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Case Report

Menetrier's disease

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ABSTRACT

A clinical case of a 57-year-old female patient with history of epilepsy diagnosed in childhood, currently being treated with magnesium valproate, she presented with oral intolerance, ascites and weight loss. The initial laboratory workup was significant for hypoalbuminemia (serum albumin 18 g/L), but her liver and renal function were normal and urinalysis was negative. Panendoscopy was performed with a microscopic report: histopathology reporting findings related to hypertrophic gastropathy, gastric foveolar hyperplasia, with chronic diffuse moderate edematous gastritis without data of acute activity, with *Helicobacter pylori* bacilli in small quantity (+), negative for metaplasia, dysplasia and/or malignancy compatible with Menetrier disease. The patient was evaluated by the general surgery service to perform a jejunostomy and/or total gastrectomy as an adjuvant treatment for the patient. The extent of the gastrectomy to be performed is the subject of debate. The patient refused surgical treatment, so she was voluntarily discharged to an outpatient clinic to begin monoclonal treatment. The patient died 3 weeks before starting monoclonal treatment. Therefore, untreated patients have a high mortality rate due to complications.

Keywords: Menetrier syndrome, Jejunostomy, Gastrectomy, Ascites

INTRODUCTION

Menetrier's disease is a rare clinical entity of unknown etiology, considered a preneoplastic condition, which predominantly affects the male sex with a usual presentation between 30 and 60 years of age, although isolated cases have been reported in childhood associated with infection by cytomegalovirus (CMV) that usually presents spontaneous recovery.[1-2]

Although Menetrier's disease has not been determined, it has been suggested that the epidermal growth receptor of the gastric mucosa as well as transforming growth factor alpha (TGF-α) play an important role, associated with *H. pylori* infection.[3] Currently etiopathogenesis of Menetrier's disease is not completely known, the overexpression of TGF-α in superficial gastric epithelium may play a role in the etiopathogenesis since it is thought that an increase in the signaling of this receptor that leads to proliferation of mucosal epithelial cells, this TGF-α receptor being a ligand that activates EGFR and stimulates growth of epithelial cells, which in turn produces a decrease in production of gastric acid and hyperplasia of superficial mucosal cells, atrophy oxytic and increased mucin production.[1,4,5] However, it has been suggested that this disease may have hereditary behavior with autosomal dominant pattern.[3]

Clinical presentation were oral intolerance, vomiting, weight loss, peripheral edema. Diagnosis is made through an endoscopic procedure in which diffuse thickening of gastric mucosa is characteristic, accompanied by histopathological findings of foveolar hyperplasia and diffuse inflammatory infiltrate.[4] Mucosal hypertrophy is associated with protein loss, giant mucosal folds in proximal part of stomach with decreased acid secretion and protein-losing state with hypo-albuminemia.[6]

Typical histopathological studies therefore include foveolar hyperplasia, tortuous and dilated gastric glands, with a decrease in parietal cells, a mild inflammatory infiltrate and an increase in mucin-producing cells.[7]

CASE REPORT

A case is reported of 57-year-old female patient with the only history of epilepsy diagnosed in childhood, currently being treated with magnesium valproate, without presenting epileptic seizure events for more than 2 years.

He reports that approximately 1 month ago he began to experience hyporexia secondary to oral intolerance characterized by vomiting of gastrointestinal content, intermittent crampy abdominal pain in the mesogastrium that increased when eating food, and weight loss of approximately 7 kg since onset of symptoms. On February 16, 2023, she was admitted to emergency room due to ascites with dysphagia to solids and liquids. On physical examination, she had grade II ascites, with no evidence of peritoneal irritation. Laboratory studies are reported in normal parameters.

You are admitted to our service where a diagnostic paracentesis is performed with cytochemical report in normal parameters. Negative cytological study, as well as culture of ascites fluid without bacterial development, as well as USG of the liver and bile ducts with a report without structural abnormalities, so simple and contrasted abdominopelvic CT was performed on March 21, 2023 with report of moderate ascites without added alterations.

