



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

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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%).

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

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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%).

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.

Keywords: helicobacter pylori, sensitivity, specificity, rapid urease test, gastric cancer, ELISA test, IgG to H. pylori.

I. INTRODUCTION

Studies 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, 4-8). The prevalence in children was found to range between 20-68% (9-15). 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% (16-30). 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.

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).

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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.

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 (31-33).

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 (34-44). 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, 45). It has been revealed that the rate of infection of H. pylori in the United States is influenced by many factors like social-economic, 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, 46). 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, 46-48).

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

low sensitivity (63%) of our ELISA test compared to histology and culture, table-3.1. Our results of high sensitivity and specificity (89.5% and 96.8%) of rapid urease test agreed with Carvalho and others (15, 49-56), but disagreed with the results of Nichols and others (23, 57). 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, 31-33). 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.

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.

REFERENCES RÉFÉRENCES REFERENCIAS

1. Dwyer B, Kaldor J, Tee W, et al. The prevalence of *Campylobacter pylori* in human populations. In Rathbone BJ & Heatley RV (editors). *Campylobacter pylori and Gastrointestinal Disease*. Blackwell Science Publ. 1989: 190-196
2. Dixon MF. *Helicobacter pylori* and peptic ulceration: histopathological aspects. *J Gastroenterol Hepatol*. 1991; 6: 125-30
3. Heatley RV. *Helicobacter pylori* Infection and Inflammation. *Scand J Gastroenterol*. 1991; 26(suppl. 187): 23-30
4. Perez- Perez GI, Taylor DN, Bodhidatta L, et al. Seroprevalence of *Helicobacter pylori* infection in Thailand. *J Infect Dis*. 1990; 161: 1237-1241
5. Przykrend B, Bauernfeind A, Bornschein W, et al. The role of *Campylobacter (Helicobacter) pylori* in disorders of the gastrointestinal tract. *Infection*. 1990; 18 (1): 3/9-7/13
6. Siurala M, Sipponen P, Kekki M. Chronic gastritis: dynamic and clinical aspects. *Scand J Gastroenterol*. 1985; 20(suppl 109): 69-76
7. Siurala M, Varis K, Kekki M. New aspects on epidemiology, genetics, and dynamics of chronic gastritis. *Front Gastrointest Res*. 1980; 6: 148-65
8. Sipponen P, Hyvarinen H. role of *Helicobacter pylori* in the pathogenesis of gastritis, peptic ulcer, and gastric cancer. *Scand J Gastroenterol*. 1993; 28 suppl 196: 3-6
9. Drumm B, Sherman P, Cutz E, et al. Association of *Campylobacter pylori* on the Gastric Mucosa with Antral Gastritis in Children. *N Eng J Med*. 1987; 316: 1557-1561
10. Czinn SJ, Carr H. Rapid diagnosis of *Campylobacter pylori* associated gastritis. *J Ped* 1987; 110: 569-70
11. Oderda G, Vaira D, Holton J, et al. Non-specific abdominal pain in childhood: Can serology detect those with *Campylobacter* associated gastritis? *Gut*. 1988; 29: A 1475
12. Hill R, Pearman J, Worthy P. et al. *Campylobacter pylori* and gastritis in children. *Lancet*. 1986; i: 387
13. Cadranet S, Goossens H, Boek de M, et al. *Campylobacter pyloridis* in children. *Lancet*. 1986; i: 735-6
14. Eastham EJ, Elliott TSM. *Campylobacter pyloridis* in children. *Arch Dis Child*. 1987; 62: 652

