Study Of The Relation Between Helicobacter Pylori Infection And Gastric Interleukin (8) In Patients With Chronic Liver Disease

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Abstract

Helicobacter pylori (H pylori) is a gram negative spirally shaped bacterium. It is known to be the most common important cause of gastritis, peptic ulcer, non ulcer dyspepsia and gastric carcinoma. The frequency and importance of gastric mucosal lesion in patients with chronic liver disease (CLD) have been increasingly recognized in recent years. IL-8 a potent leukocyte chemo–attractant cytokine produced by H pylori. It promotes polymorphnuclear leucocytes (PMNs) and mononuclear cells (MNCs) accumulation in gastric mucosa.This work aimed to clarify the relation between H pylori and IL-8 production in various chronic liver disease (CLD) lesions. Eighty patients were included in this study, 50 with CLD and 30 dyspeptic patients without CLD. Gastric mucosal biopsies were examined histopathologically for H pylori, cellular infiltration and associated pathology, together with culture of H pylori and assessment of IL-8 level in gastric tissue supernatant.

30/50 (60%) CLD patients,10/15 (66.6%) patients with non gastric dyspepsia, and 14/15 (93.3%) patients with gastric dyspepsia were positive for H pylori. There was no relationship between the prevalence of H pylori and the aetiology of CLD.

No significant difference was observed in CLD patients' group as regard to H pylori and Child grading, degree of varices, gastric or liver histopathology. Statistical difference in H pylori prevalence between patients with CLD and those with gastric dyspepsia was significant. IL-8 showed significant increase in H pylori positive vs H pylori negative patients. Positive correlation was found between H pylori density and tissue IL-8 and cellular infiltration. In conclusion the liver status does not play a role in the prevalence of H pylori infection, further studies to investigate the relation between virulent H pylori and IL-8 are needed.

Introduction

H pylori associated gastritis is fundamentally a bacterial infection of the gastroduodenal mucosal surface and as such is characterized by mucosal infiltration of PMNs and MNCs **(Yamaoka, et al., 1998)**. In H pylori infection, the cellular infiltrate contains effectors of the immune response, including CD4+ and. CD8+ T-lymphocytes, B-lymphocytes, plasma cells, monocytes, mast cells and eosinophils **(Dixon, et al., 1996)**.

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Neutrophil activity is an almost universal phenomenon in H pylori gastritis *(Davies, et al., 1994)*. It is a very sensitive indicator of the presence or absence of H pylori and disappears within days of cure of H pylori infection *(Graham, 1994)*. The density of intraepithelial neutrophils has been correlated with the extent of mucosal damage and with the intensity of H pylori infection *(Fiocca et al, 1992, Edit and Stolte, 1994)*. The presence of neutrophil polymorphs in a background of chronic inflammation is a measure of continuing acute inflammation. The normal gastric mucosa contains only individual scattered chronic inflammatory cells (mononuclear cells) in the lamina propria. Thus, any increase indicates chronic gastritis.

Inflammatory cytokines are playing critical roles in inflammation associated with gastrointestinal infections specially H pylori infection of gastric mucosa *(Yamaoka 1998)*. The migration and activation of inflammatory cells into the mucosa is thought to be related to expression of various cytokines *(Crabtree et al., 1993 and Yamaoka et al., 1995, 1996)*. Over the past 15 years a new family of cytokines has been characterized whose members have chemoattractant activity for leukocytes and fibroblast. (chemokines). They appear to play a crucial role in attracting specific types of cells into sites of tissue injury and inflammation *(Daniel 2001).*

Interleukin 8 (IL-8) is secreted by monocytes, macrophages, T. lymphocytes, neutrophils, fibroblast, keratinocyte and hepatocytes. It acts as a chemotactic factor for neutrophils, T lymphocytes subsets and basophils. It activates neutrophils to release lysosomal enzymes and primarily induces neutrophils adherence to endothelial cells *(Martin et al 2002).* A number of studies have investigated IL-8, which is known to be a neutrophil chemotactic factor, and have shown an association between levels of H pylori infection and IL-8 mRNA *(Yamaoka et al., 1995, 1996)* or IL-8 protein *(Crabtree et al., 1993, Yamaoka et al., 1997)*.

