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Case Report

Heterotopic and metaplastic gastric mucosa in the duodenum: a case report

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Abstract: We report a rare case of heterotopic and metaplastic gastric mucosa in the duodenum. Observations through histology, immunochemical analysis as well as special staining methods showed mural cells, advocate cells, endocrine cells, and mucus cells which are different in the duodenal bulb compared to the stomach glands. This results in the secretion of gastric acid, pepsin, and chymotrypsin, as well as the possession of gastric tissue structure and function, which can lead to local tissue digestion. Therefore, this report suggests that the base of the duodenum in the duodenal bulb may be one of the underlying causes of duodenal ulcers and patients with chronic inflammation of the duodenal bulb may have ectopic gastrocnemius. Due to this reason, pathological biopsy should be considered as an appropriate, clear etiology and timely ectopic fundic gland tissue local resection to prevent the occurrence of duodenal ulcer.

Keywords: Duodenal bulb, fundic gland, ectopia

Introduction

The fundic gland is normally found in the stomach, consisting of host cells, parietal cells, cervical mucus cells, endocrine cells, and stem cells. They secrete pepsinogen, hydrochloric acid, internal factors, and acidic mucus [1]. Ectopic gastric glandular tissue is when gastric tissue is located outside the stomach, commonly in the esophagus. Notably, ectopic to the duodenal bulb is rarely reported [2]. When the bottom of the stomach gland is found in the duodenal bulb, according to pathology naming methods, it is described as heterotopic and metaplastic gastric mucosa in the duodenum. At this point in the duodenal mucosa lamina propria, good differentiation can be observed and the main cells present are parietal cells and cervical mucus cells of the intact stomach. Pathological examination showing non-atypical gastric fundic tissue in duodenal tissue can be diagnosed as duodenal gastrocnemius. In 1970, Belber first described the morphological characteristics of the disease under endoscopy as nodular, polypoid, granular or flat uplift changes [3]. The disease in duodenal uplift or nodular lesions makes up about 5% [4, 5].

Case report

Clinical materials

The patient is a 54-year-old woman with a history of chronic gastritis for 20 years. She was taken to hospital complaining of acid regurgitation and eructation for 1 week. Admission examination: upper abdominal tenderness, rebound tenderness, and no muscle tension. Gastroscopy examination: bottom of the stomach gastric mucosal hyperemia, edema with funicular erosion but no active bleeding. Mucosal congestion and edema in the duodenal bulb was present along with scattered follicular hyperplasia. The boundary was clear without erosion and ulcers (**Figure 1**), and a small biopsy was taken at the lesion. The admitting diagnosis was chronic gastritis and duodenal bulb inflammation.

Methods

The surgical specimen was fixed in 10% formalin and embedded in paraffin. Hematoxylin

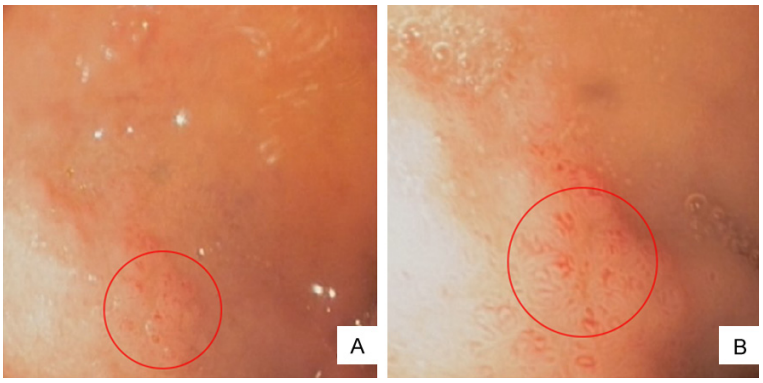

Figure 1. Duodenal bulb gastroscope observation: mucosal hyperemia edema and scattered follicular hyperplasia (A, B).

