# COMPLICATIONS OF PEPTIC DUODENAL ULCER AND THEIR TREATMENT

**ESSAY**

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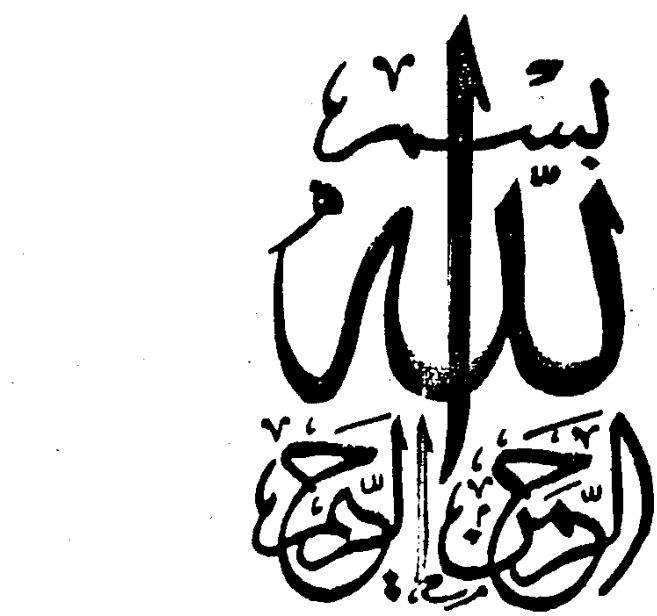


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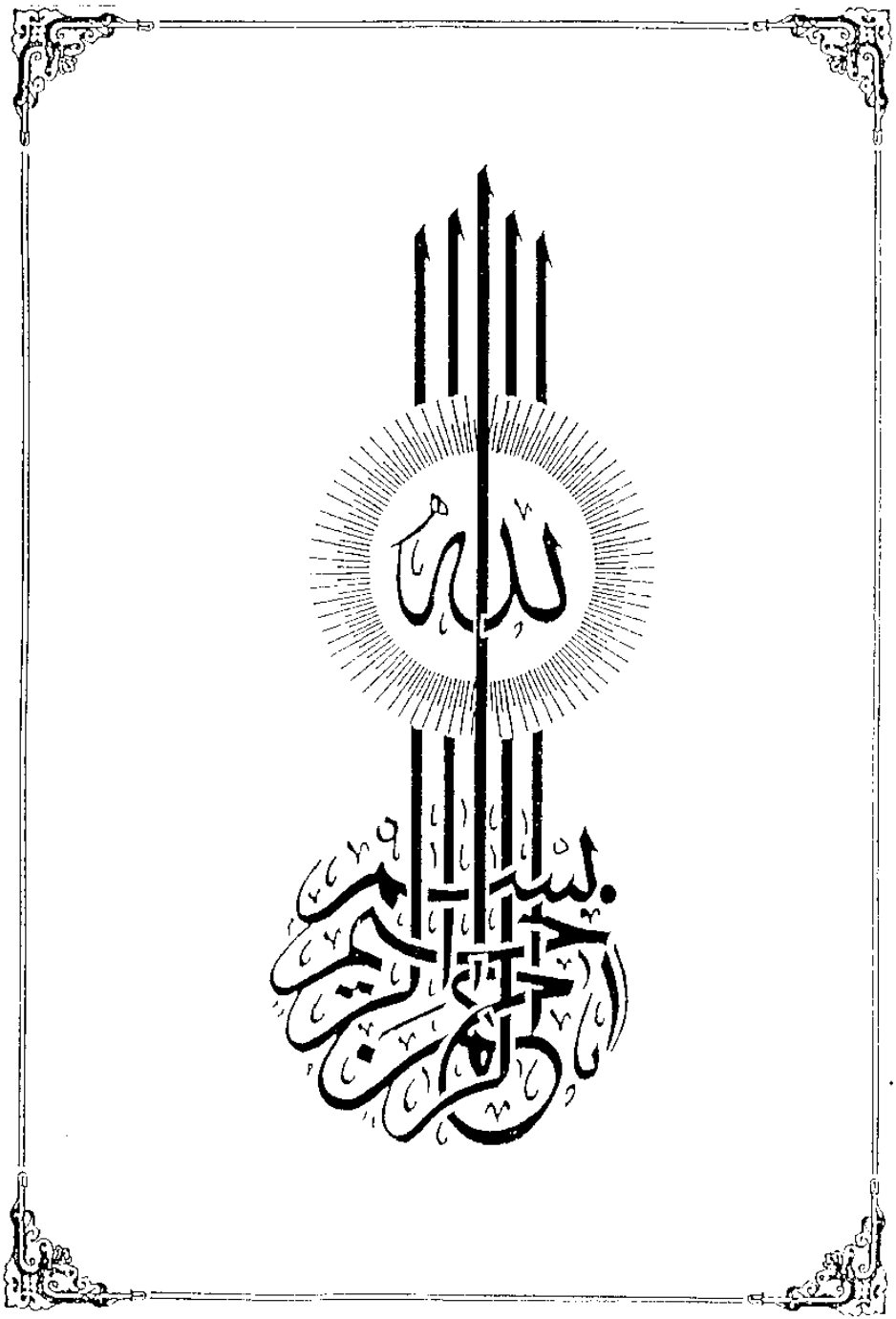

