Global Journal of Medical Research: F
Diseases
Volume 16 Issue 3 Version 1.0 Year 2016
Type: Double Blind Peer Reviewed International Research Journal
Publisher: Global Journals Inc. (USA)
Online ISSN: 2249-4618 & Print ISSN: 0975-5888

Systemic and Local Immune Response to H. Pylori Infection and their Correlation with the Degree of Antral and Duodenal Inflammation

By Awad Magbri & F. Stevens
University College Hospital Galway, United States

Abstract- *Design, setting, participants & measurements:* One hundred and eight patients presented to the University College Hospital Galway, Ireland (UCHG) with upper gastro-intestinal symptoms were included in the study. There were 52 males with age range 18-82 years, mean age 49 years, and 56 females, age range 20-83 years, mean age 52 years. They were grouped according to the endoscopic findings into 4 groups (i) Duodenal Ulcer (DU), (ii) Gastric Ulcer (GU), (iii) Gastritis (GS), & (iv) Non-Ulcer Dyspepsia (NUD). Five milliliter of venous blood was taken in a sterile plain tube; the serum was separated and stored at -70 C0 for ELISA assay. Endoscopy was carried out and at least 4 biopsies were obtained, 2 from the antrum and 2 from the first part of the duodenum (duodenal bulb). One biopsy each from the antrum and duodenum was transferred immediately into a sterile container containing 2 mL of RPMI 1640. The other biopsy specimens one from the antrum and one from the duodenum were processed for histological examination. The serum IgG antibodies to H. pylori and local IgA and IgG were measured by an ELISA test.

GJMR-F Classification : *NLMC Code: WD 308*

Strictly as per the compliance and regulations of:

© 2016. Awad Magbri & F. Stevens. This is a research/review paper, distributed under the terms of the Creative Commons Attribution-Noncommercial 3.0 Unported License http://creativecommons.org/licenses/by-nc/ 3.0/), permitting all non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

Systemic and Local Immune Response to H. Pylori Infection and their Correlation with the Degree of Antral and Duodenal Inflammation

Awad Magbri [α] & F. Stevens [σ]

Abstract- Design, setting, participants & measurements: One hundred and eight patients presented to the University College Hospital Galway, Ireland (UCHG) with upper gastro-intestinal symptoms were included in the study. There were 52 males with age range 18-82 years, mean age 49 years, and 56 females, age range 20-83 years, mean age 52 years. They were grouped according to the endoscopic findings into 4 groups (i) Duodenal Ulcer (DU), (ii) Gastric Ulcer (GU), (iii) Gastritis (GS), & (iv) Non-Ulcer Dyspepsia (NUD). Five milliliter of venous blood was taken in a sterile plain tube; the serum was separated and stored at -70 C^0 for ELISA assay. Endoscopy was carried out and at least 4 biopsies were obtained, 2 from the antrum and 2 from the first part of the duodenum (duodenal bulb). One biopsy each from the antrum and duodenum was transferred immediately into a sterile container containing 2 mL of RPMI 1640. The other biopsy specimens one from the antrum and one from the duodenum were processed for histological examination. The serum IgG antibodies to H. pylori and local IgA and IgG were measured by an ELISA test. Serum IgG antibody levels of the four groups of patients with various degrees of inflammation (0-3 degree) were compared, a highly significant association was found between the level of IgG antibodies to H. pylori and the severity of inflammatory reaction in the biopsy specimens (0 &2 and 0 & 3, p=0.0001) and between grade (1 & 2, p=0.045) and (1 &3, p=0.002) but not between (0&1, p=0.310), and (2&3, p=0.282) grades of inflammation.

Duodenal and antral IgA and IgG antibodies to H. pylori were assessed in 108 patients. A higher number of antral compared to duodenal biopsies in culture produce both IgA and IgG antibodies to H. pylori in all groups. 21/25 (84%) of DU patients had IgG antibodies to H. pylori, 22/25 (88%) had antral IgA antibodies, 19/25(76%) had antral IgG antibodies, 12/25 (48%) had duodenal IgA antibodies and 7/25(28%) had duodenal IgG antibodies to H. pylori.

