Menetrier's Disease and The Surgical Treatment

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Özet: *MENETRİER HASTALIĞI VE CERRAHİ TEDAVİSİ*

Ménétrier hastalığı (MH) midenin yüzeyel mukoza epitelinin hyperplasizi ile karakterize nadir rastlanan bir idyopatik gasatropatidir. Hastalarda azalmış acid salınımı, artmış mukus salınımının neden olduğu protein kaybı, hypoalbuminemi, ve periferik ödemler görülür. Bu çalışmada 2 ménétrier hastalığı olgusu ve bunlara uygulanan literatürde yer almayan değişik bir ameliyat şekli sunulmuştur.

Anahtar kelimeler : Ménétrier hastalığı, tıbbi tedavi, cerrahi tedavi.

Summary: *Ménétriers disease (MD) is an uncommen idiopathic gastropathy characterized by hyperplasia of superficial mucosal epithelium of the stomach. Patients with MD have low acid secretion and hypersecretion of mucus which lead to protein loss, hypoalbuminemia and peripheral edema. In this study we present two patients with MD and a different form of surgical treatment, which was not performed before.*

Key Words: Ménétrier's disease, medical treatment, surgical treatment

Two cases of Ménétrier's disease (MD) Hypertrophic Gastropathy) are described. The treatment of the disease is discussed. Case 1: A 33 year old physician presented with a 5 month history of fatigue, weight loss, occult blood in stool. Physical examination revealed a blood pressure of 120/80 mmHg, temperature 36.7 C, Pulse 72/min, and a respiration rate 12/min. The Sclerae were non icteric. The lungs were clear on auscultation and percussion. Heart sounds were normal. The abdomen was soft. There was minimal epigastric tenderness without ribound or guarding. No organomegaly or masses were detected. Rectal examination showed good tone and dark brown stools which were positive for occult blood. The neurological examination revealed no pathological finding. There were no clubbing, cyanosis or edema of the extremities. Laroratory findings yielded Hb.: 11.6 g.Hct.:33.8% platelet count 240000, a WBC 13600 of potassium 3.9, chloride 101 meq/L, BUN 29 mg/dl, glucose 96 mg/dl, GOT 13U/1, GPT 12 U/L, Total protein was 6.3 g/dl (albumin 3.7 g/dl, Globulin 2.6 g/dl.) EKG, chest x-ray was normal. Upper GI x-ray showed thickened gastric folds in cardia along the lesser curvature. Esophagogastroduodenoscopic examination was performed. A submucosal 5x2 cm polypoid mass was detected along the lesser curvatur from which multiple biopsies were taken. The histopatholocical examination yielded normal mucosa Colonoscopic examination revealed no pathology.

We performed a celiotomy with a upper midline epigastric incision on the 24th of February 1992. The cardia and the corpus was edematous, a mobile, fragile mass was palpated. A gastrotomy was performed. A very fragile cerebroid pattern due to the thickened and irregular fold resembeling cerebral convolutions were seen in the body of the stomach along the lesser curvature. (Fig 1). There was no abnormalty of the esophagogastric junction or of the duodenum. These cerebral convolutions resembeling irregular thickened folds were excised with a Y-shape incission. (Fig 2). The excision is performed without any deffect of the wall of the stomach. Gastrotomy is closed with 2-0 chrome catgut and 3-0 polyglactin 910 sutures. After placing a penrose drain,

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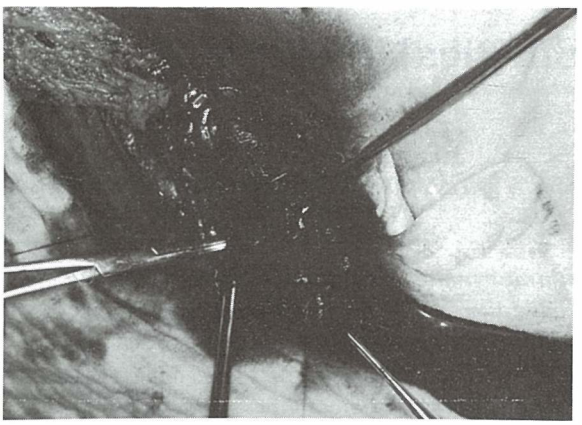

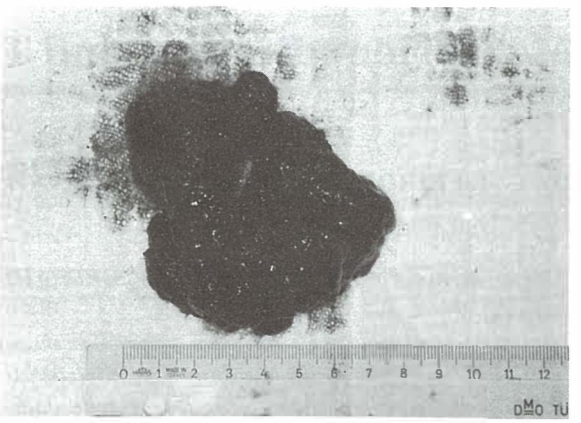

the abdomen is closed with a 2-0 polypropylene suture. The histopathological examination of the specimen yielded MD without any dysplasia or intestinal metaplasia.

