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 foreroot. 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].

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. 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.

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References

1. Shehata B, Chang T, Greene C, et al. Gastric heterotopia with...
extensive involvement of the small intestine associated with congenital short bowel syndrome and intestinal malrotation. 


