The Role of *Helicobacter pylori* in Chronic Gastritis

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Abstract

This study aimed to determine the relationship between *H.pylori* and chronic gastritis. It was carried out on 90 patients with chronic gastritis admitted to Hilla surgical teaching hospital and Merjan hospital during the period May/1999 to May/2000, normal controls were represented by 50 normal subjects. Three antral biopsy samples were taken from each subject. The study parameters were the detection of urease production, demonstration of bacteriological culture, and studying the histopathological changes in the gastric mucosa. The results revealed that 79 /90 (87.7%) patients were suffering from *H.pylori* infection associated with marked inflammatory cells and polymorphnuclear cells infiltration in the intraepithelial regions and lamina propria and there is no atrophy of gastric gland and intestinal metaplasia. The control group showed only 12/50 (24%) positive evidence of *H.pylori.*

الخلاصة

هدفت هذه الدراسة إلى تحديد العلاقة بين بكتريا *Helicobacter pylori* والتهاب المعدة المزمن وقد تضمنت 90 مريضا يعانون من هذا الالتهاب من المراجعين إلى مستشفى مرجان خلال الفترة أيار 1999 إلى أيار 2000 بينما تمثلت عينات السيطرة بخمسين شخصا سليما أخذت ثلاث خزع معدية من كل شخص وتضمنت معايير الدراسة (1) فحص إنتاج اليوريز (2) الفحوصات الزرعية البكتيرية (3) دراسة التغيرات النسجية المرضية في الطبقة المخاطية المعدية. أظهرت النتائج إن 90/79(87.7 %)من المرضى كانوا مصابين ببكتريا *Helicobacter pylori* مترافقة مع وجود واضح للخلايا الالتهابية والخلايا متعددة الانوية المترشحة في مناطق داخل النسج ألطلائي والصفيحة الأصيلة مع عدم وجود ضمور في الغدد المعدية أو التحول الخلوي المعوي، بينما لم تظهر بكتريا *Helicobacter pylori* في عينات السيطرة إلا بمعدل 50/12 (24 %)فقط.

Introduction

Several epidemiological studies have revealed that the infection with *H.pylori* is more common in developing countries and among individuals with socioeconomic status [1,2]. Although is contracted in childhood, the prevalence of organism increases with age, cohort effect reflecting a lower standard of living and higher childhood infection rate in previous generation [3,4]. The adult infection and re-infection rate is thought to be about (0.5 –1.0) % per year [5]. The main rout of infection is from person to person either by fecal-oral or oral - oral mean [6]. The proposed sequence for development of gastric adenocarcinoma is well correlated to *H.pylori* infection of gastric mucosa ,likewise *H.pylori* infection is the main environment factor causing active chronic atrophic gastritis with loss of chief cells and parietal cells and making media susceptible to the effect of carcinogens which are generated by an

over –growth of nitrifying bacteria and some other dietary factor including high salts intake and low vitamin C consumption [7,8]. *H.pylori* can cause an acute gastritis immediately after initial infection with the organism [9]. This work aimed to detect the relationship of *H.pylori* and chronic gastritis.

Materials and Methods

This study was carried out in the endoscopy unit of Hilla surgical teaching hospital and Merjan hospital over a period of one year May/1999-May/2000. The samples consisted of 90 patients (56 males and 34 females), those patients were of mean of age 56.5 year. Fifty subjects who were apparently healthy individuals were also studied as normal controls. Tissue biopsies were performed by using endoscopy forceps. Three antral biopsies specimens were taken and subjected to urease test, bacteriological culture and histological studies. The urease test was performed by mixing the gastric antrum mucosal biopsy specimens with test reagent in a test tube and the test was judged to be positive if a color changed from yellow to red within 24 hour [10].

Bacteriological culture was carried out by inoculating blood agar plates by the second biopsies and cultivated under microaerophilic conditions for 24-48 hour at 37 oC [11].

The last sample was fixed in Pouin's solution and stained with haematoxylin and eosin stain. The histological changes and grading of its severity were assessed by two pathologists and according to Sydney system (table-1) [12] . Patients with two positive parameters were considered to be infected with *H.pylori*[13] .

Table 1 Sydney system classification of the severity of inflammation

Cells per field	Degree of severity
>10*	Mild
10-20	Moderate
<20	severe

*scale of Sydney system is (0-2 mild,2-4 moderate , < 4 severe)

Results

The result of this work revealed that *H.pylori* infection was significantly higher in patients with chronic gastritis when compared to that of control group (table 2). The detection rates of *H.pylori* by using of the three different techniques were shown in table (3). It is clearly detected that *H.pylori* was significantly higher by using of urease and histological techniques in comparison to that of bacterial culture technique. Patients infected with *H.pylori* were characterized by marked inflammatory cells and polymorphnuclear cells infiltration in the intraepithelial regions and lamina propria (figure 1) and there was no atrophy of gastric gland and intestinal metaplasia (figure 2- a), on the other hand no histological changes were noted among control group (figure 2- b).

