LONG TERM NON STEROIDAL ANTI-INFLAMMATORY DRUG USE AND HELICOBACTER PYLORI INFECTION.

THESIS

Submitted for partial fulfillment

of Master Degree

(GENERAL MEDICINE)

By

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M.B.,B.Ch.

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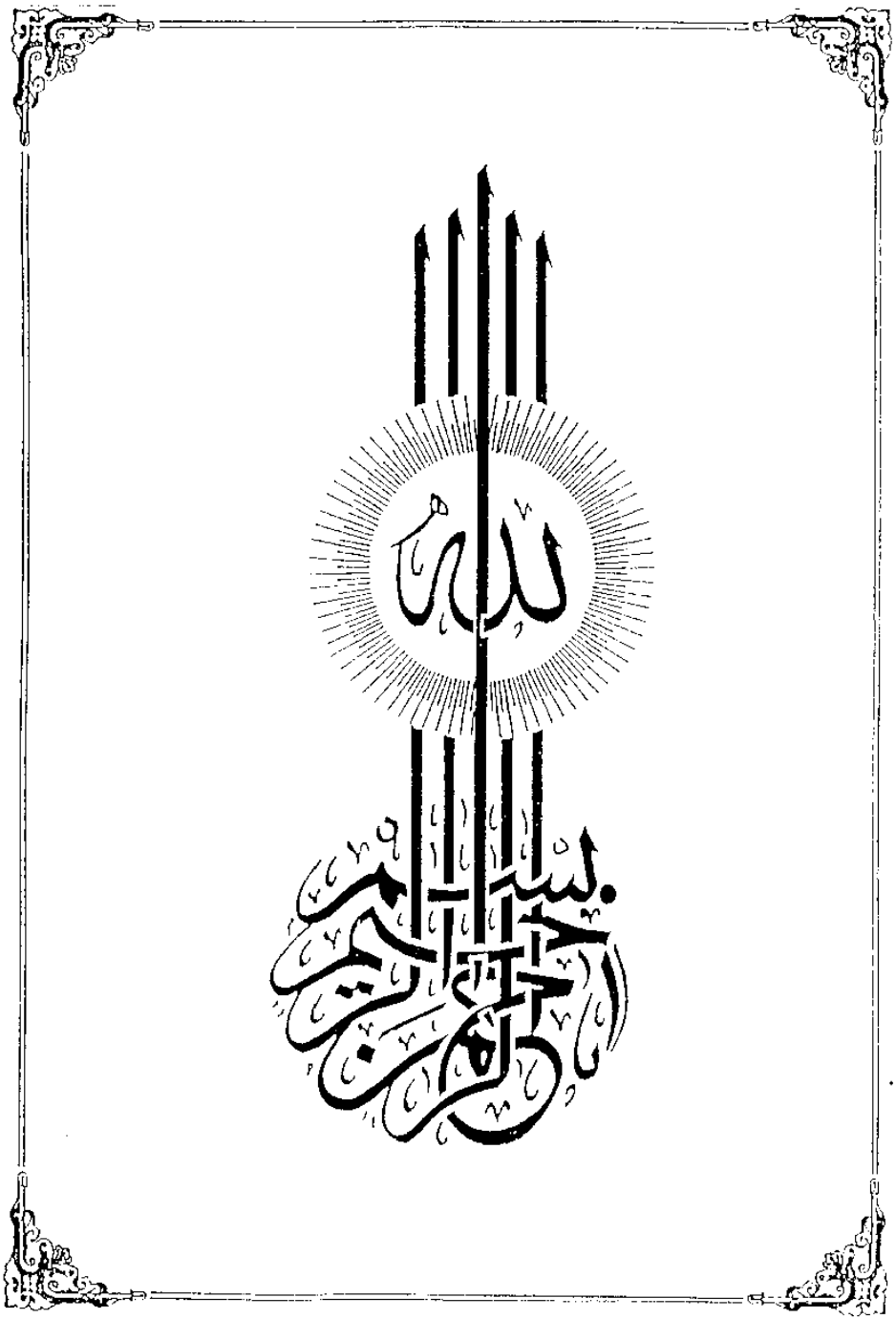

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I wish to express my deep appreciation and sincere gratitude to Professor Dr. **MOHAMED ABD EL-FATAH TAHA** Professor of Internal Medicine, Ain Shams University, for his kind encouragement valuable advice and supervision throughout the whole work.

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Ahmos Chowky Aziz

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INTRODUCTION

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Helicobacter pylori (H. pylori) is a recently rediscovered gram negative bacterium that lives in the human stomach and duodenum. Infection with this organism is strongly associated with type B antra l gastritis and peptic ulcer disease. (Blaser, 1990).

