# Papers

## Helicobacter Pylori Associated with Peptic Ulcer Disease and Chronic Antral Gastritis in Diwania City

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## Abstract

**A**im: To study the prevalence of Helicobacter Pylori organism as a cause of peptic ulcer disease and chronic antral gastritis in patients    in Diwania city.

**Methods**: In this study 90 patients patients complaining from classical symptoms of chronic dyspepsia including upper abdominal pain or discomfort, often accompanied by bloating, abdominal distention, nausea, or early satiety were included in this study. Peptic ulcer and antral gastritis are diagnosed on both endoscopic findings and histological results of biopsy . 4–6 punch biopsies were taken from gastric antrum in all the individuals and in case of gastric ulcer an additional biopsy was taken from the edge of the ulcer to exclude its malignant nature. Helicobacter pylori (H. pylori) organism was diagnosed using Histology (using Giemsa Stain). Histological diagnosis of H. pylori was taken as the "gold standard" for the presence of H. pylori organism. Histological diagnosis of gastritis was made using Hematoxylin and Eosin Stain and the gastritis was classified as active chronic gastritis and superficial chronic gastritis.

**Results**:  From 90 patients were included in this study, 50 patients were proved to have peptic ulcer disease, 45(90%) of them have positive results of H pylori while 40 patients proved to have chronic antral gastritis, 33(82.5%) of them were positive for H. pylori on histological examination.

**Conclusion**: A highly significant association between H. pylori infection with peptic ulcer disease patients and chronic antral gastritis in Diwaniya city.

**List of abbreviations**

*PU = peptic ulcer*
PUD = peptic ulcer disease
CAG = chronic antral gastritis
OGD = oesophagogastrodoudenscopy
DU = duodenal ulcer

الخلاصة

**الغاية من البحث :**هو لمعرفة العلاقة بين البكتريا الهلاليه البوابيه كسبب للإصابة بقرحة المعدة والاثنى عشري والإصابة بالتهاب المعدة ألبوابي المزمن في مدينة الديوانية

**طريقة البحث:**في هذه الدراسة كان عدد المرضى المشتركين 90 مريض يعانون من أعراض اضطراب الهضم المزمنة (نوبات متكررة من الالام المعدة مصحوبة بغثيان ورغبة بالتقيؤ مع انتفاخ البطن والشعور بالشبع المبكر) اجري لهم ناظور المرى والمعدة والاثني عشري .اعتمدت هذه الدراسة على فحص الناظور مع نتيجة الزرع النسيجي للعينات التي تم أخذها من كل مريض عن طريق ناظور المعدة والاثني عشري كأساس لتشخيص قرحة المعدة وتفريقها من التهاب المعدة ألبوابي المزمن,وقد أخذنا 4-6 عينات من جدار المعدة البوابي وكذلك أخذنا عينة إضافية للمرضى المصابين بقرحة المعدة من الحافة السليمة والقريبة من مكان القرحة للتاكد من عدم وجود قرحة سرطانية المنشأ,ولتشخيص البكتريا الهلاليه البوابيه استخدمنا صبغة الكمزا ,وكان الزرع النسيجي للكشف عن وجود

البكتريا هو الأساس الثابت لتأكيد الإصابة بينما كان الزرع النسيجي باستخدام صبغة الايوسين والهيماتوكسلين هو الإجراء الذي استخدمناه لتشخيص التهاب المعدة المزمن والذي تم تقسيمه إلى التهاب سطحي مزمن والتهاب فعال مزمن .

**النتائج :**كان عدد المرضى المشاركين في هذا البحث 90 مريض وقد أظهرت النتائج إصابة 50 مريض بقرحة المعدة والاثني عشري وكان نسبة المصابين بالبكتريا الهلالية البوابية    (45) مريض (90%) , بينما كان عدد المرضى المصابين بالتهاب المعدة المزمن 40 مريض وكان نسبة المصابين بالبكتريا الهلالية البوابية    (33) (82.5%) .

**خلاصة البحث :**اظهرت هذه الدراسة علاقة وثيقة بين الإصابة بالتهاب البكتريا الهلالية البوابية  والإصابة بكل من قرحة المعدة والاثني عشري والتهاب المعدة المزمن .

