Study Of Helicobacter Pylori In Duodenal Ulcer Patients In The Presence Or Absence of Reflux Oesophagitis

Thesis
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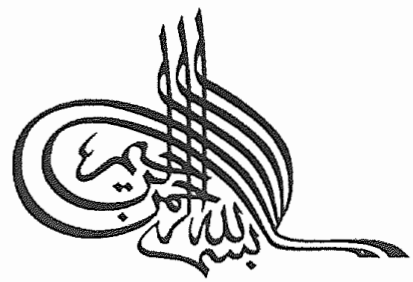

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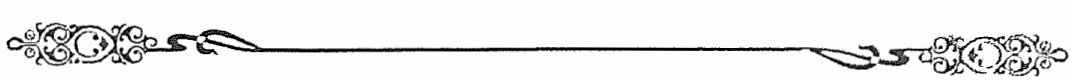

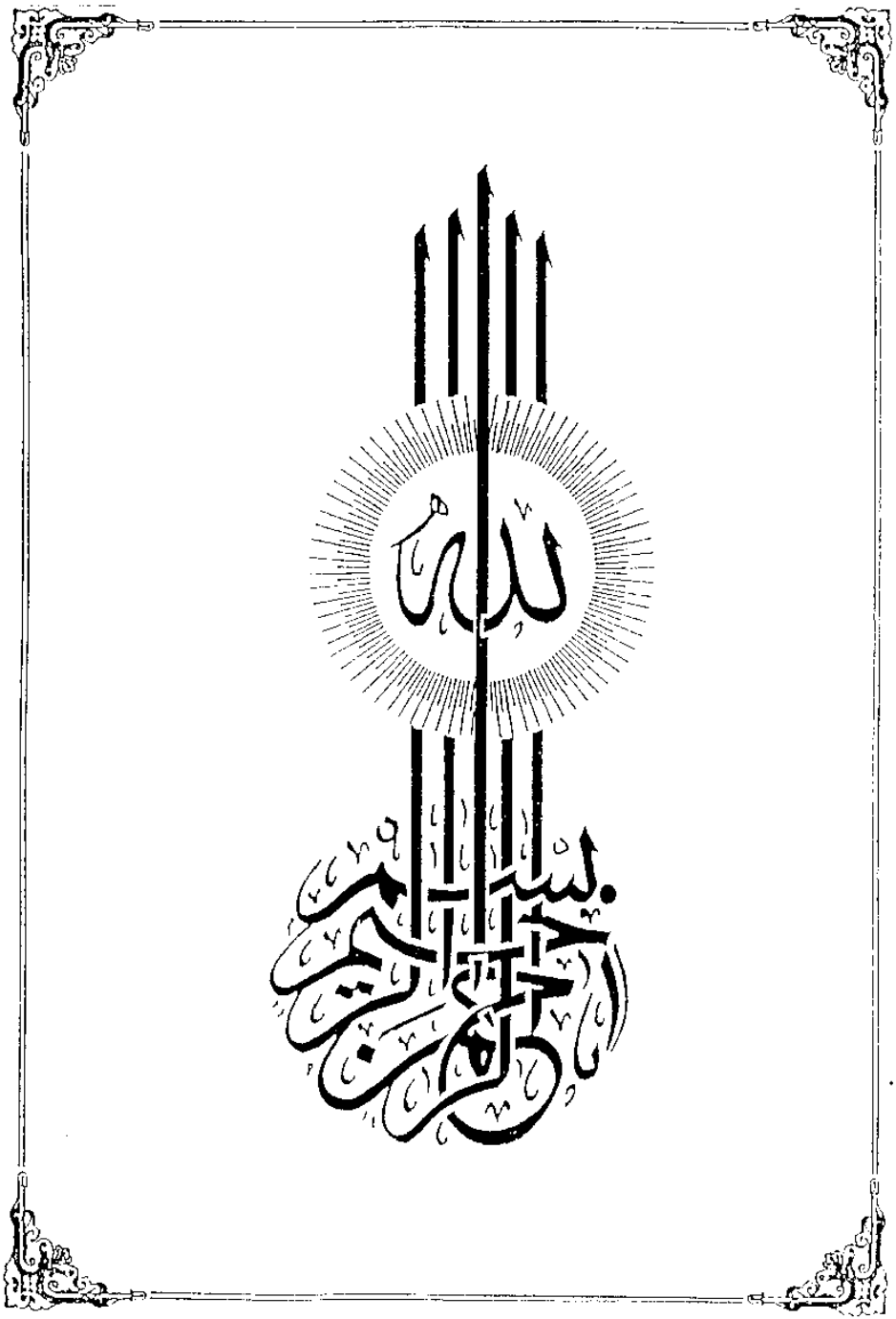

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* 53	1	Coghlan	Coughhlan
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Introduction and Aim of work

Study Of Helicobacter Pylori In Duodenal Ulcer Patients In The Presence Or Absence of Reflux Oesophagitis

Introduction :-

Numerous studies reported over the last several years have established a close association between antral Helicobacter pylori (H. pylori) and peptic ulcer disease. In a combination of data from 15 studies, 92% of patients with duodenal ulcer have H. pylori identified in antral samples ***(Tytgat and Ravws, 1990).***

The presence or absence of H. pylori associated gastritis is a crucial predictive factor for peptic ulcer. In a ten year study of asymptomatic subjects, only one of 133 persons without gastritis at the initial evaluation, developed peptic ulcer, compared with an 11% occurrence (18 duodenal ulcer, 16 gastric ulcer) in 321 subjects with gastritis ***(Sipponen et al., 1990).***

On the other hand, reflux oesophagitis is common in duodenal ulcer patients and many duodenal ulcer patients complain of typical heart burn. This lead Earlam to argue that the esophagus is a major source of symptoms in duodenal ulcer ***(Earlam, 1985).***

Aim Of The Work :-

The aim of this work is to study the H. pylori in duodenal ulcer patients (both antral and lower oesophageal mucosa) in relation to the presence or absence of reflux oesophagitis.

