocarcinoma has been reported on rare occasion.

Unfortunately, due to its rarity in pediatric population, it tends to be misdiagnosed. This case report presents a case of seven year old boy with Brunner’s gland hyperplasia presented with recurrent vomiting. A seven year-old boy was admitted to the hospital due to of worsening of intractable vomiting since five months prior to admission. He also felt abdominal discomfort, bloating, and early satiety since he was 3 years old. The patient had been treated with several medications without significant improvement. Epigastric pain was observed on abdominal palpation. Gastroscopy showed partial gastric outlet obstruction (pyloric obstruction), erosive gastritis, multiple ulcers, and erosive esophagitis. Histopathologic results from biopsy revealed chronic gastritis and positive for Helicobacter pylori.

Abdominal CT scan showed protrusion solid lesion intraluminal with diameter 1.18 x 1 x 1.25 cm in part II duodenum. Patient was consulted to pediatric surgeon for laparotomy exploration and duodeno-duodenostomy. Excision of Brunner’s gland dan biopsy were performed. Surgical report showed gastric dilatation with duodenal wall hypertrophy on part I-II of duodenum. Histopathological from biopsy revealed Brunner’s gland hyperplasia with lymphoid tissue. Post surgery patient recovered well and eventually discharged without any complains. The prognosis was good.

CASE DESCRIPTION

A seven year-old boy was admitted to the hospital due to worsening of recurrent vomiting since five months prior to admission. Patient had episodes of vomiting since he was three years old. Mostly he had vomiting once or twice a week. Since five months prior to admission, it was getting worse. Vomiting occurred at any time, more frequent at night. Vomits contain his drinks or foods that he consumed previously. He had been treated with several medication without significant improvement. The prenatal and natal history were normal. Patient was diagnosed with congenital cataract on 1 month old. None of his family had the same complaints.

On admission, vital signs were normal. His body weight was 16 kg, body height was 111 cm, and Body mass index (BMI) was 111 cm, and Body mass index (BMI)
was 13 kg/m². His nutritional status was moderate malnutrition. Patient had leukocoria on his right eye. On abdominal palpation, intermittent epigastric pain was observed during abdominal examination. Other physical examinations were within normal limit. Laboratory investigations showed IgG anti-rubella 37.2 IU/ml and IgG anti-cytomegalovirus (CMV) 44 IU/ml. Other laboratory results were normal. Patient was initially assessed as partial gastric outlet obstruction. No medications were given.

During his hospital stay, patient had poor appetite, occasional episode of non-projectile, clear, yellow vomiting, with episodes of bilious vomiting. It occurred mostly two hours following meals. Patient also had occasional abdominal pain. Gastroscopy was performed (Figure 1) and showed partial gastric outlet obstruction (pyloric obstruction), erosive gastritis, multiple ulcers, and erosive esophagitis. Biopsy revealed chronic gastritis and positive for Helicobacter pylori. The patient got treatment for Helicobacter pylori for two weeks. Abdominal CT scan was performed and showed protrusion solid lesion intraluminal with diameter 1.18 x 1 x 1.25 cm in part II duodenum without sign of malrotation. (Figure 2)

Patient was consulted to pediatric surgeon for laparotomy exploration and duodenal-duodenostomy. Excision of Brunner's gland dan biopsy were performed. Surgical report showed gastric dilatation with duodenal wall hypertrophy on part I-II of duodenum. Histopathological from biopsy revealed Brunner's gland hyperplasia with lymphoid tissue proliferation (Figure 3 and 4). Post surgery patient recovered well and eventually discharged. During follow up, patient had no complains of vomiting and in good condition.

**DISCUSSION**

Brunner's gland are mucosal and submucosal glands that secreting alkaline and commonly located in the duodenum. It also can be found in small amounts in pylorus and jejunum. The highest concentrations of Brunner's gland can be found in the first part of duodenum. It is gradually decreasing in number in the second and third portions. The main function of Brunner's gland is to secrete alkaline substances and bicarbonate to neutralize acidic chyme and gastric. Brunner's gland also secretes mucus, pepsinogen, and urogastrone, in response to the presence of acid. In a day, these glands are secreting approximately 200 milliliters of alkaline mucus with pH 8.1-9.3 to neutralize chyme. Thus, it can protect the integrity of duodenal mucosal epithelium and maintain alkaline environment in the small intestine for intestinal absorption.

Brunner's gland hyperplasia (BGH) is a benign proliferative lesion of the duodenum. The lesion mostly less than 0.5 cm, which is characterized as neutral mucin-containing glands expanding at least 50% of duodenal mucosa in a biopsy specimen. Endoscopically, Brunner's gland hyperplasia appears as submucosal nodules in the first or second portion of duodenum. Histologically, Brunner's gland hyperplasia is a single or multiple nodular lesion of excessive Brunner's
CASE REPORT

Brunner's gland hamartoma. Lymphocyte infiltration was found on inflammation also plays an important role in pancreatic exocrine insufficiency. This might be attributed to an adaptive response factor of Brunner's gland hyperplasia. This Chronic pancreatitis is a contributing factor of Brunner's gland hyperplasia. This might be attributed to adaptive response to pancreatic exocrine insufficiency. Inflammation also plays an important role in Brunner's gland hyperplasia since vast lymphocyte infiltration was found on Brunner's gland hamartoma. In our case, the biopsy of gaster revealed Helicobacter pylori infection was positive.