In GU group 6/6(100%) patients had serum IgG antibodies to H. pylori, only 4/6(66.7%) had antral IgA antibodies, and 3/5(60%) had antral IgG antibodies. Duodenal antibodies were much less common than in the DU group, only 1/5(20%) had duodenal IgA antibodies and 0/5(0.0%) had IgG antibodies to H. pylori infection. In GSs group 20/31(65%) had serum IgG antibodies to H. pylori. 19/31(61.3%) had IgA antibodies, 13/28(46.43%) had antral IgG antibodies, 4/29 (13.8%) had duodenal IgA antibodies and 3/29(10.34%) had duodenal IgG antibodies to H. pylori. In the NUD group 23/46 (50%) had serum IgG antibodies to H. pylori, 27/46 (58.7%) had antral IgA antibodies, 17/43(39.5) had antral IgG antibodies, 8/44(18.2%) had duodenal IgA antibodies and 7/44(15.9%) of patients had duodenal IgG antibodies to H. pylori. The level of serum IgG to H. pylori was significantly different between the GS and NUD (p=0.013).

Conclusion: H. pylori is capable of inflecting local and systemic immune response with production of local (antral and duodenal) IgA and IgG and systemic IgG that correlate with the degree of inflammation in the antrum and duodenum. Successful treatment of the infection with antibiotics and proton pump inhibitors may influence the degree of inflammation, the local and systemic immune response to the organisms.

I. Introduction

Helicobacter pylori are now recognized as a primary cause of active chronic gastritis in humans. Most infected humans remain asymptomatic, but are at increased risk for developing peptic ulcer disease and possibly gastric cancer (1-8). The pathogenesis of this infection is well understood and the motility and urease activities are virulent factors in an animal model (9, 10). The urease enzyme also elicits a strong immune response during acute infection, suggesting that this abundant antigen is readily available to the immune system. An increase in serum IgG titers is predictive of ongoing infection (11, 12). About 50% of H. pylori isolates produce vacuolating toxins in vitro, which may be an important determinant of virulence (12-15). The epidemiology of the infection by H. pylori correlates with the prevalence of superficial type B gastritis. Infection is associated with active (presence of neutrophils), chronic (presence of lymphocytes and plasma cells), or active-chronic (neutrophils, lymphocytes, and plasma cells) inflammatory reactions (16-18). Gastric inflammation nearly always precedes the development of peptic ulceration, and is an important component in initiating the multi-step progression towards gastric carcinogenesis (19). In this study we have demonstrated that the severity of inflammation in the antrum is strongly associated with the presence and density of the bacterial colonization of the antral mucosa and the level of the serum IgG antibody to H. pylori.

II. Subjects and Methods

One hundred and eight patients presented to the University College Hospital Galway, Ireland (UCHG)

Author α: Dialysis Access Center of Pittsburgh, PA.
e-mail: elmagbri@hotmail.com
Author σ: University College Hospital Galway, Ireland.

© 2016 Global Journals Inc. (US)

with upper gastro-intestinal symptoms were included in the study. There were 52 males with age range 18-82 years, mean age 49 years, and 56 females, age range 20-83 years, mean age 52 years. They were grouped into 4 groups according to the endoscopic findings; (i) Duodenal Ulcer group (DU), (ii) Gastric Ulcer group (GU), (iii) Gastritis group (GS), and (iv) Non-Ulcer Dyspepsia group (NUD).

Endoscopy was carried out and at least 4 biopsies were obtained, 2 from the antrum and 2 from the first part of the duodenum (duodenal bulb). One biopsy each from the antrum and duodenum was transferred immediately into a sterile container containing 2 mL of RPMI 1640 (Gibco Life Technology Ltd. UK) supplemented with 10% fetal calve serum (FCS) and 40 ug/ml gentamicin (sigma). The biopsy specimens were cultured for 3 days at $37C^0$; the unspun supernatants were removed and stored at $-70C^0$ for estimation of IgA and IgG antibodies against H. pylori (34). The other biopsy specimens one from the antrum and one from the duodenum were placed in formalin and processed for histological examination. Five milliliter of venous blood was taken in a sterile plain tube; the serum was separated and stored at -70 C^0 for ELISA assay.

The serum IgG antibodies to H. pylori were measured by a commercially available ELISA kits (Biometra, Germany). Local IgA and IgG antibodies to H. pylori in the supernatants were measured by ELISA kit (KPL Kierkegaard & Perry Laboratories, Inc.) and the results were read on Dynatech MR 5000 automatic micro-plate reader at wave length 410 nm.

