Summary

PUD remains one of the most prevalent gastrointestinal diseases, which is directly caused by infection with *H. pylori* or by NSAIDs. There are a variety of mechanisms whereby *H. pylori* may produce mucosal injury. NSAIDs can injure the gastro-duodenal mucosa through both local and systemic effects.

Abdominal pain is the cardinal symptom of PUD. The pain occurs before meals or overnight, relieved by antacids, milk, food, and acid suppressing treatments. Moreover, serology is a test for initial diagnosis of *H. pylori*, and the urea breath test is the method of choice for follow up. Diagnosis of PUD by UGI radiography requires the demonstration of barium within the ulcer crater, so endoscopy is the most reliable method of diagnosing a gastric ulcer, with accuracy higher than 97%.

Drugs may heal ulcers by eradication of *H. pylori* infection, or neutralization of acid secretion. PPIs provide more complete inhibition of acid secretion than the H2 receptor antagonists. Life style modifications are also in order. The need for elective surgery to reduce gastric acid secretion as a mean for treating PUD has been virtually abolished by the development of safe and highly effective antisecretory medications and by the recognition that ulcer recur uncommonly if *H. pylori* infection and NSAID use are eliminated. The indications of surgery in PUD are bleeding, perforation, obstruction, and intractability or non-healing. One of three basic operations is used: highly selective vagotomy, vagotomy and drainage, and vagotomy with distal gastrectomy.

Peptic ulcer is the commonest cause of acute upper gastrointestinal bleeding. The combination of *H. pylori* infection and NSAID use may increase the risk of ulcer hemorrhage. The primary diagnostic modality for evaluation of UGIB is currently OGD. The major nonthermal therapy

used for control and prevention of ulcer bleeding is injection. Endoscopic injection therapy is the first-line therapy for non-variceal UGIB. Thermal therapy includes: LASER, monopolar electrocoagulation, and transendoscopic bipolar electro coagulation and heater probe therapy. Application of a metal hemoclip to a bleeding vessel is an alternative to the currently available techniques. Embolotherapy performed after diagnostic angiography was demonstrated. Surgery is required in 10% of patients with bleeding ulcer. Bleeding duodenal ulcer requires direct exposure of the ulcer in the duodenum by way of duodenotomy. Moreover, suture ligation of bleeding duodenal ulcer with pyloroplasty and truncal vagotomy is successful in acutely controlling hemorrhage in 90% of patients. Ulcers of the incisura, antrum, and distal body of the stomach should be managed with distal gastrectomy and Bilroth I or II reconstruction.

Perforation is the second most common complication of peptic ulcer, and is a major cause of death in the elderly people. The perforations are associated with NSAID use in up to one half of cases. Endoscopy should be avoided when perforated peptic ulcer is suspected. Non-operative management for perforated duodenal ulcers consists of resuscitation with intravenous fluids, intravenous antibiotics, nasogastric suction and acid reducing drugs. The option for surgical treatment of perforated duodenal ulcer are simple patch closure, patch closure and HSV, or patch closure and vagotomy plus drainage. Laparoscopic surgery especially the simple repair of the perforation has became routinely used.

Penetration occurs when a peptic ulcer burrows through the wall of the stomach or duodenum into an adjacent organ e.g. pancreas, the left lobe of the liver, rarely can result in choledocho-duodenal fistula and gastrocolic fistula.

The majority of these fistulae heal spontaneously with intensive medical management. However; when surgery is recommended, the most common management for the fistula is excision which can be carried out laparoscopically.

Ulcers within the pyloric channel and first portion of the duodenum usually are responsible for gastric outlet obstruction. OGD has the advantage of biopsy in order to aid in the differentiation of the origin of the obstruction. Obstruction may be treated medically if results of imaging studies or endoscopy determine that acute inflammation and edema are the principle causes of the outlet obstruction. However, endoscopic dilation can be done, failed balloon dilations have been associated with long "tortuous" strictures, along with severe fibrosis. Performing dilation via gastrostomy or duodenostomy can be also used.

The relation between peptic ulcer and gastric carcinoma has long been a matter of controversy. Gastric carcinogenesis is a process that proceeds from normal gastric mucosa with epithelial hyperproliferation to chronic gastritis with variable degrees of atrophy, intestinal metaplasia, dysplasia, and ultimately carcinoma. *H. pylori* infection is accepted as the major causal factor for the histological features leading to intestinal-type gastric cancer and gastric MALT lymphoma. It is widely accepted that *H. pylori* eradication heals active gastritis and dramatically reduces peptic ulcer disease. When multiple endoscopic biopsies are performed, the probability of diagnosing a malignancy is also in excess of 97%.

Treatment strategy is related to stage. Dysplasia and early gastric cancer are amenable to endoscopic treatment. Reconstruction of gastrointestinal continuity is preferably by a Roux-en-Y jejunal loop. Jejunal interposition pouches have been constructed to improve nutritional intake.