

Prevalence of Helicobacter Pylori Infection among the Whole Spectrum of Age and the Performance of the Different Diagnostic Tests

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Abstract

UCHG) with upper gastrointestinal symptoms. The age range is 21-90 years with a median age of 52 years, 62

Index terms— helicobacter pylori, sensitivity, specificity, rapid urease test, gastric cancer, ELISA test, IgG to H. pylori.

Three hundred and thirty eight patients were included in the study. They presented to the endoscopy suite at University College Hospital Galway, Ireland (UCHG) with upper gastrointestinal symptoms. The age range is 21-90 years with a median age of 52 years, 62% females, and 24% diabetics. They were divided into 3 groups 18-30 years, 31-60 years, and 61-90 years. The prevalence of H. pylori among the different spectrum of age is calculated using different methods of diagnosing H. pylori.

Reliabilities of the diagnostic tests: The sensitivity, specificity, positive and negative predictive values for (i) Rapid urease test (RUT), (ii) ELISA, (iii) Histology and (iv) Culture. The rapid urease test was found to have a high sensitivity and specificity (89.5% and 96.8%), respectively. Although estimation of serum IgG H. pylori antibody by ELISA is relatively non-invasive procedure, unfortunately, it lacks sufficient sensitivity (63%) to be used as a sole diagnostic test for H. pylori infection. Histology on the other hand is widely available in most hospitals and has a relatively high sensitivity (77.4%) and specificity (75%). Culture of H. pylori was found to be highly specific (100%) and sufficiently sensitive (86.2%).

1 Prevalence of H. pylori infection:

The prevalence of H. pylori is assessed in the different age groups. There was a substantial increase in the prevalence of H. pylori infection with increasing age up to the age 61 years. In this study the highest prevalence of infection was found in the age group 31-60 years. The overall prevalence of H. pylori infection in patients with upper gastrointestinal symptoms as assessed by histology (73%), culture (53%), serum IgG ELISA (56%), and rapid urease test (65%).

2 Conclusion:

The prevalent of Helicobacter pylori infection are worldwide and the infection rate is intimately related to age, ethnicity, and socio-economic factors. The sensitivity and specificity of the different methods used to detect H. pylori infection vary considerably and depends on the inherent characteristics of the test used. A test with high sensitivity and specificity is needed to capture the majority of patients with infection so they can be treated early and cured of the bacteria this will prevent the development of gastric and duodenal ulcer and the late consequences of gastric malignancy.

3 I. Introduction

tudies from Western Europe, New Zealand, Australia, and United States have shown that the prevalence of H. pylori infection in symptomatic patients undergoing endoscopy is very high. The rate of H. pylori infection in

patients with upper gastro-intestinal symptoms ranges from 40-60%. In benign gastric ulcer the organism is found in about 70% (1). No the other hand, the rate of infection in duodenal ulcer patients is 85-95% (2,3). The rate of infection of *H. pylori* related to several factors, for example the rate of infection increases with age (1,(4)(5)(6)(7)(8). The prevalence in children was found to range between 20-68% (9)(10)(11)(12)(13)(14)(15). The rate of infection also correlate to underlying disease process; in duodenal ulcer and duodenitis the rate of *H. pylori* infection may be as high as 95%, and in gastritis the rate of infection ranged from 62-97% (16)(17)(18)(19)(20)(21)(22)(23)(24)(25)(26)(27)(28)(29)(30). The sensitivity and specificity of the various tests used to diagnose *H. pylori* infection have wide variation ranging from (63% to 89.5% for sensitivity) and (41.7% to 100% for specificity). The prevalence of *H. pylori* infection in different age groups was carried out in this study and was found to be highly variable in different age groups. The highest prevalence was found in the age group of 31-60 years.