## Introduction

Peptic ulcer disease (PUD) and chronic gastritis are most common disorders throughout the whole world. Helicobacter pylori (H. pylori) infect more than half of the world population making it the most prevalent infection worldwide [1]. It is regarded as the main etiological factor for peptic ulcer disease [2, 3, 4] The prevalence rate of H. pylori infection in the West is less than 50% [5,6], while in the developing countries this rate is high and range between 70-90% [5,6,7,]. The main risk factors for H. pylori acquisition are childhood, low socio-economic status, low living standards, poor sanitation, and the presence of H. pylori-positive family members [8, 9, 10, 11, 12, 13, 14, 15, 16, and 17]. In addition there is a possible role for genetic factors [18] Chronic antral gastritis is a common finding in PUD especially in duodenal ulcer (DU). The prevalence of Helicobacter pylori infection increases with age and may be present in around 75% of patients above 65 years of age [19]. H. pylori is known to be the commonest cause of antral gastritis[20] and is associated with duodenal ulcer relapse [21]. H. pylori infection in elderly people is receiving increased attention, and studies have confirmed H. pylori gastritis to have a high prevalence in older dyspeptic individuals [22, 23]. The age-related increase in gastric antral H. pylori carriage has been shown to parallel the age-related increase in the prevalence of chronic antral gastritis (CAG) [24,25].One study has reported association between chronic gastritis

and active duodenal ulcer as 100% compared to 50 % in nonulcer patients , about 75% of patients with chronic gastritis have been found to have H.pylori compared to  10% in those without gastritis. The role of H. pylori in the histological progression of CAG, however is unclear. Helicabacter pylori has been found to be associated with chronic type B gastritis (nonimmune) [18,19]. This type of chronic gastritis is associated with patchy gastric atrophy . chronic gastritis type A thought to be of autoimmune etiology is rarely associated with H. pylori infection. Also a strong association has been found between the number of antral H. pylori organisms and severity of chronic gastritis [20, 21, 22, and 23]. A definite pathological association between H.pylori infection and gastritis is supported by (1) a positive correlation between chronic gastritis and H. pylori infection, (2) a significant correlation between the number of H. pylori organism and the degree of polymorphonuclear (PMN) leukocyte infiltration in gastric mucosa, (3) absent or decreased number of PMN infiltration in portions of gastric mucosa not associated with H. pylori infection, (4) an improvement of chronic gastritis following eradication of H. pylori and (v) development of gastritis in healthy volunteers (in whom pre-ingestion antral biopsies were normal) following ingestion of H. pylori [24,25].

## Patients and Methods

In this study 117 patients complaining from classical symptoms of chronic dyspepsia

including upper abdominal pain or discomfort, often accompanied by bloating, abdominal distention, nausea, or early satiety.OGD had been done for all patients ,27 of them show normal OGD so they were excluded from the study and the remaining (90) were enrolled in this study .The diagnosis of PUD and CAG were done by both OGD and histological results of biopsy, 4-6 antral biopsies were taken from each participant using sterilized biopsy forceps the procedure was done in the endoscopy unit of DIWANIA TEACHING HOSPITAL.50 patients were proved to have PU and 40 patients have AG. Individuals with history of ingestion of steroids, non-steroidal anti-inflammatory drugs antibiotics 4-6 weeks prior were not included in this study. An additional biopsy was taken from the edge of gastric ulcers to exclude its malignant nature.The definitive diagnosis of PUD and CAG depend on both OGD and histological results of biopsy taken.

## Histopathology

For histopathological study, paraffin sections of 3-5 mm thickness were taken of the biopsy material and these were stained with Hematoxylin and Eosin Stain (to study the histology of gastric antrum) and Giemsa Stain (For identification of H. pylori organism) separately. Detection of H. pylori on Giemsa Stain was taken as "Gold Standard" for the presence of H.pylori infection.

## Statistical Analysis

Chi-square test was used to analyze the results and a P value of less than 0.05 was taken to be significant.

## Results

In this study total patients enrolled are 90 patients 72 patients are male and 18 are female and the mean age group of $30.88\pm 12.80$ and $29.80\pm11.22$ respectively as shown in Table ( I).50 patients from them are proved to have PU disease (42 are male,8 are female),while 40 patients have CAG (30 male and 10 female) as shown in table (2).72 patients from the sample of this study are male (80%) while 18 of them are female (20%) as shown in figure (1). Of the 50 peptic ulcer disease patients (DU 46,GU 2 and combined DU and GU 2),45 (90%) of them have positive results of H pylori infection and 8 patients (10%) are negative and this results is statistically highly significant (P- value < 0.05), while in those patients with CAG (40), 33 (82.5%) of them have positive H pylori results and 7 (17,5%) have negative results ,also this have significant statistical results (p value <0.05).

