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Case Study
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Hypertrophic Gastric Mucosa Related to Gastrin-Producing Neuroendocrine Tumor of Pancreas

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Abstract

Major manifestation of gastrinoma (NET-G1) is frequently gastro-duodenal ulcers and hypergatrinemia. Additionally, hypertrophic gastric mucosa in the areas with oxyntic glands is commonly observed. Because gastrin itself has no direct trohophic action to the gastric epithelial cells in the stomach, the other growth factors are contributing to the gastric mucosal hypertrophy. Here, a high expression of TGF-α was observed in the gastric epithelial cells, which suggests that the produced TGF-α may partially contribute to mucosal hypertrophy via an autocrine or paracrine mechanism in human tissues with NET-G1.

Case Study

A 71-year-old man was admitted for anorexia with body weight loss of 16 kg. A physical examination did not show any significant findings suggesting his symptoms. An upper gastroduodenal endoscopy revealed the multiple scars of gastric ulcer at angular site or the other sites and marked hypertrophic mucosa of the body of stomach (Figure 1). *Helicobacter pylori* (*H.pylori*) infection was negative by a rapid urease test and serum antibody test. Abdominal Computed Tomography (CT) have revealed a hypervascular tumor in the head of the pancreas measuring approximately 7 cm (Figure 2a). Laboratory examination demonstrated extremely high levels of fasting serum gastrin (FSG) (66,110 pg/ml). A biopsy of the pancreas via an endoscopic ultrasound-guided fine-needle aspiration, revealed a typical neuroendocrine tumor (NET). Immunohisotologic study confirmed the tumor was positive for gastrin (Figure 2b). Therefore, the final diagnosis was a gastrin-producing pancreatic NET-G1 with associated mucosal hypertrophy of the gastric body (Figure 2c). The patient did not have a family history of multiple endocrine neoplasia type 1, evidence of hyper-parathyroidism, or pituitary diseases. After the tumor was surgically resected, FSG level was normalized and the prominent gastric folds were improved.

Discussion

Major manifestation of NET-G1 (gastrinoma) is recurrent gastro-duodenal ulcers and hypergatrinemia. Beside gastro-duodenal ulcers or the scars, hypertrophic gastric mucosas in areas with oxyntic glands are frequently observed. However, gastrin itself has no direct trophic action

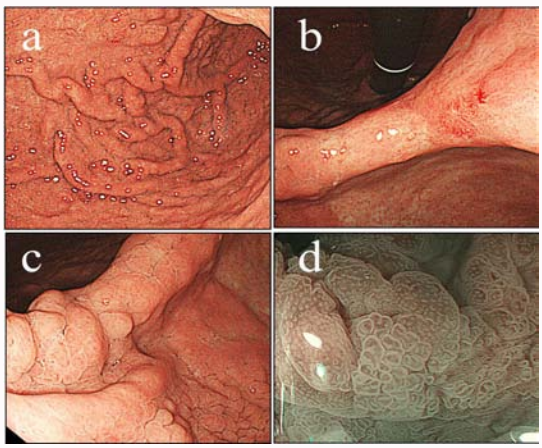

Figure 1: The gastric endoscopy (a, c) and the magnified narrow-band imaging (b) showed marked hyperplastic mucosa of the gastric body and the scar of the stomach (b).

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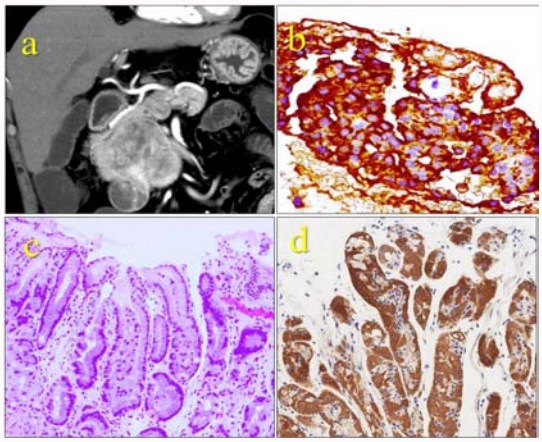

Figure 2: The contrast-enhanced CT revealed a hypervascular tumor in the head of the pancreas (a). The gastrin production in the tumor cells was confirmed by immunostaining (b). Pathological examination demonstrated hyperproliferative epithelium in HE staining (c) and the epithelial cells have shown TGF-α production by immunostaining (d).

to gastric epithelial cells in the stomach [1]. In mouse models with hypergastrinemia, a high level of gastrin has induced the gastric mucosal hypertrophy. Gastrin binds to gastrin/cholecystokinin-B receptors (CCKB-Rs) restrictedly on ECL cells, parietal cells and mucous neck cells or gastric pit cells in the oxyntic glands and TGF-α and/or HB-EGF are produced from the cells [2]. In addition, Reg proteins are mainly producing from ECL cells [3]. Because gastric pit cells are expressing TGF-α receptor and EGF receptor [4], these growth factors might have a proliferative activity directly or indirectly to the pit cells in the oxyntic glands. In our case, a high expression of TGF-α was observed in the gastric epithelial cells besides parietal cells (Figure 2d), which suggests that the produced TGF-α may partially contribute to mucosal hypertrophy partially via an autocrine or paracrine mechanism. This case clearly demonstrated the relation between hypergastrinemia and hypertrophic gastric mucosa via TGF-α production in human tissues with NET-G1 gastrinoma.

References

1. Hakanson R, Oscarson J, Sundler F. Gastrin and the trophic control of gastric mucosa. Scand J Gastroenterol. 1986;118:18-30.
2. Majumdar AP. Regulation of gastrointestinal mucosal growth during aging. J Physiol Pharmacol. 2003;54:143-54.
3. Kinoshita Y, Ishihara S, Kadowaki Y, Fukui H, Chiba T. Reg protein is a unique growth factor of gastric mucosal cells. J Gastroenterol. 2004;39:507-513.
4. Dufresne M, Seva C, Fourmy D. Cholecystokinin and gastrin receptors. Physiol Rev. 2006; 86(3): 805-847.