IL-8 and other interleukins attract neurtrophils which migrate from the capillaries, through the lamina propria and emerge between the epithelial cells. The most common cause of peptic ulcer may be the migration of these many neutrophils with subsequent release of their digestive products *(Ge and Taylor 1999)*. **Wu et al., (1998)** added that H pylori infection causes almost all uncomplicated duodenal ulcers and more than 80% of gastric ulcers. **Yoshikawa et al., (1999)** found that The Cag A genome in gastric Juice was detected in 98% of patients with peptic ulcer.

In the last few years, a higher prevalence of H pylori infection has been described in patients with liver cirrhosis than in age and sex matched controls *(Siringo, et al., 1997,)*. Further studies, however ,are necessary in order to verify whether the association is causal or occasional. Furthermore, it is unclear whether the association has a clear pathological significance as the available data show no relation between H pylori infection status and the severity of liver disease *(Farinati, et al., 1998)*. However, the fact that infected patients have higher blood concentrations of ammonia and that eradication of the bacterium results in a significant reduction in ammonia levels is interesting *(Miyaji, 1997)*. H pylori urease activity in the stomach of cirrhotic patients has been proposed to represent a significant source of ammonia, contributing to the

development of hepatic encephalopathy *(Attili, et al., 1994, Gubbins, et al., 1993).*

The aim of this work is to clarify the relation between H pylori infection and chemokine (IL-8) in different gastric lesions in patients with different forms of liver disease compared to those with normal liver status.

Subjects, Materials and Methods

This study was conducted on 80 patients admitted to Tropical Medicine Department, Menoufiya University Hospitals in the period from January 1999 to December 2000. They were divided into 50 patients with chronic liver disease (CLD) and 30 dyspeptic control subjects with normal liver status. The diagnosis of CLD was confirmed by a combination of clinical, biochemical, imaging methods and liver biopsy if indicated. The CLD patients were included in the study regardless of the presence or absence of upper gastro-intestinal symptoms. They comprised group I, their mean age was 40.7±9.28 ranging from 21-59 years. They were 38 (76%) males and 12 (24%) females.

Thirty age and sex matched dyspeptic subjects with no evidence of previous liver disease were included in this study as a control group. They comprised group II. They were classified according to their endoscopic findings into: Group IIa: gastric dyspepsia group (abnormal endoscopic findings). Group IIb: non gastric dyspepsia group (normal endoscopic findings). Their mean age was 37±7.12 ranging from 23-60 years. They were 26.(86.7%) males and 4 (13.3%) females.

The severity of CLD in group I was determined and each patient was classified using Pugh's modification of Child's classification (Table 1).

Table 1: Pugh's modification of Child's classification *(Pugh, 1973)*

	Score 1 point	Score 2 point	**Score 3 point**
Serum bilirubin	Below 2	2-3	Over 3
Prolongation of prothrombin time (second)	1-4	4-6	>6
Albumin (gm/dl)	Over 3.5	3-3.5	Under 3
Ascites	None	Slight	Moderate or more
Portosystemic encephalopathy (grades)	None	1 and 2	3and 4

Grade A=5 or 6 points   Grade B=7-9 points   Grade C=10-15 points

All patients underwent eosphago-gastroduodenoscopy where four biopsy specimens were taken from both antrum and corpus, two were used for histological examination, one for H pylori culture and one for IL -8 measurement. The presence of H pylori was determined by culture and

histological examination. Patients were classified as H pylori positive if at least one of the examinations gave a positive result.

Histology

The biopsy specimens were fixed in 10% buffered formalin, embedded in paraffin wax, Sections were stained with haematoxylin and eosin and modified Giemsa stain and were examined for small curved bacilli on the mucosal surface and the density of H pylori was scored based on average density on surface and graded from 0 to 3+; G0: no characteristic bacilli; G1+: occasional curved bacilli; G2+: Scattered bacilli in most high power field; G3+: numerous bacilli in most high power field *(Yamaoka et al., 1998)*.

Also MNCs and PMNs density were evaluated and graded from 0 to 3 using visual analogue scale, according to the Updated Sydney system *(Dixon et al., 1996)*. For analysing the relation between the histological findings and the expression patterns of chemokines, we used the biopsy specimens from a mucosal site adjacent to the site where the biopsy specimens for chemokine measurements were obtained.