Table 2 Distribution of *H.pylori* infection in patients and controls

subject	**Total number**	***H.pylori* infection**
Controls	50	12 (24 %)
Patients	90	79 (87.7 %)

Table 3 Results of the three parameters using in the demonstration of *H.pylori*

Test	Control		Patients	
	Positive	Negative	Positive	Negative
Urease	17(34%)	33(66%)	84(93.33%)	6(6.7%)
Bacterial Culture	2(4%)	48(96%)	33(36.67%)	57(63.33%)
Histological changes	12(24%)	38(76%)	70(77.77%)	20(22.23%)

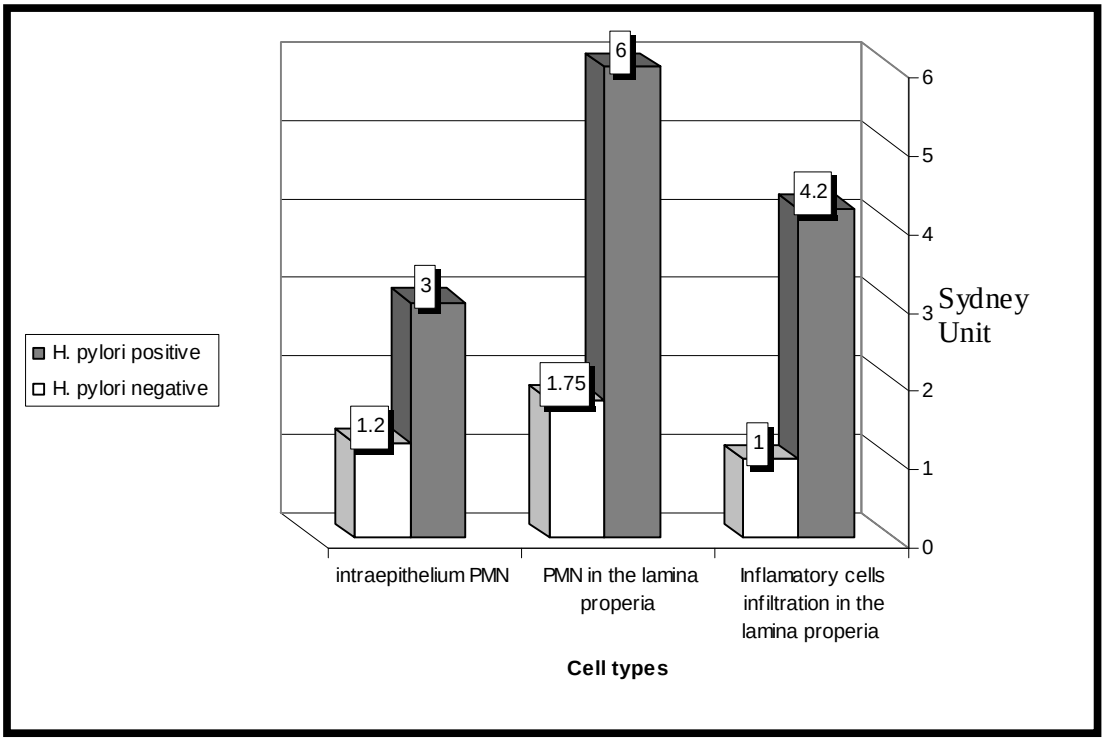

Figure 1 Histological differences between *H. pylori positive* and *H. pylori* negative infection in gastritis patients.