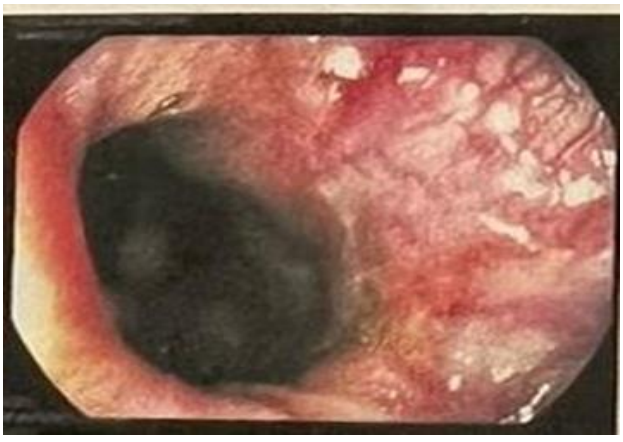

Figure 1: Panedoscopy: Lateralized pylorus, edematous pyloric canal. On retroflexion: incisura angularis edematous, background mucosa with diffuse hyperemia.

The evolution of the patient without clinical or imaging findings with persistence of intolerance to the oral route, it was decided to perform endoscopy with a report of edematous pangastropathy to rule out probable Menetrier syndrome (Figure 1), a biopsy was taken in the gastric body and pylorus with histopathological findings. compatible with hypertrophic gastropathy gastric foveolar hyperplasia, diffuse moderate edematous chronic gastritis, without data of acute activity with *Helicobacter pylori* bacilli in small quantity (+), negative for metaplasia, dysplasia and/or malignancy (Figure 2).

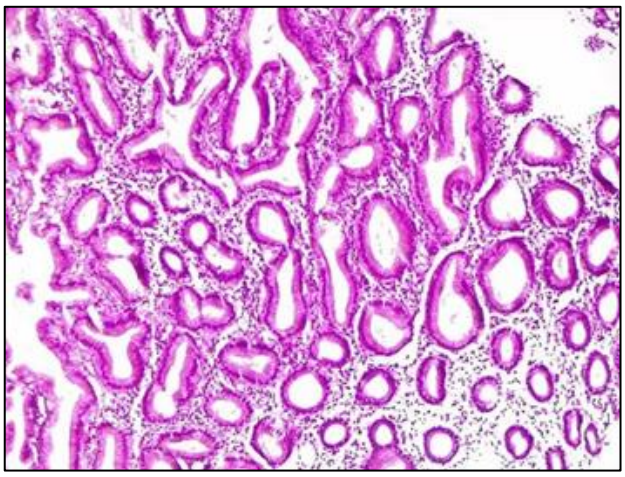

Figure 2: Biopsy of gastric body/pylorus: foveolar epithelium with hyperplastic and tortuous changes. Gastric and tortuous and hyperplastic foveolae. Deep mucosa with fundic glandular. Glands not equidistant and in variable shapes and size chronic inflammatory reaction and edema.

DISCUSSION

Supportive treatments have described a high protein diet, as well as symptomatic management with proton pump inhibitors and micronutrient replacement. Treatment of *H. pylori* and CMV is recommended.[2,8] Treatment is based on surgical management with total or partial gastrectomy, as well as symptomatic management, in which there has not been solid evidence of a reduction in clinical symptoms.[9]

Recently, the use of cetuximab, a recombinant IgG1 monoclonal antibody, has been considered.[10] It has the ability to bind to the extracellular portion of the EGF receptor, which inhibits the binding of TGF-alpha, which has shown good results and promises to be a treatment of choice. However, the treatment that has currently shown the greatest results has been partial and total gastrectomy, which continues to be an acceptable means of treatment for cases with persistent symptoms.[9]

CONCLUSION

The need to perform a total or partial gastrectomy was debated, since it is commented in some articles that depending on the patient's symptoms, the procedure to be performed is performed to eliminate the risk of malignancy. The patient refused surgery and was sent to an outpatient clinic to start a protocol for treatment with monoclonal antibodies. Unfortunately, after 3 weeks the patient died before starting monoclonal treatment. It is concluded that patients with menetrier disease have a

high mortality if is not a monoclonal or surgical treatment. Diet is not enough to reduce symptoms and signs in this case.

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