H pylori Culture

One gastric biopsy was placed in thioglycolate broth and transferred to the laboratory. The biopsy specimens were immediatly homogenized using tissue homogenizer to ensure equal distribution of tissue fragments. The minced homogenate was cultured on Dent medium, formed of: Colombia Blood agar base with 10% defibrinated sheep blood ii-Helicobacter pylori selective supplement (Dent) (SR 147 E) having the following formula: (Vancomycin 5.0 mg, Trimethoprim

2.5mg, Cefsulodin 2.5mg. Amphotericin B 2.5mg) and incubated at 37C° under microaerophilic conditions for 3-5 days, using the anaerobic jar with a catalyst and the Gas Generating Kit Campylobacter System (Campy Gen™-Oxoid-BR 056A). H Pylori colonies appear as non-haemolytic, tiny, circular, 1 to 2 mm. in diameter, grey translucent, discrete and convex colonies on blood agar plates. Colonies that exhibited characteristics of colonial morphologies were identified as H Pylori by: Gram stain, positive catalase, oxidase and urease reactions *(Cheesbrough M, 2000)*.

IL-8 Measurement

The biopsy specimens were placed in 1.5 ml of phosphate buffered saline (PBS) pH 7.4 and were immediately homogenized using ultrasonic homogenizer. Aliquots of homogenate supernatants were obtained by centrifugation at 10.000 rpm for 10 minutes and were stored at -70ºC until assay for total proteins by a modified Lowry method using commercially available kit (Spinreact - Spain) and for IL-8 by ELISA technique using kits supplied by **(Diaclone - France)** & following the manufacturer instructions. The mucosal level of IL-8 was expressed as pg /mg biopsy protein and data were expressed as median and range.

Statistical Analysis and Data Presentation

Two tailed students' t and χ^2 tests were used to compare different parameters. Correlations were determined using (r) coefficient of variation. Probability (p) values of less than or equal to 0.05 were considered significant.

Results

The presence of H pylori was determined by culture and histopathology. Patients were classified as H pylori positive if at least one of the examinations gave a positive result (Fig 1 a & b). It was 54/80 (67.5%) where 30/50 (60%) CLD patients, and 24/30 (80%) control group were H pylori positive. H pylori positivity showed no significant difference between both groups as regard to sex (P>0.05) while age distribution showed a significant high positivity of H pylori in older than middle aged patients of both groups (P<0.01) (Table 2).

The main complaints of H pylori positive CLD patients were epigastric pain and sense of fullness. While H pylori positive subjects in control group were mainly suffering from epigastric pain and heartburn. No significant difference between H pylori positive patients with and without CLD as regard to symptoatology could be detected (P>0.05).

Endoscopic findings studied in both groups showed that in GI there were 12 (24%) with normal endoscopic findings, while 38 (76%) showed overlapping findings in form of gastritis 4 (8%), deudenitis 7 (14%), gastric ulcer 9 (18%), duodenal ulcer 6 (12%), oesphageal varices 25 (50%), lower end oesphagitis 13 (26%), reflux oesphagitis 2 (4%), and gastric mass one (2%) while in GII there were 6 (20%) with gastritis, 3 (10%) with deudenitis, 3 (10%) with gastric ulcer, 3 (10%) with duodenal ulcer, 2 (6.6%) with lower end oesphagitis , no one showed gastric mass and one only (3.3%) with reflux oesphagitis , those comprised group IIa (Gastric dyspepsia group). While there were 15 (50%) with normal endoscopic findings. They were considered non gastric dyspeptic group (group IIb) (Table 3).

Histopathological grading of H pylori positivity in different studied groups showed that H pylori positivity is highly significant in GIIa (gastric dyspepsia group) while moderate increase was detected in GI and GIIb (Table 4). A high significant difference could be detected as regard to PMNs & MNCs infilteration in gastric tissue, where they were increased in GIIa P<0.01 (Table 5).

Looking for the aetiology of liver disease, clinical, laboratory and histopathological findings showed that 17 (34%) had schistosomiasis, 9 patients (18%) had cirrhosis. 12 (24%) had chronic hepatitis and lastly 12 (24%) showed mixed picture. No significant difference could be detected between H pylori positive and H pylori negative patients as regard to liver disease aetiology P> 0.05 (Table 6). Statistical analysis of H pylori positivity with the severity of liver disease in CLD patients following Pough modification of Child classification, showed no statistical difference in H pylori positivity and different grades of liver affection P>0.05 (Table 7).