4 II. Subjects and Methods

Three hundred and thirty eight patients were included in the study. They presented to the endoscopy suite at University College Hospital Galway, Ireland (UCHG) with upper gastrointestinal symptoms. The age range is 21-90 years with a median age of 52 years, 62% females, and 24% diabetics. They were divided into 3 groups 18-30 years, 31-60 years, and 61-90 years. The prevalence of *H. pylori* among the different spectrum of age is calculated using different methods of diagnosing *H. pylori*, table 3.2.

Formal written consent was obtained and the procedure was explained to each patient included in the study.

Blood was collected in a plain tube before endoscopy for estimation of serum IgG antibodies to *H. pylori* using ELISA test (Biometra, Germany). Specimens were taken from antrum and duodenum from each patient enrolled in the study, for histological examination and culture. They were forwarded to the bacteriology laboratory in a separate containers containing a transport medium (Nutrient Broth Code: CMI oxide) for direct smear and culture on Colombia blood agar base (code: CM331) containing cefoperazone selective supplement (code: SR125) mixed together into sterile Petri dishes prepared according to the specifications of the supplier (Oxide, Ireland).

The specimens for the histological examination were labeled with reference numbers, and were formalin fixed and prepared according to the standard methods. Sections 5 μ m thin and stained with haematoxylin and eosin (H&E) or modified Giemsa stain and read by an experienced pathologist (RS) without previous knowledge of clinical or microbiological information.

A biopsy specimen was examined by commercially available rapid urease test kindly provided by (Jatrox HP-Test, Rohm Pharma Waterston, Germany).

The performance of the different tests used to diagnose *H. pylori* was outlined in table 3.1.

5 III. Results

Reliabilities of the diagnostic tests: The sensitivity, specificity, positive and negative predictive values for (i) Rapid urease test, (ii) ELISA, (iii) Histology and (iv) Culture are shown in table 3.1.

The rapid urease test (RUT) was found to have a high sensitivity and specificity (89.5% and 96.8%) respectively. However, it needs an endoscopic procedure to obtain an antral biopsy for the assay which is not available in all hospitals. Endoscopy is an invasive and expensive procedure.

Although estimation of serum IgG *H. pylori* antibody by ELISA is relatively non-invasive procedure, unfortunately, it lacks sufficient sensitivity (63%) to be used as a sole diagnostic test for *H. pylori* infection, table -3.1.

Histology on the other hand is widely available in most hospitals and has a relatively high sensitivity (77.4%) and specificity (75%), table 3.1. It is relatively quick, cheap and easy to perform but is requires endoscopic examination which is an invasive technique and needs a well trained histological expertise and therefore, it cannot be utilized as a screening test for *H. pylori* diagnosis.

Culture of *H. pylori* was found to be highly specific (100%) and sufficiently sensitive (86.2%), table -3.1. However, it takes a few days for the results to come through and it cannot be used for quick diagnosis of *H. pylori* infection. Culture of *H. pylori* is, however, needed to determine the sensitivity of the bacterium to the antimicrobial agents, especially to metronidazole. Obtaining the biopsy for culture is an invasive procedure.

Prevalence of *H. pylori* infection: The prevalence of *H. pylori* is assessed in the different age groups, table -3.2. There was a substantial increase in the prevalence of *H. pylori* infection with increasing age up to the age 61 years. This result agrees with the results of previous workers. However, in the age group 18-30 years the prevalence of *H. pylori* infection in patients with dyspepsia ranges from 45-53% depending on the mode used for diagnosis.

In this study the highest prevalence of infection was found in the age group 31-60 years, table-3.2. Usually the colonization of the bacteria is most prevalent in the elderly. The reason for this finding is not entirely clear. However, the frequent use of drugs like NSAIDS, and corticosteroids could contribute to the relatively low prevalence in this age group.

The overall prevalence of *H. pylori* infection in patients with upper gastrointestinal symptoms as assessed by histology (73%), culture (53%), serum IgG ELISA (56%), and rapid urease test (65%) table-3.2 agree with the previous studies from the developed countries (31)(32)(33).

