INTRODUCTION

Menetrier’s disease (also called hypertrophic gastropathy) is a rare condition characterized by the presence of giant gastric folds mainly involving the body of the stomach (1), which results in decreased gastric acid production, and a protein losing gastropathy causing hypoalbuminemia (2). Patients usually present with epigastric pain, vomiting, anorexia, peripheral edema, and weight loss (3).

The etiology of Menetrier’s disease is unknown, although infections with certain organisms, such as *Helicobacter pylori* (4,5) and cytomegalovirus(6) have been associated with this disease. CMV-induced Menetrier’s disease is primarily seen in the pediatric population, but it has been rarely documented in immunocompetent adults.

We report a case of an immunocompetent young female who presented with abdominal pain, nausea, and vomiting. Upper gastrointestinal endoscopy revealed thickened and enlarged gastric folds, while gastric biopsy showed foveolar hyperplasia with evidence of Cytomegalovirus (CMV) infection. CMV immunoglobulin G and immunoglobulin M antibodies were detected in the serum.

CMV infection can induce Menetrier’s disease in immunocompetent adults, and should be considered in the differential of both pediatric and adult patients who present with a protein losing enteropathy.

A CASE TO REMEMBER

CMV Induced Menetrier’s Disease in an Immunocompetent Adult

Amer Kassar, M.D., and Mohammed Barawi, M.D., St. John Hospital and Medical Center, Affiliated with Wayne State University School of Medicine, Detroit, MI.
A CASE TO REMEMBER

CMV Induced Menetrier’s Disease in an Immunocompetent Adult

We present a case of 20-year-old immunocompetent female, who was found to have Menterier’s disease induced by CMV infection, proven by biopsy (8).

CASE REPORT

A previously healthy 20-year-old African American female presented to the hospital with complaints of fever, abdominal pain, nausea and vomiting. Five days prior to admission, she had a decreased appetite associated with seven to eight episodes of vomiting. She denied having hematemesis, dysphagia or regurgitation. The abdominal pain that had started three days prior, was described as a constant, dull sensation, located mostly in the epigastric and left upper quadrant areas. She denied having diarrhea or constipation. No hematochezia, or mucous in the stool. These symptoms were associated with fever and rigors during the same period of time.

The patient did not have a significant past medical history, except for a dilatation and curettage for a miscarriage two years previously. She was not on any medications. She denied smoking cigarettes, drinking alcohol, or using illicit drugs.

On physical examination, her temperature was 99.2°F, heart rate was 124 beats per minute, respiratory rate was 20 breaths per minute, and blood pressure was 147/97 mmHg. The head and neck examination was normal. The lungs were clear. The heart was regular in rate and rhythm, without murmurs or gallops. The abdomen was soft, with moderate tenderness in the epigastric and left upper quadrant areas, bowel sounds were present, no guarding or rigidity appreciated. Peripheral pulses were present in the periphery, without edema. No focal neurologic deficit was detected.

Liver function tests showed elevated alanine aminotransferase and aspartate aminotransferase at 161 and 170, respectively. The levels of conjugated and total bilirubin, alkaline phosphates, lipase, blood urea nitrogen, creatinine, chloride, and bicarbonate were within normal limits. The urine was positive for occult blood, with trace leukocyte esterase; the sediment contained 6 white cells, 9 red cells, and a few amount of bacteria per high power field.

The acute abdominal series was nonspecific. The abdominal ultrasound showed probable hemangioma in the spleen with splenic enlargement, otherwise negative. An esophagogastroduodenoscopy showed erythematous distal esophageal mucosa. The gastric folds were prominent and friable.

The biopsy showed active chronic inflammation in the gastric as well as duodenal mucosa, with hyperplastic polyps in the gastric mucosa; no *Helicobacter pylori* was detected. The patient was diagnosed with gastritis, and was treated with proton pump inhibitors. Her symptoms improved during her hospital stay, and her diet was advanced with good toleration. Liver enzymes trended down gradually. Computed tomography of the abdomen and pelvis showed splenomegaly, which was felt to be an incidental finding. The patient (continued on page 103)
was discharged home after five days, and was asked to follow-up with the gastroenterologist as an outpatient.

Two days later, the patient presented to the hospital with complaints of persistent epigastric pain, associated with intractable nausea and vomiting. She denied left upper quadrant pain and fever at this presentation.

