Chronic Pancreatitis with Brunner’s Gland Hyperplasia of Ampulla Vater and Duodenal Obstruction

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Hyperplasia or tumors of Brunner’s glands in the duodenum are rare and many of them have been incidental findings. The most common symptom was hemorrhage. Duodenal obstruction and its resulting pancreatitis are extremely rare. Herein, we reported a 33-year-old male alcoholic presented with recurrent chronic pancreatitis and progression of bowel obstruction. Image and clinical surveillance revealed duodenal obstruction at the level around the Ampulla Vater. Although biopsy did not yield malignancy, he underwent the Whipple operation with uneventful course and the patient resumed active work 2 months after the operation. The pathologic test results showed Brunner’s gland hyperplasia at the Ampulla Vater with destruction of the pancreatic duct.

Key words: Brunner’s gland hyperplasia, chronic pancreatitis, duodenal obstruction

Introduction

Brunner’s glands are located almost exclusively between the pylorus and the Ampulla Vater. The pathogenesis of Brunner’s gland hyperplasia (BGH) remains completely unknown and many of its symptoms are nonspecific. BGHs are not premalignant and surgery should usually be conservative. Our case was complicated with obstruction and four previous episodes of chronic pancreatitis with acute exacerbation resulting in a hard induration in the pancreatic head. The Whipple operation was a feasible option in our case.

Case Report

A 33-year-old man was admitted for evaluation of abdominal pain, vomiting and general weakness. The patient had a 15-year history of alcohol abuse. During the 6 months prior to this admission, he had been hospitalized four times for acute and chronic pancreatitis, duodenal ulcer with bleeding, and electrolytes imbalance. On physical examination, the patient was anicteric, and mild abdominal distension and epigastric tenderness were noted. Blood tests revealed the following: serum amylase 387 u/l (normal, 25-125 u/l); lipase 309 u/l (normal, 7-58 u/l); alkaline phosphatase 64 Iu/l (normal, 40-120 Iu/l); blood sugar 169 mg/dl; GOT 23 Iu/l; GPT 7 Iu/l; total bilirubin 0.5 mg/dl, and direct bilirubin 0.1 mg/dl. Upper GI barium meal showed a distended stomach and outlet obstruction due to focal circumferential stenosis involving the post-bulbar region (Fig. 1).
The second portion of the duodenum was found to have mucosal swelling and near total obstruction under panendoscopy. Biopsy yielded mucosal erosion and inflammation. Abdominal computed tomography revealed duodenal wall thickening and an edematous mass lesion arising from the pancreatic head. The pancreatic body and tail were well demarcated (Fig. 1). The patient underwent conservative treatment for 2 weeks including nasogastric tube decompression, adequate hydration and proton-pump inhibitor administration, however, the symptoms persisted. Under the impression of duodenal obstruction and periampullary tumor with pancreatic head lesion, surgery was considered. During the operation, a firm 5-6 cm mass was noted around the periampullary region causing obstruction. The pancreatic body and tail were soft. A pancreaticoduodenectomy with a pancreaticogastrostomy and an end-to-side hepaticojejunostomy were performed. Grossly, the periampullary duodenal mucosa showed segmental stricture, focal hemorrhage and granularity circumferentially, and there were areas of necrosis and fibrosis in the pancreatic head (Fig. 2). The postoperative course was uneventful and the patient was discharged on postoperative day 18 and resumed active work 2 months after the operation. The pathologic examination showed circumferential diffuse BGH with multiple sessile projections in the duodenum around the Ampulla Vater, and the most thickened wall was measured 12 mm (Fig. 3). Some glands of the Ampulla Vater and the pancreatic ducts were destroyed, and there was extensive fibrosis in the pancreatic head indicating chronic inflammation (Fig. 4).

**Discussion**

_Brunner’s glands are located almost_
Fig. 2  A firm mass in the pancreatic head including the duodenum (white arrow). Soft and smooth in the pancreatic body and tail (white arrow head). Segmental stricture, focal hemorrhage and granularity circumferentially in the duodenum around the Ampulla Vater (black arrow), and areas of necrosis and fibrosis in the pancreatic head (black arrow head).

