

# Comparison between IgG Antibodies and Urease Activity to *Helicobacter pylori* in patients with different Gastroduodenal conditions

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## ABSTRACT

**Background:** Many non-invasive and invasive tests are in clinical practice for the diagnosis of *Helicobacter pylori* infection that is one of the most common infections in the world due to its association with contaminated water, poor housing and socioeconomic pitfalls. Many problems influence the choice of a diagnostic test most notably the sensitivity and specificity of the tests, the clinical circumstances and the cost-effectiveness of the testing strategy.

**Aim:** To compare the urease test with IgG antibody titre in *H. pylori* infection diagnosis.

**Methods:** A total number of 84 subjects were included in the study. Gastric endoscopy and histopathological examination was done in all the subjects. ELISA for IgG antibody and urease activity was measured by standard method and by using standard kits.

**Results:** The results of our study propose that patients with Active Chronic Gastritis had shown 100% raised antibody titre in comparison to urease test (71.59%). Furthermore, 55 out of 74 showed the raised antibody titre to *H. pylori* with prevalence of 74.32% and 51 cases (68.9%) revealed urease activity.

**Conclusion:** In view of the results of this study, IgG antibody titre is better test for screening of *H. pylori* patients for start of therapy.

**Keywords:** IgG antibodies, helicobacter pylori, urease activity

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## INTRODUCTION

The human stomach is regarded as the principal host and reservoir of *Helicobacter pylori*. *Helicobacter pylori* invade human stomach in early childhood. *Helicobacter pylori* infection results in gastritis, ulcers and extragastric complications upon exposure to appropriate host and environmental factor, by producing low grade inflammatory changes in the gastric mucosa<sup>1,2,3,4</sup>. The prevalence of infection caused by *H. pylori* is 20-50% in developed countries and 70%-90% in developing countries<sup>5,6</sup>. Its transmission is from person-to-person spread and through oral and fecal-oral route<sup>7</sup>.

Different invasive and non-invasive techniques are in clinical practice for the diagnosis of *H. pylori* infection. Invasive methods include histology, rapid urease test (RUT), microbiological culture and polymerase chain reaction (PCR). These are called invasive as endoscopy is required for sample collection while non-invasive tests are stool antigen test, serology and urea breath test (UBT)<sup>8</sup>. Either

these tests are difficult for the patient or are not cost effective and also have limitations<sup>9</sup>. We planned this study to compare the efficacy of biopsy based test urease with cost effective serology test.

## MATERIAL AND METHODS

The study was conducted at microbiology department, basic medical sciences institute, Jinnah Postgraduate Medical Centre, Karachi. A total number of 74 patients were included in the study among which 45 were males with a mean age of 41 years and 29 were females with a mean age of 37 years. The subjects included in the study were patients who had the history of dyspepsia, epigastric pain or both. Ten control cases included in the study had symptoms of gastritis and peptic ulcer but normal gastric and duodenal mucosa on histopathological examination.

Approximately, 4 ml of blood was taken for IgG antibodies to *H. pylori* and two biopsy specimens were taken one for histopathology and one for urease activity. Histopathological examination was carried out by using Giemsa stain for organism's morphology and by H&E stain for histopathology. Commercially prepared kit of Adamjee Company was used for estimation of urease activity while ELISA kit of

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AMRAD Company was used for estimation of IgG antibodies to *H. pylori*.

## RESULTS

Specimens were considered reactive for IgG antibodies to *H. pylori* if it contained greater than 50units/ml and considered non-reactive if they contained less than 30units/ml titre at the test dilution. The units of measurement of infection rate were the number of *H. pylori* reactive cases in 100 patients. Confidence limit for 95% probability was derived by Barker and Hall formula. Table 1 shows results of urease activity and IgG antibodies to *H. pylori* in patients and controls. Among 74 patients 51 (68.9%) had shown the urease activity and 55 (74.32%) had shown the positive antibody titre. Among ten controls, two samples (20%) had shown the urease activity and only one (10%) had shown the positive antibody titre.

