Polypoid and hyperplastic heterotopic gastric mucosa in the jejunum as a cause of recurrent subocclusive episodes

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Gastric heterotopia occurs throughout the entire gastrointestinal tract, from the oral cavity to the anorectum, and also involves the gallbladder, biliary tract, umbilicus and scrotum [1-10]. The presence of this lesion beyond the ligament of Treitz with recurrent intestinal subocclusive episodes is uncommon [1-10].

We report a case of a 21-year-old woman who had a one-year history of intermittent hypogastric abdominal pain, vomiting and nausea. A physical examination revealed abdominal tenderness with reduced bowel sounds. An abdominal X-ray and a CT scan showed gastric and proximal small bowel distention with multiple air-fluid levels.

An abdominal laparotomy was performed with the following findings: small bowel adhesions and the presence of a large intraluminal tumor affecting the jejunum. The tumor was totally resected.

Upon a macroscopic examination, the specimen was a 25-cm segment of the jejunum containing a large and soft polypoid mass of 15 cm length (Fig. 1). A histological examination revealed that the tumor consisted of gastric type epithelium with hyperplastic foveola and oxyntic glands covered by parietal, chief and neuroendocrine cells.

To analyze cell cycle proteins we counted positive cells per 100 epithelial cells in five randomly selected microscopic fields. p27 was positive in 97% of foveolar cells, in 68% of mucous neck cells and in 20% of glandular cells. p21 was positive in 82% of foveolar cells, in 10% of mucous neck cells and in 2% of glandular cells. p16 and p57 were negative. Cyclin D1 was positive in 87% of foveolar cells, in 75% of mucous neck cells and in 2% of glandular cells. Ki67 was positive in 20% of foveolar cells and 99% of mucous neck cells and was negative in glandular cells.

Several hypotheses have been suggested to explain the origin of gastric heterotopia. Wacrenier *et al* [4] and Soule [5] believed that gastric heterotopia arose from the epithelium of the primitive gut, which was separated from the primordial stomach and underwent hyperplasia over time due to unknown pathways. Skandalakis *et al* [6] proposed that heterotopic gastric mucosa originated from the metaplasia of pluripotent endodermal cells of the foregut. Abel *et al* [7] proposed that this lesion was of vitellointestinal tract origin. Other authors proposed the ability of endodermal cells of the primitive gut throughout the gastrointestinal tract to differentiate and undergo hyperplasia or physical movement of the gastric epithelia due to unknown pathways [2,7,8].

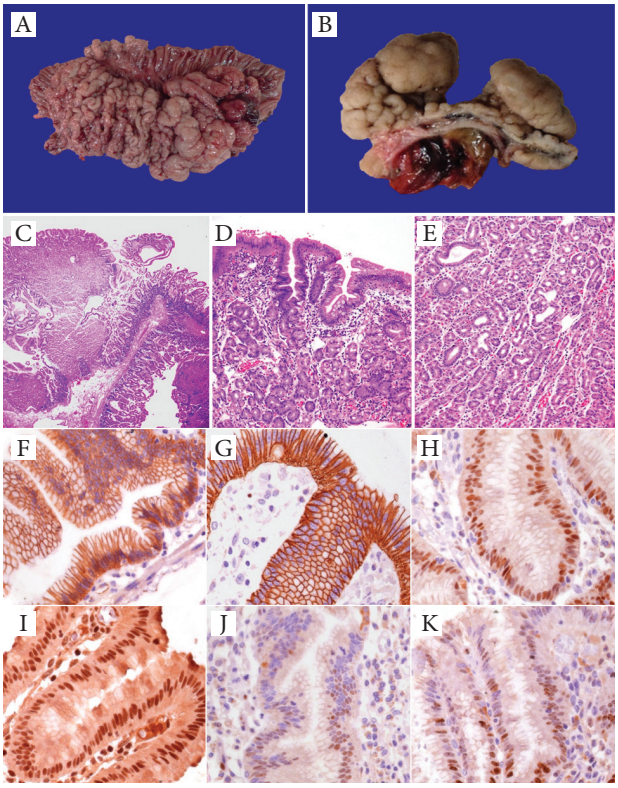

Figure 1 (A, B) A multilobulated mass in the jejunum that in cross-section affected only the intestinal mucosa. (C) The histological examination showed the transition between intestinal (right) and gastric mucosa (left) (40x, H & E). (D) Heterotopic gastric mucosa with hyperplastic foveolar epithelium (100x, H & E). (E) Gastric mucosa with oxyntic glands covered by parietal cells and chief cells with focal glandular dilatation (400x, H & E). (F) β-catenin-positive areas in the epithelial cell membrane (400x). (G) E-cadherin-positive areas in the epithelial cell membrane (400x). (H) Cyclin D1-positive areas in foveolar and mucous neck cells (400x). (I) p27 nuclear positivity in foveolar cells (400x). (J) p21 nuclear positivity in foveolar cells and an expression reduction in mucous neck cells (400x). (K) Ki67 positivity in mucous neck cells and in sparse superficial cells (400x)

In the immunohistochemical analysis of cell cycle molecule expression, we showed that p21, p27 and cyclin D1 were highly positive on the foveolar surface. In the neck, a site of cellular replication, p21 was low, and p27 and cyclin D1 were high; p16 and p57 were negative throughout the gastric mucosa. Ki67 was positive in the neck zone, with few positive cells in the foveolar region.

We propose that heterotopic gastric mucosa is associated with an alteration in the expression of cell cycle molecules (the Cip/Kip family), stimulated by unknown growth factors that induce high cellular proliferation.

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Conflict of Interest: None

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