6 IV. Discussion

Infection with *H. pylori* is rampant and worldwide. The rate of infection is influenced by age, race, geographical and socio-economic factors, as well as dietary practices (34)(35)(36)(37)(38)(39)(40)(41)(42)(43) (??44). The rate of infection is found to be higher in China and India than in North America. On the other hand, the infection rate is similar in Mexico and the United States (37, ??5). It has been revealed that the rate of infection of *H. pylori* in the United States is influenced by many factors like socioeconomic, ethnicity, age, and gender. These findings suggest that the rate of *H. pylori* infection is modified by geographical and host factors. The type and severity of gastritis associated with *H. pylori* colonization are also influenced the rate of infection.

The epidemiology of *H. pylori* infection has been extensively studied and was found to be closely correlated with superficial type-B gastritis. Infection with *H. pylori* is associated with active and chronic inflammation of gastric mucosa (32, ??6). The density of the bacteria in the tissue is also correlated to the severity of inflammation and the local and systemic immune response mounted against the bacteria (32, ??46) (??47) (??48).

H. pylori infection can be detected with various methods e. g histological examination and culture of the gastric biopsy specimens which takes several days, serology, rapid urease test etc. Serum IgG/IgA ELISA and Rapid Urease test were compared to the gold standard tests (histology and culture) and evaluated in this study. The organism was detected in 76 of 107 dyspeptic patients attending GI unit at UCHG. We found a relatively good correlation between serology and histological findings in the antral biopsies despite the (15, ??49) (??50) (??51) (??52) (??53) (??54) (??55) (??56)), but disagreed with the results of Nichols and others (23, ??7). The advantages of quick test e.g. RUT is that treatment can be given to the patients within one hour of their endoscopic procedures. In the Amsterdam study (15) the results of culture and histology were analogous to ours.

We found that, histological examination, culture, ELISA and RUT revealed an increased prevalence of *H. pylori* infection and the rate of infection are rising with increasing age up to the age of 61 years. This is in parallel with the findings that the prevalence of infection is related to the prevalence of gastritis (6,(31)(32)(33). However, the prevalence of *H. pylori* in the elderly age group is lower than the reported prevalence from Western countries, table-3.2. We noted that the prevalence of *H. pylori* infection in the age group 61-90 years was below that of 30-60 years. This agreed with the findings of Newell et al (57).

Previously, it has been suggested that the age discrepancy is due to progressive atrophic gastritis with hypochlorhydria in the body and fundus of the stomach of the elderly patients. This environment is hostile to the existence of the organism. Another possible explanation to the low infection rate in the elderly could be related to the increased use of the antibiotics in this age group which may clear the organism. The histological sections from the mucosa of the 25 patients from the group 61-90 years old were reviews, none were taken NSAIDs concurrently. We found 14(56%) of these patients had either normal or mild inflammation and of them 6(43%) were *H. pylori* positive. Moderate inflammation was present in 9(36%) all were *H. pylori* positive. Only 2(8%) had severe inflammation and both were *H. pylori* positive. Atrophy of specialized cells in the fundus and body of the stomach cannot be inferred from examination of antral biopsies. Thus in this study we have no evidence to support the notion that atrophic gastritis is associated with a decreased prevalence of *H. pylori* in the elderly. We concluded that the majority if the elderly (>61 years) had a prevalence of *H. pylori* ranging from (45-64%), table-3.2, depending on the mode used for detection of *H. pylori* infection. More than 50% of them had only mild inflammation; in these group *H. pylori* was isolated in 43%. However, moderate to severe degree of inflammation was present in 44% of them and *H. pylori* were always associated with gastritis.

7 V. Conclusion

The prevalent of *Helicobacter pylori* infection are worldwide and the infection rate is intimately related to age, ethnicity, and socio-economic factors. The sensitivity and specificity of the different methods used to detect *H. pylori* infection vary considerably and depends on the inherent characteristics of the test used. A test with high sensitivity and specificity is needed to capture the majority of patients with infection so they can be treated early and cured of the bacteria this will prevent the development of gastric and duodenal ulcer and the late consequences of gastric malignancy.