Table 1: Relationship of urease activity and IgG antibodies to *H. pylori* in patients and control subjects

No. of Cases	Urease activity	IgG antibody
Patients (74)	51 (68.9%)	55 (74.32%)
Controls (10)	02 (20%)	01 (10%)

Table 2 shows the urease activity versus IgG antibody against *H. pylori* in gastritis and peptic ulcer patients. Among 51 cases of chronic gastritis, 35 (68.67%) had shown urease activity while 33 (66.70%) cases had shown significant IgG antibody titre. Among 13 cases of active chronic gastritis, 11(71.59%) had shown the urease activity while all 13(100%) cases had shown significantly raised IgG antibody titre. In gastric ulcer patients, 2 out of 5 patients (40%) had shown urease activity while 4 out of five (80%) had shown significantly raised IgG antibody titre. In duodenal ulcer patients, 3 out of 5 patients (60%) had shown urease activity while all five cases (100%) had shown raised IgG antibody titre.

Table 2: Urease activity VS IgG antibody titre to *H. pylori* in gastritis and peptic ulcer patients

Patients	Urease +ve	Antibody+ve
Chronic gastritis (n=51)	35(68.67%)	33(66.70%)
Active ch. gastritis(n=13)	11(71.59%)	13(100%)
Gastric ulcer(n=5)	02(40%)	04(80%)
Duodenal ulcer (n=5)	03(60%)	05(100%)

## DISCUSSION

The results of our study propose that patients with Active Chronic Gastritis had shown 100% raised antibody titre in comparison to urease test (71.59%). Furthermore, 55 out of 74 had shown the raised

antibody titre to *H. pylori* with prevalence of 74.32% and 51 cases (68.9%) had shown the urease activity. The results of our study are comparable to a study conducted on 102 patients that shows a significantly raised antibody titre compared to histopathological examination<sup>10</sup>. Prevalence of *H. pylori* in patients with chronic gastritis was also studied by Loffeld et al., 1989 that also showed relation of antibodies to *H. pylori* to that of histopathology<sup>11</sup>. A similar result is documented by Perez-Perez et al., 1990 who compared antibody titre to culture and histopathological examination and declared that antibody measurements are of equal importance<sup>12</sup>. Literature shows that Detection of antibodies is useful for detecting past or present exposure but the serological tests fail to distinguish between past and current *H. pylori* infection<sup>13</sup>.

Our results are in line with other studies regarding the urease activity. Our study results propose that antibody titre is better as compared to rapid urease activity and equally comparable to histopathological examination<sup>14</sup>. Another study was made on 200 patients who were diagnosed endoscopically and on the basis of urease activity; the prevalence of urease activity was 91.6%<sup>15</sup>. It is proposed that urease tests are faster, cheaper and have comparable sensitivity and specificity when compared with histology and culture but their sensitivity decreases in patients with bleeding peptic ulcers (67%-85%) or patients with partial gastrectomy(79%)<sup>16, 17, 18, 19</sup>. We recommend that serological estimation of antibodies to *H. pylori* are equally comparable to other tests and must be used for the initial screening of *H. pylori* infection as compared to invasive tests like urease test and histopathology.

## REFERENCES

- Pietrojusti A, Galante A, Magrini A, Bergamaschi A. *Helicobacter pylori* interference with micronutrients and orally administered drugs: a new mechanism explaining its role in extra gastric disorders. *Mini Rev Med Chem.* 2008; 8:135-41.
- Kuipers EJ, B MJ. Acid peptic disease: Epidemiology and Pathobiology. In: Goldman L, Ausiello D, eds. *Cecil Medicine.* 23<sup>rd</sup> ed. Philadelphia: Elsevier. 2007: 1009-13.
- Palmer KR, Penman ID, Paterson- Brown S. Alimentary Tract and Pancreatic Diseases. In: Boon NA, Colledge NR, Walker BR, Hunter JAA, eds. *Davidson's Principles and Practice of Medicine.* 20<sup>th</sup> ed. London. Elsevier. 2005: 849-934.
- Kumar P, Clark M. Kumar and Clark's Clinical Medicine. 7<sup>th</sup> ed. London: WB Saunders. 2002:253-334.