Studying the relation between H pylori positivity and degree of oesophageal varices in CLD patients showed no significant difference (P>0.05).

Gastric histopathological findings in group I showed that there were 8 (16%) with normal mucosa, 25 (50%) with chronic atrophic gastritis, 9 (18%) with atrophic dysplasia, 7 (14%) with reactive gastritis while only one patient (2%) with gastric carcinoma. When the positivity of Hp was statistically analysed in relation to gastric histopathology no significant difference could be detected P>0.05 (Figure 2). As expected relation between H pylori positivity and PMNs & MNCs in CLD

patients was highly significant (Table 8).

IL-8 showed significant difference between GI and GIIa P< 0.01 (Table 9). And it showed a significant increase in H pylori positive vs H pylori negative patients in G I&IIa (Table 10). A significant positive correlation was found between H pylori positivity, PMNs, MNCs and IL-8 level in CLD patients P< 0.05 (Figur 3 a,b,c). On the other hand, no significant relation could be detected between mucosal IL-8 level and gastric histopathology or endoscopic findings in CLD patients P> 0.05 except for, significant increase in patients with deudenal ulcer p< 0.05.

Table (2): Statistical analysis of demographic data in relation to H pylori in the studied patients

Sex	GI					GII				
	HP+	HP-	Total	X^2	P	HP+	HP-	Total	X^2	P
Male	26	12	38	3.28	>0.05	21	5	26	0.07	>0.05
Female	4	8	12			3	1	4		
Age										
20 -40	9	14	23	7.73	<0.01	7	4	11	2.91	<0.01
40 -60	21	6	27			17	2	19		
Total	30	20	50			24	6	30		

Table (3): Endoscopic findings in the studied patients.

Endoscopic Findings	GI No=50		GII No=30	
	No	%	No	%
Normal findings	12	24	15	50
Gastritis	4	8	6	20
Deudenitis	7	14	3	10
Gastric ulcer	9	18	3	10
Deodenal ulcer	6	12	3	10
Oesphageal varices	25	50	--	--
Lower end oesphagitis	13	26	2	6.6
Reflux	2	4	1	3.3
Mass	1	2	--	--

Table (4): Statistical analysis of histopathological grading of H pylori postivity in the studied patients.

Hp Grading		GI	GIIa	GIIb	Total	X2	P
Hp	-ve	20	1	5	26	23.47	<0.01*
	+	1	0	5	6		
	++	22	9	5	36		
	+++	7	5	0	12		
Total		50	15	15	80		

* Highly significant

Table (5): Statistical analysis of gastric cellular infiltration in the studied patients

MNL Grading		GI	GIIa	GIIb	Total	X^2	P
MNL	0	7	0	1	8	19.69	<0.01
	1	21	1	7	29		
	2	18	7	6	31		
	3	4	7	1	12		
PMN	0	10	0	1	11	13.21	<0.01
	1	14	0	5	19		
	2	18	10	7	35		
	3	8	5	2	15		
Total		50	15	15	80		

Table (6): Statistical analysis of H pylori postivity and aetiology of liver diseases in CLD patients

Cause of disease	H p positivity		Total	X^2	P
	H p+	H p-			
Schistosomiasis	11	6	17	0.06	>0.05
Cirrhosis	6	3	9	0.2	>0.05
Mixed	8	4	12	0.29	>0.05
Chronic hepatitis	5	7	12	1.4	>0.05
Total	30	20	50		

Table (7): Statistical analysis of H pylori postivity and severity of liver affection in CLD patients

Child grade	H p postivity		Total	X^2	P
	H p+	H p-			
Child A	17	13	30	0.35	>0.05
Child B	9	5	14	0.15	>0.05
Child C	4	2	6	0.13	>0.05
Total	30	20	50		

Table (8): Statistical analysis of gastric cellular infiltration and H pylori positivity in CLD patients

Cells		H p				Total	X^2	P
		-ve	+	++	+++			
PMNs	0	10	0	0	0	10	47.77	<0.01
	1	8	1	5	0	14		
	2	1	0	15	2	18		
	3	1	0	2	5	8		
MNCs	0	7	0	0	0	7	56.62	<0.01
	1	13	1	7	0	21		
	2	0	0	15	3	18		
	3	0	0	0	4	4		
Total		20	1	22	7	50		