Table1. Characteristics of peptic ulcer disease patients and chronic antral gastritis

|  | PU | CAG |
|---|---|---|
| NUMBER | 50 | 40 |
| MALE | 42 | 30 |
| FEMALE | 8 | 10 |
| *MEAN AGE 4- S.D* | 30.88 4- 12.80 | 29.80 4- 11.22 |

Table 2. Show characteristics of patients and relation with H pyl

| Site of ulcer | sex | | H. pyloric | | p- value |
|---|---|---|---|---|---|
|  | Male | Female | +ve | -ve |  |
| P.U | 42 | 8 | 45 | 5 | <0.05 |
| CAG | 30 | 10 | 33 | 7 | <0.05 |
| Total | 72 | 18 | 63 | 27 |  |
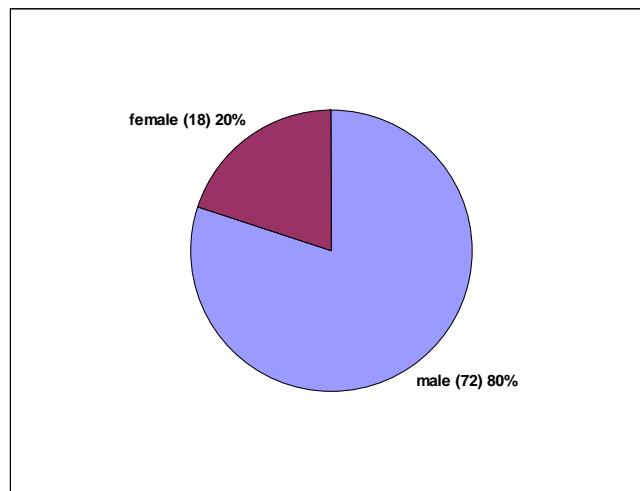
P – Value = <0.05

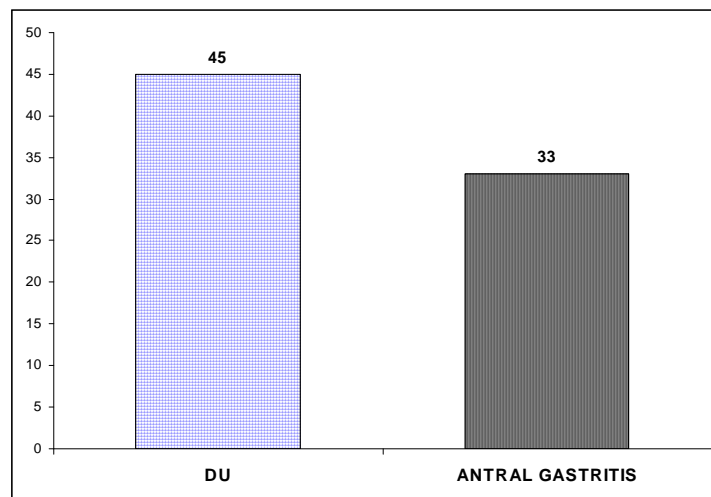Figure 1. Show distributions of peptic ulcer and CAG according to the sex



Figure 2. Show distributions of H. pyloric according the site of pathology

## Discussion

Helicobacter pylori is a gram negative bacillus responsible for one of the most common infection found in human world wide. Warren and Marshall first cultured the organism in as Campylobacter pylori in 1982.By 1989 it was renamed and recognized to be closely associated with antral gastritis (gastric and duodenal ulcers in adults and children ).

Helicobacter pylori organisms has been found to play an important pathogenic role in peptic ulcer disease (especially in duodenal ulcer), gastritis and in gastric cancer special gastric lymphoma and adenocarcinoma [2, 4, 6, 7]. The antral colonization with H. pylori organism has been reported from every corner of the globe especially from developing and underdeveloped countries [2,4,6].In this study the total number of the sample 90 patients and this match with other studies,( Al-Moagel MA, Evans DG, Abdulghani ME, et al. , Dominici P, Bellentani S, Di Biase AR, et al ) [13,23,25] Although PU and CAG usually associated

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with elderly patients this study show increase incidence in young age group , other studies show increase incidence of PU and CAG disease among young age group [13,23,25].This study show increase incidence of infection in male more than female, other studies show increase incidence in both sex [13,23,25].The study show a significant association among H pylori infection and development of both PU and CAG , other studies show the same statistical significant of  association [13,23,25]. This study confirms the data published from different parts of the world (12,17) .