The association between helicobacter infection and peptic ulcer disease is strongest for the presence of duodenal ulcer. The use of nonsteroidal antiinflammatory drugs (NSAIDs) is also associated with peptic ulcer disease (Graham et al., 1991).

Recently, interest has focused on the possible relation- ship between helicobacter infection and non ulcer dyspepsia. Graham and his colleagues studied the prevalance of H. pylori among patients on longterm NSAID and came to the conclusion that the NSAID induced damage to the gastric mucosa does not increase the susceptibility to helicobacter pylori infection.

AIM OF THE WORK

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The aim of this work is to evaluate the association between non steroidal anti-inflammatory drugs users and helico bacter pylori infection.

REVIEW OF LITERATURE

HELICOBACTER PYLORI (H.pylori)

Steer and Colin Jones (1975) reported the presence of gram negative bacteria on gastric mucosa deep to the mucous layer in 80 % of patients with gastric ulceration. They also, described the association of this bacteria and active gastritis. Morphologically and in respect to their atmospheric requirment and DNA base composition these organisms were most closely related to the genus campylobacter. The term campylobacter pyloridis was applied to this new species of bacteria, perhaps because "the pyloric campylobacter" would do to define the site where these organisms were commonly found (Marshall and Warren, 1984). The organism was named campylobacter pyloridis but was changed into campylobacter pylori (CP),(Corneluis et al., 1988). Morerecently they were named helicobacter pylori (H. pylori). (Maddocks, 1990).

Mode of transmission of H. Pylori:

Little is known about the natural reservoir or the mode of transmission of H. pylori in humans. When and how an individual becomes colonized by H. pylori remains to be elucidated (Rauws and Tytgat, 1989),. Evidence for person to person spread came from a study of the relatives of four children with proven H. pylori infection. The family members were found to have a significantly higher incidence of H. pylori infection (64 %) than 166 age matched controls (13 %) measured by serology (Mitchell et al., 1987). In addition to spread by PH electrodes during experiments, endoscopy and biopsy equipment have been incriminated (Rokks, et al., 1988). Frickner (1984) found that H.pylori adherent to the plastic used in endoscope channels.

Repeated washing with phosphate - buffered saline failed to remove the bacteria completely stressing the need for thorough cleaning of endoscope between examination. Other similar tightly coiled spirochaetes found in cats dogs and apes. Compylobacter Mustelae, an organism similar to H. pylori is found in many ferrets, stomachs and infection can be associated with ulcers (Maddocks, 1990). Lee et al., (1988) have recently isolated a spiral shaped bacterium from a cat stomach. The isolate was urease,

catalase and oxidase positive. They also, observed a tightly coiled spiral organisms in the gastric biopsies of 2 patients and inferred that dogs (which also harbour spiral shaped organisms in their stomachs) and cats can transmit these bacteria to man.

Prevalance of helicobacter infection:

Epidemiological studies using serological tests have shown that a large proportion of healthy people have antibodies against helicobacter pylori (anti HP). It is uncertain whether the presence of anti HP indicates active infection or only past exposure to the microorganism. In 100 healthy volunteers detection of anti HP with a specific enzyme linked immuno sorbant assay and investigation foractive H pylori infection were done, 49 % had a high anti HP titre but only 24 % had active H. pylori infection (Meyer, 1991).

Loffeld et al.,(1987) have reported the results of a prospective study in which the incidence of chronic gastritis and H. pylori infection in 100 non ulcer patients was determined. They concluded that:

1. Compylobacter associated gastritis was present in 55 % of non ulcer dyspepsia patients.

2. A modified giemsa stain was the most sensitive way to establish the presence of H. Pylori.

3. H.Pylori. was present in 42 % of patients without gastritis histologically.

In a study to determine the prevalance of campylo bacter like organism (CLO) in nonulcer dyspepsia (NUD) and to relate this to histological abnor malities, 40 consecutive patients with upper gastrointestinal dyspeptic symptoms and no recent history of NSAIDs consumption were studied all had normal abdominal ultra sound and in all upper gastrointestinal endoscopy revealed no significant abdominal pathology. They found that CLO were cultured in 16 (40 %) of the patients (Rokkas et al., 1986).

In patients undergoing endoscope there is strong association of H.pylori with duodenal ulcer (85 %), but only a weak association with gastric ulcer (53 %) and duodenitis (50 %). There is no increased prevalance in patients with gastroesophageal reflux disease (28 %) non ulcer dyspesia (43%) and endoscopic gastritis (29 %). There is 2 fold increased prevalnce in blacks compared to whites but no increased prevalance with age or gender predilection. Un answered is the variability in H.pylori prevalance amongethnic group. It is also unclear how to determine in a particular individual if H.pylori is an innocent Bystander or isa pathogen causing significant changes and symptoms (Schubert and Schnell, 1989).

Preliminary studies have reported that the prevalnce of