Review of Treatment Strategies. *Journal of Gastroenterology and Hepatology Research* 2015; 4(7): 1698-1701 Available from: URL: http://www.ghrnet.org/index.php/joghr/article/view/1173

**INTRODUCTION**

Brunner’s gland hyperplasia is a benign lesion arising from the duodenum. It accounts for 10.6% of benign tumors in the duodenum and is often an incidental finding on endoscopy with the majority of patients being asymptomatic\(^1\). Most lesions are less than 1cm in size and account for about 6.9% of endoscopically removed polyps in the duodenum\(^1\). Rarely Brunner’s gland hyperplasia can result in gastrointestinal bleeding, obstruction and abdominal pain\(^2\). We report an unusual case of Brunner’s gland hyperplasia leading to severe gastric outlet obstruction requiring surgical intervention.

**CASE REPORT**

A 59 year old female with history of hypertension presented with acutely worsening oral intolerance for 3 days. Patient reported symptoms initially began with early satiety and progressed to vomiting 3 months prior to presentation. Review of symptoms was otherwise negative. On presentation patient was having non-bilious non-bloody episodes of vomiting daily, occurring immediately after meals for 3 days and reported a 25lb weight loss over 2 months. Physical exam was notable for mild discomfort in epigastric region upon deep palpation. Labs were notable for a creatinine of 3.32 (normal, 0.6-1.3mg/dL). CT done at that time showed gastric distension possibly secondary to gastric outlet obstruction. Upper GI series showed distension of the stomach with retention of contrast material most suggestive of a near complete gastric outlet obstruction (Figure 1). Esophagogastroduodenoscopy (EGD) showed pyloric channel narrowing and a clean-based antral ulcer (Figure 2).

Biopsies were negative for malignancy. Patient underwent exploratory laparotomy and distal gastrectomy with gastrojejunostomy.
Gross examination of the distal gastrectomy was described as a 14cm × 7cm × 2cm portion of distal stomach and duodenum. The serosal surface was unremarkable, smooth with a pink tan. When opened, the mucosal surface exhibited a 2cm × 2cm polypoid mass that was 0.5cm from the pylorus. Pathology was consistent with Brunner’s gland hamartoma (Figures 3 and 4). Patient recovered well and remained symptom free at 6 month follow up.

DISCUSSION

The Brunner’s gland is found in the proximal duodenum and secretes alkaline-based mucus to protect the duodenal lining from gastric acid. The etiology of Brunner’s gland hyperplasia is not known. It is hypothesized that excess gastric acid secretion or increased inflammation may lead to hyperplasia[2]. Previously Brunner’s gland hyperplasia was divided into circumscribed nodular hyperplasia, diffuse nodular hyperplasia and adenomatous hyperplasia, however recently it is hypothesized that these represent progression of a single clinical entity[3]. Conditions associated with Brunner’s gland hyperplasia include uremia, chronic pancreatitis and Helicobacter pylori infection[4-6]. On extremely rare occasions adenocarcinoma has been shown to arise from Brunner’s gland hyperplasia with immunohistochemistry evidence of increased p53 antigen expression within the gland[7]. Often Brunner’s gland hyperplasia is asymptomatic and only diagnosed incidentally on endoscopy. It is most often found in the duodenal bulb where it appears as a mucosal protrusion or polyp ranging from 0.5 to 1.5 cm in size[2]. Brunner’s gland hyperplasia becomes rarer as you get further from the duodenal bulb: duodenal bulb 57% of cases, second portion of duodenum 27%, third portion of duodenum 7%, jejunum 2%, terminal ileum 2% and 5% found on the pylorus[9]. Patients most commonly present with abdominal pain and gastrointestinal bleeding, but obstruction and intussusception are rare clinical presentations[10].

Although radiographic imaging and EUS can aid in diagnosis, definitive diagnosis requires tissue pathology[11]. It is estimated that duodenography has a reliability of 61% and a 20% false negative rate[12]. Endoscopy has a sensitivity of 72-89%, however biopsies may be non-diagnostic as lesions are submucosal and may be missed on pinch biopsy[13]. Treatment is resection via endoscopy, laparoscopy or laparotomy. Endoscopic polypectomy is a cost effective, less invasive approach that has been shown to be an effective and safe alternative to surgery[14]. In our extensive literature review we found that size and pedunculation were important factors in determining which treatment modality was pursued[15-26]. Typically, endoscopic polypectomy was...
attempted in patients with polyps of less than 5 cm in size and with a pedunculated characteristic[18,20,21,23]. Surgery was the preferred modality for polyps 5 cm or larger[16,17,22,26].

One unique aspect of our case is the finding of an antral ulcer that may have been associated with the patient’s Brunner’s gland hamartoma. To our knowledge there has only been one case report of a concurrent antral ulcer and Brunner’s gland hamartoma[27]. While the exact pathophysiology of this phenomenon is unclear, it may be that the gastric outlet obstruction leads upstream mucosal damage leading to peptic ulcers, gastritis and esophagitis. Fuse et al. measured the thickness of the Brunner’s gland in 297 cases of surgically resected peptic ulcer disease and found a negative correlation between distance of the peptic ulcer from pylorus and thickness of the Brunner’s gland[28]. The authors’ concluded that this pattern was likely due to increased gastric acid secretion leading to hyperplasia of the Brunner’s gland. Another possible relation between peptic ulcers and Brunner’s gland hyperplasia is that both have an association with Helicobacter pylori. Kovacevic et al. conducted a prospective study involving 19,100 patients of which 5 patients were found to have concurrent H. pylori infection from the 7 patients found to have Brunner gland adenoma[6]. Although it seems Helicobacter pylori infection may be common in patients with Brunner’s gland adenoma, the exact pathogenesis is not known. The patient in this case tested negative for Helicobacter pylori. The severity of the patient’s presenting symptoms, with near complete gastric outlet obstruction, also makes our case distinct. The obstruction was so severe that the hamartoma could not be accessed through endoscopy. Thus the patient underwent laparotomy and is the setting of her very severe gastric outlet obstruction, distal gastrectomy with gastrojejunostomy was performed.

CONCLUSION

In summary, we presented an unusual case of Brunner’s gland hyperplasia that led to severe gastric outlet obstruction requiring surgical intervention. Our case supports the notion that in evaluation of patients with gastric outlet obstruction, a benign pathology of Brunner’s gland hyperplasia should be included into the differential diagnosis. Furthermore, on review of the literature we conclude that management of gastric outlet obstruction from Brunner’s gland hyperplasia depends on severity of symptoms and endoscopic accessibility.

CONFLICT OF INTERESTS

There are no conflicts of interest with regard to the present study.

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