Brunner’s gland adenoma of duodenum: A case report and literature review

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Abstract
AIM: To analyze the clinicopathological features of Brunner's gland adenoma of the duodenum.

METHODS: A rare case of Brunner's gland adenoma of the duodenum was described and related literature was reviewed.

RESULTS: Brunner's gland adenoma of the duodenum appeared to be nodular hyperplasia of the normal Brunner's gland with an unusual admixture of normal tissues, including ducts, adipose tissue and lymphoid tissue. We suggested that it might be designated as a duodenal hamartoma rather than a true neoplasm.

CONCLUSION: The most common location of the lesion is the posterior wall of the duodenum near the junction of its first and second portions. It can result in gastrointestinal hemorrhage and duodenal obstruction. Endoscopic polypectomy is a worthy treatment for benign Brunner's gland adenomas, as malignant changes in these tumors have never been proven.

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INTRODUCTION
Brunner’s gland adenoma, also known as Brunneroma or polypoid hamartoma, is a rare, benign, proliferative lesion arising from the Brunner’s glands of the duodenum, accounting for 10.6% of benign tumors of the duodenum. The time patients are usually asymptomatic and lesions are discovered incidentally. These lesions manifest occasionally as a rare cause of duodenal obstruction or upper gastrointestinal hemorrhage, and require surgical excision[1]. This article reports a case of Brunner’s gland adenoma, and reviews briefly its clinical presentations, pathological features and therapy.

CASE REPORT
A 32-year-old Chinese man complained of two episodes of melena and a two-month history of vague epigastric discomfort. The patient had in October 2001 his first tarry stool episode without nausea, vomiting and epigastralgia. Endoscopic examination revealed a mild ulcer in the duodenal bulb. The symptoms were relieved by antacids and H2 blockers. He reported however another tarry stool in September 2002 and complained about epigastric pain without nausea and vomiting during the preceding 2 mo. Antacids were taken and the pain was relieved. Vital signs were normal and no anemia was reported when he was admitted. The physical examination had no remarkable finding. The abdomen was soft without palpating pain and jumping pain.

However, X-rays barium radiological examination of the upper gastrointestinal in October 2002 revealed a nodular, polypoid-filling defect mass measuring 3 cm×2.5 cm with a smooth surface and no ulceration in the duodenal bulb. A tumor was suspected. Follow-up endoscopy disclosed a lobulated, red-color tumor, occupying the anterior wall of the bulb. The surface of the tumor was smooth with mild depression at the top. The mucosa had no erosions and ulcers. Multiple biopsy specimens were taken and interpreted as “mucosal mild-medium atypia.” CT scan was negative. A preoperative diagnosis of malignant tumor was made and the patient prepared for operation. On October 25, 2002, at the time of operation, a pedunculated polyp on a short broad-based stalk, 3.5 cm×3 cm×2 cm in size, was found in the anterior wall of the duodenal bulb. The common bile duct was normal. The lesion was amputated at the base of the stalk. The resected specimen showed a lobulated, polypoid mass measuring 3.3 cm×2.5 cm×2.5 cm projecting into the duodenum. The stalk was measured 1.8 cm in diameter and 0.5 cm in length. The tumor was completely enveloped by the intact thin duodenal mucosa. The surface of the tumor was smooth without erosions and ulcers. The cut surface of tumor had a gray-red or gray-yellow color, revealing lobules. The consistency was moderate. On microscopic examination, the tumor was composed of hyperplasia of Brunner’s glands. The hyperplasia formed lobules that were separated by intervening bands of fibrous tissue, adipose tissue, ducts and well developed aggregated lymphoid. No sign of malignancy was found in the hyperplasia, Brunner’s glands as well as the surrounded duodenal mucosa. The frozen sections and the final pathologic diagnosis were assessed as Brunner’s gland adenoma. The patient had an uneventful postoperative course and was discharged on the tenth postoperative day. He has remained symptom free ever since and no episode of recurrent melena has been reported.