Endoscopy: all endoscopies were performed by two experienced endoscopists (CLL and SFM). Endoscopic findings were registered and stored in a computer data base. Biopsy specimens for histological examination, culture of the organisms, and estimation of local antibodies to H. pylori in the cultured biopsy materials were taken from the antrum and duodenal bulb. The endoscopes (Olympus Gastrscope type GIF-Q10 and GIF-Q20) and biopsy forceps were cleaned and disinfected in a commercially available 2% glutaraldehyde solution (Totacide 28 Coventry Chemicals Ltd) for a period of 10 minutes between each endoscopy.

a) Estimation of local and systemic H. pylori antibodies

ELISA assay for estimation of systemic antibodies: H. pylori antibody test is an enzyme linked immune-sorbent assay (ELISA) for the quantitative determination of H. pylori IgG antibodies in human serum or plasma. The methodology is based on using the solid phase technique on the micro-wells. The purified H. pylori specific antigens are immobilized into the surface of the micro-wells. The antigens react with H. pylori IgG antibodies present in both the standards and patient samples. The resulting antigen-antibody complex binds the second enzyme-conjugate antihuman IgG antibody, creating a sandwich. The enzyme conjugated antigen-antibody complex reacts with added substrate to develop a colored solution. The intensity of the color is dependent on the amount of the enzyme coupled to the complex and proportional to the H. pylori antibody concentration in the patient's samples. The optical density (OD) of the color in each well is read using Dynatech MR 5000 automatic micro-plate reader at 450 nm. Serum IgG antibodies to H. pylori were measured using the computer constructed formula for the semi-log curve fit. $Y = a + b \log x$

Where (Y) is the mean of the OD of the sample, (a)-is the Y intercept of the graph and (b) is the constant factor for (Logx). Having obtained the value of Logx from the equation the inversion of that value represents the amount of the IgG in the sample expressed in ELISA units.ml sample (E.U/ml sample). The assay procedure is done at room temperature ($18\text{-}22C^0$), higher temp up to $35C^0$ does not interfere with the results of the test but the incubation time for the TMB enzyme substrate reaction should be reduced according to the specifications of the manufacturer.

ELISA assays for estimation of local antibodies: Local IgA and IgG antibodies in the supernatants of the cultures biopsy materials from the duodenal bulb and the antrum were assayed using the ELISA assay as specified by the manufacture (KPL Kierkegaard & Perry Lab Inc.)

b) Statistical methods

The analysis of the results of the serum IgG antibody levels to H. pylori were assessed using One Way Analysis of Variance (ANOVA) with the addition of the Tukey HSD test option to the statistics. The additions of the Tukey option to the statistic command in the one-way analysis of variance will protect from declaring pairs of means differ when they could differ by chance. Analysis of variance offers a standard method for comparing various groups when there is no presumption beforehand that they differ. The Tukey's method exemplifies those tests for difference among group means by using the difference between the largest and smallest means, often called the range, as a measure of their dispersion. We have used the SYSTAT computer package (1990, SYSTAT inc.) for ANOVA with Tukey's option. Chi square was used to analyze the difference of the local and systemic humoral immune response to H. pylori infection. Pearson's correlation method was used for statistical analysis of the relationship between the density of H. pylori and serum IgG antibody levels.

III. Results

a) Antral inflammation Vs serum IgG antibodies to H. pylori

When the serum IgG antibody levels of the four groups of patients with various degrees of inflammation

© 2016 Global Journals Inc. (US)

(0-3 degree) were compared, a highly significant association was found between the level of IgG antibodies to H. pylori and the severity of inflammatory reaction in the biopsy specimens. One was analysis of variance with Tukey HSD multiple comparisons was applied for statistical analysis. There were a significant difference of serum IgG antibody levels to H. pylori between grade (0 &2 and 0 & 3, p=0.0001) and between grade (1 & 2, p=0.045) and (1 &3, p=0.002) but not between (0&1, p=0.310), and (2&3, p=0.282) grades of inflammation, Table-1.

b) Local and systemic humoral immune response to H. pylori

In vitro production of duodenal and antral IgA and IgG antibodies to H. pylori were assessed in 108 patients with dyspepsia. There were divided into four subgroups according to the endoscopic findings (i) DU group (25 patients), (ii) GU group (6 patents), (iii) GS group (31 patients), (iv) NUD group (56 patients), Tables -2-6. The results were analyzed using the Chi square test and the p values were shown in Table -6.

