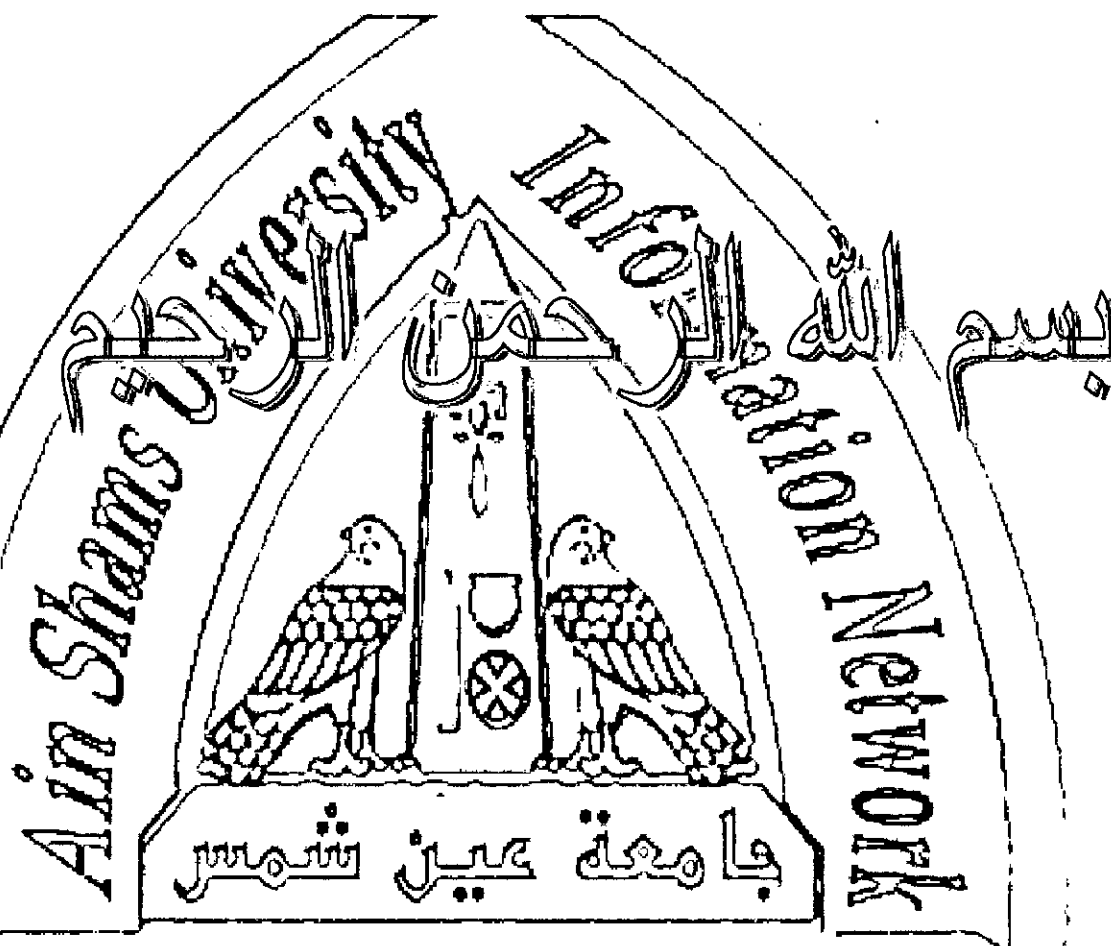




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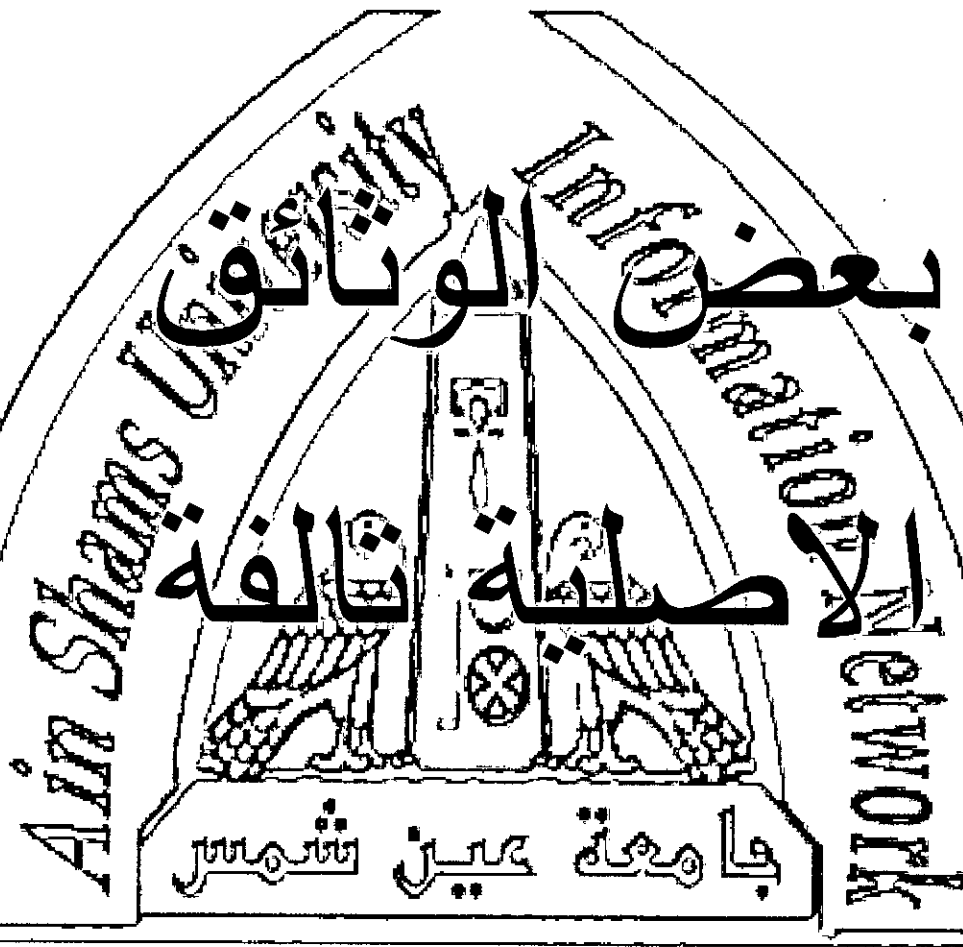
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# بالرسالة صفحات لح

## بالاحل



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**RELATION BETWEEN HELICOBACTER PYLORI  
SEROPOSITIVITY AND HYPEREMESIS GRAVIDARUM**

**THESIS**

**Submitted for partial fulfillment of Master Degree in Obstetrics and  
Gynaecology**

**Presented By**

**Abeer Saad Esawy Mesheal**

**(M. B. B. Ch.)**

**Supervisors**

**Prof. Sami Abd El-Aziem Saad**

**Prof. of Obstetrics and Gynaecology Benha Faculty of Medicine**

**Dr. Mohii El-Dien Ibrahiem Fahmy**

**Ass.Professor of Obstetrics and Gynaecology Benha Faculty of Medicine**

**Dr. Moharram Abd El-hasieb Abd El-Haii**

**Ass.Professor of Obstetrics and Gynaecology Benha Faculty of Medicine**

**Prof. Niveen Abd El-Hafiez Ahemed**

**Prof. of clinical pathology Benha Faculty of Medicine**

**Benha Faculty of Medicine**

**Benha University**

**2006**





﴿قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا  
مَا عَلَّمْتَنَا إِنَّكَ أَنْتَ الْعَلِيمُ  
الْحَكِيمُ﴾

صدق الله العظيم  
سورة البقرة (٣٢)

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## *Introduction*

Nausea and vomiting of pregnancy begins between the fourth and seventh week after the last menstrual period and cease by 12 weeks in 60% of affected women. About 9% of affected women have symptoms that persist beyond 16 weeks and may persist until 22 weeks of gestation (*Lagiou et al., 2003*).

Hyperemesis gravidarum is a severe form of nausea and vomiting in pregnancy, often associated with weight loss, ketonemia, ketonuria, dehydration, electrolyte imbalance, and possible hepatic and renal damage necessitating hospitalization (*Eliakim et al., 2000*).

The actual incidence of hyper emesis gravidarum has not yet been established, but has been reported to be between 0.3% and 2% of all pregnant women (*Davis., 2004*) .

Different biological and psychological factors may play a role in triggering hyperemesis gravidarum. Elevated serum steroid hormone and H.C.G. concentrations as well as gastric motility disturbances in early pregnancy are thought to play a role in this condition (*Broussard and Richter., 1998*).