5. Kabir S. Detection of *Helicobacter pylori* in feces by culture, PCR and enzyme immunoassay. *J Med Microbiol* 2001; 50: 1021-1029.
6. Dunn BE, Cohen H, Blaser MJ. *Helicobacter pylori*. *Clin Microbiol Rev* 1997; 10: 720-741.
7. Allaker RP, Young KA, Hardie JM, Domizio P, Meadows NJ. Prevalence of *Helicobacter pylori* at oral and gastrointestinal sites in children: evidence for possible oral-to-oral transmission. *J Med Microbiol* 2002; 51: 312-317.
8. Peng NJ, Lai KH, Lo GH, Hsu PI. Comparison of noninvasive diagnostic tests for *Helicobacter pylori* infection. *Med Princ Pract* 2009; 18: 57-61.
9. Shukla SK, Prasad KN, Tripathi A, Ghoshal UC, Krishnani N, Nuzhat H. Quantitation of *Helicobacter pylori* ureC gene and its comparison with different diagnostic techniques and gastric histopathology. *J Microbiol Methods* 2011; 86: 231-237.
10. Quintero GA, Williams JD, Wingate DL, Newell DG. A microbiological etiology for gastritis and peptic ulceration. *World JSurg*. 1988;12:718-22.
11. Loffeld RJ, Stobberingh E, Flendrig JA, van Spreeuwel JP, Arends JW. Diagnostic value of an immunoassay to detect anti *Campylobacter pylori* antibodies in non-ulcer dyspepsia. *Lancet*. 1989; 1: 1182-5.
12. Perez-Perez GI, Taylor DN, Bodhidatta L, Wongsrichanalai J, Baze WB, Dunn BE, Echeverria PD, Blaser MJ. Seroprevalence of *Helicobacter pylori* infections in Thailand. *J Infect Dis*. 1990;161:1237-41.
13. Choi J, Kim CH, Kim D, Chung SJ, Song JH, Kang JM, Yang JI, Park MJ, Kim YS, Yim JY, Lim SH, Kim JS, Jung HC, Song IS. Prospective evaluation of a new stool antigen test for the detection of *Helicobacter pylori*, in comparison with histology, rapid urease test, (13)C-urea breath test, and serology. *J Gastroenterol Hepatol* 2011; 26: 1053-1059.
14. Montgomery EA, Martin DF, Peura DA. Rapid diagnosis of *Campylobacter pylori* by Gram's stain. *Am J Clin Pathol*. 1988; 90: 606-9.
15. Nanivadekar SA, Bhat PP, Sawant PD, Shroff CP, Patel HD. Unusual features of *Helicobacter* (*Campylobacter*) *pylori*-associated gastritis in India, a study of 200 cases. *J Assoc Physicians India*. 1990; 38: 695-8.
16. Choi YJ, Kim N, Lim J, Jo SY, Shin CM, Lee HS, Lee SH, Park YS, Hwang JH, Kim JW, Jeong SH, Lee DH, Jung HC. Accuracy of diagnostic tests for *Helicobacter pylori* in patients with peptic ulcer bleeding. *Helicobacter* 2012; 17: 77-85.
17. Tian XY, Zhu H, Zhao J, She Q, Zhang GX. Diagnostic performance of urea breath test, rapid urea test, and histology for *Helicobacter pylori* infection in patients with partial gastrectomy: a meta-analysis. *J Clin Gastroenterol* 2012; 46: 285-292.
18. Tseng CA, Wang WM, Wu DC. Comparison of the clinical feasibility of three rapid urease tests in the diagnosis of *Helicobacter pylori* infection. *Dig Dis Sci* 2005; 50: 449-452.
19. Gisbert JP, Abaira V. Accuracy of *Helicobacter pylori* diagnostic tests in patients with bleeding peptic ulcer: a systematic review and meta-analysis. *Am J Gastroenterol* 2006; 101: 848-863.