Fig. 3  Photomicrograph of duodenum showing pale-staining hyperplastic Brunner’s glands with lobular architecture separated by fibrous septa occupying submucosa and mucosa and extending toward luminal surface and Ampulla Vater (H&E, x20) Inset, Brunner’s glands diffuse nodular hyperplasia with multiple sessile projections
Brunner's glands are exclusively located between the pylorus and the Ampulla Vater, being found only sporadically distal to the papilla\(^1,2\). They are disseminated within the submucosa, which has a mean thickness of 0.42 mm\(^3\). The first detailed morphological differentiation of BGH was undertaken by Feyerter\(^4\) in 1934. In his classical study of BGH in a series of 2800 consecutive autopsies, three types of pathologic changes of Brunner’s glands were described: type 1, diffuse nodular hyperplasia, in which multiple sessile projections are found throughout the duodenum; type 2, circumscribed nodular hyperplasia with sessile projections limited to the duodenal bulb, and type 3, glandular adenoma with polypoid tumor-like projections.

The pathogenesis of BGH remains unknown. However, many researchers have associated this condition with uremia, chronic pancreatitis, and hyperacidity\(^5,6\). Although an increased acid load on the duodenal mucosa has been linked with this entity, it does not by itself explain the development of BGH, which is not present in many duodenal ulcer patients. In a series by Stolte et al., hyperplasia of Brunner’s glands, found in 75.7% of chronic pancreatitis cases, was exclusively of the diffuse type\(^7\). Their hypothesis for the cause of BGH in chronic pancreatitis was the result of the exocrine insufficiency of the pancreas, where the glands were stimulated not only functionally, but also trophically. Other cases of pancreatitis due to obstruction have been reported\(^8,9\). Yamaguchi et al\(^10\) reported an association between groove pancreatitis and BGH in five of eight patients undergoing pancreaticoduodenectomy. However, this phenomenon may have also been caused by chronic alcohol ingestion, as four of the BGH patients in their series were chronic alcoholics. In our case, the pathophysilology was chronic pancreatitis associated BGH which resulted in duodenal obstruction.

A variety of symptoms have been described in patients with BGH. Many of these symptoms are nonspecific and include mild epigastric pain,
gastrointestinal bleeding, and obstruction. Only seven cases associated with obstruction have been reported in the literature, and only one case was reported with pancreatitis as the presenting symptoms\(^9\). Our case of BGH was relatively rare because he had both pancreatitis and duodenal obstruction.

The definite management of BGH is somewhat controversial. All authors agreed that these tumors are not premalignant and surgery should be conservative and determined by the symptoms produced. Most solitary polyps can be managed with polypectomy. In those patients with hyperacidity refractory to proton-pump inhibitors, some form of ulcer operation might be considered, usually vagotomy with either antrectomy or pyloroplasty. A duodenal bypass procedure may be required for a patient with diffuse or circumferential hyperplasia with obstruction. In a case reported by Mayoral et al.\(^9\), a biliary stent was placed to resolve the biliary obstruction and pancreatitis resulting from BGH of the Ampulla Vater. However, the stent became clogged as the disease progressed, and the patient eventually underwent the Whipple operation. Our case was complicated with obstruction and four previous episodes of chronic pancreatitis with acute exacerbation resulting in a hard induration in the pancreatic head. The Whipple operation by an experienced surgeon may be a feasible option to manage obstruction and pancreatic head mass at the same time while avoiding further operations.

In conclusion, we presented a patient with chronic pancreatitis with BGH of the Ampulla vater and duodenal obstruction. Initial histological findings were misleading. Definitive diagnosis was difficult and required expert pathologic review. Cure was accomplished after a Whipple operation. Diffuse type of BGH, related to chronic pancreatitis, should be added to the list of entities causing ampullary masses and duodenal obstruction. Surgical resection appears to provide the best management option for symptomatic cases at this time.

References

慢性胰臟炎併十二指腸壟腹Brunner氏腺體增生及十二指腸阻塞：病例報告

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Brunner氏腺體主要生長於幽門與十二指腸壟腹之間，大多數Brunner氏腺體產生的腫瘤大多數無症狀，或僅輕微出血，至於造成胰臟炎與腸阻塞實屬罕見。本文報告一例23歲男性反覆上腹痛與胰臟炎發作，經檢查為十二指腸壟腹處腫瘤造成腸阻塞及胰頭處胰臟炎，藉由十二指腸胰頭切除術治癒。病理證實為十二指腸壟腹處Brunner氏腺體增生造成腸阻塞與胰頭腺體破壞。查閱文獻記載，類似案例僅有少數，手術方式以症狀治療為主，本案例則藉以Whipple氏手術處理十二指腸壟腹處阻塞及胰頭處胰臟炎。僅以此案例，作相關文獻分析與討論。

關鍵詞：Brunner氏腺體增生，慢性胰臟炎，十二指腸阻塞