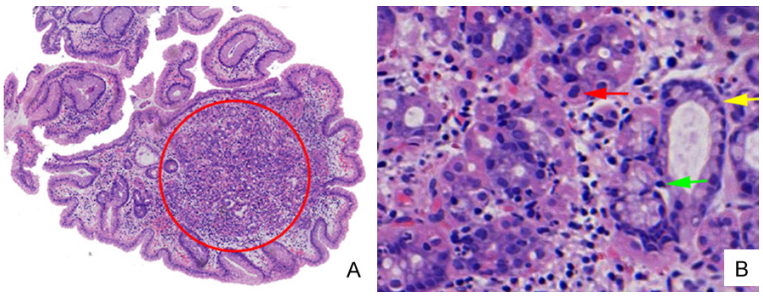

Figure 2. Duodenal bulb microscopic observation: Duodenal bulb mucosa in the lamina propria with stomach gland ectopic tissue. A. Duodenal bulb mucosa in the lamina propria with a nodular lesion (Obj.2×); B. Typical primary cells (green arrow), parietal cells (red arrow), and mucous neck cells (yellow arrow) are seen in the lesion (Obj.40×).

and eosin staining as well as immunohistochemistry were performed for light microscopy on 4 μm sections. Immunostaining was performed using an autostainer (Leica Microsystems, Tokyo, Japan). Antibodies against the following antigens were used at the indicated dilutions: α1-antichymotrypsin (AACT) (2051-98, Abcam; 1:2000), α1-antitrypsin (AAT) (18-0492, Abcam; 1:500), neural cell adhesion molecule (CD56) (R&D Systems; 1:1000), chromogranin A (CgA) (212287, Abcam; 1:1000), synaptophysin (Syn) (18258, Abcam; 1:100), and gastrin (22623, Abcam; 1:100).

Pathological examination

A sesame-sized gray, yellow mass and two pieces of tissue approximately the size of a rice grain were found upon pathological examination. Microscopically, duodenal biopsy tissue was covered with a monolayer of columnar epithelium and the cells were arranged regularly without atypia. Within the lamina propria fundic gland tissue, parietal cells and cervical mucus cells can be seen. In the edema intracellular substance, there appeared to be small scattered lymphocytes, plasma cells, and minor neutrophil infiltration (**Figure 2**).

Immunohistochemical examination

Positive staining was observed for α1-antichymotrypsin (AACT), α1-antitrypsin (AAT), Neural cell adhesion molecule (CD56), chromogranin A (CgA), synaptophysin (Syn), and gastrin (**Figure 3**).

Special staining examination

Periodic acid-Schiff staining (PAS) was performed and showed positive staining (**Figure 3**).

Pathological diagnosis

Micro-ectopic gastrectomy polyps and chronic inflammation with mild acute activity in the duodenal bulb were observed.

Discussion

The diagnosis of ectopic gastric glands

The pathological changes observed under gastroscope were visible follicle hyperplasia. In addition, the main cells, parietal cells and cervical mucus cells observed under the microscope constituting the stomach glandular tissues. Immunohistochemical examination showed positive staining for neuroendocrine markers CgA, CD56, and Syn, suggesting that there are neuroendocrine cells in the stomach and gland. Special staining (PAS) showed positive cervical mucus cells. Therefore, the ectopic stomach gland showed the same integrity as a stomach gland, indicating that lesion was an ectopic fundic gland in the duodenal bulb.

The pathogenesis of ectopic gastrocnemius in duodenal bulb

The duodenum lies between the stomach and jejunum, where it accepts pancreatic juice, gas-

