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THE RELATION BETWEEN GASTRIC ACIDITY & CAMPYLOBACTER PYLORIDIS IN CASES OF DUODENAL ULCER

THESIS

Submitted for Partial Fulfillment of
Master Degree in
General Medicine

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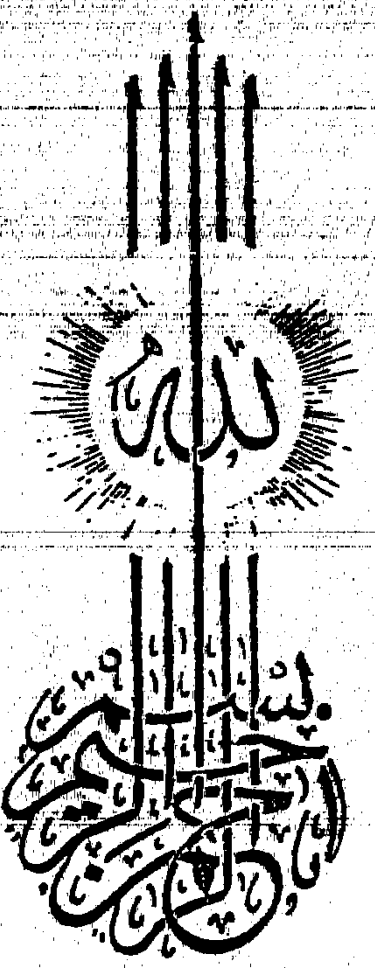
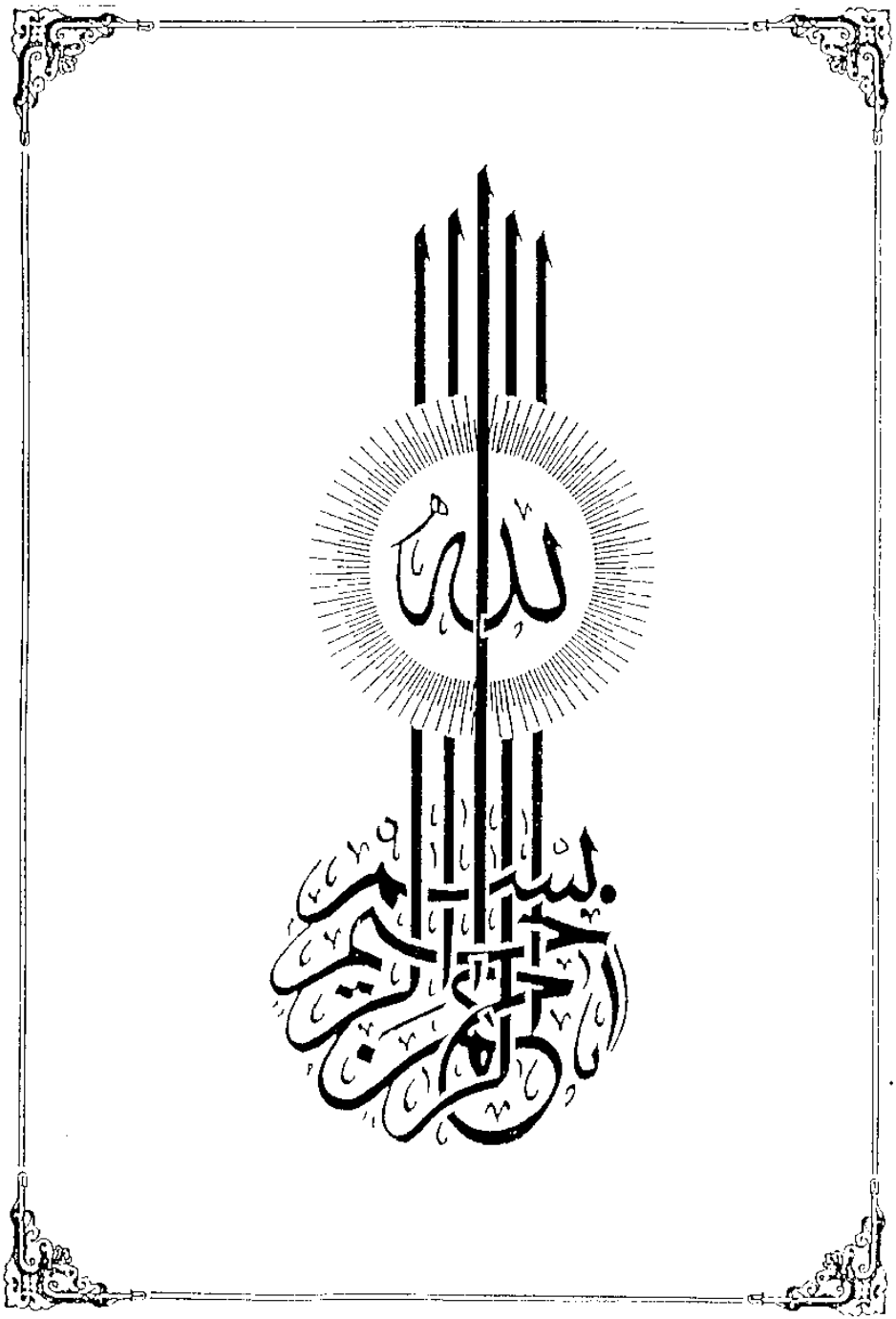
1987





