Menetrier's Disease: Case Report

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ABSTRACT

A 25 year old man presented with epigastric pain, postprandial vomiting which was non projectile and non bilous, progressive weight loss and body swelling for 2 years. The only remarkable findings were wasting and epigastric tenedreness and positive succussion splash. A gastroduodenoscopy revealed polypoid gastric mucosa involving the fundus and body of the stomach but sparing the antrum; there was no ulceration. Histology os endoscopic biopsy specimen showed features suggestive of Menetrier's disease. Treatment with oral cimetidine was continued along with high protein and calorie diet but the symptoms persisted. Subsequently, an exploratory laparotomy was done and the findings at surgery were a hypertrophied stomach with polypoid mucosa involving the fundus and body. The antroduodenal region was grossly normalTotal gastrectomy with direct esophagoduodenostomy was done The second histology report confirmed Menetrier's disease. Menetrier's disease is a rare form of hypertrophic gastropathy. It is a premalignant disorder of the stomach. Cytomegalovirus and Helicobacter pylori infections, and transforming growth factor alpha , have been implicated in the aethiopathogenesis of the disease. Conservative treatment by aggressive eradication of H.pylori improves patients symptoms, and abrogates the protein losing enteropathy characteristic of the disease. Surgery by total gastrectomy offers the best definitive treatment as it removes the risk of gastric cancer in a gastric remnant.

KEY WORDS: Menetrier's Disease, Helicobacter Pylori, Total Gastrectomy

Introduction

Menetrier's disease was described by Menetrier in 1888. It is a rare form of hypertrophic gastropathy characterised by thickening of gastric mucosal folds due to foveolar (pit) hyperplasia.[1-3] Associated biochemical features include hypochlorhydria, increased mucus secretion, and hypoalbuminaemia due to a protein losing gastroenteropathy.[4,5] The aetiology is unknown but infec

tions by Cytomegalovirus, [6-7] and Helicobacter pylori, [8-9] have been implicated. Also transforming growth factor alpha may have a possible role in the pathogenesis. [9-10] This is a report of our experience with a case of Menetrier's disease.

Case report

A 25 year old man presented with epigastric pain , postprandial vomiting which was non projectile and non bilous, progressive weight loss, and body swelling for 2 years. He passed well formed stools.There were no symptoms referable to the cardiorespiratory and urinary systems.He was on empiric treatment for peptic ulcer disease with various forms of antacids, and H-2 receptor antagonists without any appreciable improvement before referal.

Physical examination showed wasting, dehydrationb, no pallor and no peripheral lymphadenopathy. There was facial puffiness and bilateral pitting pedal oedema up to the knees. The vital signs were stable. The cardiorespiratory systems were essentially normal.The abdomen was tender in the epigastrium but there was no palpable intraabdominal mass. The liver span was within normal limits. Succussion splash was positive. There was no ascites. The bowel sounds were normal. Rectal examination was normal. A working diagnosis of gastric outlet obstruction due to chronic duodenal ulcer was made.

Figure 1: Histolgy of Endoscopic Specimen Showing Mrked Hyperplasia of Gastric Glands

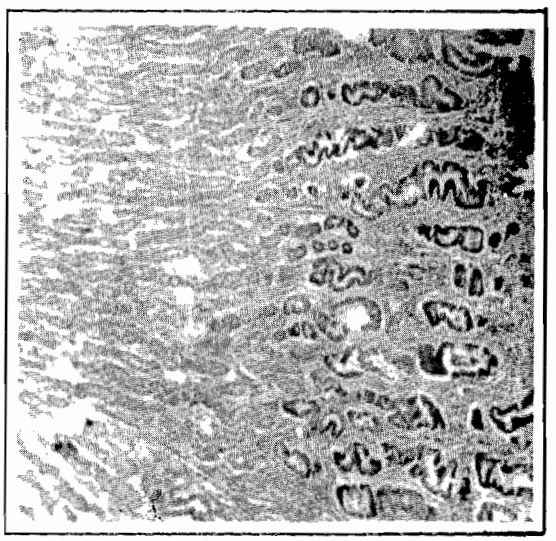
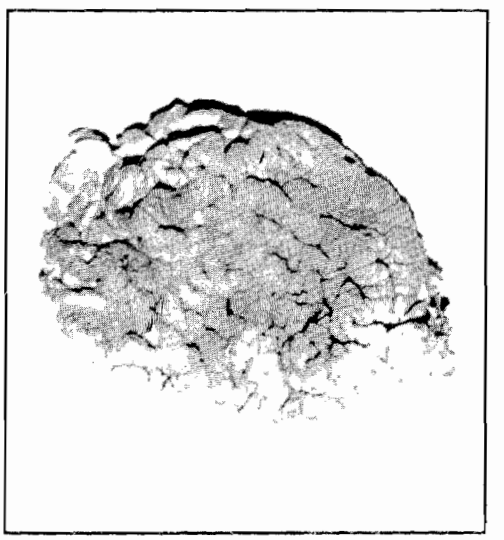


Figure 2: Mucosal Appearance of Resected Stomach



The results of relevant investigations showed normal heamoglobin level, urea and electrolytes. There was hypoproteinaemia with serum proteins of 40.0g/dl (normal value = 62 - 82g/dl). There was no proteinuria. A gastroduodenoscopy revealed polypoid gastric mucosa involving the fundus and body of the stomach but sparing the antrum ; there was no ulceration or gastric outlet obstruction. The histopathological report of the endoscopic biopsy revealed hyperplastic mucosa with foveolar hyperplasia, suggestive of Menetrier's disease.

