THE ROLE OF CAMPYLOBACTER IN AETIOLOGY OF GASTRITIS

THESIS

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BY

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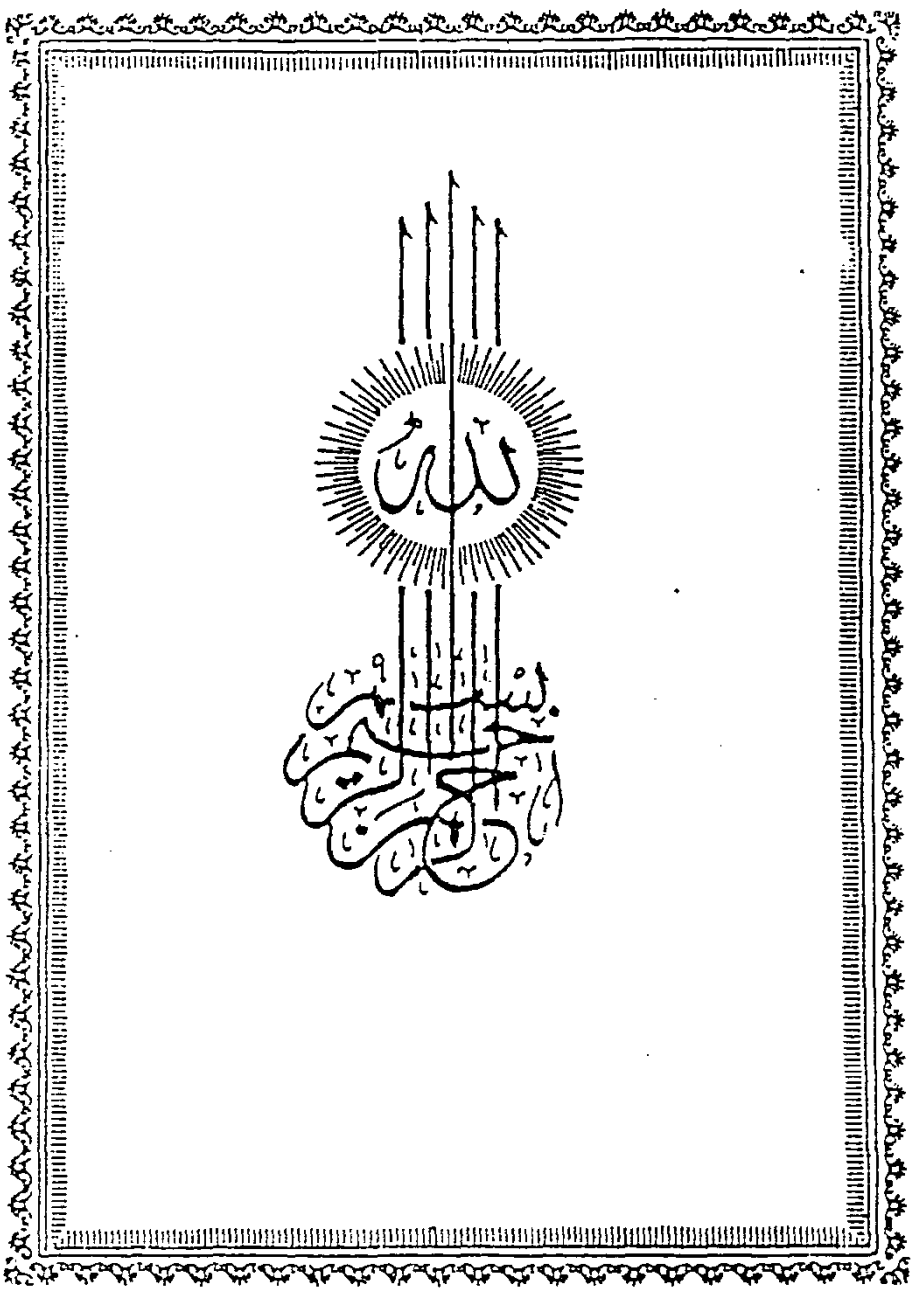