A higher number of antral compared to duodenal biopsies in culture produce both IgA and IgG antibodies to H. pylori in all groups (tables 2-6). This is an expected finding as H. pylori only colonize duodenum if gastric metaplasia is present. 21/25 (84%) of DU patients had IgG antibodies to H. pylori, 22/25 (88%) had antral IgA antibodies, 19/25(76%) had antral IgG antibodies, 12/25(48%) had duodenal IgA antibodies and 7/25(28%) had duodenal IgG antibodies to H. pylori, Table 2.

In the GU group although 6/6(100%) patients had serum IgG antibodies to H. pylori, only 4/6(66.7%) had antral IgA antibodies, and 3/5(60%) had antral IgG antibodies. Duodenal antibodies were much less common than in the DU group, only 1/5(20%) had duodenal IgA antibodies and 0/5(0.0%) had IgG antibodies to H. pylori infection, Table 3.

In the GS group 20/31(65%) had serum IgG antibodies to H. pylori. 19/31(61.3%) had IgA antibodies, 13/28(46.43%) had antral IgG antibodies, 4/29(13.8%) had duodenal IgA antibodies and 3/29(10.34%) had duodenal IgG antibodies to H. pylori, Table 4.

In the NUD group 23/46(50%) had serum IgG antibodies to H. pylori, 27/46(58.7%) had antral IgA antibodies, 17/43(39.5) had antral IgG antibodies, 8/44(18.2%) had duodenal IgA antibodies and 7/44(15.9%) of patients had duodenal IgG antibodies to H. pylori, Table 5.

The difference between the local (antral and duodenal) IgA and IgG antibodies in the DU, GU, GS, and NUD groups are shown in Tables 2-6.

The level of serum IgG to H. pylori was significantly different among the 4 groups when tested by the Kruskal-Wallis One Way Analysis of variance (p=0.0008). However, when the multiple comparisons using the Tukey HSD One way analysis of variance was applied the significant difference was found to be residing in the GS and NUD groups (p=0.013). There were no significant difference between the other groups.

IV. Discussion

Chronic gastritis with a prominence of lymphocytes and plasma cells is considered to be a morphologic indicator of an immune response to H. pylori infection (175). Indeed, a local immune response and production of antibodies against H. pylori have been demonstrated in patients with gastritis by Grabtree and others (19-21) and have been confirmed in this study also (Tables 2-6). Berstad et al (22) have demonstrated that the characteristics (severity and cell type) of gastritis associated with H. pylori infection are influenced by geographical factors that may be similar to those that modify infection rates for different geographical locations. The pathogenesis of the bacterium depends on the production of several virulence factors. The most important ones are CagA (cytotoxic associated gene A) and VacA (vacuolizing cytotoxin A) (23-27). We have found a relatively good correlation between the serology and the histological findings in antral biopsies despite the low sensitivity (63%) of our ELISA test compared to histology and culture. Our results agreed with Bertram et al (28, 29) that atrophic gastritis was associated with low detection rate of H. pylori by histology.

The reliability of culture of H. pylori as a diagnostic test varies considerably from center to center. Many workers found a sensitivity of 85% as we did. Our results showed lower sensitivity and specificity (77.4% and 75%) of histology. ELISA assay has been widely utilized in the sero-epidemiological studies and in the follow up of treated patients with H. pylori infection (30). We recommended determining IG antibody as a pre-endoscopic screening test. Further improvement and refinement of H. pylori antigens and proper exploitation of this test may have a great implication on the work load of busy endoscopic units and on the economy of health services. In H. pylori both IgA and IgG antibody titers are significantly elevated. On the other hand, the IgM antibodies are similar in H. pylori positive and negative subjects (31, 32). Probably because the IgM sero-conversion in the early phase of infection is rapidly transit and usually will not be detected. Acid-glycin extraction and more purified antigens to H. pylori raise the sensitivity and specificity of ELISA to H. pylori to 95-96% and 74-83% respectively (33-37). Although the antigen we used for ELISA assay was purified antigen, our ELISA lacks a sufficient sensitivity (63%, but has a high specificity (97%) for H. pylori.

Talley et al (38) evaluated the sensitivity and specificity of different ELISA and found them with high

© 2016 Global Journals Inc. (US)

sensitivity and specificity 96 and 94% respectively. Newell et al (39) also reported high sensitivity and specificity of ELISA (100%) in 47 patients who had endoscopy and confirmed by histology and culture.