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²Prevalence of *Helicobacter Pylori* Infection among the Whole Spectrum of Age and the Performance of the Different Diagnostic Tests



Figure 1:

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Figure 2:

44. The Gastrointestinal Physiology Working Group. Helicobacter pylori and Gastritis in Peruvian Patients: Relationship to Socioeconomic Level, Age, and Sex. The Am J Gastroenterol. 1990; 85(7): 819-23	51. Ching CK, Buxton C, Holgate C, et al. brushing urea broth test: a highly sensitive specific test for Helicobacter pylori infection. Gastrointestinal Endoscopy. 1991: 550-551
45. Bertram TA, Murray PD, Morgan DR, et al. Gastritis associated with infection by Helicobacter pylori in humans: Geographical differences. Scand J Gastroenterol. 1991; 26 (suppl. 181): 1-8	52. McNulty CAM, Dent JC, Uff JS, et al. Campylobacter pylori by the biopsy urease assessment in 1445 patients. Gut. 1989; 30: 1062
46. Karttunen T, Niemela S, Lehtola J. Helicobacter pylori in dyspeptic patients: quantitative association with severity of gastritis, intragastric pH, and serum gastrin concentrations. Scand J Gastroenterol. 1991; (suppl. 186): 124-34	53. Thillainayagam AV, Arvind AS, Cook DG. Diagnostic efficiency of an ultrarapid endoscopy room test for Helicobacter pylori. Gut. 1991; 42: 467-69
47. Mollenkopf C, Steinger H	
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Histology	77.4%
Culture	86.2%
Serum IgG	63%
Local IgA	79.2%
Urease test	89.5%

Figure 3:

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	18-30 years	31-60 years	61-90 years	Overall
Histology	52.9%	85.3%	56%	72.8%
Culture	47.1%	57.4%	45.5%	53%
ELISA	45.8%	61.2%	52.6%	55.8%
Rapid Urease test	47.06%	69.8%	64%	64.8%

Figure 4: Table 3 .

