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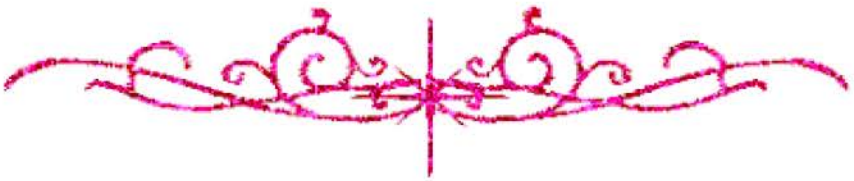
التوثيق الالكتروني والميكروفيلم



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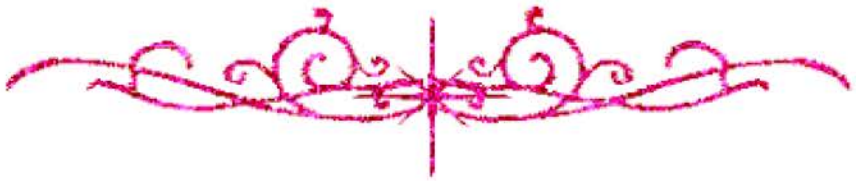

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بالرسالة صفحات

لم ترد بالأصل

HOSSAM MAGHRABY

ROLE OF HELICOBACTER PYLORI IN DYSPEPSIA WITH AND WITHOUT NSAIDs INTAKE AND THE EFFECT OF THERAPY

THESIS
Submitted in Partial Fulfillment for the M.D. Degree in Tropical Medicine

PRESENTED BY
MAGDY ABD EL- MAWGOOD GAD
(M.B.; B. Ch., M. Sc. Tropical Medicine)

SUPERVISED BY
PROF. SAMIR MOHAMED KABIL
Prof. and Head of the Department of Hepatology, Gastroenterology and Infectious Diseases
Benha Faculty of Medicine
University of Zagazig

PROF. JEAN FRANCOIS ROSSIGNOL
Clinical Professor at The University of South Florida USA. Visiting Professor at the Department of Hepatology, Gastroenterology and Infectious Diseases, Benha Faculty of Medicine, University of Zagazig

PROF. MOHAMED MAGDY ATTA
Prof. of Hepatology, Gastroenterology and Infectious Diseases
Benha Faculty of Medicine
University of Zagazig

PROF. ASHRAF KHAMIS NASSAR
Ass. Prof. of Hepatology, Gastroenterology and Infectious Diseases
Benha Faculty of Medicine
University of Zagazig.

BENHA FACULTY OF MEDICINE
UNIVERSITY OF ZAGAZIG

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وقل رب زدنی علما

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ABSTRACT

In 600 patients with dyspepsia with and without NSAIDs intake and/or CLD, the overall incidence of H. pylori was 92%. Diagnosis of H. pylori was made by ELISA finger prick test, rapid urease test, histopathology and culture methods. The highest incidence of H. pylori (97.6%) was found in peptic ulcer and the lowest incidence (85%) in NUD. CLD and NSAIDs does not constitute an independent risk factor for contracting H. pylori. Duodenal ulcer was diagnosed in 104 patients (17.3%) of which 101 (97.1%) were found to be colonized with H. pylori. High colonization density of H. pylori was found in patients with DU as well as in patients with dysplasia and intestinal metaplasia with high significant correlation between the degree of colonization and activity of gastritis. This finding enforces the role played by this organism in the aetiopathogenesis of DU and adds a further confirmatory evidence that patients with high grade of colonization are more liable for changes in the surface mucosa with its possible consequences. H. pylori positive patients were randomly allocated to one of two treatment strategies. Either dual therapy with omeprazole (or lansoprazole) and nitazoxanide (or roxithromycin) or triple therapy with lansoprazole, roxithromycin and metronidazole (or nitazoxanide). Patients treated with triple therapy of lanzop., roxithro. and nitazoxanide showed almost 92% cure rate judged by breath test (in a subset of patients) 4 to 6 weeks after therapy. The relapse rate of H. Pylori was found to be 2.7% (2/73). It is concluded, therefore, that H. pylori is widely prevalent in patients with dyspepsia in Egypt and H. pylori eradication is a stable phenomenon over time.

