A 49-year-old man presented with a 5-month history of epigastric distension after meals. He had no history of abdominal pain, nausea, vomiting, or diarrhea and no family history of cancer. The physical examination was negative for edema, ascites, and superficial lymph nodes. Helicobacter pylori infection was detected with the carbon 13 urea breath test. Gastroscopy showed diffuse hypertrophic gastric mucosa covered by excessive mucus and limited to the stomach body and fundus (Fig. 1a, b). A regular pit pattern of the hypertrophic mucosa was observed on narrow-band imaging (Fig. 1c). Ultrasonic endoscopy revealed a thickened mucosal layer (Fig. 1d). Computed tomography confirmed these findings and showed no palpable abdominal lymph nodes (Fig. 1e). Histology of full-thickness biopsy specimens showed marked foveolar epithelial hyperplasia and no malignant cells (Fig. 2). The patient’s total serum protein, albumin, gastrin, and immunoglobulin G (subtypes IgG1–IgG4) were normal, as well as the blood cell count. Therefore, Ménétrier’s disease was diagnosed.

Ménétrier’s disease is a rare, idiopathic hypertrophic gastropathy characterized by hyperproliferative foveolar epithelium of the stomach body and fundus and

Fig. 1 Ménétrier’s disease. a, b Endoscopic views of the stomach under white light. c Narrow-band imaging of the hypertrophic mucosa. Ultrasonic (d) and computed tomographic (e) views of the stomach.

Fig. 2 Histology of the biopsy specimen.
hypoproteinemia [1]. Ménétrier’s disease should be distinguished from gastric polyposis syndrome, hyperplastic gastritis, Zollinger–Ellison syndrome, and gastric malignancy [1,2]. In this case, the patient had a normal albumin level, indicating that the secretion of mucus had not resulted in hypoproteinemia. In addition, the Ménétrier’s disease was associated with *H. pylori* infection [3,4]. Therefore, treatment to eradicate *H. pylori* was initiated. After 1 month, the patient’s symptoms were alleviated, his body weight increased 2.5 kg, and the secretion of mucus decreased; however, there was no regression of the hypertrophic gastric mucosa.

Endoscopy_UCTN_Code_CCL_1AB_2AD_3AD

**Competing interests:** None

**Lang Yang, Xiaojun Zhao, Ai-qin Li, Jian-qiu Sheng**

Department of Gastroenterology, General Hospital of Beijing Military Command, Beijing, China

**References**


**Corresponding author**

Jian-qiu Sheng, MD, PhD

Department of Gastroenterology
General Hospital of Beijing Military Command
Nannencang 5#, Dongcheng District
Beijing 100700
China
Fax: +86-10-66721299
jianqiu@263.net

**DOI** http://dx.doi.org/10.1055/s-0034-1377983

Endoscopy 2015; 47: E9–E10

© Georg Thieme Verlag KG
Stuttgart · New York
ISSN 0013-726X