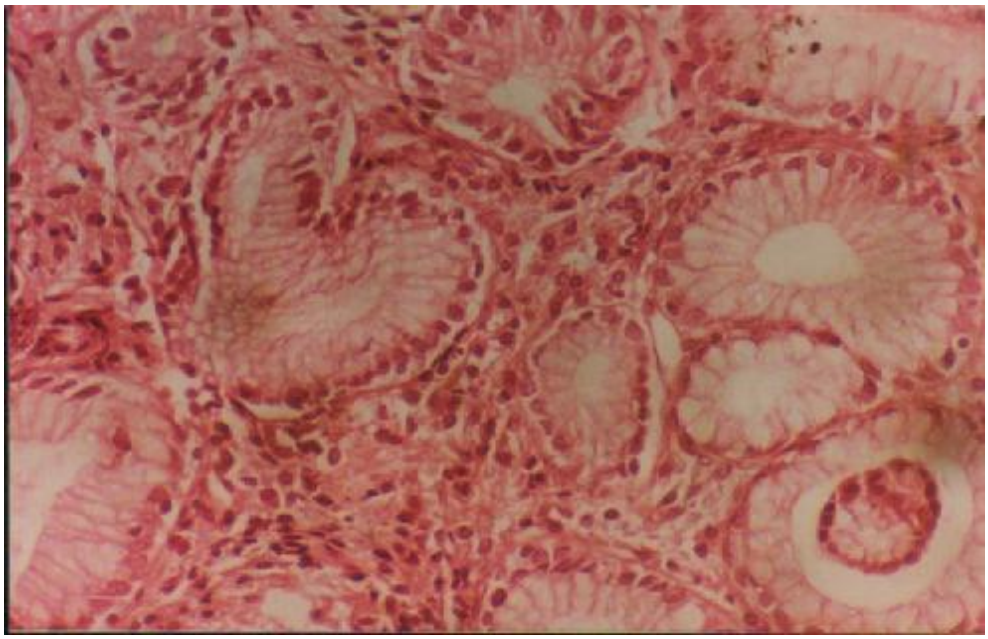

Figure 2-a A photomicrograph of cross section in gastric antrum of gastric patients

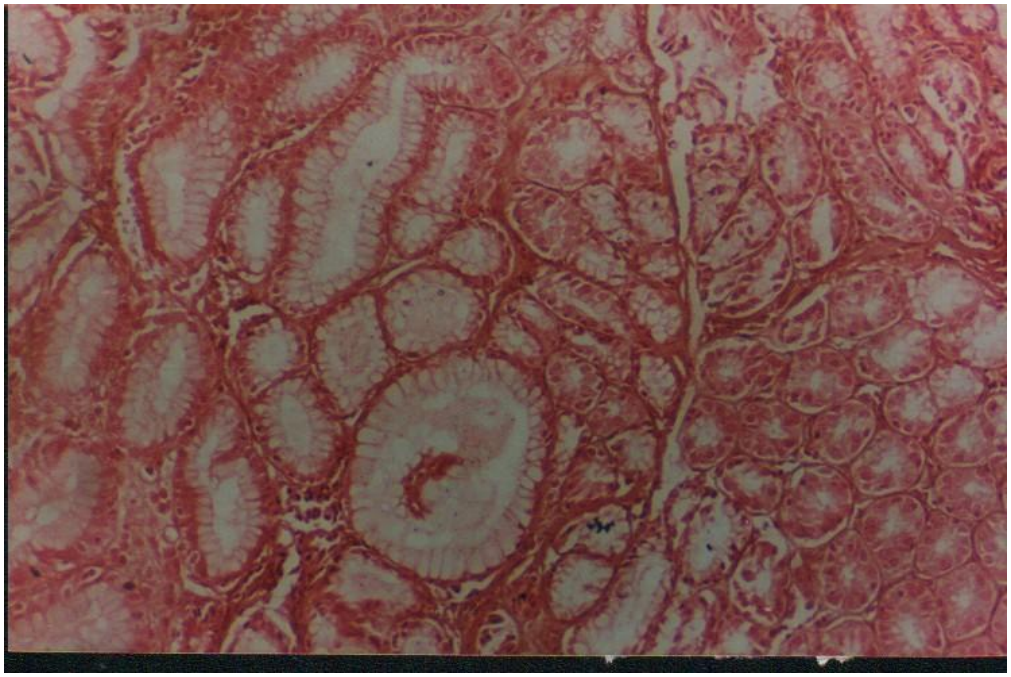

Figure 2-b A photomicrograph of cross section in gastric antrum of control group

Discussion

The results expressed above were agreed to a large extent with other studies that have been focused on the role of *H.pylori* as a causative agent of gastritis .Initial ingestion of *H.pylori* is followed by an acute illness characterized by acute upper gastro-intestine symptomatology ,rapid proliferation of organisms and transient chlorhydria [14] .Many patients infected with less virulent *H.pylori* remains asymptomatic for life .The body develops a weak humoral immune response that fails to eliminate the bacteria from the stomach [15] .In subset of patients ,decades of untreated infection may lead to gastric atrophy ,intestinal metaplasia and ultimately gastric adenocarcinoma [16] .The means by which *H.pylori* causes mucosal inflammation are still under intensive investigation .Most *H.pylori* are free living in the mucus layer overlying the gastric mucosal epithelium .Their survival in the stomach despite peristaltic activity and at low pH must depends on effective rapid replication and ineffective host humoral and cellular immune responses [17] . The inflammation that caused by *H.pylori* is characterized by infiltration of the lamina propria by PMNs ,plasma cells, lymphocytes and monocytes [18] .Several factors influence the ability of this organism to colonize and inflame the gastric mucosa of which the production of urease is the most important [19] .Other virulent factors include vacuolating cytotoxin , lipopolysaccharides of outer membrane, and production of catalase and other enzymes [20] .

The high rate of *H.pylori* detection in this work indicates that this bacterium type play an important role in the development of gastritis among the studied patients.

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