Prieto et al (40) found that nodular anteritis was a frequent (67%) and a specific finding in H. pylori infection. Their presence was associated with lymphoid follicles in histopathological examination. Our results agreed with Prieto et al. Moderate inflammation were present in 9/25(36%) of our patients, all were H. pylori positive. We concluded that the majority of the elderly (>61 yrs) had a prevalence of H. pylori ranging from 45-64% depending on the mode used to detect H. pylori infection. More than 50% of this group had only mild inflammation and H. pylori was isolated in 43%, however, moderate to severe degree of inflammation was present in 44% of them and H. pylori was always associated with gastritis. If we interpret this association in the light of serum IgG antibodies to H. pylori we will find that 95% of the elderly people with moderate to severe degree of inflammation will have an elevated serum IgG antibody levels of 220-250 EU for moderate inflammation and >250 EU for severe inflammation.

The evolving knowledge of the immune reaction to H. pylori infection allows us to study the inter-relationship between (i) the presence of H. pylori and the density of the organisms on the histological section from the antral biopsies, (ii) the severity of inflammation, and (iii) the serum IgG antibodies to H. pylori as a marker of established infection. The presence of the bacteria was associated with increased amounts of mononuclear inflammatory cells, neutrophilic and eosinophilic leukocytes in the antral biopsies, thus confirming the results of Karttunen et al (41) and Alam et al (42). In these patients the infection with H. pylori produces a higher number of local IgA and IgG antibodies to H. pylori in the antral compared to duodenal biopsies in culture, in all groups (p=0.0296), Tables 2-6.

V. Conclusion

Helicobacter pylori are now recognized as a primary cause of active chronic gastritis in humans. Most infected humans remain asymptomatic, but are at increased risk for developing peptic ulcer disease and possibly gastric cancer. H. pylori is capable of inflecting local and systemic immune response with production of local (antral and duodenal) IgA and IgG and systemic IgG that correlate with the degree of inflammation in the antrum and duodenum. Successful treatment of the infection with antibiotics and proton pump inhibitors may influence the degree of inflammation, the local and systemic immune response to the organisms.

References Références Referencias

1. Weck MN, Gao L, Brenner H. Helicobacter pylori infection and chronic atrophic gastritis: associations according to severity of disease. Epidemiology. 2009; 20(4): 569-574
2. Dixon MF, Genta RM, Yardley JH, et al. Classification and grading of gastritis. The update Sydney System. International Workshop on the Histopathology of Gastritis, Houston 1994. Am J Surg Pathol. 1996; 20(10): 1161-1181
3. Blaser MJ, Atherton JC. Helicobacter pylori persistence: biology and disease. J Clin Invest. 2004; 113(3): 321-333
4. Ciociola AA, McSorley DJ, Turner K, et al. Helicobacter pylori infection rates in duodenal ulcer patients in the United States may be lower than previously estimated. Am J Gastroenterol 1999; 94: 1834
5. Parsonnet J, Friedman GD, Vandersteen DP, et al. Helicobacter pylori infection and gastric lymphoma. N Eng J Med. 1994; 330: 1267-1271
6. Wen S, Moss SF. Helicobacter pylori virulence factors in gastric carcinogenesis. Cancer Letters. 2009; 282(1): 1-8
7. Kusters JG, van Vliet AH, Kuipers EJ. Pathogenesis of Helicobacter pylori infection. Clinical Microbiology Reviews 2006; 19(3): 449-490
8. Sagaert X, Van Cutsem E, De Hertogh G, et al. Gastric MALT lymphoma: A model of chronic inflammation-induced tumor development. Nature Reviews Gastroenterology & Hepatology 2010; 7(6): 336-346
9. Blaser MJ. Hypotheses in the pathogenesis and natural history of Helicobacter pylori induced inflammation. Gastroenterology. 1992; 102: 720-727
10. Smooth DT, Mobley HL, Chippendale GR, et al. Helicobacter pylori urease activity is toxic to human gastric epithelial cells. Infec. Immuno. 1990; 58(6): 1992-1994
11. Mobley HLT, Hu LT, Foxall PA. Helicobacter pylori: properties and role in pathogenesis. Scand J Gastroentrol. 1991; 26(suppl 187): 39-46
12. Newell DG. Virulence factors of Helicobacter pylori. Scand J Gastroentrol. 1991; 26(suppl. 187): 31-38
13. Cover TL, Vaughn SG, Cao P, et al. Potentiation of Helicobacter pylori vacuolating toxin activity by nicotin and other weak bases. J Infect Disease 1992; 166: 1072-1078
14. Cover TL, Dooley CP, Blaser MJ. Characterization and human serology response to proteins in Helicobacter pylori broth culture supernatants with vacuolzing cytotoxin activity. Infec. Immu. 1990; 58(3): 603-610
15. Heatley RV. Helicobacter pylori infection and inflammation. Scand J Gastroenterol 1991; 26(suppl 187): 23-30
16. Kartunen T, Niemela S, Lehtola J. Helicobacter pylori in dyspeptic patients: quantitative association with severty of gastritis, intragastric pH, and serum

