INCIDENCE OF CLINICAL MANIFESTATIONS OF HELICOBACTER PYLORI INFECTION IN EGYPTIAN CHILDREN

Thesis
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By

Ihab Samy Mohammed
(M.B.B.CH)

Supervised by

Prof. Dr. Mona Ahmed Abu-Zekry
Professor of Pediatrics
Faculty of Medicine, Cairo University

Prof. Dr. Mohammed El-Sayed Hashem
Professor of Pediatrics
Faculty of Medicine, Cairo University

Dr. Aliaa Adel Ali
Lecturer of Pediatrics
Faculty of Medicine, Cairo University

Cairo University
Faculty of Medicine
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Contents

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 endscopic 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

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).**

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).**

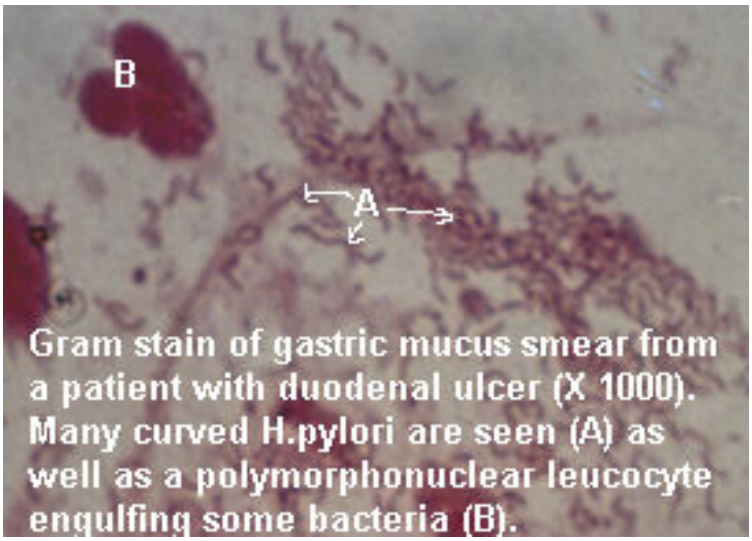

Fig. (1): Gram stain of gastric mucus smear from a patient with duodenal ulcer. (Holston 2006)

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).**

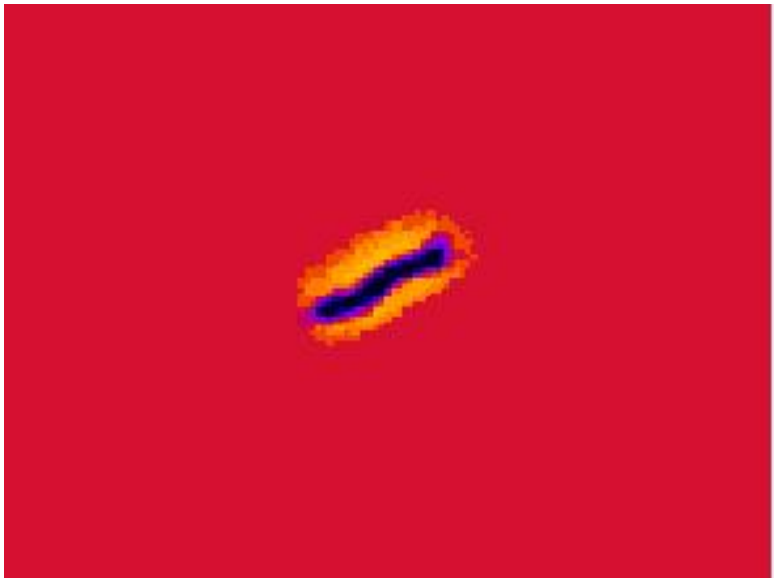

Fig. (2): Helicobacter pylori can be detected by high power microscopy as a spiral-shaped rod. (Holston 2006)

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).**

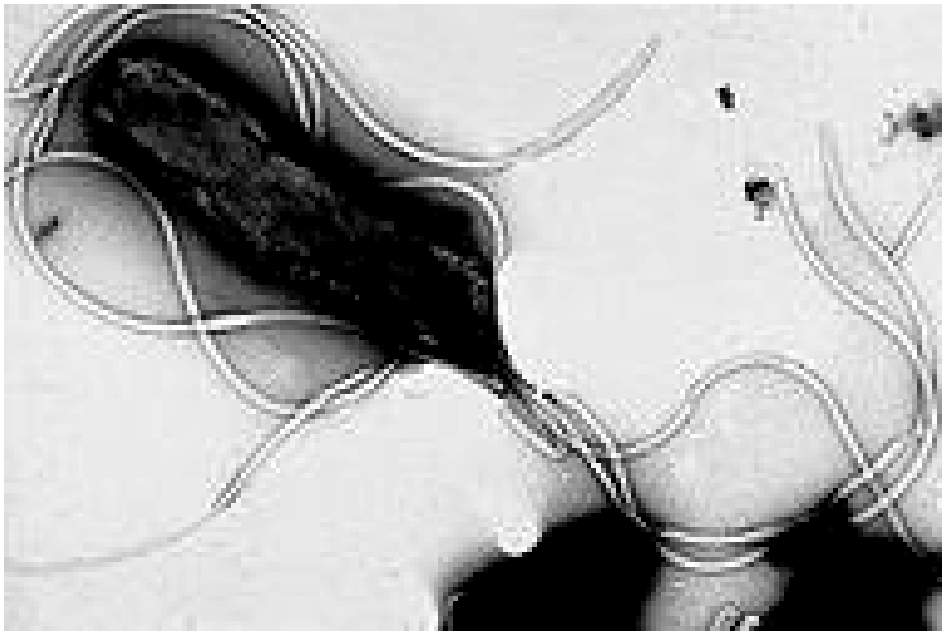

Fig. (3): Helicobacter pylori by electromicroscope. (Holston 2006)