Treatment with oral cimetidine was continued along with high protein and calorie diet but the symptoms persisted . Subsequently , an exploratory laparotomy was done and the findings at surgery were a hypertrophied stomach with polypoid mucosa involving the fundus and body. The antroduodenal region was grossly normal(figure 1).Total gastrectomy with direct esophagoduodenostomy was done after an extended Kocherisation of the duodenum. The second histology report showed that the stomach weighed 665g. The mucosal surface showed prominent rugae. There was marked foveolar hyperplasia of the mucosal cells, with moderate lymphocytic infiltration, findings consistent with Menetrier's disease (figure 2).

Barium swallow was done seven days postoperatively and it showed an intact anastomosis. The nasogastric tube was then removed and he was commenced on graded oral sips, and subsequently continued on kwashpap of 2000 - 2500mls daily along with tablets of vitamin c and b-complex. He was discharged home two weeks after surgery on small meals, at a time of high protein and calorie diet. He has remained well at follow up.

Discussion

Menetrier's disease or polyadenomes en nappe is a rare form of hypertrophic gastropathy characterised by giant gastric ruggae and foveolar hyperplasia usually confined to the gastric body and fundus, sparing the antrum.[1-4] There is associated widening of gastric tight junctions which is responsible for the non-selective protein loss and hypoalbuminaemia seen in this disease.[5] It is a rare disease world wide, and most of the publications in literature are case reports. Our patient presented with clinicopathological features of Menetrier's disease.

The aetiology,pathogenesis and natural history are mostly unknown. Epidemiologic and molecular studies have shown that cytomegalovirus (CMV) infection may be involved in the pathogenesis in both adult and paediatric Menetrier,s disease.[6,7,11] Evidence for this include isolation of CMV inclusion bodies in gastric biopsies, CMV specific immunoglobulin M antibody in serum, and CMV-DNA in patients with Menetrier,s disease;[7,11] All these strongly suggest a pathogenic role of the virus. Similarly there are supportive evidences implicating Helicobacter pylori infection in Menetrier,s disease;[8-9,13-16] In a retrospective analysis, H. pylori was found in more than 90% of patients with Menetrier,s disease;[13] Also eradication of H. pylori with prolonged antibiotic treatment in patients with H. pylori colonisation with omeprazole, metronidazole and clarithromycin has

resulted in improved patients' condition and abrogation of protein loss.[13-16] In some instances there has been resolution of enlarged gastric folds and mucosal histology.[15] The improvements observed above support an aetiological role for H.pylori infection in Menetrier,s disease.Transforming growth alpha is an epithelial mitogenproduced by gastric mucosa;it inhibits acid secretion and increases mucsu secretion.[17] Studies have shown that its altered expression may be involved in the pathogenesis of Menetrier,s disease.[9- 10] TGF- alpha immunostaining of gastric mucosal biopsies showed intense staining in mucus cells of patients with Menetrier,s disease relative to the normal control;Also data from transgenic mice model that over produces TGF-alpha, showed a number of features characteristic of Menetrier,s disease,including decreased acid production,increased hyperplasia of surface mucus cells and increased mucus production[9-10] at the expense of other cell lineages. Taken together, these observations from human material and correlation with data from transgenic mouse model support an important role for TGF-alpha.

Menetrier,s disease can be managed non-operatively and operatively. Spontaneous remissions are rare, except in CMV-associated Menetrier,s disease.[12] It could also be succesfully managed conservatively by eradication of H.pylori[8,13-16] This has been found to control the protein loss and associated lessions of Menetrier,s disease, and should therefore be aggressively eradicated in any patient with this condition. H.pylori was not tested for in our patient nor was he on any empirical treatment for its eradication,so one cannot categorically comment on the effect of its eradication in this particular patient. Surgery by partial or total gastrectomy offers the definitive treatment for this problem especially for those with debilitating disease, and for cases in which there is concern over the development of gastric cancer.[1] The risk of gastric cancer in this disorder is difficult to quantify but retrospective analyses have shown to be in 10%-15% of reported cases.[1] Total gastrectomy is preferable because it removes the risk of cancer if a gastric remnant is left behind.[1] Our patient had total gastrectomy with direct esophago-duodenostomy after a well kocherised duodenum;This form of reconstruction was chosen in preference to better options like the Roux-en y,in order to avoid multiple anastomoses because of the patient,s poor nutritional state. This procedure despite its known shortcomings has been well tolerated by the patient.

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