Table (9): Statistical analysis of gastric IL-8 in the studied groups

IL8 (pg/mg)	N	Mean	SD	t.	P value
GI	50	70.994	18.448	- 4.168	P* <0.01
GII a	15	103.333	43.985		
GII b	15	70.713	11.798	0.055	P >0.05

Table (10): Statistical analysis of gastric IL-8 level and H pylori positivity in the studied patients

| Group	H p(+)	H p (-)	P value
GI	80.3 ± 7.9	62.2 ± 10.2	< 0.05
GII a	114 ± 24.3	85 ± 18.1	< 0.01
GII b	74.6 ± 10	68.5 ± 9.2	> 0.05
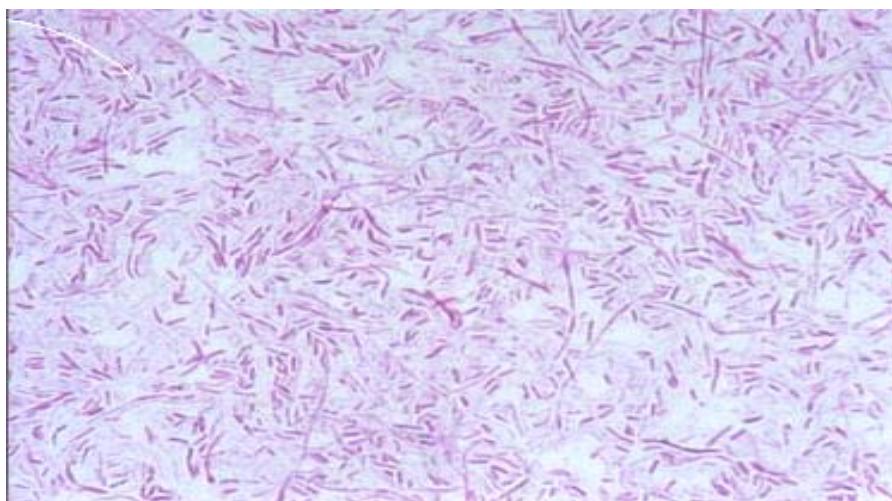