Case 2: A 22 year patient is admitted to the hospital after a celiotomy was performed for 4 days in his hometown, where a suspicious mass filling cardia and duodenum was detected. He was emaciated with a blood pressure of 100/60 mmHg, pulse 88/min, temperature 37°C, and respiratory rate of 14/min. The sclerae were nonicteric, the lungs were clear to auscultation. Heart sounds were normal. The abdomen was soft, had clean epigastric midline incision wound. He had minimal epigastric tenderness without guarding or rebound. No organomegaly was detected. The neurological examination showed that the patient was alert, well orientiert without any neurological deficit. Laboratory examinations yielded Hb of 12.3 g, Hct 35.3%, WBC 17200 Normal value of electrolits, SGOT 18 U/L, SGPT 23 U/L, BUN 23 mg/dl, Total protein 6.2 g/dl, (albumin 2.8 mg/dl, globulin 3.4mg/dl.) EKG and chest x-ray graphies were normal. Esophagogastroscopy was performed, bipsies were taken which yielded no pathological finding. We performed a celiotomy on the 21st of May 1992 A polypoid mass was detected in the esophagogastric Junction which is excised totaly and the specimen sent for the pathological examination yielded MD without any displasia or metaplasia. Because the patient had deep peptic ulcus in bulbus which was resistant to the medical treatment with H_2-receptor blockes and H^+-K^+ ATP'ase inhibitors we performed truncal vagotomy (TV) and a drainage procedure in form of gastrojejunostomy and took multiple biopsies. The histopathological examination of these biopsy specimens yielded gastritis. The patient was discharged on 28th of may 1992 following a primary wound healing.

DISCUSSION

Ménétrier's disease (MD) originally described by Ménétrier in 1888 (1,2) is an uncommen idiopathic gastropathy charactherized by hyperplasia of superficial mucosal ephitel (3). Patients with MD have low acid secretion and hypersecretion of mucus which lead to protein loss, hypoalbuminemia and sometimes asscia-ted with peripheral edema. (1,3) Two forms of MD exists (4). The commen form involves thickened gastric rugau while the rare form involves the discreate gastric polyps. The pathogenesis of MD is still unresolved. The association between MD and severe neuroendocrin tumors including acromegaly, carcinoid tumor, pancreatic islet cell tumor, chemodectoma, amine precursor uptake and decarboxylation (APUD) cell adenomas and multiple endocrine adenomatosis (MEA) raises the possibility that the products of these neuroendocrin neoplasms may stimulate the stomach therby contributing to the development of MD (4). MD occuring in children is generally selflimited, transient benign condition and been accompained by eosinophilia suggesting an allargic reaction or a viral infection (1). The

main clinical symptoms are epigastric pain, weight loss, fatigue and anemia. Occult blood in stoohl is the most commen laboratory finding. Acid output is decreased or absent in 50% to 70% of the patients with MD. Approximately 50% have a slightly increased serum gastrin levels (5). Upper GI barium meal x-rays suggest the diagnosis of MD. Biopsies of the mucosa and submucosa performed on upper endoscopy confirm the diagnosis of MD. The differential diagnosis of the MD includes Zollinger Ellison syndrom, amyloidosis, infectious disease including tubercilosis, syphilis, Cronkhide-Canada syndrom which is described in 1956, characterized by diffuse gastrointestinal polyposis and assciated with dystrophic changes in the fingernails, alopecia, high blood pressure, diarrhea, weight-loss, abdominal pain and cutaneous pigmentations. Treatment is directed to relieve the abdominal pain, to render the blood loss and hypoproteinemia and to abolish the occurence of gastric carcinoma. Anticholinergics, antacids, histamine-H_2 receptor blockers, H^+-K^+ ATP'ase inhibitors may be helpfull in some cases. If pharmacologic therapy fails, surgery is appropriate.

Although subtotal distal gastric resection with a BII reconstruction reduces gastric acid secretion by removing gastrin-secreting antralcells and indirectly decreases loss of albumin as pH rises, it entails anastomosis of thick, edematous proximal stomach to the normal jejunum. The risk of leakage is high. Distal gastric resection with BI reconstruction is even more susceptible to these problems (6). Truncal vagotomy (TV) seems to have advantage of avoiding these complications of the dangerous anastomosis but have no physiological advantage over BI or BII typ of reconstructions. Proximale selective vagotomy (PSV) is not more efficacious than the treatment with the Histamine-H_2 receptor blockers. Total gastrectomy seems to be the best solution to the MD. Recunstruction of Roux-Y loop of jejunum avoids the occurence of reflux esophagitis and prevents the malignant complications of MD.

Although the gastric carcinoma has been associated 10-15% of the reported cases of MD there is not any prospective study which certainly gives the prevelance of the gastric carcinoma associated with MD. So we did not perform total gastric resection in our first case but prefered the excision of the giant hypertrophic mucosa of the stomach. We regularly perform upper GI endoscopy and take biopsies from the suspicious tissues. In case of an intestinal metaplasia or gastric adenoma both of which are accepted as a premalignant conditions we will perform total gastric resection and a recunstruction with a Roux-Y jejunal loop. In our second case we excised the polipoid submucosal tissue and performed TV+ Drainage procedure in form of gastrojejunostomy and took biopsies from the ulcus. Which was infact resistant to the medical therapy with H_2-reseptor blockers and H^+-K^+ ATP'ase inhibitors. The biopsies taken from the ulcus yielded gastritis. The histopathological examination of the submucosal polypoid mass showed MD without any metaplasia or displasia. We compleat esophagogastroduodenoscopy with regular intervals and take biopsies from the suspicious tissues. In case of an intestinal metaplasia, displasia, or gastric adenoma all of which are accepted as a premalignant conditions we will perform total gastrectomy and a reconstruction with a Roux-Y jejunol loop.

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