15. Parsonnet J, Friedman GD, Vandersteen DP, et al. Helicobacter pylori infection and the risk of gastric carcinoma. *N Eng J Med.* 1991; 325: 1127-31
16. Booth L, Holdstock G, MacBride, et al. Clinical importance of Campylobacter pyloridis and associated serum IgG and IgA antibody responses in patients undergoing upper gastrointestinal endoscopy. *J Clin Pathol.* 1986; 39: 215-9
17. Burnett RA, Forrest JAH, Girdwood RWA, et al. Campylobacter-like organisms in the stomach of patients and healthy individuals. *Lancet.* 1984; i: 1349
18. O'Connor HJ, Dixon MF, Wyatt JI, et al. Effect of duodenal ulcer surgery and enterogastric reflux on Campylobacter pyloridis. *Lancet.* 1986; ii: 1178-81
19. Fiocca R, Villani L, Turpini F, et al. High incidence of Campylobacter-like organisms in endoscopic biopsies from patients with gastritis, with or without peptic ulcer. *Digestion.* 1987; 38: 234-44
20. Haruma K, Sumii K, Okamoto S, et al. Helicobacter pylori infection causes low antral somatostatin content: pathogenesis of inappropriate hypergastrinemia. *Gastroenterology.* 1992; 102 (pt 2): A80
21. Jones DM, Lessells AM, Eldridge J. Campylobacter-like organisms on the gastric mucosa: culture, histological, and serological studies. *J Clin Pathol.* 1984; 37: 1002-6
22. Langenberg ML, Tytgat GNJ, Schipper MEI, et al. Campylobacter-like organisms in the stomach of patients and healthy individuals. *Lancet.* 1984; i: 1348-9
23. Marshall BJ and Warren JR. Unidentified curved bacillus in the stomach of patients with gastritis and peptic ulcerations. *Lancet.* 1984: 1311-1315.
24. Marshall BJ, McGeachie DB, Rogers PA, et al. Pyloric Campylobacter infection and gastroduodenal disease. *Med J Austr.* 1985; 142: 439-44
25. McNulty CAM, Watson DM. Spiral bacteria of gastric antrum. *Lancet.* 1984, i: 1068-9
26. Price AB, Levi J, Dolby JM. Et al. Campylobacter pylori in peptic ulcer disease: microbiology, pathology, and scanning electron microscopy. *Gut.* 1985; 26: 1183-8
27. Rauws EAJ, Langenberg W, Houthoff HJ, et al. Campylobacter pyloridis-associated chronic active antral gastritis. *Gastroenterology.* 1988; 94: 33-40
28. Rathbone BJ, Wyatt JI, Worsley BW, et al. Systemic and local antibody responses to gastric Campylobacter pylori in non-ulcer dyspepsia. *Gut.* 1986; 27: 642-7
29. Von Wulffen H, Heesemann J, Butzow GH, et al. Detection of Campylobacter pylori in patients with antrum gastritis and peptic ulcer by culture, complement fixation test, and immunoblot. *J Clin Microbiol.* 1986; 24: 716-20
30. Wyatt JI, Rathbone BJ, Dixon MF, Heatley RV. Campylobacter pyloridis and acid induced gastric metaplasia in the pathogenesis of duodenitis. *J Clin Pathol.* 1987; 40: 841-848
31. Ihamki T, Saukkonen M, Siurala M. Longterm observation of subjects with normal mucosa and with superficial gastritis. Results of 23-27 years follow up examination. *Scand J Gastroenterology.* 1979; 13: 771-6
32. Prieto G, Polanco I, Larrauri J, et al. Helicobacter pylori infection in children: clinical, endoscopic, and histological correlations. *J Ped Gastroenterol & Nutrit.* 1992; 14(4): 420-25
33. Siurala M, Varis K. Gastritis In: Sircus W, Smith AN, eds, Scientific foundations of gastroenterology. Philadelphia: WB Saunders. 1980; 375-96
34. Craanen ME, Dekker W, Blok P, et al. Intestinal metaplasia and Helicobacter pylori: an endoscopic bioptic study of the gastric antrum. *Gut.* 1992; 33: 16-20
35. Dooley CP, Cohen H, Fitzgibbons PL, et al. Prevalence of H pylori infection and histological gastritis in asymptomatic persons. *N Eng J Med.* 1989; 321 (23): 1562-66
36. Graham DY, Klein PD, Opekun AR, 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. *J Infect Disease.* 1988; 157: 777-780
37. Graham DY, Adam E, Reddy GT, et al. Seroepidemiology of Helicobacter pylori infection in India. Comparison of developing and developed countries. *Dig Dis Scien.* 1991; 36 (8): 1084-8
38. Hopkins RJ, Russell RG, O'Donnoghue JM, et al. Seroprevalence of Helicobacter pylori in Seventh-Day Adventists and Other Groups in Maryland. *Arch Int Med.* 1990; 150: 2347-2348
39. Kang JY, Wee A, Math MV, et al. Helicobacter pylori and gastritis in patients with peptic ulcer and non-ulcer dyspepsia: ethnic differences in Singapore. *Gut.* 1990; 31: 850-853
40. Kosunen TU, Hook J, Rautelin HI, et al. Age-dependent increase of Campylobacter pylori antibodies in blood donors. *Scad J Gastroenterol.* 1989; 24: 110-114
41. Malaty HM, Evans DG, Evans DJ, et al. Helicobacter pylori in Hispanics: Comparison with blacks and whites of similar age and socioeconomic class. *Gastroenterology.* 1992; 103: 813-816
42. Mendall MA, Goggin PM, Molineaux N, et al. Childhood living conditions and Helicobacter pylori seropositivity in adult life. *Lancet.* 1992; 339 (8798); 896-7
43. Tytgat GN, Axon ATR, Dixon MF, et al. Helicobacter pylori: Causal agent in peptic ulcer disease? Working Party Reports. (1990): 36-45