Various expression of mucin protein expressions has been involved in the pathogenesis of many non-neoplastic and neoplastic conditions in the gastrointestinal tract. In normal condition, Brunner's glands express MUC6, a mucin secreted by gastric pyloric gland mucin meanwhile MUC 5AC are expressed in gastric foveolar epithelium. In Brunner's glands hyperplasia, changes in morphologic are thought to be reactive changes that can be described in two ways. The changes in surface epithelial or large size frequently exhibited sclerotic glandular foci is the first mechanism. When it is enlarged, the outlet of the Brunner's gland may be obstructed by destruction of surface mucosa and glandular complexity that cause changes in morphology of Brunner's glands.

Later, dilatation of Brunner's glands tends to mimic to that of sclerotic glandular foci related with changes in mucin protein expression. The expression of MUC6 and MUC5 was loss in both, implied that they have a similar origin.

Most patients with Brunner's gland hyperplasia are asymptomatic as it is generally an incidental histologic finding on routine biopsies performed during endoscopies of the upper gastrointestinal tract. Clinical manifestations will depend on the size, type, and location. A large-size intraluminal mass or circumferential hyperplasia can cause intestinal obstruction. Erosion or ulceration of can lead to upper gastrointestinal bleeding. Brunner's gland hyperplasia usually presents in middle age with no sex predominance; however, cases have been described from early infancy to 80 years of age. More than half of the patients present with abdominal pain, 43% have melena, and 12% experience hematemesis. Hemorrhage is more likely to occur in distal tumors than those from the first part of the duodenum. Duodenal obstruction and intussusception are less common presentations. Gastrointestinal obstruction occurs when the nodules of Brunner's gland hyperplasia is large enough. In adults, the average diameter should be greater than 2.1 cm. In our case, the clinical manifestations was intractable vomiting and signs of gastric outlet obstruction. The dimension of Brunner's gland hyperplasia was 1.18 x 1 x 1.25 cm. Other pediatric case, an 8-year-old boy with Brunner's gland hyperplasia showed epigastric pain associated with significant non bilious vomiting without significant weight loss.

Diagnosis of Brunner's gland hyperplasia is usually confirmed by imaging studies and gastroscopy. Endoscopy can provide direct visualization and accurate location of Brunner's gland hyperplasia. However, when it is located in the posterior wall of the duodenal bulb, transitional part and the beginning of descending part, it can be missed during endoscopy. The use of barium x-ray and CT scan can be complimentary approaches to minimize the missed diagnosis. The exact diagnosis requires pathologic findings. Endoscopy can localize the lesions, however biopsies usually negative. A deep endoscopic or surgical biopsies will provide adequate tissues because Brunner's gland hyperplasia is usually covered by normal mucosa. Its shows a wide range of histopathological findings ranging from extensive proliferation of mucinous glands arranged in the form of lobules, pedunculated adenomatous or rarely as hamartomatous lesions. It characterized by small polyloid lesions of excessive Brunner's gland separated by fibrous septa. In the case, endoscopy couldn't localize the lesion. It was found by abdominal CT scan and biopsy from surgical approach could confirm the diagnosis.

The management of Brunner's gland hyperplasia depends on the size and the clinical manifestations. For asymptomatic or small lesion, conservative treatment is performed. While excision is recommended for the large lesion. For symptomatic patients, endoscopic or surgical resection should be considered. In our case, the surgery was performed to remove the glandular hyperplasia. Generally, Brunner's gland hypertrophy is benign and has a good prognosis. However, with the growth of benign proliferative lesions of Brunner's glands, mucosal ulcers may develop, thereby leading to the repair of gastric foveolar metaplasia with papillary architecture and then malignant transformation. It has been reported that 2.1% of the Brunner's gland hyperplasia evaluated had dysplasia and 0.3% invasive carcinoma. There are several warning signs of potentially malignant transformation of Brunner's gland hyperplasia. The size of polypoid lesions increases with a change in the morphologic is the first warning...
sign. Second, a submucosal tumor-like lesion that accompanied with shallow central depression. 9

CONCLUSION
Our case didn’t show warning signs and had no signs of obstruction and bleeding after surgery, so we conclude the prognosis was good.

CONFLICT OF INTEREST
The authors declare that there is no competing interest regarding the manuscript.

ETHICAL CONSIDERATION
This research was conducted based on the ethical conduct of research from the Ethics Committee of the Medical Faculty, Universitas Airlangga

FUNDING
The authors are responsible for the study’s funding without the involvement of a grant, scholarship, or any other funding resource.

AUTHOR CONTRIBUTION
All authors contributed to the study from the conceptual framework, data gathering, and analysis until the study’s results were interpreted upon publication.

REFERENCES
6. Buchanan EB. Nodular Hyperplasia of Brunner’s the Duodenum Glands of. 1961;101

This work is licensed under a Creative Commons Attribution