ACKNOWLEDGEMENT

I would like to express my deep thanks and profound gratitude to Professor Dr. Mohamed AbdéEl-Fattah Taha , Professor of Medicine, Ain Shams University, Faculty of Medicine who assigned the work, helped me to overcome all the obstacles and continously guiding me to the essential points for the success of this work.

I also would like to express may sincere gratitude to Dr. Ibrahim Khalil Ali Assistant Professor of Clinical Pathology, for his encouragement and helpful directions during this work.

I am sincerely grateful to Dr. Mohsen Moustafa Maher Lecturer of Medicine for his unfailing support and continuous advice.

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INTRODUCTION

I N T R O D U C T I O N

Although much is known concerning the factors which contribute to the development of duodenal ulcer. We do not completely understand its pathogenesis.

Acid secretion by stomach is required for the production of duodenal ulcer. But the factors which renders the acid secretory subject susceptable to duodenal ulceration, are not completely understand. More than one half of duodenal ulcer patients have gastric acid secretory rates within normal range.

Aim of Work

Whether this bacterium has any role in the pathogenesis of duodenal ulcer, is a matter of various investigations.

Can there be any relation between gastric acidity secretory rates and the presence of such organisms in gastric mucosa of duodenal ulcer patients.

REVIEW OF LITERATURE

DUODENAL ULCER

A duodenal ulcer is a distinct break in the mucosa of the duodenum, almost invariably in the duodenal bulb. The ulcer may be superficial or may be penetrate to the serosa. Gastric acid secretion is an important factor in ulceration, but there is considerable overlap between the amount of acid secreted by healthy individuals and duodenal ulcer patients (Pounder, 1985).

Acid secretion by the stomach is required for production of a duodenal ulcer, but the factors which render the acid secreting subject to duodenal ulcer are not completly understood. As a group duodenal ulcer patient secrete more acid than normal, however, one half to two thirds of duodenal ulcer patients have gastric acid secretory rates, both basal acid output (BAO) and maximum acid output (MAO) within the normal range (McGuigan,1984).

The aggressive factors in the gastric juice eroding or digesting its way into (and through) the duodenal mucosa. While the defective factors are stated to include bicarbonate (which neutrilize acid, inactivate pepsin , and the duodenal lumen) act as a barrier to acid and

pepsin to maintain the state of duodenal mucosa. When the scales are tipped invafour of aggression ulcer develops (Wormsley, 1983). Traditional veiws of the pathology of duodenal ulcer disease consider the disease as being of excessive acid secretion. Duodenal ulcer patients have higher maximal rates of acid secretion because of their large parietal cell masses. However, the average duodenal ulcer patients secrete at maximal rate for only a short period after meal, otherwise secretion is clearly submaximal (Walsh, 1979).