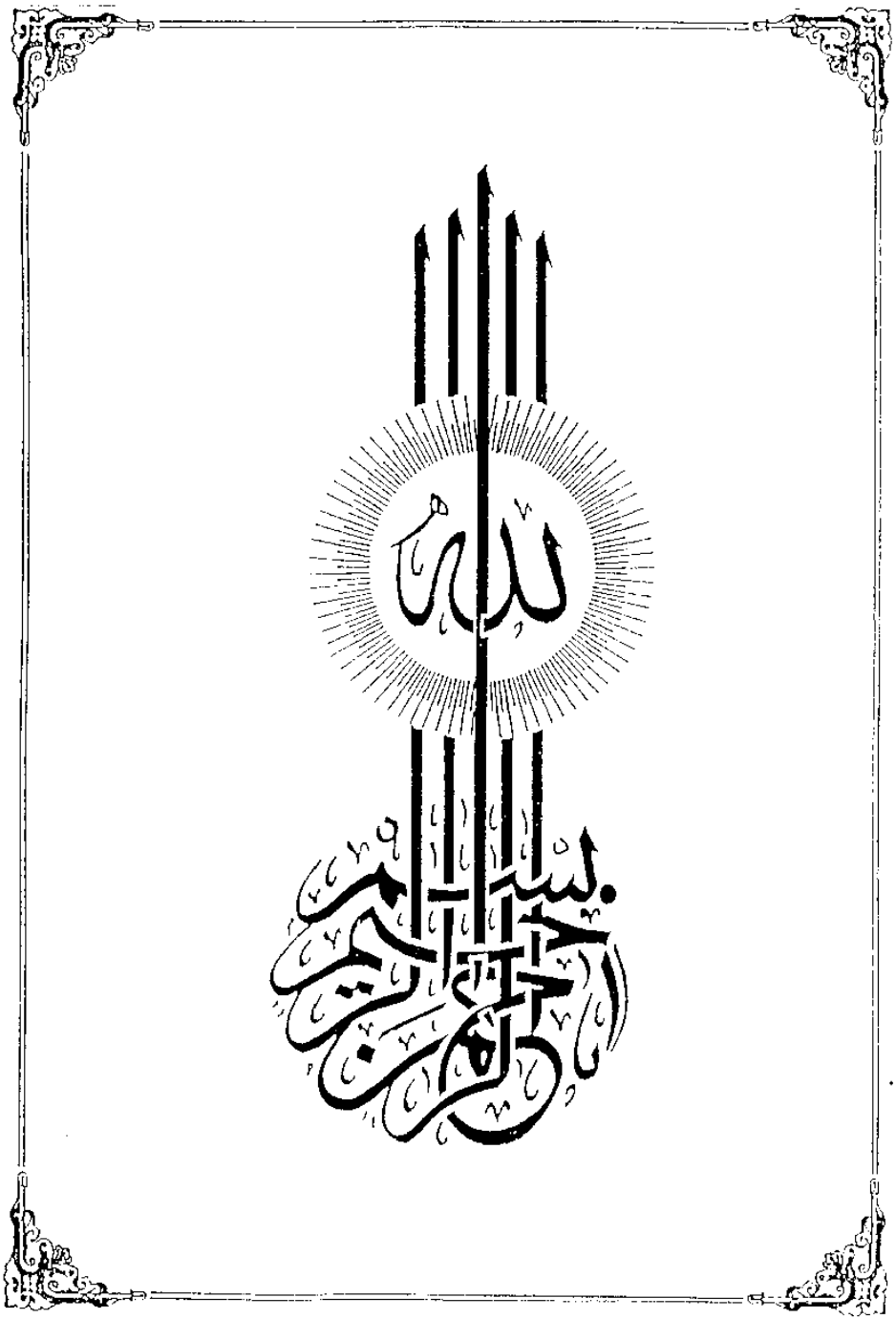

DEDICATION

To my Father's Soul

who tried always to pave my way ...

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"First and Foremost, Thanks are due to GOD"

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INTRODUCTION

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Gastric spiral bacteria have been repeatedly observed, reported and then forgotten for at least 45 years **(Doenges, 1938 & Ito, 1967).**

In **(1940) Freedburg and Barron** stated that "spirochaetes" could be found in up to 37 % of gastrectomy specimens, but examination of gastric suction biopsy material failed to confirm these findings **(Palmar, 1954).**

Since that time, the spiral bacteria have rarely been mentioned, expcet as curiosities **(Ito, 1967),** and the subject was not reopened with the advent of gastroscopic biopsy until 1983 .

Recently, spiral organisms have been seen in the stomach and they are closely associated with gastritis and duodenal ulceration **(Rollason et al., 1984; Price et al., 1985).** The provisional name of campylobacter pyloridis has been assigned to this new organism and its cultural requirements were established **(Marshall and Warren, 1984).**

Gastritis however, is a common finding in patients with dyspepsia referred for gastroscopy. An aetiological

diagnosis is frequently required. So the aim of this study is to find a correlation between gastritis as diagnosed by endoscopic picture and histopathological examination and campylobacter infection and to show the effect of its eradication (by antibiotics) on gastritis.

REVIEW OF LITERATURE

GASTRITIS

This term is extremely confusing subject and the term was often loosly used to describe gastroscopic picture or involved to explain indigestion.

Inflammation of the stomach may be diffuse and involve all parts of the stomach or localized to the fundus and body or antrum. Even within a specific area (e.g. the antrum), inflammation may be diffuse or localized.