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# INTRODUCTION

Diseases of the gastrointestinal tract are common accounting for almost one-third of medical refrrals to hospitals. A large part of these consists of stomach and duodenum disorders. In a typical general practice of 2500 approximately 380 patients per annum consult the doctors for gastrointestinal disorders **(Fry, 1979).**

Peptic ulceration has a high rate of disability and is responsible for great costs to society in loss of production time and in medical care **(Thompson, 1980).**

The main cause of peptic ulceration is still uncertain, but many theories explain it, duodenal ulceration is that lesion which results from a relative or absolute excess of acid-peptic gastric secretions **( Menguy, 1976; Way, 1981).**

By definition peptic ulceration is a break in the mucosa exposed to acid-pepsin secretion. Ulceration usually occurs at mucosal junctions for example:

* Duodenal ulcers usually occur in duodenal mucosa adjacent to antral mucosa.
* Anastmotic ulcers occur in jejunal mucosa.
* Oesophageal ulcers occur in squamous epithelium at the oesophagogastric junction.
* Peptic ulceration can also occur in a Meckel's diverticulum.

Peptic ulceration is a world-wide disease. the incidence of peptic ulceration and its associated complications reached a peak between 1920 and 1950, and now appears to be declining. In developed countries the incidence in people over the age of 15 years is 2/1000/years.

Duodenal ulceration is more common than gastric ulceration with a ratio of 4:1 and there is a strong male predominance in each disorder. The prevalence of peptic ulcer disease in western countries is approximately 12% for men and 9% for women, ulceration has a mortality of approximately 5/100,000. The risk is greater in men and patients with a chronic gastric ulcer and increases with age. The major cause of death is gastrointestinal haemorrhage.

Although both duodenal and gastric ulcers are examples of peptic ulceration, they have separate aetiologies. Indeed, there is now considerable evidence that duodenal and gastric ulceration have a variety of causes. It is assumed that all patients with peptic ulcers have a disturbance in the ratio of gastric acid-pepsin secretion to mucosal resistance.

Many patients with duodenal ulceration have a gastric secretory capacity higher than normal. Duodenal ulceration does not occur in patients with a gastric acid secretory rate of less than 12.5 m mol/hour.

In patients with Zollinger-Ellison syndrome, duodenal ulceration is due to hypersecretion of acid. Mucosal resistance is also important. The cause of the gastric hypersecretion associated with duodenal ulceration is not known, but may be due to a hypothalamic abnormality resulting in increased vagal stimulation **(Byrns and Hugh, 1982).**

Genticaly there appears to be a strong predisposition towards duodenal ulceration, first relatives are affected approximately three times more often than the general population. This has been confirmed by studies which showed that 50% of identical twins but only 14% of dizygotic twins were affected. Other factors associated with duodenal ulceration include group 0, non-secretion of salivary blood group antigens, hyperpepsinogenaemia and HLA-B5 antigen.

Smoking causes a small but definite increase in the incidence of duodenal ulceration.

A hormonal factor can be suspected by the fact that duodenal ulceration is uncommon during pregnancy and perforated ulcer is uncommon in premenopausal women. Two studies have shown that oestrogens accelerate ulcer healing, but side effects preclude their use.

In the presence of chronic disease, it is claimed that duodenal ulceration is more common, for example in patients with chronic

obstructive airway disease, alcoholic cirrhosis and chronic renal failure. The relationship has been questioned because the presence of two diseases may increase the chance of a patient seeking medical treatment.

