Table 17-5 Hypertrophic Gastropathies and Gastric Polyps

Parameter	Ménétrier Disease (adult)	Zollinger-Ellison Syndrome	Inflammatory and Hyperplastic Polyps	Gastritis Cystica	Fundic Gland Polyps	Gastric Adenomas
Mean patient age (yr)	30-60	50	50-60	Variable	50	50-60
Location	Body and fundus	Fundus	Antrum > body	Body	Body and fundus	Antrum > body
Predominant cell type	Mucous	Parietal > mucous, endocrine	Mucous	Mucous, cyst-lining	Parietal and chief	Dysplastic, intestinal
Inflammatory infiltrate	Limited, lymphocytes	Neutrophils	Neutrophils and lymphocytes	Neutrophils and lymphocytes	None	Variable
Symptoms	Hypoproteinemia, weight loss, diarrhea	Peptic ulcers	Similar to chronic gastritis	Similar to chronic gastritis	None, nausea	Similar to chronic gastritis
Risk factors	None	Multiple endocrine neoplasia	Chronic gastritis, *H. pylori*	Trauma, prior surgery	PPIs, FAP	Chronic gastritis, atrophy, intestinal metaplasia
Association with adenocarcinoma	Yes	No	Occasional	No	Syndromic (FAP) only	Frequent

FAP, Familial adenomatous polyposis; PPIs, proton pump inhibitors.

Treatment of Ménétrier disease is supportive, with intravenous albumin and parenteral nutritional supplementation. In severe cases gastrectomy may be needed. More recently, agents that block TGF-α-mediated activation of the epidermal growth factor receptor have shown promise.

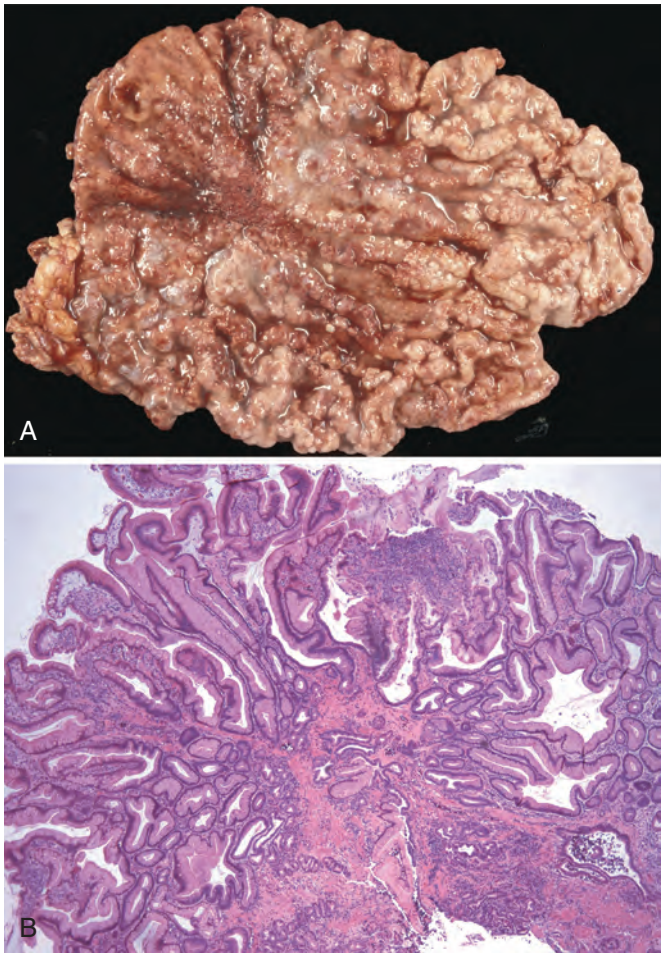

Figure 17-15 Ménétrier disease. **A,** Marked hypertrophy of rugal folds. **B,** Foveolar hyperplasia with elongated and focally dilated glands. (Courtesy Dr. M. Kay Washington, Vanderbilt University, Nashville, Tenn.)

Zollinger-Ellison Syndrome

Zollinger-Ellison syndrome is caused by gastrin-secreting tumors. These gastrinomas are most commonly found in the small intestine or pancreas. Patients often present with duodenal ulcers or chronic diarrhea. Within the stomach, the most remarkable feature is a doubling of oxyntic mucosal thickness due to a five-fold increase in the number of parietal cells. Gastrin also induces hyperplasia of mucous neck cells, mucin hyperproduction, and proliferation of endocrine cells within oxyntic mucosa. In some cases these endocrine cells can form small dysplastic nodules or, rarely, true carcinoid tumors.

Treatment of individuals with Zollinger-Ellison syndrome includes blockade of acid hypersecretion. This can be accomplished in almost all patients with proton pump inhibitors. Acid suppression allows peptic ulcers to heal and prevents gastric perforation, allowing treatment to focus on the gastrinoma, which becomes the main determinant of long-term survival.

Although they grow slowly, 60% to 90% of gastrinomas are malignant. Tumors are sporadic in 75% of patients. These tend to be solitary and can be surgically resected. The remaining 25% of patients with gastrinomas have multiple endocrine neoplasia type I (MEN I). These individuals often have multiple tumors or metastatic disease and may benefit from treatment with somatostatin analogues. Detection of tumors may be enhanced by using somatostatin receptor scintigraphy or endoscopic ultrasonography.

Gastric Polyps and Tumors

Polyps, nodules or masses that project above the level of the surrounding mucosa, are identified in up to 5% of upper GI endoscopies. Polyps may develop as a result of epithelial or stromal cell hyperplasia, inflammation, ectopia, or neoplasia. Only the most common types of polyps will be discussed here (Peutz-Jeghers and juvenile polyps are discussed with intestinal polyps). This is followed by a presentation of gastric tumors, including adenocarcinomas, lymphomas, carcinoid tumors, and stromal tumors.