Figure (1a): H pylori organisms in Gram stain under light microscopy (oil immersion lens X 100)
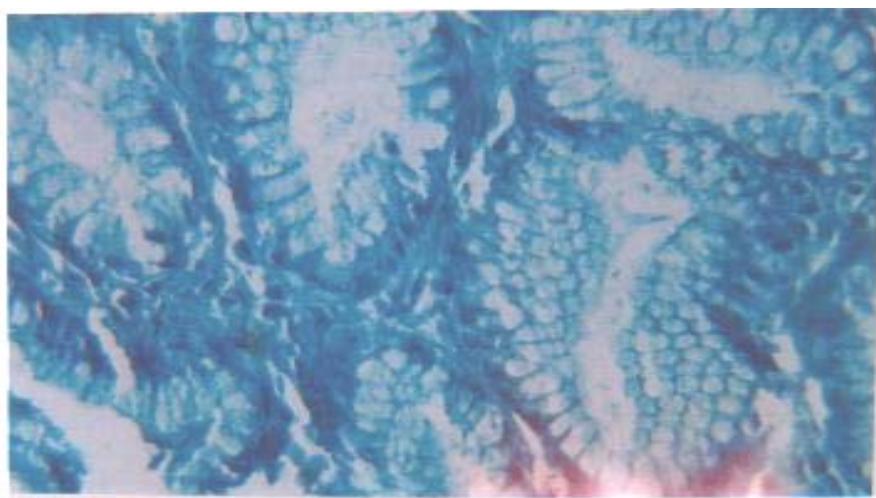
Figure (1b): Few number of H pylori organisms inside the Lamina in some gastric tubules (modified Giemsa stain x400)
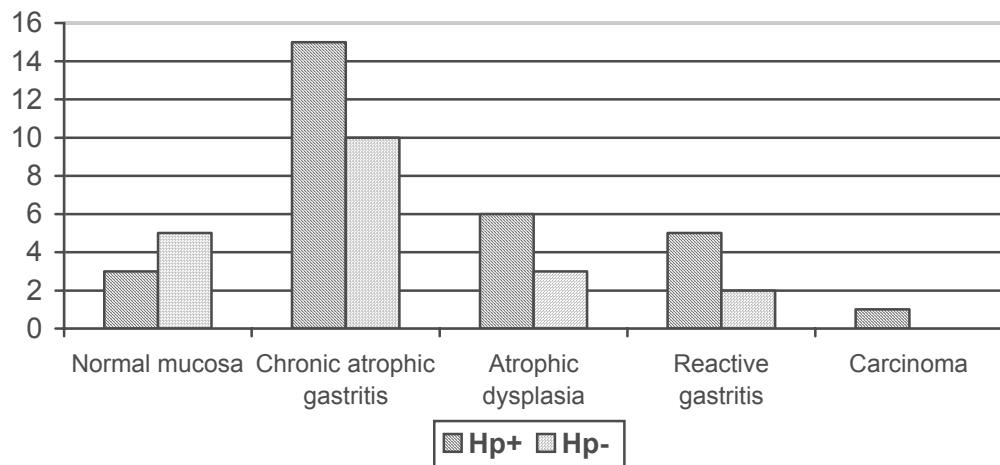
Figure (2): Relation between H pylori positivity and gastric histopathology in CLD patients
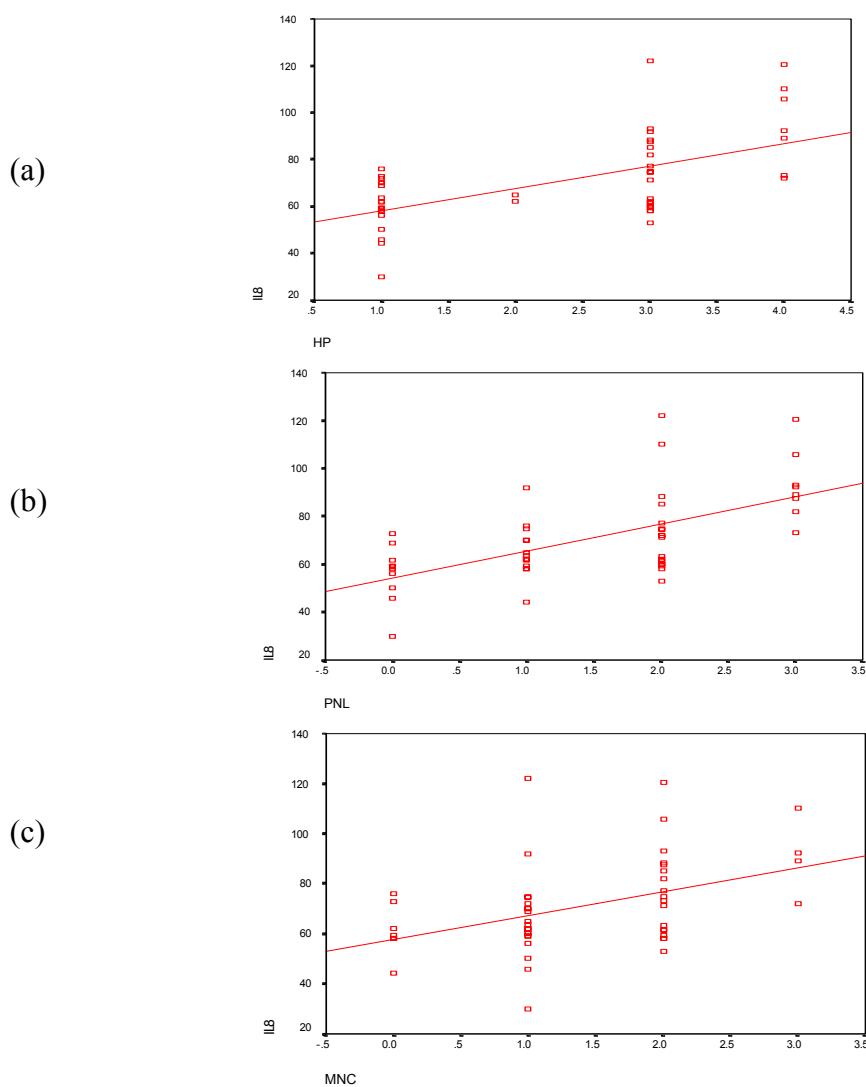

Figure (3): Correlation between H pylori positivity (a) PMNs (b) and MNCs (c) and IL-8
in CLD patien

