Hyperplasia of Brunner's glands

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ABSTRACT

A case of tumor like hyperplasia of Brunner's glands surgically removed from the oral end of the duodenum of 52-year-old woman was reported and the pathogenesis of the lesion was briefly discussed.

Key Words: duodenal polyp; tumor of the duodenum; hamartoma of the dumodenum; duodenal adenoma.

INTRODUCTION

Benign tumors of the duodenum attract attention because of their infrequent occurence and low prevalence among gastrintestinal neoplasms ¹⁻⁴. Pathologic proliferation of Brunner's glands has been variously described as adenoma, adenomatous polyp, polyp, circumscribed nodular hyperplasia ¹⁻³. The pathogenesis and cause of these tumors are still in doubt. In this paper, a case of hyperplasia of Brunner's glands of the duodenum is presented.

CASE

A 52-year-old female was admitted to Saiseikai Shimonoseki Hospital with chief complaint of nausea and vomiting. She had right lower abdominal pain associated with low grade fever. Physical examination revealed no abnormality. There were no episodes of melena, hematoemesis, diarrhea or jaundice. She had histerectomy for uterine myoma about ten years ago. Family history was not remarkable. Laboratory examination was within normal limits (Table I).

Examination of the gastrointestinal tract disclosed a polypoid lesion at the duodenal bulb. By endoscopic examination, it was hemispherical in shape and the surface was covered by the smooth intact mucosa. No

Table	Τ.	aboratory	Data
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RBC	$404\times10^3/\text{mm}^3$
Hb	13.7g/dl
Ht	41%
WBC	$6500/\text{mm}^3$
Protein	6.3g/dl
(Albumin	2.9g/dl)
(Globulin	3.4g/dl)
GTP	4 units
GOT	2 units
Cholesterol	166mg/dl
Cholesterol	166mg/dl
Glucose	94 mg/dl
Urea N	7 mg/dl
NPN	26mg/dl

significant obstruction was noted secondary to this duodenal lesion. Partial gastro-duodenectomy was performed. Her postoperative course was uneventful. Gastric analysis could not be performed.

GROSS APPEARANCE

A sessile polypoid tumor $2 \times 2 \times 2$ cm. with rubbery consistency on the anterior wall of the duodenal bulb. Its external surface was smooth and there was no evidence of ulceration on the mucosal aspect. The cut surface revealed that the bulk of the tumor was composed of a tan yellow, finely lobulated parenchyma.

MICROSCOPIC APPEARANCE

The tumor was in the submucosa and covered by intact normal duodenal mucosa which was hemispherically elevated. There were no distinct capsule and invasion. It consisted of mucous secreting epithelial cells (PAS stain positive) similar in appearance to normal Brunner's glands cells. The strands of smooth muscle fiber and fibrous connective tissue divided the tumor into lobules. Ducts were scattered throughout the glandular lesion. Occasionally the duct could be traced into their termination in the individual acinus. Some of these glands lined with flattened or cuboidal epithelium showed cystic dilatation. In parts, focal collections of lymphocytes resembling rudimentary lymph follicles were noted. Paneth cells were absent in the tumor. There was no evidence of atypia or of malignant change, and no mitotic figures were present.

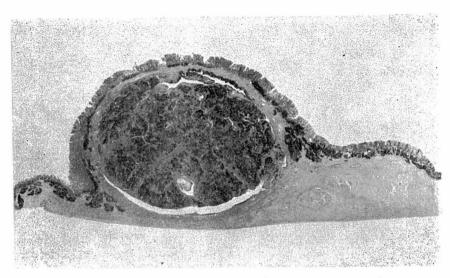


Fig. 1. Low powered view of the tumor. The darker area represents the Brunner's glands. (PAS $stain \times 2.5$)

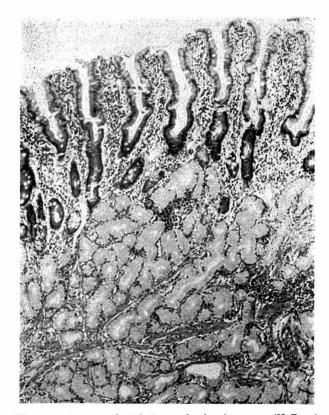


Fig. 2. The tumor is covered with intact duodenal mucosa. (H-E stain, \times 100)

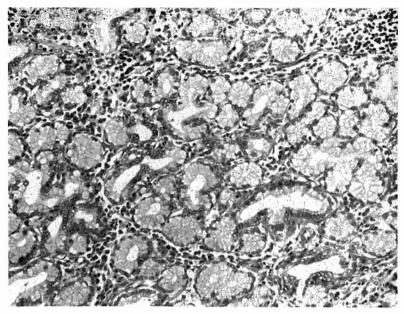


Fig. 3. The tumor consists of well-differentiated Brunner's glands subdivided into lobules by fibro-muscular septa with marked infiltration of lymphocytes. (H-E stain, ×2(0)

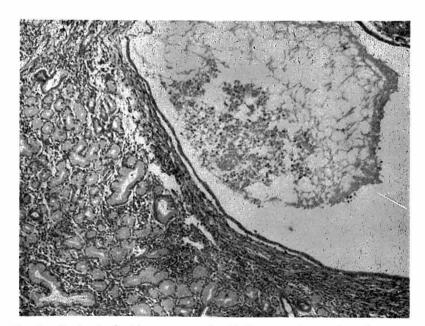


Fig. 4. Cystic gland with an attenuated epithelium contains mucin and neutrophils. (H-E stain, $\times 100$)

COMMENT

Benign tumors of the duodenum are infrequent and in paticular the tumor of the Brunner's glands are scarce^{3,5)}. Brunner's gland tumors are usually found in the first portion of the duodenum. Feyrter⁶⁾ classified these lesions into three types, i.e., circumscribed nodular hyperplasia, diffuse nodular hyperplasia and adenoma. Most of the cases reported as Brunner's gland adenoma are not true adenoma^{5,7}). Several authors have postulated that the changes might be hamartomatous rather than neoplastic4,8). However, it is still controversial whether it is hyperplasia or hamartoma. The tumor is similar to structurally normal Brunner's gland with no demonstrable atypism although the inflammation is evident. These histological findings seem to favor the view that the lesions are hyperplasia of Brunner's glands. The lesion might be induced in most of the cases by some abnormal stimuli, e.g., neural or hormonal and some authors considered possible implication of local irritation^{3,6)}. A theory that hyperplasia of the Brunner's glands occurs as a response to hypersecretion of gastric acid had been proposed⁹⁾. However, Robertson could not find correlation of incidence between peptic ulcer and hyperplasia of Brunner's glands¹⁰. In some reported cases, there are smooth muscle, Paneth cells and adipose tissue in addition to Brunner's glands^{4,8,11)}. The exact pathogenesis is not well understood.

Although the tumor is usually very small, there is a report of a case with large tumor measuring $12 \times 10 \times 8$ cm¹²⁾. A common clinical manifestation is a slow and persistent loss of blood due to ulceration of the overlying mucosa and it leads to iron deficiency anemia, but sometimes the patient dies from massive hemorrhage³⁾. This picture may be confused with bleeding from peptic ulcer. Large tumor may cause pyloric obstruction. Epigastric distress, bloating, vomiting and weight loss were frequent³⁾. However, in many instances the complaints of the patients were nonspecific and their relation to the pathologic lesions were questionable.

The surgical treatment should be conservative and partial gastrectomy lead to a satisfactory outcome since the lesion is not malignant³⁾. In fact, the existence of carcinoma of Brunner's glands is doubtful.

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