

**INCIDENCE OF CLINICAL
MANIFESTATIONS OF HELICOBACTER
PYLORI INFECTION IN EGYPTIAN
CHILDREN**

Thesis

Submitted for Partial Fulfillment of Master Degree in Pediatrics

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Abstract

Background: *Helicobacter pylori* colonizes over 50% of world population, yet less than 20% of those infected individuals will develop a gastroduodenal disease. *H.pylori* colonization is the most common cause of chronic gastritis & is etiologically associated with duodenal ulcer, gastric ulcer, gastric adenocarcinoma, and mucosa-associated lymphoid tissue (MALT) lymphoma. Gastroduodenal diseases associated with *H.pylori* are manifested principally in adults. However, it is usually during childhood that the infection is acquired.

Key words:

Clinical manifestation

Helicobacter Pylori

The endoscopic biopsy & UBT

List of Abbreviations

UBT	Urea Breath Test
MALT	Mucosa-Associated Lymphoid Tissue
WK	Week
H. pylori	Helico Pacter Pylori
TNFα	Tumor Necrosis Factor Alpha
IL-1b	Interluken-1b
IL-10	Interluken-10
COX-2	Cyclooxygenase-2
RAP	Recurrent Abdominal Pain
IgG	Immunoglobulin G
ELISA	Enzyme-Liked Immunosorbent Assay
GERD	Gastro Eosophogial Reflux Disease
Yr.	Year
14C	Carbon 14
13C	Carbon 13
US	United States
DNA	DeoxyriboNucleic Acid
Rt	Right

Lt	Left
IL-8	Interluken-8
IL-6	Interluken-6
ICAM-1	IntraCellular Adhesion Molecule-1
VEGF	Vascular Endothelial Growth Factor
NF-kB	Necrosis Factor-β
vs	Versus

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INTRODUCTION

Helicobacter pylori is a spiral-shaped, Gram-negative rod and catalase-positive organism (O'Rourke et al, 2004).

Helicobacter pylori is a highly successful bacterial pathogen that persistently colonizes the mucosa of the human stomach (Covacci et al, 1999, Cover & Blaser , 1999 & Ernst & Gold, 2000). The bacterium has been recognized as the causative agent of chronic gastric inflammation, which can progress to a variety of other gastroduodenal diseases, such as peptic ulcers, mucosa-associated lymphoid tissue lymphoma, or even gastric cancers (Forman et al, 1991, Rappuoli, 2001, Peek & Blaser, 2002, & Gatti et al, 2005).

Aim of the work

The aim of this study is to determine the most common clinical presentation of *Helicobacter pylori* infection in Egyptian children.

Methods:

This is a retrospective study aiming at identifying the most common clinical manifestations of *H.pylori* infection in children. The study included 150 patients – ages 5-15 yrs, who presented to the GE Unit, Cairo University's Pediatric Hospital with abdominal complaints. Diagnosis of *H.pylori* infection was done by the C13 urea breath test, and confirmed by visualizing the bacterium in biopsy specimens obtained by upper gastrointestinal endoscope. Eighty six point seven per cent of the patients who had a positive urea breath test complained of abdominal pain, while 13.3% had no abdominal pain.

INTRODUCTION

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There is increasing evidence that distinct variants of *H. pylori* exist and that these may be associated with the pathogenicity of the bacterium (Van Doorn et al, 1998 & Gatti et al, 2006). Several virulence-associated genes have been identified (Covacci et al, 1993 & Atherton et al, 1995).

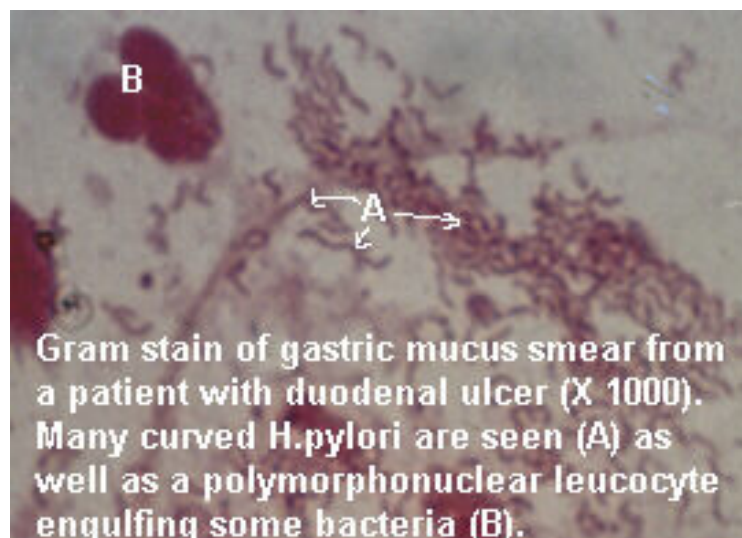


Fig. (1): Gram stain of gastric mucus smear from a patient with duodenal ulcer. (Holston 2006)

There are 2 phenotypically distinct *H.pylori* groups: type 1 bacteria, which express the cytotoxin-associated gene antigen (*cagA*) and the vacuolating cytotoxin associated gene antigen (*vacA*), and type 2 bacteria, where *cagA* is absent and vacuolating cytotoxin activity is not manifested although *vacA* gene is present. The type 1 bacteria are more strongly pathogenic than the type 2 and induce a more intense inflammatory response **(Xiang et al, 1995)**. Virtually, the presence of *vacA* gene has been reported in all *H. Pylori* strains; various strains show marked differences in production of vacuolating cytotoxins **(Mahboob et al, 2005)**.

