
شبكة المعلومات الجامعية


# بسم الله الرحمن الرحيم


# شبكة المعلومات الجامعية

# التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

# جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

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# بالرسالة صفحات لم ترد بالاصل

# STUDY OF GASTRIC AND GALL BLADDER EMPTYING USING REAL TIME ULTRASONOGRAPHY IN CASES OF HELICOBACTER PYLORI GASTRITIS



## Thesis

Submitted to the Faculty of Medicine
University of Alexandria
In partial fulfillment of the
Requirements of the degree of

## Master in Internal Medicine

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2006

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# ACKNOWLEDGEMENT

" Thanks to ***Allah*** for giving me the strength and means to do this work."

I would like to express my deep gratitude to ***Prof. Dr. Noha Mohamed EL Sabbagh***, Professor of Internal Medicine, Faculty of Medicine, University of Alexandria, for her great encouragement, constant support. Without her continuous help this work would never have been accomplished.

Special thanks to ***Dr. Mohamed Samy Gad,*** Ass. Professor of Internal Medicine, Faculty of Medicine, University of Alexandria, for his help, guidance, and continuous encouragement in this study.

I am also grateful to ***Prof. Dr. Ahmed Mohamed Mohalell,*** Professor of Radiodiagnosis, Faculty of Medicine, University of Alexandria, for his personal attitude, his support and sincere help. I am forever thankful.

# LIST OF ABBREVIATION

| | |
|---|---|
| H.PYLORI | Helicobactor pylori |
| NSAIDS | Non steroidal anti inflammatory drugs |
| MALT | Mucosa associated lymphoid tissue |
| NUD | Non ulcer dyspepsia |
| GERD | Gastro-esophageal reflux disease |
| GE | Gastric emptying |
| GB | Gall bladder |
| FAA | Fasting antral area |
| MAA | Maximum antral area |
| AA30 | Antral area after 30 minutes |
| GE 30% | Gastric emptying% at 30 minutes |
| FV | Fasting volume |
| RV | Residual volume |
| EF% | Percent of ejection fraction |
| EPIG.PAIN | Epigastric pain |
| AO | Aorta |
| SMV | Superior mesenteric vein |

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# INTRODUCTION

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## Helicobacter pylori

### Historical background

The demonstration of spiral organisms were first found incidentally in the stomach of dogs in 1893[1] and later in cats and rats in 1896.[2]

The interest in the role of gastric urease and its association with ulcer disease was emphasized by Fitzgerald and Murphy.[3]

Interest in gastric bacteria waned until 1975 when Steer and Collin reported the presence of bacteria on gastric mucosa, deep in the mucus layer of the normal individuals.[4]

In 1983 Warren reported the finding of an unidentified curved bacillus in close contact with the gastric epithelium in biopsy samples showing active chronic gastritis.[5]

Biochemical analysis and enzyme capabilities have shown that campylobacter pylori do not belong to the genus campylobacter, a new genus name was required and thus the bacterium was renamed Helicobacter pylori.[6]

## Microbiology of H. pylori

H. pylori is a curved gram-negative organism, it is non-capsulated, non-spore forming bacillus with a length of about 3 um and a width of 0.5 um.[7]

It is a motile, curved bacterium which fails to multiply under aerobic conditions. It colonizes in the gastric epithelium beneath the mucus layer and in areas of gastric metaplasia. It is a urease producing bacteria and it is found in greatest numbers under the mucus layer in gastric pits in close opposition to gastric epithelial cells.[8]

H. pylori is predominately an extra cellular organism either living in or attached to the epithelial cells but rarely intracellular penetration can occur.[7]

H. pylori is catalase positive and strongly urease positive. H. pylori has the highest urease activity of all bacteria.[9]

## Characteristics

H. pylori strains are homogeneous with regard to their cultural requirements: media, atmosphere and temperature [10]. It is incubated in an atmosphere of 5% $O_2$ and 5-10% $CO_2$ on chocolate or blood agar plates at 37°C.[11]

The identification of H. pylori species is based on the presence of several enzymes which are always present: urease, oxidase, catalase, alkaline phosphatase, γ-glutamyl transferase, DNAase and various esterases.[12]

H. pylori is primarily found in the antrum of the stomach but is also seen in the duodenum within areas of gastric metaplasia and in the fundus.[13]

The organism avoid contact with gastric acid, presumably because of their susceptibility to pH below 3.5.[14] The pH rises within the gastric mucus layer and approaches neutrality at the brush border, a function of bicarbonate secretion. This may explain the ability of H. pylori to survive in hypersecretory conditions.[15]

## Epidemiology

H. pylori is one of the most common chronic infections known and has a world wide distribution in normal population, with 7 out of 10 people infected globally.[16]

Low socioeconomic status predicts infection and the prevalence of infection differs between developed and developing countries. [17]

H. pylori has been found in all age groups from infants to the elderly but the prevalence increases strikingly with age.[18]

An Egyptian serological study on a group of asymptomatic persons, all aged below 30 years revealed an overall prevalence of H. pylori of 87.6% with a prevalence of 53% in those below 10 years. [19]