Duodenal ulcer disease results from an imbalance between duodenal acid load and injerous force and mucosal prostaglandin generation, protective factor. The prostaglandins maintain mucosal integirty and accelerate ulcer healing (Hillar et al., 1985).

Mucosal prostaglandin generation in the duodenum is induced postcibum in relation to duodenal acid load. This may be a physiologic example of adaptive cytoprotection but in duodenal ulcer there may be a defect in such mechanism (Ahlquist et al., 1983).

Duodenal bulbar pH: the evidence most often interpreted as shown disequilibrium resulting from aggression by gastric juice is drived from human studies. If gastric juice is indead etiologically implicated in ulcerogenesis, the intraluminal contents of the duodenal bulb of patients with duodenal ulcer should be more acid than normal. The pH was measured at different sites in the duodenum for short periods in patients with duodenal ulcer, there were striking differences in pH were observed, adjacent to the pylorus the pH was predominantely acid wheres in the second part of the duodenum it was predominantely neutral. Moreover there was a steep gradient of pH across the first part of the duodenum. In the middle of the bulb the fluctuation of pH were unlike those seen else where in the duodenum, there the pH often fluctuating, approximately equally between extremes of acidity and neutrality. So in patients with duodenal ulcer the acidity tended to be high at all sites in the duodenum for a greater proportion of the time than in normal subjects (Rhodes and Prestwich, 1966).

The decreased duodenal bulb pH, despite an apparently normal antral pH in duodenal ulcer patients can be

explained in several ways:

1- In the low pH range seen in the antrum, minor changes in pH reflect relatively great changes in H^+ activity.

2- The volume of gastric secretions in ulcer patients may increase more than the acid concentration, so that the increased total acid output may exceed duodenal neutralizing reserves.

3- In ulcer patients, duodenal neutralizing mechanisms are less effective than in normal persons (Archambault et al., 1967).

No significant difference was found between the dudenal pH values in normal controls and in patients with duodenal ulcer (Rune and Viskum, 1969).

Gastric hypersecretion

The evidance that duodenal ulcers are caused by hypersecretion of gastric juice of nervous origin. Duodenal ulcer patients usually secrete 3 - 10 times as much hydrochloric acid during basal fasting as do healthy

people. When hypersecretion of this degree is reported expremintal animals, ulcers exactly resempling the clinical lesion usually appear. Complete division of the vagus nerve to the stomach in duodenal ulcer patients abolishes the fasting hypersecretion, indicating that it is of nervous origin and is mediated by the vagus nerve. It seems probable than in some way the tensions and strains of modern life produce this secretory hypertonus in the vagus nerve and it is in this way that the central nervous system (C.N.S) plays its role in the cause of the disease:

a. When the fasting hypersecretion in duodenal ulcer patients is abolished by vagotomy, the ulcer usually heals and if an adequate drainage operation has also been performed to prevent stasis of food in the gastric antrum, it remains healed.

b. The wid spread adaption of vagotomy and drainage in surgical treatment of refractory duodenal ulcer and the resultant inform healing of these lesions is additional evidence that this view of there development is correct (Dragstedt, 1978).

Vagotomy reduces basal and stimulants such as gastrin (Blair, 1985).

In duodenal ulcer nocturnal acid secretion is higher than normal. Acid secretion during the day time hours and nocturnal are significantly higher (Feldman, 1986).

Nocturnal and day time acid secretions are important in duodenal ulcer patients. Single dose of Cimetidine or Ranitidine can reduce recurrences of duodenal ulcer and can facilitate healing of active duodenal ulcer by blocking H_2 receptors of the stomach (Ireland, 1984).

In duodenal ulcer healing may be achieved by reducing nocturnal acid secretion alone (Lam et al., 1985).