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- [Kosunen et al. ()] 'Agedependent increase of Campylobacter pylori antibodies in blood donors'. T U Kosunen , J Hook , H I Rautelin . *Scand J Gastroenterol* 1989. 24 p. .
- [Drumm et al. ()] 'Association of Campylobacter pylori on the Gastric Mucosa with Antral Gastritis in Children'. B Drumm , P Sherman , E Cutz . *N Eng J Med* 1987. 316 p. .
- [Hill et al. ()] 'Campylobacter pylori and gastritis in children'. R Hill , J Pearman , P Worthy . *Lancet* 1986. i p. 387.
- [Price et al. ()] 'Campylobacter pylori in peptic ulcer disease: microbiology, pathology, and scanning electron microscopy'. A B Price , J Levi , J M Dolby . *Gut* 1985. 26 p. .
- [Wyatt et al. ()] 'Campylobacter pyloridis and acid induced gastric metaplasia in the pathogenesis of duodenitis'. J I Wyatt , B J Rahthbone , M F Dixon , R V Heatley . *J Clin Pathol* 1987. 40 p. .
- [Cadranet et al. ()] 'Campylobacter pyloridis in children'. S Cadranet , H Goossens , M Boek De . *Lancet* 1986. p. .
- [Eastham and Elliott ()] 'Campylobacter pyloridis in children'. E J Eastham , Tsm Elliott . *Arch Dis Child* 1987. 62 p. 652.
- [Rauws et al. ()] 'Campylobacter pyloridis-associated chronic active antral gastritis'. Eaj Rauws , W Langenberg , H J Houthoff . *Gastroenterology* 1988. 94 p. .
- [Burnett et al. ()] 'Campylobacter-like organisms in the stomach of patients and healthy individuals'. R A Burnett , Jah Forrest , Rwa Girdwood . *Lancet* 1984. p. 1349.
- [Langenberg et al. ()] 'Campylobacter-like organisms in the stomach of patients and healthy individuals'. M L Langenberg , Gnj Tytgat , Mei Schipper . *Lancet* 1984. p. .
- [Jones et al. ()] 'Campylobacterlike organisms on the gastric mucosa: culture, histological, and serological studies'. D M Jones , A M Lessells , J Eldridge . *J Clin Pathol* 1984. 37 p. .
- [Mendall et al. ()] 'Childhood living conditions and Helicobacter pylori seropositivity in adult life'. M A Mendall , P M Goggin , N Molineaux . *Lancet* 1992. (8798) p. 339.
- [Siurala et al. ()] 'Chronic gastritis: dynamic and clinical aspects'. M Siurala , P Sipponen , M Kekki . *Scand J Gastroenterol* 1985. 20 (109) p. . (suppl)
- [Booth et al. ()] 'Clinical importance of Campylobacter pyloridis and associated serum IgG and IgA antibody responses in patients undergoing upper gastrointestinal endoscopy'. L Booth , G Holdstock , Macbride . *J Clin Pathol* 1986. 39 p. .
- [Wulffen et al. ()] 'Detection of Campylobacter pylori in patients with antrum gastritis and peptic ulcer by culture, complement fixation test, and immunoblot'. Von Wulffen , H Heesemann , J Butzow , GH . *J Clin Microbiol* 1986. 24 p. .
- [Graham et al. ()] 'Effect of Age on the Frequency of Active Campylobacter pylori Infection Diagnosed by the (C13) Urea Breath Test in Normal Subjects and Patients with Peptic Ulcer Disease'. D Y Graham , P D Klein , A R Opekun . *J Infect Disease* 1988. 157 p. .
- [O'connor et al. ()] 'Effect of duodenal ulcer surgery and enterogastric reflux on Campylobacter pyloridis'. H J O'connor , M F Dixon , J I Wyatt . *Lancet* 1986. ii p. .
- [Kang et al. ()] 'Helicobacter pylori and gastritis in patients with peptic ulcer and nonulcer dyspepsia: ethnic differences in Singapore'. J Y Kang , A Wee , M V Math . *Gut* 1990. 31 p. .
- [Dixon ()] 'Helicobacter pylori and peptic ulceration: histopathological aspects'. M F Dixon . *J Gastroenterol Hepatol* 1991. 6 p. .
- [Malaty et al. ()] 'Helicobacter pylori in Hispanics: Comparison with blacks and whites of similar age and socioeconomic class'. H M Malaty , D G Evans , D J Evans . *Gastroenterology* 1992. 103 p. .
- [Heatley ()] 'Helicobacter pylori Infection and Inflammation'. R V Heatley . *Scand J Gastroenterol* 1991. 26 p. . (suppl. 187)
- [Parsonnet et al. ()] 'Helicobacter pylori infection and the risk of gastric carcinoma'. J Parsonnet , G D Friedman , D P Vandersteen . *N Eng J Med* 1991. 325 p. .
- [Haruma et al. ()] 'Helicobacter pylori infection causes low antral somatostatin content: pathogenesis of inappropriate hypergastrinemia'. K Haruma , K Sumii , S Okamoto . *Gastroenterology* 1992. 102 (2) p. A80.
- [Prieto et al. ()] 'Helicobacter pylori infection in children: clinical, endoscopic, and histological correlations'. G Prieto , I Polanco , J Larrauri . *J Ped Gastroenterol & Nutrit* 1992. 14 (4) p. .
- [Tytgat et al. ()] 'Helicobacter pylori: Causal agent in peptic ulcer disease?'. G N Tytgat , Atr Axon , M F Dixon . *Working Party Reports* 1990. p. .
- [Fiocca et al. ()] 'High incidence of Campylobacter-like organisms in endoscopic biopsies from patients with gastritis, with or without peptic ulcer'. R Fiocca , L Villani , F Turpini . *Digestion* 1987. 38 p. .