## Conclusion

This study found a highly significant association between Helicobacter pylori infection and peptic ulcer disease (duodenal ulcer). Also we found a highly significant association between H.pylori infection in chronic antral gastritis. This leads me to a conclusion that Diwaniya city is a highly endemic area with H.pylori and it is worthwhile to consider treating and eradicating H,pylori  for every patient complaining from chronic dyspeptic symptoms.

## References

1. Pisani P, Parkin DM, Munoz N, Ferlay J. Cancer and infection: estimates of the attributable fraction in 1990. Cancer Epidemiol Biomarkers Prev 1997; 6: 387-400.
2. Crabtree JE, Taylor JD, Wyatt JI, et al. Mucosal IgA recognition of Helicobacter pylori 120 kDa protein, peptic ulceration, and gastric pathology. Lancet 1991; 338: 332-5.
3. Graham DY, Lew GM, Klein PD, et al. Effect of treatment of Helicobacter pylori infection on the long term recurrence of gastric and duodenal ulcer. A randomized, controlled study. Ann Intern Med 1992; 116: 705-8.
4. Karita M, Morshed MG, Ouchi K, Okita K. Bismuth-free triple therapy for eradicating Helicobacter pylori and reducing the gastric ulcer recurrence rate. Am J Gastroenterol 1994; 89: 1032-5.
11. Graham DY, Malaty HM, Evans DG, Evans DJ Jr, Klein PD, Adam E. Epidemiology of Helicobacter Pylori in an asymptomatic population in the United States. Effect of age, race, and socioeconomic status. Gastroenterology 1991; 100: 1495-501.
12. Mégraud F, Brassens-Rabbe MP, Denis F, Belbouri A, Hoa DQ. Seroepidemiology of Campylobacter pylori infection in various populations. J Clin Microbiol 1989; 27: 1870-3.
13. Al-Moagel MA, Evans DG, Abdulghani ME, et al. Prevalence of Helicobacter Pylori (formerly Campylobacter) infection in Saudi Arabia, and comparison of those with and without upper gastrointestinal symptoms. Am J Gastroenterol 1990; 85: 944-8.
14. Bani-Hani KE, Hammori SM. Prevalence of Helicobacter pylori in Northern Jordan. Endoscopy-based study. Saudi Med J 2001; 22: 843-7.
15. Rosenstock S, Jorgensen T, Andersen L, Bonnevie O. Seroconversion and seroreversion in IgG antibodies to Helicobacter pylori: a serology based prospective cohort study. J Epidemiol Community Health 2000; 54: 444-50.
16. Malaty HM, Graham DY. Importance of childhood socioeconomic status on the current prevalence of Helcobacter pylori infection. Gut 1994; 35: 742-5.
17. Pounder RE, Ng D. The prevalence of Helicobacter pylori infection in different countries. Aliment Pharmacol Ther 1995; 9(suppl 2): 33-9.
18. Vandenplas Y, Badriul H. Helicobacter pylori infection. Taiwan Erh Ko I Hsueh Hui Tsa Chih 1999; 40: 212-24.
19. Opekun AR, Gilger MA, Denyes SM, et al. Helicobacter pylori infection in children of Texas. J Pediatr Gastroenterol Nutr 2000; 31: 405-10.
20. Deltenre M, de Koster E. How come I've got it? (A review of Helicobacter pylori transmission). Eur J Gastroenterol Hepatol 2000; 12: 479-82.
21. Luzza F, Mancuso M, Imeneo M, et al. Evidence favouring the gastro-oral route in the transmission of Helicobacter pylori infection in children. Eu J Gastroenterol Hepatol 2000; 12: 623-7.
22. Drumm B, Perez-Perez GI, Blaser MJ, Sherman PM. Intrafamilial clustering of Helicobacter pylori infection. N Engl J Med 1990; 322: 359-63.
23. Malaty HM, Kumagai T, Tanaka E, et al. Evidence from a nine-year birth cohort study in Japan of transmission pathways of Helicobacter pylori infection. J Clin Microbiol 2000; 38: 1971-3.
24. Dominici P, Bellentani S, Di Biase AR, et al. Familial clustering of Helicobacter pylori infection: population based study. Br Med J 1999; 319: 537-41.
25. Replogle ML, Glaser SL, Hiatt RA, Parsonnet J. Biological sex as a risk factor for Helicobacter pylori infection in healthy young adults. Am J Epidemiol 1995; 142: 856-63