Some studies have recently suggested that there is an association between hyperemesis gravidarum and helicobacter pylori infection (*Frigo et al., 1998; Kazerooni et al., 2002; Shirin et al., 2004*).

Helicobacter Pylori is curved or spiral shaped, Gram negative, non-capsulated non spore-forming bacillus. The organism measures (0.5-1) nm. in width and (2.5-5.0) nm. in length. Helicobacter pylorus plays a major role in abdominal symptoms and gastro-duodenal pathology (*Suerbaum and Blaser., 2001*).

# BACTERIOLOGY OF HELICOBACTER PYLORI

## *Historical introduction*

Pathologists noted the presence of spiral bacteria in the human stomach as early as **1906**. Although similar observations were repeatedly reported during the subsequent decades, they did not receive much attention because organisms could not be cultured until Warren and Marshall, in 1982, isolated a campylobacter like bacterium from patients with gastritis, (*Warren and Marshall, 1983*).

The bacterium was initially classified as campylobacter, but was placed in a new genus *Helicobacter*, on the basis of its ultra structure and morphology, fatty acid composition, growth characteristics, respiratory quinones and enzymatic properties (*Goodwin et al., 1989*).

The importance of the pathogenic role of *H. pylori* came from clinical trials showing that the elimination of *H. pylori* substantially changes the clinical course of ulcer disease. The elimination of *H. pylori* with antibiotic-containing regimens significantly reduced the high relapse rate of gastroduodenal ulcer disease (*Hentschel., 1993*).

In the early **1990**, three large prospective seroepidemiological studies each indicated that *H. pylori* is a major risk factor for the development of gastric non cardiac adenocarcinomas and *H. pylori* was classified as a definitive carcinogen by the world health organization in 1994. Subsequently *Helicobacter pylori* has been strongly associated with malignant non-Hodgkin's lymphoma of the stomach. (*Suerbaum and Blaser, 2001*).

### ***Habitat***

*Helicobacter pylori* are most frequently found deep to the mucus layer overlying the gastric epithelial cells of the antral, body and cardiac regions but tend to be numerous in the antrum. The organism has occasionally been detected in gastric juice, saliva, dental plaque, bile and faeces (*Owen, 1995*).

### ***Morphology***

*H. Pylori* are curved or spiral shaped Gram negative bacteria. The organism measures (0.5-1) nm. in width and (2.5-5.0) nm. in length and is unipolar with four to six sheathed flagella attached to one pole; each flagellum measures approximately 30 nm. in length and 2.5 nm. in thickness. (*Reigg et al., 1995*). The outer membrane is smooth and adheres closely to the cytoplasmic membrane (*Goodwin et al., 1985; Goodwin et al., 1989*).



***Figure I: Helicobacter pylori has polar flagella and a spiral shape that enable the bacterium to be motile. Magnification x 16,000. Taken from Rathbone (1992).***

## ***Growth Requirements***

### **1-Atmospheric condition:**

H. Pylori are microaerophilic organisms that require atmosphere with a reduced oxygen content about 5%, elevated CO<sub>2</sub> (5-10%), and die if exposed to air with 21% oxygen for a short period (*Skirrow., 1990*).

A suitable atmosphere can be obtained in aerobic jars by the use of commercial Campy Pak (*Goodwin et al., 1985*). It is possible to grow H.pylori on subculture in a humidified CO<sub>2</sub> incubator containing 8-10 % CO<sub>2</sub> (*Marshall et al., 1984*). But this is not recommended for optimal recoveries on primary isolation (*Goodwin et al., 1985*).

### **2-Temperature:**

Optimal growth of H.pylori occurs at 37°c with limited growth over the range 30- 42°c (*Goodwin et al., 1989*). No growth occurs at 25°c (*Nachamkin and Skirrow, 1998*).

### **3-PH:**

Ranges from 5.5-8.5(*Goodwin et al., 1989*). In vivo: H.pylori are able to survive at a PH of less than 3 due to massive secretion of urease that hydrolyzes urea with production of ammonia which forms a buffering layer around the organism (*Marshall et al., 1990*).

### **4-Transport media:**

H.pylori is a fastidious organism and should be maintained in transport media to prevent the organism from drying out and also to protect them from the effect of atmospheric oxygen (*Tompkins, 1992*).