Fellows' Corner

by Robert J. Bonasera and Vineet Korrapati

CASE REPORT

A 57-year-old man was evaluated for a complaint of six months of intermittent loose, non-bloody stools, nausea, and abdominal discomfort. Furthermore, he had developed swelling in his lower extremities over the last two months. He had a history of ulcerative colitis and thyroiditis. On physical examination he had pitting edema in his lower extremities, his abdominal exam was without abnormality. Stool cultures were negative; a colonoscopy performed one year earlier was unremarkable. Most notable was an albumin level of 2.9 gm/dL (3.5–4.8 normal range). A CT scan of the abdomen (Figure 1) showed evidence of diffuse thickening of the gastric wall. Subsequent EGD performed revealed thickened gastric folds in the proximal stomach (Figure 2), from which biopsies were taken. There was a distinct transition to normal appearing gastric mucosa at the antrum.

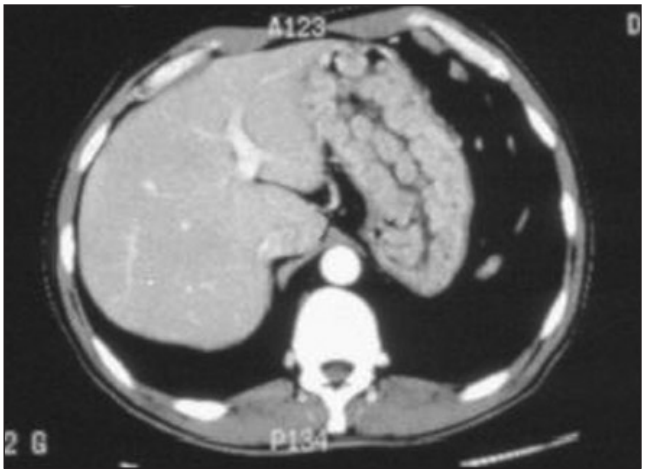

Figure 1.

Questions

1. What is the diagnosis?
2. Is the disease considered to be premalignant?
3. What are the treatment options?

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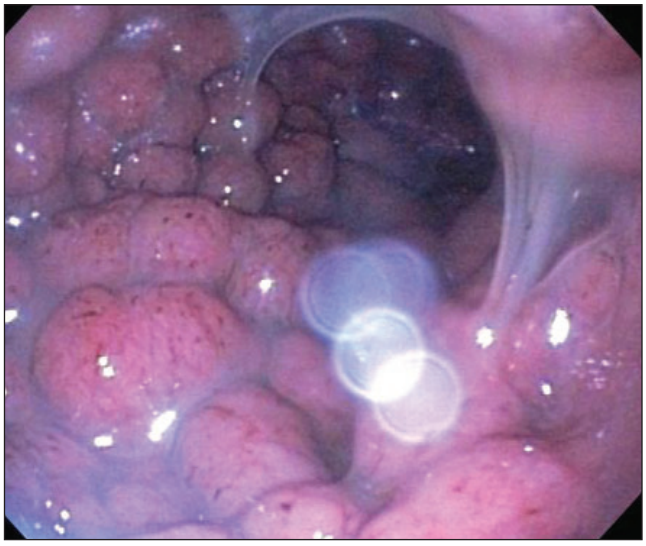

Figure 2.

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DISCUSSION

The patient was diagnosed with Menetrier's disease based on the clinical scenario, endoscopic appearance and characteristic pathology: foveolar hyperplasia, glandular cystic dilatation and smooth muscle proliferation within the lamina propria.

First described in 1888, to date there are roughly 300 case reports of Menetrier's in the literature. It is a rare, acquired condition in which there are hypertrophic gastric folds, excessive mucous production (picture), hypochlorhydria (50%–70%) and hypoproteinemia (80%). The non-selective loss of proteins across the gastric mucosa is thought to occur as a result of impaired intercellular junctions (1,2). The etiology of the disorder remains unclear. There is some data to suggest that increased signaling of the epidermal growth factor receptor plays a role. Other explanations include a food antigen-hypersensitivity response, *H. pylori*, and cytomegalovirus. The endoscopic appearance is typically that of giant mucosal folds in the fundus with a normal antrum. Often there is a sharp demarcation as in our patient. The endoscopic appearance is often suggestive; however, biopsies are necessary for the diagnosis. Routine cold biopsy forceps may acquire tissue which is too superficial for a diagnosis. Suction or snare biopsies can be used to obtain greater tissue depth. Whether or not Menetrier's can be considered premalignant is debatable. Of the cases in the literature, roughly 15% were documented as progressing to a gastric malignancy (2). Recommendations regarding surveillance do not exist, however, most authors subscribe to the more vigilant approach of repeat endoscopies every one-to-two years. Guidelines pertaining to treatments are equally ill defined. There are reports of success with acid suppressive therapy, most notably with H_2-blockers and anticholinergics which may give an added benefit of tightening the gastric intercellular junctions. Furthermore, monoclonal Ab against EGF receptor, octreotide, antifibrinolytics, parietal cell vagotomy and gastric resections have been reported. All with varying success, and very little in the way of controlled trials looking at these different treatment options (2,3).

Our patient was placed on an H_2-blocker and was instructed to increase his daily protein intake. He has responded well both clinically and biochemically with less discomfort and nausea and almost complete resolution of his peripheral edema. ■

References

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2. Fordtran J, Sleisenger M. Menetrier's disease (hypertrophic gastropathy). 8th edition. *Gastrointestinal and Liver Diseases,* 2006; 1082-1083.
3. Bdick S, Chung E. Treatment of Menetrier's disease with a monoclonal antibody against the epidermal growth factor receptor. *NEJM*, 2000; (23)343: 1697-1701.