Helicobacter pylori is the primary cause of gastric and peptic ulcer disease **(Matthews et al, 2005)**. This organism has been shown to infect more than 50% of the world's population with the incidence up to 80% in developing countries **(Gonzalez et al, 2004)**.

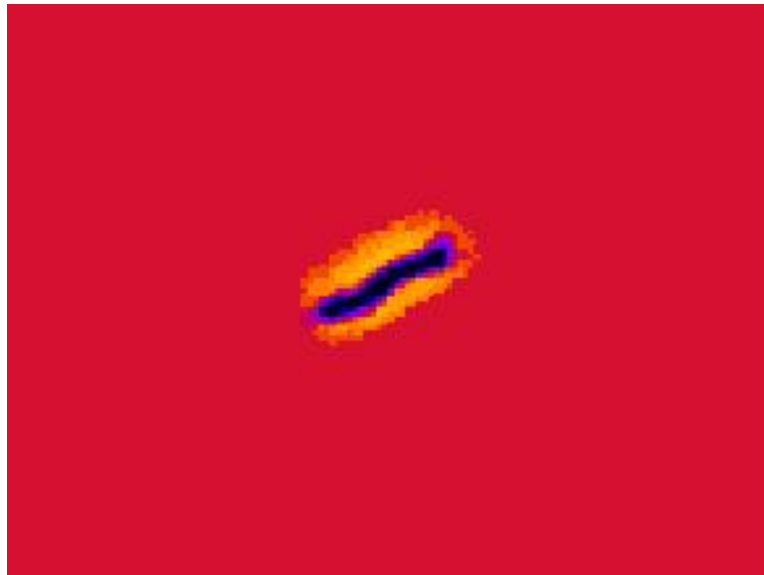


Fig. (2): *Helicobacter pylori* can be detected by high power microscopy as a spiral-shaped rod. (Holston 2006)

Helicobacter pylori can be detected at endoscopy by histology, culture, or urease test. All these biopsy based methods for detection of *Helicobacter pylori* are liable to sampling error because infection is patchy. In addition after partially effective eradication treatment, low levels of infection can easily be missed by endoscopic biopsy (**Robert & Marjorie, 2001**).

Carbon 13 urea breath test is simple, robust, noninvasive, accurate and inexpensive (**Ather et al, 1994**). With a sensitivity of 98% and specificity of 97%, the urea breath test is rapidly becoming the test of choice in detection of *H. Pylori* infection (**Matthews et al, 2005**).

The role of *Helicobacter pylori* in the colonization of the stomach in adults and children with chronic gastritis, peptic ulcer, and possibly gastric carcinomas is well-documented (**JAMA, 1994**), and eradication of the bacteria is very effective in preventing peptic-ulcer relapses in both adults and children (**Hentschel et al, 1993**).

Recurrent abdominal pain (RAP), according to Apley's criteria (i.e., at least 3 discrete episodes of abdominal pain of sufficient severity to interrupt normal daily activities or performance, occurring over a period of 3 months)(**Apley, 1958**) and non ulcer dyspepsia (NUD), which refers to pain or discomfort centered in the upper abdomen (**Rasquin-Weber, 1999**) are difficult conditions to define in children, because many have imprecise symptoms.

Moreover, the role and clinical manifestation of *H pylori* remain unclear in such children. Vomiting and acute abdominal pain related to ulcer disease may be associated with *H pylori* infection, whereas the role of this bacterium in children with RAP and NUD is the subject of conflicting reports (**Mahony , 1988**).

Localized epigastric pain and nocturnal awaken (**Gormally , 1995 & Goggin , 1998**) have been reported occasionally in children with this infection. However, a high incidence of H.Pylori infection in the course of RAP does not prove a causal relationship between this infection and abdominal pain (**Gormally , 1994**).

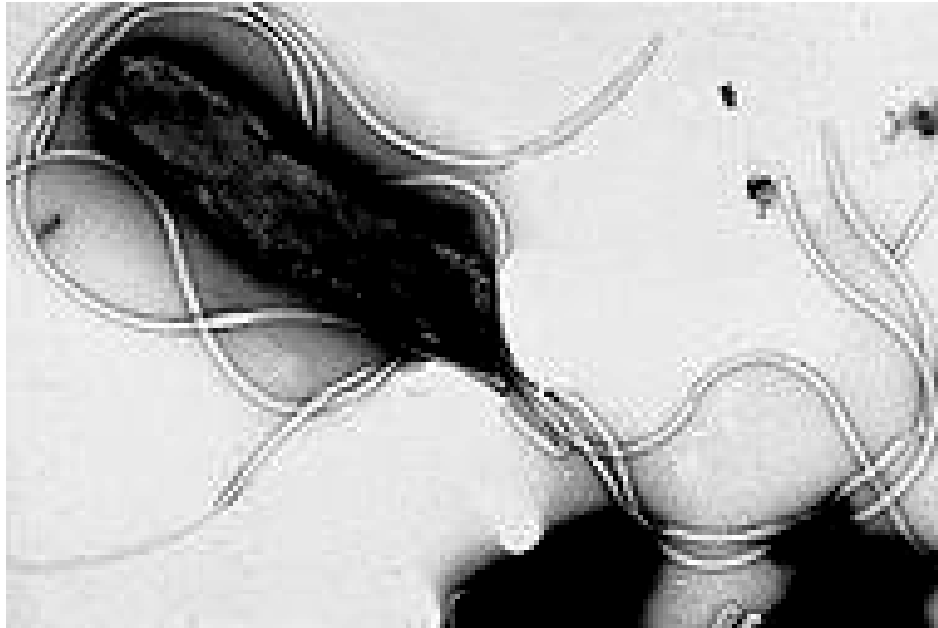


Fig. (3): Helicobacter pylori by electromicroscope. (Holston 2006)