Another Egyptian study showed that H. pylori infection is more prevalent in low socioeconomic class [20]

In developed countries, there is high prevalence (50%) of H. Pylori infection in the general population of Austria, as in other epidemiological studies in Spain and other European countries, the distribution according to age shows a clear tendency to increase from childhood to adolescence and adult life.[21]

## Risk factors

Studies have shown that socioeconomic factors as poor housing and sanitation are closely associated with H. pylori infection.[22]

It was found that the size of the family, close contact and crowding are risk factors for H. pylori infection. Also; it was found that molecular studies of H. pylori in members of the same family show genetically identical strains.[23]

Other studies have shown that people with blood group A or O are easier to be infected with the organism. It has been reported that such individuals posses specific receptors for H. pylori adhesion on their gastric epithelial cells which allows H. pylori to adhere and colonize in the stomach more rapidly, but these findings were not confirmed by other studies.[24]

## Modes of transmission

The major mode of transmission is still unknown. Possible routes of infection include either oro-oral or feco-oral, iatrogenic by the use of unsterile pH probes and endoscopy. The only significant reservoirs of infection are humans themselves.[25]

## Sexual transmission

Sexual transmission was potentially implicated by a study of homosexuals and heterosexuals. [26]

# Mechanism of H. pylori induced damage

The exact mechanism by which H. pylori contributes to the pathogenesis of gastro duodenal diseases is unclear but may include both direct and indirect components. [27]

When H. pylori enters the gastrointestinal tract it colonizes with gastric acid barriers. H. pylori overcomes this through the activity of urease enzyme, which is present on the surface of the organism and in its cytoplasm. Via this urease, it produces ammonia around the bacterium, thus protecting it by local neutralization of gastric acid. On the other hand H. pylori is microaerophilic organism abling it to survive at low oxygen level.[28]

H. pylori has the ability to change to coccoid forms, which have been served in biopsy specimens. These may represent transitory dormant states which are able to resist unsuitable environment.[29]

## Urease

Urease accounts for over 5 percent of the organism's total protein weight. Urea, when hydrolyzed by bacterial urease, can form compounds such as ammonium chloride and monochloramine that can directly damage epithelial cells. In addition, the urease enzyme itself is antigenic, activates the host immune system and indirectly produces injury by stimulating inflammatory cells.[30]

Ammonia produced via the organism's urease activity may cause histological damage and vaculation of epithelial cells. Vaculation may also occur independently via the activity of vaculating cytotoxins (vac A) which is expressed in 50% to 60% of H. pylori strains. Production of the toxin is associated with the presence of an antigenic protein encoded by cytotoxin associated gene A (cage A). The majority of H. pylori strains can be divided into two distinct phenotypic groups based on the presence (type 1) or absence (type 2) of expression of vacA (vacA*) and cagA (cagA*). Furthermore while all strains of H. pylori are potentially pathogenic type 1 strains are associated with greater inflammatory activity and more severe gastroduodenal disease. [29]

# Inflammatory response

Although H. pylori is a noninvasive organism, it stimulates an inflammatory and immune response. [31] A variety of factors may contribute to these changes.

Cellular disruption especially adjacent to epithelial tight junctions, undoubtedly enhances antigen presentation to the lamina propria and facilitates immune stimulation. The net result is increased production of inflammatory cytokines such as IL-1, IL-6, IL-8 and tumor necrosis factor alpha (TNFα).

Bacteria that express cag A and vac A are more potent inducers of IL-8: the gene primarily responsible for IL-8 induction is picB. CagA/ VacA positive strains are also more found in patients with clinical manifestations of H. pylori infection suggesting that IL-8 play an important pathophysiologic role in duodenal disease.[33]

H. pylori itself is in part able to survive this inflammatory response by producing the enzyme catalase. This enzyme neutralizes the damaging reactive oxygen metabolites liberated by neutrophils. With time, the host appears to down regulate the inflammatory response, making it easier for the organism to persist and proliferate. [34]

A study reported that H. pylori elicits chlorinated toxic oxidant production from neutrophils and that these substances play a critical part in H. pylori associated gastric mucosal damage. Also, reactive oxygen metabolites and degranulation products released from the inflammatory cells stimulated with H. pylori play a part of the mucosal tissue damage associated with H. pylori infection. [35]

## Antibody response

Most infected individuals systemically produce antibodies to a variety of H. pylori antigens. The antibody response changes as infection progresses from an acute to a chronic stage. [36]

Detection of IgM antibodies is an insensitive indicator of acute infection and generally not clinically useful even in children. [37]

IgA and IgG antibodies are produced in response to infection remain present as long as infection is active and quantitatively decrease after infection is cured. [38]

Antibodies to cagA protein are detectable in gastric tissue and serum and permit the identification of infection with presumably more virulent organisms.[37]

## Virulence factors

### Urease

H. pylori produce large amounts of urease. H. pylori possesses an intrinsic ability to maintain nearly neutral internal pH in experimental setting down to pH 3. [37]