Ménétrier disease: A rare cause of hypertrophic gastropathy☆

Enfermedad de Ménétrier: una causa rara de gastropatía hipertrófica

Ménétrier disease (MD) is an enteropathy characterized by gastric fold hyperplasia and a state of protein loss. Pharmacologic treatment has shown varying results, and in cases of refractoriness, gastrectomy is a therapeutic option.[1,2]

A 58-year-old woman with 4-month progression of the disease presented with mild epigastralgia, nausea, and lower limb edema. Laboratory work-up reported Hb 10 g/dl, total protein 4.8 g/dl, and albumin 2.9 g/dl. Endoscopy revealed multiple hypertrophic folds in the gastric body, resembling ''circumvolutions'' with mucus (Fig. 1A and B), and erosions at the level of the antrum (Fig. 1C). The anatomopathologic study reported marked foveolar hyperplasia, tortuous foveolar glands, and multiple cystic dilations (Fig. 1D), as well as the presence of *Helicobacter pylori*. Extended-release octreotide was administered at a dose of 20 mg IM monthly, for 4 months, along with 1 g of amoxicillin every 12 h and 500 mg of levofloxacin every 24 h for 14 days, for *Helicobacter pylori* eradication, and 40 mg of esomeprazole every 12 h for 4 months. The eradication of the bacterium was verified. Given the lack of clinical and endoscopic improvement, monoclonal antibodies were considered the best option. However, those medications are not available at our hospital, and so total gastrectomy was performed. The patient's progression was favorable at the follow-up at one year.

MD is a gastropathy characterized by gastric fold hypertrophy, with protein loss. It affects middle-aged men[3] and is associated with gastric adenocarcinoma in 6–10% of cases.[4]

The disease is related to higher alpha-TGF production and epithelial cell proliferation that produce abundant mucus. The hypersecretion of mucus causes malabsorption of nutrients, electrolytes, and vitamins. Ninety percent of cases are associated with *Helicobacter pylori*.[3,4]

The clinical manifestations are epigastralgia (65%), anorexia (45%), weight loss (45%), edema (38%), and vomiting (38%); diarrhea and gastrointestinal bleeding are less frequent.[3,5]

Laboratory findings include hypoalbuminemia, elevated serum gastrin levels, and iron-deficiency anemia.[5] Endoscopy identifies hypertrophic gastric folds that do not affect the antrum. Histologic study describes foveolar hyperplasia, oxyntic gland atrophy, and a decrease in parietal cells, as well as edema and smooth muscle hyperplasia in the lamina propria.[6,7] The differential diagnosis in hypertrophic gastropathy should include infections due to cytomegalovirus and syphilis; tumors, such as adenocarcinoma, lymphoma, and Zollinger-Ellison syndrome; infiltrative diseases, such as sarcoidosis and amyloidosis; and other causes, such as eosinophilic gastroenteritis.[6] Treatment includes a protein-rich diet, proton pump inhibitors, and macronutrient replacement. Octreotide has been shown to be beneficial in some patients, but in refractory cases, total gastrectomy is recommended, as in the case of our patient.[8,9]

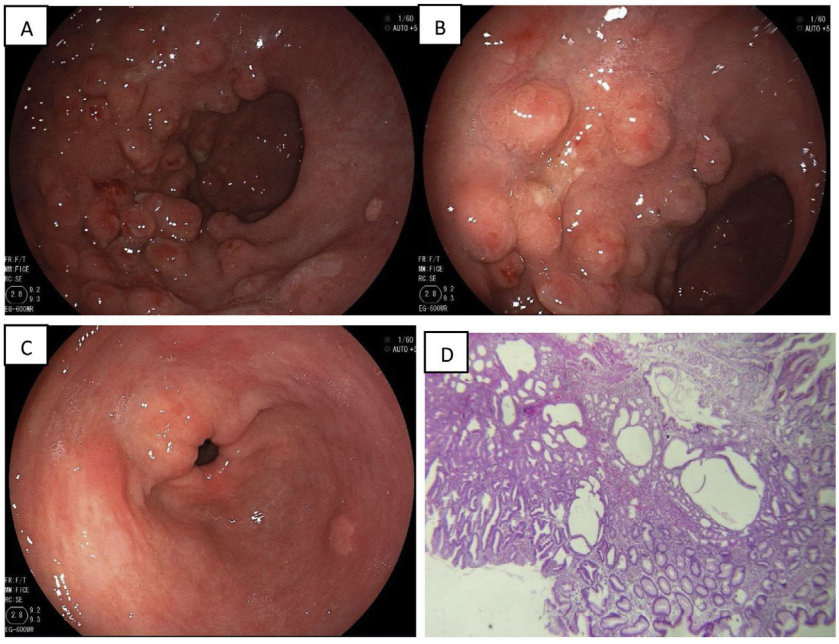

Figure 1 (A) Gastric body showing giant hypertrophic folds. (B) Folds with mucus secretions. (C) Gastric antrum with erosions. (D) Histologic study showing marked foveolar hyperplasia and cystic dilations.

