سامية محمد مصطفى

شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم

سامية محمد مصطفى

شبكة المعلومات الجامعية

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التوثيق الالكتروني والميكروفيلم

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سامية محمد مصطفى

جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها علي هذه الأقراص المدمجة قد أعدت دون أية تغيرات

يجب أن

تحفظ هذه الأقراص المدمجة بعيدا عن الغبار

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بعض الوثائق الأصلية تالفة

سامية محمد مصطفى

شبكة المعلومات الجامعية

بالرسالة صفحات لم ترد بالأصل

STUDY THE EFFECT OF TRETMENT OF HELICOBACTER PYLORI INFECTION ON PLASMA FIBRINOGEN LEVEL IN PATIENTS WITH ISCHEMIC HEART DISEASE

Thesis

Submitted in partial fulfillment of the Master degree

In

Internal Medicine

By

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(M.B.B.Ch.)

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FACULTY OF MEDICINE
TANTA UNIVERSITY
2002

ACKNOWLEDGMENT

THANKS TO **"ALLAH"** *FOR HE'S SO GREAT.*
AND HIS LOVE IS GRAND AND SWEET.
AND HIS HELP GIVES LIFE ITS TASTE.
AND SUPPORTS US HEARTS TO FEET.

****

To the father of my skill
To the father of my joy.
Prof. Dr. Abd El-Rasheed Ali Wafy.
Prof. of Internal Medicine ,
Who I'm proud to be his boy.

****

To **Dr. Hegazy M. Hegazy**
Assistant Prof. of Internal Medicine ,
Who planted my own career.
And who started this work too
And who was ever sincere.

****

Thanks to **Dr. Desouky E. Abo Ammo.**
Assistant professor of Clinical Pathology,
For his precious guidance and care.
For his professorial advice.
Which I was lucky to share.

****

To my Father the father of the smiles.
That were given to all free.
And the help sincerely offered.
To my family and to me.

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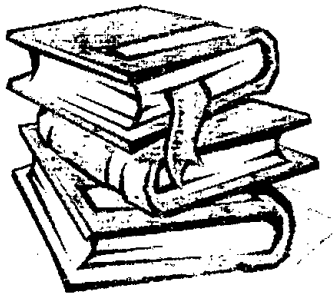
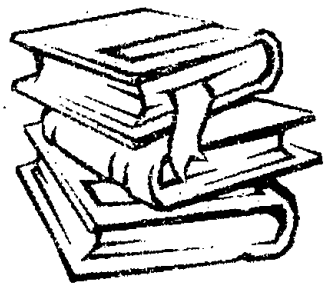


INTRODUCTION



INTRODUCTION

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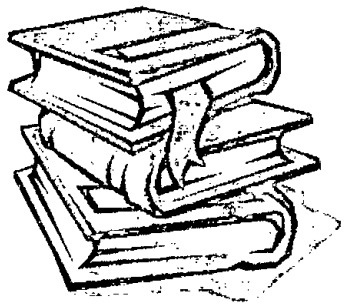
Coronary heart disease is the primary cause of mortality in western countries. The well-established risk factors cannot fully explain epidemiological variations of this disease. Form several years infections have been linked to ischemic vascular events [1].

Major factors associated with an increased risk of ischemic heart disease (IHD) are well known, yet they do not completely explain the pathogenesis of the disease. Recent data suggest that active inflammation and/or infection, possibly in the coronary arteries, may play a role in IHD[2].

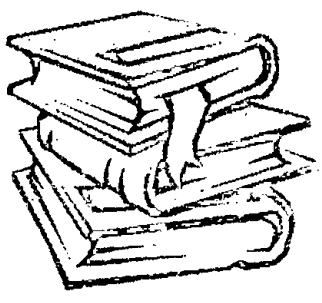
Helicobacter pylori infection, usually acquired in childhood, has also been recently associated with an increased risk of developing IHD. In other reports concerning normal subjects, some authors found that chronic infection with H. pylori are associated with higher fibrinogen plasma levels than in noninfected subjects, suggesting that fibrinogen could be a link between chronic infection and increased risk for IHD[2].

Recent studies have suggested that chronic infections with H. pylori may be associated with the risk of coronary heart disease [3].

The possible mechanisms by which H.pylori may influence cardiovascular risk are unknown. Chronic infections which are accompanied by a persistent inflammatory response, may contribute to the risk of coronary heart disease by increasing the concentrations of acute phase reactions such as fibrinogen "and sialic acid" which are predictors of coronary heart disease [3].



REVIEW OF LITERATURE



REVIEW OF LITERATURE

HELICOBACTER PYLORI

Historical background:

The genus Helicobacter is composed of at least nine species which share common properties, especially those related to life in stomach. H. pylori has the peculiarity of being a homogeneous species when it is considered phenotypically while its genomic diversity is probably one of the widest ever found. These microaerophilic bacteria resembled Campylobacters by light microscopy and in guanine plus cytosine content, and were thus named Campylobacter pyloridis [4].

Later studies on the ultrastructure and fatty acid profile of Campylobacter pylori, as well as the 16s subunit ribosomal RNA sequences of this organism, clearly revealed that it did not belong to the genus Campylobacter and thus the bacterium was renamed Helicobacter pylori[5].

The new name Helicobacter pylori (H. pylori) reflects the helical appearance of this organism in vivo as well as the most common isolation place, the pylorus of the stomach [6].

Morphology:

H. pylori is a small curved, S-shaped or slightly spiral gram-negative rod 2.5-3.5 mm in length and 0.5-1.0 mm width. It has a smooth coat and 1-6 sheathed unipolar flagella with a terminal bulb[4].

H. pylori is oxidase and catalase positive and hippurate negative. A striking feature is the extremely potent urease activity of H. pylori [7].

H. pylori is a fastidious organism, which does not grow aerobically or anaerobically, isolates left for longer than 45 minutes on a laboratory bench may die. It is incubated in an atmosphere of 5% O_2 and 5-10% CO_2 on chocolate or blood agar plates at 37°C [4].