© 2016 Global Journals Inc. (US)

gastrin concentration. Scand J Gastroentrol. 1991;)suppl 186): 124-134
17. Mollenkoph C, Steininger 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
18. Prieto G, Polanco I, Larrauri J, et al. Helicobacter pylori infection in children: clinical, endoscopic, and histologic correlations. J Paed gastroentrol & Nutrit. 1992; 14(4): 420-425
19. 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): 1266-1273
20. Stromberg E, Edebo A, Svennerholm AM, et al. Decreased epithelial cytokine responses in the duodenal mucosa of Helicobacter pylori-infection duodenal ulcer patients. Clin Diagn Lab Immunol. 2003; 10: 116-124
21. Hamlet A, Thoreson AC, Nilsson O, et al. Duodenal Helicobacter pylori infection differs in cagA genotype between asymptomatic subjects and patients with duodenal ulcers. Gastroenterology 1999; 116: 259-268
22. Berstad AE, Holbjorn K, Bukholm G, et al. Complement activation directly induced by Helicobacter pylori. Gastroenterology 2001; 120: 1108-1116
23. Mosbley HL. Defining Helicobacter pylori as a pathogen: strain heterogeneity and virulence. Am. J Med. 1996; 100; 2S-9S, discussion 9S-11S
24. Rohde M, Puls J, Buhrdorf R, et al. A novel sheated surface organelle of Helicobacter pylori cag type IV secreation system. Moecular Microbiology 2003; 49(1): 219-234
25. Backert S, Selbach M, Role of type IV secretion in Helicobacter pylori pathogenesis. Cellular Microbiology 2008; 10(8): 1573-1581.
26. Odenbreit S, Puls J, Sedlmaier B, et al. translocation of Helicobacter pylori CagA into gastric epithelial cells by IV secretion. Science 2000; 287(5457): 1497-1500
27. Higashi H, Tsutsumi R, Muto S, et al. SHP-2 tyrosine phosphatase as na intracellular target of Helicobacter pylori Cag A protein. Science 2002; 295: 683-686
28. Bertram TA, Murray PD, Morgan DR, et al. Gastritis associated with infection by Helicobacter pylori in humans: Geographical differences. Scand J Gastroentrol. 1991; 26(suppl 181): 1-8
29. Klein PD, Graham DY, Gaillour A, et al. Water sources as risk factor for Helicobacter pylori infection in Peruvian children. Lancet. 1991; 337(8756): 1503-1506
30. Kosunen TU, Seppala K, Sarna S, Sipponen P. Diagnostic value of decreasing IgG, IgA, and IgM antibody titers after eradication of Helicobacter pylori. The Lancet. 1992; 339: 893-895
31. Maddocks AC. Helicobacter pylori (formerly campylobacter pylori) 1986-1989: A review. J Clin Pathol. 1990; 43: 353-356
32. Rauws EAJ, Tytgat GNJ. Campylobacter pylori. Gist-brocades Pharmaceuticals. Division of Royal Gist-brocades NV, Delft, The Netherlands. 1989.
33. Dent JC, McNullty CAM, Uff JS. Et al. Campylobacter pylori urease: a new serological test. Lancet. 1988; 1002
34. Goodwin CS, Blincow E, Peterson G, et al. Enzyme-Linked Immunosorbent Assay for Campylobacter pyloridis: Correlation with presence of C. pyloridis in the Gastric Mucosa. J Infect Dis 1987; 155(3): 488-494
35. Newell DG. An Enzyme-Linked Immunosorbent Assay for the serodiagnosis of Campylobacter pylori-associated gastritis. Scand J Gastroenterol. 1988; 23(suppl 142): 53-57
36. Rathbone BJ, Wyatt JI, Worsley BW, et al. Immune response to Campylobacter pyloridis. Lancet. 1985; 25: 1217
37. Steer HW, Hawtin PR, Newell DG. An ELISA technique for the serodiagnosis of Campylobacter pyloridis infection in patients with gastric and benign duodenal ulceration. Serodiag Immunother. 1987; 1: 253-259
38. Talley NJ, Newell DG, Ormand JE, et al. Serodiagnosis of Helicobacter pylori: comparison of enzyme-linked Immunosorbent assays. J Clinical Microbiol. 1991; 29(8): 1635-1639
39. Newell DG, Hawtin PR, Stacey AR, et al. Estimation of prevalence of Helicobacter pylori infection in an asymptoatic elderly population comparing [14C] urea breath test and serology. J Clin Pathology. 1991; 44(5): 385-387
40. Prieto G, Polanco I, Larrauri J, et al. Helicobacter pylori infection in children: endoscopic, and histology correlations. J Paed Gastroenterol & Nutri. 1992; 14(4): 420-425
41. Karttunen T, Niemela S, Lehtola J. Helicobacter pylori in dyspeptic patients: quantitative association with severity of gastritis, intragastric pH, and serum gastrin concentration. Scand J gastroenterol. 1991; (suppl 186): 124-134
42. Alam K, Schubert TT, Bologna SD, Ma CK. Increased density of Helicobacter pylori on antral biopsy is associated with severity of acute and chronic inflammation and likelihood of duodenal ulceration. Amer J Gastroenterol. 1992; 87(4): 424-428

