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MENETRIER'S DISEASE AND LIMITED SURGICAL APPROACH

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We describe a 33 year old man who presented with fatigue, weight loss, and dark brown stool. The upper gastrointestinal X-ray series and endoscopy revealed thickened gastric folds in the cardia and corpus of the stomach. Gastic juice analysis after pentagastrin stimulation and albumin levels of serum brought to mind Menetrier's disease but histopathologic examination not. We performed celiotomy and then gastrotomy. A very fragile cerebroid pattern due to thickened and irregular folds resembling cerebroid convolutions were seen along on the cardia and corpus of the stomach. These irregular thickened mucosal folds were excised without any defect in the wall of the stomach. Histopathologic examination of the operation specimen confirmed Menetrier's disease. In the seven years since the operation the patient has had no complaint and was normal in endoscopic controls. If the giant rugae in the stomach involve a limited area then a limited resection, which is described in this study, is both effective and free from the complications of the extensive gastrectomy.

***Key words**: Menetrier's Disease, Hypertrophic Gastropathy, Protein-Losing Gastropathy, Surgery.*

Menetrier Hastalığı ve Sınırlı Cerrahi Yaklaşım

Bu çalışmada sunulan olgu yorgunluk, kilo kaybı ve melena yakınmalarıyla başvurdu. Üst gastrointestinal radyolojik ve endoskopik incelemeler mide kardia ve korpusunda kalınlaşmış mukozal kıvrımları ortaya koydu. Pentagastrin uyarısı sonrası mide sıvı analizi ve serum albumin seviyeleri Menetrier hastalığını düşündürdü fakat histopatolojik inceleme Menetrier hastalığını teyit etmedi. Laparatomi ve gastrotomi sonrası kardia ve korpusta frajil, kalınlaşmış, düzensiz mukozal kıvrımlar görüldü. Bu mukozal kıvrımlar mide duvarında herhangi bir defekt yaratmaksızın eksize edildi. Operasyon materyalinin histopatolojik incelemesi, Menetrier hastalığını teyit etti. Operasyondan yedi yıl sonra hastanın herhangi bir yakınması yoktu ve endoskopik incelemeler normaldi. Menetrier hastalığında hipertrofik gastrik mukozal kıvrımlar sınırlı bir alandaysa, bu olgudaki gibi sınırlı mukozal rezeksiyon hem hastalığın ortadan kaldırılmasında etkilidir hem de gastrektominin komplikasyonlarından sakınılmış olur.

***Anahtar kelimeler:** Menetrier Hastalığı, Hipertrofik Gastropati, Protein Kaybettiren Gastropati, Cerrahi.*

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Menetrier`s disease (MD) is characterized by hypertrophic gastric rugae, gastrointestinal upset, protein losing gastropathy, and normal or hyposecretion of gastric acid. There is not yet a standardized medical or surgical treatment for this disease. The operative procedures performed in MD consist of subtotal and total gastrectomy [1-3] or exploratory laparotomy and biopsy.[1] Our purpose here is to record a case, paying particular attention to the surgical management of MD.

A 33 year old physician man presented with a 4 month history of fatigue, weight loss, and dark brown stool. Past medical and family history were non-contributory. Physical examination revealed normal vital signs. Deep palpation in the epigastrium produced minimal tenderness without rebound or guarding. Otherwise, the physical examination was within normal limits, excluding rectal examination showed dark brown stool.

Abnormal laboratory values included a hematocrit of 33.8 %, hemoglobin of 11 gm/dL, white blood cell count of 13,600/mm^3, albumin of 2.9 gm/dL, and total protein of 4.5 gm/dL. Gastric juice analysis after pentagastrin stimulation is shown in Table 1.

All of 15-minutes collection fractions had alkaline pH (7-7.6) and contained no free acid. Helicobacter pylori test was negative.

The upper gastrointestinal x-ray series showed thickened gastric folds in the cardia and corpus of the stomach. Gastroscopy revealed large, redundant, and friable gastric rugae, measuring 8 x 5 cm in the cardia and corpus of the stomach. Multiple biopsies were taken. The histopathologic examination yielded normal mucosa. Colonoscopic examination revealed no pathology.

We performed a celiotomy with the initial diagnosis of MD. The cardia and corpus of the stomach were edematous on inspection with a mass feeling on palpation. A vertical gastrotomy was performed. A very fragile cerebroid pattern, measuring 8x5 cm, due to thickened and irregular folds resembling cerebral convolutions were seen along the lesser curvature on the cardia and corpus of the stomach (Figure 1).