Review of Literature

Review of Literature

Peptic Ulcer

Pathogenesis of peptic ulcer :-

Peptic ulcer is an ulcer that occurs in those portions of the alimentary tract which come into contact with gastric juice. So, it could occur in the lower part of the oesophagus, in the stomach, in the duodenum, in the jejunum after gastroenterostomy and in the duodenum and jejunum in the patient not operated upon in Zollinger Ellison syndrome.

There is no single etiologic factor responsible for peptic ulcer, and each factor that influences the final outcome acts as a contributor. The development of chronic peptic ulcer is determined by the algebric sum of two groups of forces acting upon the gastrointestinal mucosa; the one defensive, and the other aggressive (destructive) ***(Hollander, 1954).***

• Aggressive factors involved in the disturbance of the mucosal integrity:

1- HCL hypersecretion :-

For peptic ulcer to occur, there should be Hcl and pepsin. The evidences are;

a) The absence of duodenal ulcer in conditions in which achlorhydria is common.

b) The high incidence of duodenal ulcer in conditions associated with high rates of gastric secretion as Zollinger Ellison syndrome.

c) The healing of duodenal ulcer when gastric secretory capacity reduced.

A- Basal acid secretion :-

An increased basal acid output suggests the presence of an increased basal drive and is compatible with the concept of increased vagal tone ***(Dragstedt, 1956).***

Most investigations agree that duodenal ulcer patients have an increased basal acid output. ***Feldman et al., (1980)*** defined that an increase in basal acid output does not appear to be a common abnormality in patients with duodenal ulcer and occurs in about 10 - 20% of patients studied.

B- Maximal acid output :-

In any given population of duodenal ulcer patients, there are both hypersecretors and normosecretors, the proportion of hypersecretors varies in different parts of the world.

Lam and Sircus, (1975) suggested that patients with a positive family history of ulcer dyspepsia may hypersecrete when compared with and without such history.

C- Nocturnal acid secretion :-

Acid is continuously secreted by the normal human stomach during sleep ***(Stacher et al., 1975).*** Because food buffers is not available, the duodenum is exposed to long hours of acidity and hypersecretion may take place in those with duodenal ulcer because of an abnormally large parietal cell mass, hyperfunction of G cells and increased vagal derive.

A recent study aimed at studying the effect of controlling the nocturnal acid secretion (with a single night-time dose of H_2-receptor antagonist) on duodenal ulcer healing showed that healing could be achieved in the majority of patients ***(Lam et al., 1983).***

2- Pepsin hypersecretion :-

Pepsin output is correlated to acid output, and in general the two are secreted in response to the same stimuli. Pepsin hypersecretion is as important as acid hypersecretion in ulcer pathogenesis. A radioimmunoassay for group I pepsinogens has been established, which gives a good correlation between maximal acid output and serum pepsinogen I. These results indicate that 2/3 of duodenal ulcer patients have concentrations greater than 175 ngm / ml while only 8% of normal subjects have levels in excess of that figure ***(Pearson et al., 1986).***

3- Increased parietal cell mass :-

The concept of increased parietal cell mass was suggested by the finding that many patients with duodenal ulcer had very large stomachs at autopsy and large number of parietal cells on microscopy.

Card and Marks, (1960) supported this concept by relating the difference between pre and post operative acid output to the actual number of parietal cells removed at gastrectomy. It is estimated that duodenal ulcer patients have an average of 1.9 billion parietal cell compared to 1 billion in normal subjects and 0.8 billion in gastric ulcer.

Administration of exogenous gastrin increases parietal cell mass and acid secretory capacity ***(Johnson, 1976).***

4- Mechanical defects :-

The rate of emptying the gastric contents in the duodenum and expulsion of acid from the duodenum may play a role in the pathophysiology of duodenal ulcer. It is believed that the majority of patients with duodenal ulcer empty both fluids and solid gastric contents abnormally rapid. Indeed, acid in the duodenum increases the rate of emptying in many patients with duodenal ulcer, while slowing emptying in normal subjects ***(Fordtran and Walsh, 1973).***

It has been suggested that high energy meals are emptied rapidly in duodenal ulcer patients as a result of dysfunction of the duodenal receptors controlling the rate of gastric emptying ***(Stubbs and Hunt,***

1975). Also, the abnormally rapid rate of gastric emptying in the duodenal ulcer patients may be a consequence of an abnormal motor response to hormones.

5- Mucosal circulation abnormalities :-

Abnormalities of the mucosal circulation resulting in ischaemia have been repeatedly proposed as the basis for the discrete nature of both gastric and duodenal ulcers. It has been shown that some parts of the duodenal mucosa are supplied by end arteries, particularly in the ulcer prone parts of the duodenum, this led to the conclusion that focal mucosal ischaemia contributes to mucosal ulceration of the duodenum and was, perhaps, responsible for tendency to chronicity ***(Piasecki, 1977 & Kirk, 1982).***

6- Immunologic abnormalities :-

Duodenal ulceration may have an immunologic basis. Peptic ulcers have been described in children seen at an allergy clinic, and allergic disorders have been reported in patients of children with coeliac disease who also have an increased incidence of peptic ulcer ***(Rotter and Heiner, 1982).***

Patients with duodenal ulcer are reported to show more frequent occurrence of antibody to secretory immunoglobulin A (45%) of peptic ulcer patients compared with 11% of controls ***(Wilson, 1972 & Kwitko et al., 1980).***