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
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
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
47. Mollenkopf C, Steinger H, Weineck G, Meyer M. Gastritis: Immunohistochemical detection of specific and nonspecific immune response to Helicobacter pylori. Zeitschrift Fur Gastroenterol. 1990; 28 (7): 327-34
48. Crabtree JE, Shallcross TM, Wyatt JI, et al. Mucosal humoral immune response to Helicobacter pylori in patients with Duodenitis. Dig Dis Scien. 1991; 36 (9): 1266-73
49. Carvalho AS, Queiroz DM, Mendes EN, et al. Diagnosis and distribution of Helicobacter pylori in the gastric mucosa of symptomatic children. Brazilian J Med & Biolog Resea. 1991; 24(2): 163-66
50. Borromeo M, Lambert JR, Pinkard KJ. Technical Methods; Evaluation of "CLO-test" to detect Campylobacter pyloridis in gastric mucosa. J. Clin. Pathology. 1987; 40: 462-468
51. Ching CK, Buxton C, Holgate C, et al. Cytological brushing urea broth test: a highly sensitive and specific test for Helicobacter pylori infection. Gastrointestinal Endoscopy. 1991: 550-551
52. McNulty CAM, Dent JC, Uff JS, et al. Detection of Campylobacter pylori by the biopsy urease test: an assessment in 1445 patients. Gut. 1989; 30: 1058-1062
53. Thillainayagam AV, Arvind AS, Cook RS, et al. Diagnostic efficiency of an ultrarapid endoscopy room test for Helicobacter pylori. Gut. 1991; 32: 467-69
54. Vaira D, Holton J, Cairns SR, et al. Four hour rapid urease test for detecting Campylobacter pylori: is it reliable enough to start treatment? J Clin Pathol. 1988; 41: 355-6
55. Westblom TU, Madan E, Kemp J, Subik MA. Evaluation of a rapid urease test to detect Campylobacter pylori infection. J Clin Microbiol. 1988; 26: 1393-4
56. Nichols L, Sughayer M, DeGirolami PC, et al. Evaluation of diagnostic methods for Helicobacter pylori gastritis. Am J Clin Pathol. 1991; 95(6): 769-773
57. Newell DG, Hawtin PR, Stacey AR, et al. Estimation of prevalence of Helicobacter pylori infection in an asymptomatic elderly population compaing (14C) urea breath test in serology. J Clin Pathology. 1991; 44(5): 395-7

Table – 3.1 : The reliabilities of the various diagnostic tests for H. pylori infection.

Test	Sensitivity	Specificity	PP value	NP value
Histology	77.4%	75%	83.7%	66.7%
Culture	86.2%	100%	100%	76.5%
Serum IgG	63%	97%	98%	51%
Local IgA	79.2%	41.7%	57.6%	66.7%
Urease test	89.5%	96.8%	98.6%	78.9%

Table 3.2 : The prevalence of H. pylori infection: age difference.

	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%