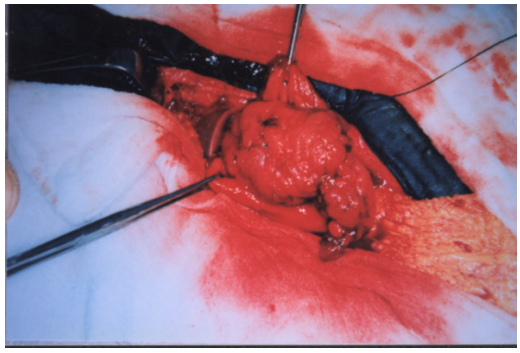

Figure 1. The appearance of giant rugae in the stomach during laparotomy.

There were no other abnormalities of the stomach or the duodenum. These irregular thickened mucosal folds were excised with a λ shaped incision sparing the muscularis coat of the stomach (Figure 2).

The excision was performed without any defect in the wall of the stomach. The mucosal opening was closed in a ⊥ form with 3-0 polyglactin 910 sutures and then the gastrotomy was closed continuously with 3-0 polyglactin 910 in the mucosal coat and

Time (min)	Sample volume(ml)	pH	Albumin (g/dl)
Basal Gastric Secretion			
0-15	50	7.1	0.23
16-30	25	7.1	0.23
Stimulated Gastric Secretion			
0-15	75	7.8	0.18
16-30	150	7.1	0.17

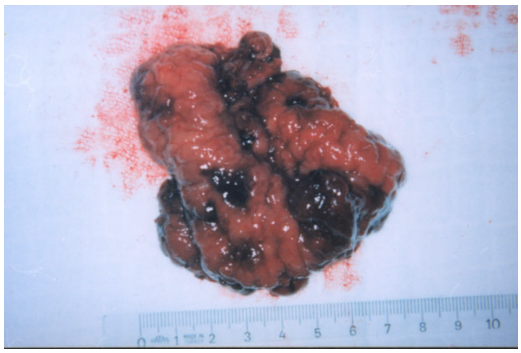

Figure 2. The appearance of giant rugae in the stomach during laparotomy.

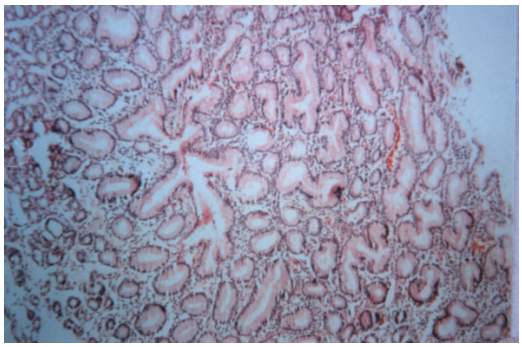

Figure 3. H.E., medium power view showing increased mucosal thickness with hyperplastic mucous glands.

polypropylene sutures in the serosal coat. The histopathologic examination of the specimen revealed increased mucosal thickness with hyperplastic mucous glands (Figure 3).

The patient made an uneventful recovery. In the seven years since the operation the patient has had no complaint, has gained weight, and has returned to full time employment. The gastroscopies performed on postoperative year 1, 3, 5, and 7 revealed completely normal mucosa of the stomach.

DISCUSSION

MD is a description usually applied to giant hypertrophy of the mucosal folds of the stomach that may or may not be associated with loss of protein from the mucosa.[4] The exudative gastric protein losses, which may occur, can cause life-threading profound hypoproteinemia despite large increases in albumin synthesis and turnover rate.[2] In MD the mucosal hypertrophy involves the fundus and body of the stomach and excludes the antrum.

Treatment of MD with protein-losing gastropathy has obviously developed by empiricism. Spontaneous remissions in a few cases were reported.[1,2] Since the etiology of the disease is unknown there is no specific therapy. Therapeutic trial of a variety of drugs including antacids,[2] anticholinergics,[3] steroids,[5] H_2-receptor blockers and H^+/K^+ ATP-ase inhibitors,[6] high protein diets,[3] repeated infusions of albumin,[3] and gastric irradiation[7] have proven inadequate when the clinical manifestations of MD and the protein losing syndrome are severe. Under such circumstances the gastric protein losses must be curtailed and resection of the site of protein loss is a logical and effective treatment. Since the hypertrophied gastric folds, which are the site of protein loss, occupy the fundus, cardia, and body of stomach, the local excision of the hypertrophied folds or total or subtotal gastric resection[8] can be performed.

The portion and amount of stomach to be resected depend on the judgement of the surgeon as well as on the location of the gastric pathology. Long term results of patients operated upon were generally satisfactory as defined by a disappearance of symptoms, weight gain, and normalization of serum albumin concentration.[2,3,7] Additionally, the 10 % chance of malignancy in MD can be eliminated by total gastrectomy[2,9] The anastomoses formed with abnormal, hyperplastic mucosa in subtotal gastrectomy may be more likely to break down than those formed with normal gastric tissue.[2] Therefore if the giant rugae in the stomach involve a limited area, then a limited resection, which is described in this study, is both effective and free from the complications of total or subtotal gastrectomy. However, the resection of the mucosa and submucosa that have MD would eliminate the chance of malignancy.

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