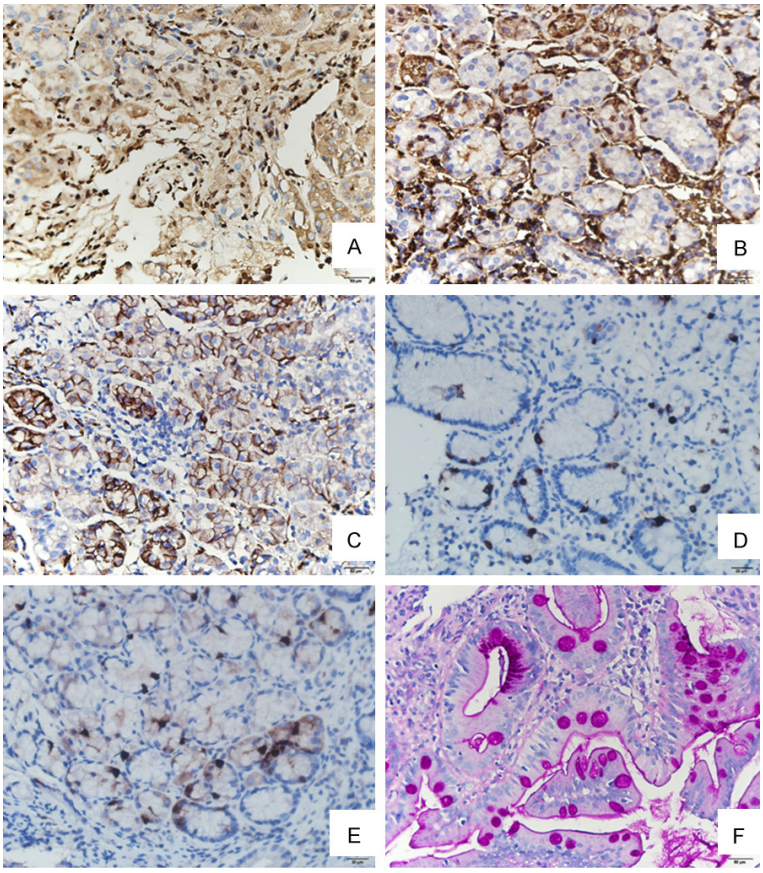

Figure 3. Immunohistochemical and special staining: AACT (A), AAT (B), CD56 (C), CgA (D), gastrin (E) and PAS staining (F). (A-F: Obj.40×).

tric juice, and bile. It is important for small intestinal digestion and absorption and is made up of four parts in structure [6, 7]. The cause of ectopic gastric mucosa in the duodenal bulb is still not clear, but is often thought to be relatively rare congenital residual embryonic tissue. Some scholars believe that the disease can be classified into congenital and acquired, where in the former, lesions are present with complete fundic gland structure and in the latter, most duodenal ulcers or erosion is caused by gastric mucosal epithelial changes without a complete fundic gland structure. Our case in the duodenal mucosa of the lamina propria can be seen within the complete structure of the fundic gland without metaplastic change. Therefore, we believe that the case is a congenital disease.

Pathological and clinical significance of ectopic gastrectomy in duodenal bulb

We believe that ectopic gastric mucosa in the duodenal bulb may lead to duodenal ulcers and inflammation. Common lesions of the duodenal bulb are peptic ulcers through *Helicobacter pylori* infection and other factors [8]. In this case, we not only observed ectopic fundic glandular tissue in the duodenal bulb but also parietal cells, cervical mucus cells, and endocrine cells. We used immunohistochemistry to confirm that these ectopic stomach glands secret α1-trypsin (AAT), α1-pancreatic protease (AACT), and gastrin. As the main cells in the fundic gland, are parietal cells as well as cervical mucus cells, they can secrete pepsinogen, hydrochloric acid, internal factors, and acidic mucus. Therefore, these ectopic features are also capable of acid secretion, digestion, and decomposition with strong corrosive and digestive ability [9]. Due to the mucin-bicarbonate barrier in the duodenal mucosa lacking the gastric mucosal epithelium, this easily leads to self-digestion of duodenal tissue and the formation of peptic ulcers or inflammation. This view has not been reported in the literature.

There is currently no uniform treatment for duodenal peptic ulcer disease at present. Based on the disease characteristics and newly proposed duodenal peptic ulcer pathogenesis, the principle of medical treatment should be to inhibit gastric acid, pepsin, and gastrin secretion. For recurrent episodes of gastrointestinal symptoms, once ectopic stomach glands are noted, there should be endoscopic dissection of ectopic tissues and complete resection of ectopic stomach glands to prevent duodenal peptic ulcers.

According to this case of clinical diagnosis, when there is a clinical experience of chronic gastritis and duodenal bulb inflammation, upon gastroscope examination, attention should be paid to the duodenal bulb for ectopic fundic glands to avoid missed diagnosis.

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Disclosure of conflict of interest

None.

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