Gastritis is classified as acute or chronic primarily on the basis of histologic and/or endoscopic findings and long term clinical follow-up. Acute gastritis is believed to be a self limited disease, whereas chronic gastritis by definition persists for long periods of time.

Acute Gastritis

Acute gastritis can be classified into two main groups; acute exogenous and acute endogenous gastritis.

1- Acute exogenous gastritis.

* Bacterial origin.
* Toxic gastritis.
* Corrosive gastritis.
* Radiation gastritis.

* Thermal, mechanical and food irritants.

2- Acute endogenous gastritis.

* Acute infectious diseases.
* Systemic disorders.
* Phlegmonous (suppurative) gastritis.
* Allergic gastritis.
* Erosions and ulcers due to stress.
* Acute erosive gastritis.

Drugs such as aspirin and other non steroidal anti-inflammatory drugs, bile salts and pancreatic enzymes damage the gastric mucosa and are believed to cause both acute and chronic gastritis. They are thought to disturb the gastric mucosal barrier, thereby allowing back diffusion of acid and pepsin. Inhibition of prostaglandin synthesis by these drugs may also be a factor **(Ivey, 1981).**

Acute gastritis also occurs in the setting of severe medical or surgical illnesses such as respiratory failure, sepsis, renal failure, hypotension or trauma. This form of acute gastritis is called "stress" ulceration and may produce stress bleeding.

Although the exact pathogenesis of "stress-related gastritis" is not known, mucosal ischaemia is believed to be an important factor **(Moody et al., 1980).**

Gastric acid is also likely to be involved, since in experimental models mucosal damage does not occur in the absence of acid.

Hastings et al. (1978) studied the effect of antacid titration in the prevention of acute gastro-intestinal bleeding by a controlled randomized trial in 100 critically ill patients. The study demonstrates that the incidence of stress-related upper gastro-intestinal bleeding in critically ill patients can be reduced by maintaining intragastric pH above 3.5 with antacid. Bile and pancreatic juice may also be contributing factors.

An epidemic form of acute gastritis of unknown etiology had been reported associated with decreased gastric acid secretion (hypochlorhydria). Since gastritis occured in a number of different persons who were in contact with each other over a relatively brief period of time, an infectious etiology was suspected; however, an organism has not been identified **(Trier et al., 1979).**

Additional causes of acute gastritis include roentgen irradiation, ingestion of corrosive substances, ingestion of staphylococcal exotoxin and bacterial infection.

Gastritis caused by bacterial infection is called acute phlegmonous gastritis. This is a rare but fulminant

form of acute gastritis. Streptococci are most commonly the cause, although staphylococci, escherichie coli and proteus have been cultured from stomachs of patients with acute phlegnomous gastritis.

Clinical Manifestations of Acute Gastritis

Patients with acute gastritis secondary to aspirin or "stress" often have hematemesis and/or melena and may have pain, nausea and vomiting. This form of acute gastritis is called acute hemorrhagic or erosive gastritis. At times, bleeding can be so severe that patients develop hypotension or shock.

Finding acute gastritis on biopsy does not necessarily mean that a patient has clinically important disease, since as many as 30 percent of otherwise healthy, asymptomatic persons can have acute gastritis on biopsy. Some forms of acute gastritis with known cause, such as acute irradiation gastritis, are not associated with symptoms. However, patients with other causes of gastritis may have symptoms. For example, some patients with epidemic gastritis with hypochlorhydria have epigastric pain, nausea and vomiting.

The physical examination in patients with acute gastritis is usually normal unless bleeding or other illness such as liver disease or arthritis is present.

If bleeding occurs,there may be reduced hematocrit,

increased blood urea nitrogen, and a positive nasogastric aspirate of stool guaiac test for blood.

Diagnosis of Acute Gastritis

Most clinically significant forms of acute gastritis are diagnosed by endoscopy. In acute gastritis secondary to aspirin or "stress", the gastric mucosa often appears congested with petechial hemorrhages, erosions and superficial ulcerations covering the mucosal surface. These changes may be diffuse although the fundus and body are most severely affected.

On biopsy inflammatory cells (usually neutrophils and mononuclear cells) infiltrate the lamina propria, whereas the glandular areas are distorted by oedema and hemorrhage. Exudates often fills the gastric pits and/or gland. In severe forms of gastritis, focal slaughing of surface epithelial cells can occur, producing superficial erosions and ulcerations.

The major feature, differentiating acute from chronic gastritis is the tendency for mucosal changes in acute gastritis to revert to normal. The time over which this occurs depends on the type of acute gastritis and on the method used to detect the end point. For example, in acute hemorrhagic gastritis the endoscopic appearance of the gastric mucosa may revert to normal within 24 to 48 hours after bleeding has stopped. Histologic reversion to normal