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In conclusion, MD is a form of hypertrophic gastropathy that presents with digestive symptoms and protein loss. In medical treatment-refractory cases, total gastrectomy should be considered an option.

Ethical considerations

The authors declare that no experiments were conducted on humans for this research. We utilized the protocols for obtaining patient information from our work center's database, preserving patient anonymity, so informed consent was not requested. This study meets the current bioethical research regulations.

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Conflict of interest

The authors declare that there is no conflict of interest.

References

1. Kamal MU, Tariq H, Mehak V, et al. A rare etiology of abnormally large gastric folds: Menetrier's disease. Case Rep Gastrointest Med. 2019:7927083, http://dx.doi.org/10.1155/2019/7927083.
2. Thapa S, Ghosh A, Pun G, et al. A misdiagnosed case of hypertrophic gastropathy. Case Rep Pathol. 2020:4562531, http://dx.doi.org/10.1155/2020/4562531.
3. Hassan IH, Soliman M, Shirazi-Nejad AR, et al. Ménétrier's disease and its atypical presentation in four siblings. Cureus. 2022;14:e30759, http://dx.doi.org/10.7759/cureus.30759.
4. Pepa P, Uehara T, Wonaga A, et al. Menetrier's disease. A diagnostic and therapeutic challenge. Medicina (B Aires). 2021;81:470–3. PMID: 34137712.
5. Waisberg DR, Sobroza-de Mello E, Tustumi F, et al. A case report of diffuse hyperplastic gastropathy with multiple polypoid formations in a patient with pernicious anemia, *Helicobacter pylori* infection, hypergastrinemia and hypoalbuminaemia: Do not forget of Ménétrier's disease. Int J Surg Case Rep. 2020;77:498–502, http://dx.doi.org/10.1016/j.ijscr.2020.11.055.
6. Agarwala R, Shah J, Dutta U. Thickened gastric folds: approach. J Dig Endosc. 2018;09:149–54, http://dx.doi.org/10.4103/jde.JDE_72_18.
7. Wang H-H, Zhao C-C, Wang X-L, et al. Menetrier's disease and differential diagnosis: A case report. World J Clin Cases. 2021;9:6943–9, http://dx.doi.org/10.12998/wjcc.v9.i23.6943.
8. Nunes G, Barosa R, Patita M, et al. Ménétrier's disease: a case of successful treatment using long-acting octreotide. Acta Gastroenterol Belg. 2019;82:429–32. PMID: 31566332.
9. Parianos C, Aggeli C, Sourla A, et al. Total gastrectomy for the treatment of Menetrier's disease persistent to medical therapy: a case report. Int J Surg Case Rep. 2020;73:95–9, http://dx.doi.org/10.1016/j.ijscr.2020.06.033.

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Liver transplantation from a donor with multidrug-resistant *Acinetobacter baumannii* infection. Is it a risk?☆

Trasplante hepático de un donador con infección por *Acinetobacter baumannii* multidrogorresistente. ¿Representa un riesgo?

Orthotopic liver transplantation (OLT) is the treatment of choice for patients with end-stage liver disease, acute liver failure, hepatocellular carcinoma, and other liver diseases.[1]

However, the mortality rate for patients on the liver transplant waiting list has increased due to high demand and organ scarcity worldwide, including Mexico.[2] To resolve this problem, efforts have been made to broaden the potential donor pool, including some donors considered ''high risk'' or marginal; for example, donors with positive blood cultures, whose organs can be safely transplanted, with the exception of those with sepsis and multiorgan failure at the time of procurement.[3]

We present herein the case of an OLT recipient whose donor had confirmed bacteremia due to multidrug-resistant (MDR) *Acinetobacter baumannii* (*A. baumannii*).

A 66-year-old man, whose current disease appeared in 2016, characterized by general malaise, variceal bleeding, ascites, and grade II hepatic encephalopathy. He was diagnosed with cirrhosis of the liver due to metabolic dysfunction-associated fatty liver disease (MAFLD), after ruling out other etiologies. Two years after his disease onset, the patient developed three lesions under 4 cm, consistent with hepatocellular carcinoma. He received three sessions

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