© 2016 Global Journals Inc. (US)

Table- 1 : the severity of inflammation Vs serum H, pylori IgG antibody levels Matrix of pairwise absolute mean difference

	0	1	2	3
0	0.000			
1	24.239	0.000		
2	59.366	35.128	0.000	
3	92.460	98.221	33.094	0.000

Tukey HSD multiple comparisons

Matrix of pairwise comparison probabilities

	0	1	2	3
0	1.000			
1	0.310	1.000		
2	0.0001*	0.045*	1.000	
3	0.0001*	0.002*	0.282	1.000

**Significant results*

Table - 2 : Systemic (serum IgG antibody) Vs local (antral and duodenal IgA and IgG antibodies) to H. pylori in D>U patients

	Serum IgG	Antral IgA	Antral IgG	Duodenal IgA	Duodenal IgG
# of patients	25	25	25	25	25
Positive	21	22	19	12	7
negative	4	3	6	13	18
% positive	84%	88%	76%	48%	28%

Table - 3 : Systemic (serum IgG antibody) Vs local (antral and duodenal IgA and IgG antibodies) to H. pylori in G.U patients

	Serum IgG	Antral IgA	Antral IgG	Duodenal IgA	Duodenal IgG
# of patients	6	6	5	5	5
Positive	6	4	3	1	0
Negative	0	2	2	4	5
% positive	100%	66.7%	60%	20%	0%

Table - 4 : Systemic (serum IgG antibody) Vs local (antral and duodenal IgA and IgG antibodies) to H. pylori in gastritis patients

	Serum IgG	Antral IgA	Antral IgG	Duodenal IgA	Duodenal IgG
# of patients	31	31	28	29	29
positive	20	19	13	4	3
Negative	11	12	15	25	26
% positive	65%	61.3%	46.43%	13.8%	10.34%

Table - 5 : Systemic (serum IgG antibody) Vs local (antral & duodenal IgA & IgG antibodies) to H. pylori in patients with NUD

	Serum IgG	Antral IgA	Antral IgG	Duodenal IgA	Duodenal IgG
# of patients	46	46	43	44	44
Positive	23	27	17	8	7
Negative	23	19	26	36	37
% positive	50%	58.7%	39.5%	18.2%	15.9%

Table - 6 : The p value for the difference between positive and negative IgA and IgG antibodies to H. pylori in all groups

	DU	GU	GS	NUD
IgA antibody	0.0064*	0.35	0.0044*	0.0002*
IgG antibody	0.0018*	0.17	0.0062*	0.026*

NUD – non-ulcer dyspepsia

**significant result*

The overall probability is 0.0296 for the IgA and IgG status in all groups.

© 2016 Global Journals Inc. (US)