- 206 [Craanen et al. ()] 'Intestinal metaplasia and *Helicobacter pylori*: an endoscopic bioptic study of the gastric
207 antrum'. M E Craanen , W Dekker , P Blok . *Gut* 1992. 33 p. .
- 208 [Ihamki et al. ()] 'Longterm observation of subjects with normal mucosa and with superficial gastritis. Results
209 of 23-27 years follow up examination'. T Ihamki , M Saukkonen , M Siurala . *Scand J Gastroenterology* 1979.
210 13 p. .
- 211 [Siurala et al. ()] 'New aspects on epidemiology, genetics, and dynamics of chronic gastritis'. M Siurala , K Varis
212 , M Kekki . *Front Gastrointest Res* 1980. 6 p. .
- 213 [Oderda et al. ()] 'Non-specific abdominal pain in childhood: Can serology detect those with *Campylobacter*
214 associated gastritis?'. G Oderda , D Vaira , J Holton . 29: A 1475. *Gut* 1988.
- 215 [Dooley et al. ()] 'Prevalence of *H pylori* infection and histological gastritis in asymptomatic persons'. C P Dooley
216 , H Cohen , P L Fitzgibbons . *N Eng J Med* 1989. 321 (23) p. .
- 217 [Marshall et al. ()] 'Pyloric *Campylobacter* infection and gastroduodenal disease'. B J Marshall , D B Mcgechie
218 , P A Rogers . *Med J Austr* 1985. 142 p. .
- 219 [Czinn and Carr ()] 'Rapid diagnosis of *Campylobacter pylori* associated gastritis'. S J Czinn , H Carr . *J Ped*
220 1987. 110 p. .
- 221 [Sipponen and Hyvarinen ()] 'role of *Helicobacter pylori* in the pathogenesis of gastritis, peptic ulcer, and gastric
222 cancer'. P Sipponen , H Hyvarinen . *Scand J Gastroenterol* 1993. 28 p. . (suppl)
- 223 [Graham et al. ()] 'Seroepidemiology of *Helicobacter pylori* infection in India. Comparison of developing and
224 developed countries'. D Y Graham , E Adam , G T Reddy . *Dig Dis Scien* 1991. 36 (8) p. .
- 225 [Hopkins et al. ()] 'Seroprevalence of *Helicobacter pylori* in Seventh-Day Adventists and Other Groups in
226 Maryland'. R J Hopkins , R G Russell , J M O'donnoghue . *Arch Int Med* 1990. 150 p. .
- 227 [Perez-Perez et al. ()] 'Seroprevalence of *Helicobacter pylori* infection in Thailand'. G I Perez-Perez , D N Taylor
228 , L Bodhidatta . *J Infect Dise* 1990. 161 p. .
- 229 [Siurala et al. ()] M Siurala , K Varis , Gastritis . *Scientific foundations of gastroenterology*, W Sircus, A N Smith
230 (ed.) 1980. p. .
- 231 [McNulty and Watson ()] 'Spiral bacteria of gastric antrum'. Cam McNulty , D M Watson . *Lancet* 1984. p. .
- 232 [Rathbone et al. ()] 'Systemic and local antibody responses to gastric *Campylobacter pylori* in non-ulcer
233 dyspepsia'. B J Rathbone , J I Wyatt , B W Worseley . *Gut* 1986. 27 p. .
- 234 [Dwyer et al. ()] 'The prevalence of *Campylobacter pylori* in human populations'. B Dwyer , J Kaldor , W Tee .
235 *Blackwell Science Publ* Rathbone BJ & Heatley RV (ed.) 1989. p. . (*Campylobacter pylori* and Gastroduodenal
236 Disease)
- 237 [Przyklend et al. ()] 'The role of *Campylobacter* (*Helicobacter*) *pylori* in disorders of the gastrointestinal tract'.
238 B Przyklend , A Bauernfeind , W Bornschein . *Infection* 1990. 18 (1) p. .
- 239 [Bj and Warren ()] 'Unidentified curved bacillus in the stomach of patients with gastritis and peptic ulcerations'.
240 Marshall Bj , J R Warren . *Lancet* 1984. p. .