DISCUSSION
Besides the duodenal gland, the duodenum has Brunner’s glands under the mucin. Its structure and function are similar to glands of the pylorus. Brunner’s glands secrete an alkaline fluid composed of viscous mucin, whose function appears to protect the duodenal epithelium from acid chyme of the stomach. Brunner’s glands consist of submucosal mucin-secreting glands located exclusively in the duodenum. They extend from the pylorus distally for a variable distance, usually stopping at the first and second portions of the duodenum, and less often stopping at the third and fourth portions.

In 1688 Brunner gave a precise anatomic description of the duodenal submucosal glands and coined the term “pancreas secundarium.” In 1846 Middeldorpf correctly identified these glands as a separate entity, which he proposed be named Brunner’s
glands. Salvioli reported the first adenoma of Brunner’s gland in 1876. Since then, 150 cases or so have been reported in literature of English language [2].

The etiology of Brunner’s gland adenoma remains obscure. It tends to present predominantly in the fifth and sixth decades of man’s life with no sex predominance. It has been found although the size of adenoma might extend from 1-12 cm, it is generally 1-2 cm in diameter [3]. The most common location for the lesion is the posterior wall of the duodenum near the junction of its first and second portions. Brunner’s gland adenoma was rarely found extending to the proximal jejunum [4].

Brunner’s gland adenoma has fallen into two categories: symptomatic tumors and asymptomatic ones that are only found incidentally. Symptomatic tumors can further be divided into hemorrhagic and obstructive tumors. The clinical manifestations of the former are gastrointestinal hemorrhage, due to ulceration or erosion of the tumor. Obstructive tumors occur when hyperplasia diffuses or a single adenoma grows too large, causing epigastric bloating, discomfort, vomiting or weight loss. Duodenal intussusception has been reported only in two patients [2], probably because of the fixation of duodenum to the posterior abdominal wall. There are also reports about patients who complained of diarrhea owing to duodenal motor disturbances [5].

Preoperative histological diagnosis at present is not always easy. In X-rays barium examination, the findings are often nonspecific because there is usually a sessile or pedunculated polypoid-filling defect in the duodenal bulb. Some doctors hold that hypotonic duodenography should play a vital role in establishing the diagnosis and should be treated as the best method to check the surface of the lesion. Endoscopy has an additional function in diagnosing and treating Brunner’s gland adenoma, since it can verify the histological diagnosis and remove the tumor simultaneously. Endoscopic pinching biopsy however usually gave a negative result because the tumor was almost covered entirely with thick intact duodenal mucosa in the biopsy sites and the biopsy was often not deep enough to reach the submucosal tumor tissue [6]. In our case, the endoscopic biopsy was negative, and the condition was diagnosed as chronic gastritis, although the final pathologic diagnosis indicated that the patient suffered from Brunner’s gland adenoma. CT examination appeared to be unrevealing.

Pathomorphological features of Brunner’s gland are characterized by the presence of nondysplastic, lobulated Brunner’s glands. Its hyperplasia is divided into diffuse hyperplasia, nodular hyperplasia and adenomatous hyperplasia with or without erosion or ulcer. In our opinion, the unusually admixture of normal tissues, including Brunner’s glands, ducts, adipose tissue, and lymphoid tissue, supports the designation of these lesions as a hamartoma or nodular hyperplasia rather than a true neoplasm. It is a tumor without malignant predisposition. The malignant type is rare. Fujimaki et al. reported recently one patient with a focus atypical gland [7].

It is still controversial whether asymptomatic Brunner’s gland adenoma found incidentally needs surgical removal. Some people think that it needs no treatment, whereas others hold that it should undergo endoscopic excision in order to prevent complications. There have been several reports [2, 4] that Brunner’s gland adenoma could give rise to acute profuse bleeding, which results in shock of patients. Symptomatic Brunner’s gland adenoma, in our point of view, usually needs surgical treatment. When the tumor is small or pedunculated, endoscopic polypectomy is the first choice. Open surgical excision is reserved for cases where snaring has failed or when tumor is too large. The outcome of operation is usually excellent and there is no recurrent ever reported.

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