There is no convincing evidence that steroids or analgesics are causally associated with duodenal ulceration, also there is no convincing evidence that psychological stress is important in causing an ulcer **(Byrnes and Hugh, 1982).**

The natural history of a duodenal ulceration is that of a chronic illness. Recent clinical trials have demonstrated by endoscopy that without treatment approximately 65% of ulcer patients relapse within one year of ulcer healing, endoscopy of duodenal ulcer patients when they are asymptomatic and receiving no treatment has demonstrated that there is active ulceration in upto 28%. Until recently, duodenal ulceration was considered to be a relatively transient illness reaching a peak after 5-10 years and then settling spontaneously. Current data do not however, confirm this impression. For most patients the major symptom of duodenal ulceration is pain, with approximately 15% of patients having a history of haemorrhage and only one per cent having a history of perforation, pyloric stenosis has an even lower incidence **(Pounder, 1985).**

This thesis aims at reviewing the complications of duodenal peptic ulcer and its different types of treatment. It is essential to discuss briefly the anatomy and physiology of the stomach and duodenum before hand.

# Anatomy of the Stomach and Duodenum

## ANATOMY OF THE STOMACH

### EMBRYOLOGY

The stomach commences as a fusiform dilatation of the foregut at about the fourth week. At this time, it has a ventral and dorsal mesentery. Very active growth takes place along the dorsal border, which becomes convex and forms the greater curvature of the stomach. The anterior border becomes concave and forms the lesser curvature. The fundus appears as a dilatation at the upper end of the stomach. At this stage, the stomach has a right and left surface to which the right and left vagus nerves are attached respectively. With the continued active growth of the dorsal border of the stomach along with the great growth of the right lobe of the liver, the ventral border of the stomach gradualy rotates toward the right so that the left surface becomes anterior and the right surface becomes posterior. The ventral and dorsal mesenteries become altered in position as a result of this rotation of the stomach and they now form the omenta of the stomach and various peritoneal ligaments. A pouch of peritoneum becomes located behind the stomach and is known as the omental bursa **(Snell, 1975).**

The stomach is a muscular bag, fixed at both ends, mobile elsewhere, and subject to great variations in size in conformity with the volume of its contents. It consists of fundus, bodoy, pyloric antrum and pylorus.

**The fundus:** is that part which projects upwards, in contact with the left dome of the diaphragm, above the level of the cardiac orifice.

**The body:** extends from the fundus to the level of the incisura angularis, a constant notch in the lower part of the lesser curvature.

**The pyloric antrum:** extends from this level, narrowing gradualy towards the pylorus.

**The pylorus:** is propably thicker than the rest of the stomach wall and the pyloric canal is held closed by the tonus of the pyloric sphincter except when the latter relaxes to allow the stomach to expel a jet of its contents into the duodenum **(Last, 1984).**

**Peritoneal attachments**

With the exception of a small area on the posterior wall below the oesophageal orifice the stomach is entirely covered with peritoneum. At the lesser curvature the peritoneal layers covering the anterior and posterior walls form the lesser omentum, which extends upwards to the liver, being attached to the margins of the porta hepatis. From most of the greater curvature the peritoneal layers sweep downwards as the anterior two layers of the greater omentum; these two layers then turn upward (as the posterior two layers of the greater omentum) and enclose the transverse colon.

At the greater curvature of the fundus the peritoneum sweeps off the stomach as the gastrosplenic and gastrophrenic ligaments.

**Blood supply of the stomach**

The blood supply of the stomach is so profuse that any one of the large vessels supplying the organ is sufficient to maintain its vitality providing the marginal anastmotic vessels are patent. **(Du Plessis, 1984).**

1. **The left gastric artery:** a branch of the coeliac trunk, runs behind the peritoneum from the upper border of the pancreas across the left crus of the diaphragm to the oesophageal hiatus. It enters between the two leaves of the lesser omentum and turns to the right along the lesser curvature. It breaks into two parallel branches which anastmose end on with the two branches of the right gastric artery.

2. **The right gastric artery:** a branch of the hepatic artery. It leaves the hepatic artery as it turns into the lesser omentum. The vessel passes to the left between the layers of the lesser omentum and divides into two branches which anastmose end on with the branches of the left gastric artery.

3. **The gastroduodenal artery** is the largest branch of the hepatic artery. It passes behind the first part of the duodenum, where it bifurcates into the superior pancreaticoduodenal artery and the right gastroepiploic artery.