Her physical exam had not changed from her previous visit, with persistent mild elevation in alanine and aspartate aminotransferases on laboratory work. The esophagogastroduodenoscopy was repeated at that time, and revealed hypertrophic rugal folds in the gastric body as well as in the fundus, with generalized gastritis and poor distensibility of the stomach with air insufflation. A large particle biopsy was performed using a hot snare after submucosal injection of 3 cc of 1/10000 epinephrine.

Endoscopic ultrasound revealed thickening of the gastric wall, especially the mucosal layer. The wall thickening was 6.6 mm, with the normal range between 3 to 4 mm. Multiple hypoechoic round lymph nodes were seen around the gastric body, especially in the splenic hilum region.

The gastric biopsy showed giant fold disease with foveolar hyperplasia (Figure 1), without Helicobacter pylori organisms, consistent with Menetrier’s disease.

Laboratory tests showed elevated IgM and IgG CMV antibodies at 1.32 and 3.46, respectively. Helicobacter IgG antibodies were within normal limits.

The patient was placed on proton pump inhibitors, and was discharged home after her symptoms improved.

Four weeks later, she was asymptomatic. Repeat upper endoscopy revealed normal appearing gastric folds with complete resolution of the enlarged gastric folds. The biopsies showed normal gastric mucosa without evidence of persistent cytomegalovirus infection.

**DISCUSSION**

Menetrier’s disease is the most common hypertrophic gastropathy disorder associated with gastrointestinal protein loss, and is characterized by large mucosal folds usually involving the body of the stomach. The pathogenesis of this disease is not completely understood, but involve transforming growth factor-alpha (9,10), which increases gastric mucous production and inhibits acid production. The levels of this factor are markedly increased in the gastric mucous cells in patients with Menetrier’s disease.

Infection with Helicobacter pylori has been associated with this disease. It has been postulated that hypertrophic gastropathy might be a form of Helicobacter pylori gastritis, and eradication of this organism may lead to complete clinical and morphologic recovery (11). A childhood form of this disease has also been associated with cytomegalovirus infection, but there are few reports of this kind of association in
adults, especially in immunocompetent patients. Close follow-up in patients with Menetrier’s disease is essential as they have a 15%–20% higher incidence of gastric-associated malignancies, primarily gastric carcinoma (12,13) and gastric lymphoma (14).

Menetrier’s disease can present with a variety of clinical features including epigastric pain, substantial weight loss, nausea, vomiting, diarrhea. Occult gastrointestinal bleeding may occur, but overt bleeding is unusual. Between 20% and 100% of patients develop a protein-losing gastropathy accompanied by hypoaalbuminemia and edema.

Diagnosis of this disease is established by the morphologic appearance of enlarged gastric folds seen on endoscopy or barium radiography; these folds are usually enlarged symmetrically and confined to the body and fundus. A full-thickness biopsy is usually required for diagnosis, which shows foveolar hyperplasia and glandular atrophy, with replacement of chief and parietal cells with mucous glands.

Multiple medical treatments including: antacids, anticholinergic drugs (15), prednisone (16), H2-receptor blockers (17,18), proton pump inhibitors (19), and prostaglandins, have been used for patients with this disease but none has proven to be consistently beneficial. A high-protein diet should be recommended to replace protein loss in patients with hypoaalbuminemia. Surgery is indicated for patients with intractable pain, hypoaalbuminemia with edema, hemorrhage, pyloric obstruction, and for those in whom malignancy cannot be excluded (20,21).

We reported a case of a young immunocompetent female who presented with symptoms of epigastric pain, nausea and vomiting. Upper endoscopy revealed typical enlarged gastric folds with severe inflammation of the associated mucosa. Menetrier’s disease was suspected, and gastric biopsy confirmed the diagnosis by showing the typical hypertrophic gastric mucosa associated with foveolar hyperplasia, evidence of cytomegalovirus infection by fluorescence staining, and the presence of inclusion bodies.

The patient was treated with proton pump inhibitors, and was asked to return in few weeks for repeat upper endoscopy. During the follow-up period, the patient was improving clinically, and the repeat endoscopy revealed normal gastric mucosa with complete resolution of the enlarged gastric folds. The biopsies were negative for cytomegalovirus or Menetrier’s disease.

References