LIST OF ABBERVIATIONS

AIDS	: Acquired immune deficiency syndrome.
CBS	: Colloidal bismuth subcitrate.
CHD	: Coronary heart disease.
CLD	: Chronic liver disease.
COX	: Cyclo-oxygenase enzyme.
DU	: Duodenal ulcer.
G17	: Gastrin 17.
GERD	: Gastroeosphageal reflux disease.
GU	: Gastric ulcer.
HBsAg	: Hepatitis B surface antigen.
HBcAb	: Hepatitis B core antibody.
HCV	: Hepatitis C virus.
H.P	: Helicobacter pylori.
H.pylori	: Helicobacter pylori.
IBS	: Irritabe bowel syndrome.
IgA	: Immunoglobulin A.
IgG	: Immunoglobulin G.
IgM	: Immunoglobulin M.
IL	: Interleukin.
LPS	: Lipopolysaccharide.
MAO	: Maximal acid output.
MALT	: Mucosa associated lymphoid tissue.
MHC	: Major histocomptability complex.
MIC	: Minimum inhibitory concentration.
NSAIDs	: Non steroidal anti-inflammatory drugs.
NUD	: Non ulcer dyspepsia.
PGs	: Prostaglandins
PHG	: Portal hypertensive gastropathy.
PMN	: Polymorph neuclear leuckocytes.
PCR	: Polymerase chain reaction.
PKa	Partition co-efficient
PPIs	: Proton pump inhibitors
RBS	: Ranitidine Bismith citrate.
UBT	: Urea breath test.
TNF	: Tumour necrosis factor.

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INTRODUCTION

INTRODUCTION

Since the first report of unidentified curved bacilli in the gastric mucosa by ***Warren and Marshall (1983),*** there has been much interest in possible link between this organism and gastroduodenal lesions. H. pylori, which colonizes the antrum of the stomach has received much scientific attention and its discovery has changed the aetiological concept of peptic ulcer disease.

H. pylori is considered to be the major aetiologic factor in peptic ulcer disease ***(Marshall, 1994)*** and a probable initiating factor in gastric carcinoma ***(Blaser and Parsonet, 1994)*** and gastric lymphoma ***(Wother Spoon et al., 1991).***

Patients with dyspepsia are shown to have H. pylori present if biopsy specimens are taken at endoscopy and eradication of H. pylori improves patient symptoms ***(O'Marain and Gilvarry, 1993).*** Moreover, patients infected with H. pylori are more likely to have a peptic ulcer rather than a non-ulcer dyspepsia if an underlying chronic liver disease is existing ***(Kabil et al., 1995 b).***

Among the pathogenic mechanisms involved in gastroduodenal mucosal injury, H. pylori and NSAIDs show both similar effects, such as the stimulation of gastric acid secretion and impairment of gastric mucus properities, as well as opposing effects, such as prostaglandin production, which is increased by H. pylori infection and decreased by NSAIDs ***(Malfertheiner and Labenz, 1998).*** H. pylori-mediated effect on NSAID-induced change in prostaglandin production would be a potential mechanism for any interaction of H. pylori and NSAIDs ***(Soll et al.,***

1992). It has therefore been suggested that an interaction of NSAIDs with this infection could lead to increased mucosal damage and perhaps explain the higher incidence of complications related to NSAIDs *(Graham and Smith, 1988).*

The eradication of H-pylori infection was found to reduce incidence of relapse and if H-pylori confirmed to be eradicated one month after treatment, the risk of recurrence is extremely low, maintenance treatment is not required and complicated peptic ulcer are less likely to rebleed *(Beeching and Harries, 1994).* It is therefore generally agreed upon that all patients with proven ulcer disease need antibiotic treatment, as long as infection with H. Pylori has been established.

It is difficult to state with assurance which regimen of eradication of H. Pylori is clinically optimal, although it is clear that eradication rates of 80 to 90% are achievable with some regimens, including triple therapy *(Perston, 1994)* and quadruple therapy *(De Boer et al., 1998).*

AIM OF THE WORK