Discussion

H pylori is a very common pathogen, infecting approximately half the world's population *(Pisani et al., 1997)*. The epidemiology of H. pylori infection differs between the developed and developing worlds. In industrialized countries, infection is acquired at a fairly constant rate of 0.5-2%/ year, reaching a prevalence of 20-40% in the adult population *(Parsonnet, 1995)*. However, in developing countries, H pylori is acquired mainly during early childhood at a very fast rate. By the age of 20 years, 70-90% of the population already infected *(Hopkins et al., 1993).* In the present study, H pylori was detected in 67.5% of the studied patients This was in accordance to previous Egyptian reports suggesting a prevalence of 60-90% of H pylori in adults *(Mounir et al., 2000, Bassily et al., 1999, Omar et al., 1997 and El Zayadi et al., 1990)*.

In the present study, H pylori was less frequent in CLD patients than in control group as its prevalence in CLD was 60% while in non gastric dyspeptic subjects it was 66.6% with no significant difference between both groups. It was only significant when comparing CLD patients with gastric dyspeptic subjects p< 0.05. **Balzano et al., (1991),** reported that the prevalence of H pylori in CLD patients was lower than normal population and suggested that the gastric mucosa of patients with portal hypertension may be inhospitable environment for H. pylori. **Farianti et al., (1998)** added that hypertensive gastropathy might not represent a favourable environment for H pylori thus making the diagnostic sensitivity of the biopsy lower than expected. However, **Calvet et al., (1997),** reported that 105 out of 209 cirrhotic patients (50.2%) had positive H pylori IgG and concluded that H pylori

infection in cirrhosis had the same epidemiological pattern as in the general population in Spain. Also, in Egypt, **Omar et al., (1997),** concluded that there was no relation between H pylori infection and chronic liver disease. On the other hand **Ponzetto et al., (2000),** found that the prevalence of H pylori infection in CLD patients was significantly higher than in age-matched male blood donors and the investigators suggested that H pylori may be implicated in the pathogenesis and prognosis of cirrhosis in CLD individuals. Also **Siringo et al., (1997),** suggested that this is due to recurrent admission to hospital and endoscopy which were well-known risk factors for spread of H pylori infection.

In the present study, there was no significant difference between males and females as regard H pylori infection either in CLD or control group. The same results were obtained by **Calvet et al., (1997), Schmulson et al, (1997) and Omar et al., (1997),** while **Siringo** and his colleagues, **(1997)** reported a significant increase of H pylori infection in male cirrhotic patients.

In this study, the prevalence of H pylori infection increases with age in both studied groups, This finding agreed with the result of **Vaira et al., (1997).** The increase in frequency of infection in older patients might be due to cumulative risk for infection **(Dubois, 1995)**. On the other hand, **Tsai (1998) Omar et al., (1997)** and **El Ansary (1994)** found no difference in H pylori prevalence between different age groups.

Analysis of dyspeptic symptoms in this study revealed no significant difference between all groups. Also, there were no specific symptoms for H pylori infection and these was in accordance to **Graham et al., (1992)**

who suggested that 20-50% of patients with positive H pylori showed dyspeptic symptoms.

In the present study, there was no significant difference between the prevalence of H pylori infection and aetiology of liver disease. This was in accordance to **Tsai (1998)** and **Siringo et al., (1997)** On the other hand, **Abd El Hamid et al., (1995**) found that, correlating H pylori status with the aetiology of liver disease revealed that patients with mixed schistosomiasis and cirrhosis had higher prevalence of H pylori compared with chronic active hepatitis patients.

In the present work, it is evident that chronic atrophic gastritis (50%) was the predominant gastric histopathology in CLD with H pylori infection. This was in accordance to **(Negrini et al., 1996;** and **Edit** and **Stolte, 1994)**. Despite the fact that H pylori is known to be non invasive, mucosal infiltration of inflammatory cells have been observed in gastric mucosa. This study showed that PMNs and MNCs infiltration was significantly high with increased H pylori density. This result was also supported by **Kim et al., (1998)** who stated that a significant correlation was observed between H pylori density and histological severity.

In the assessment of whether the positivity of H pylori varied with increasing severity of CLD. According to Child-Pugh classification, it was found that there was no significant difference in H pylori positivity in cirrhotic patients with different degrees of severity The same finding was reported by **Tsai , (1998), Siringo et al., (1997)** and **Wu et al., (1995),** who found that the prevalence of H pylori was similar in compensated and decompensated cirrhotic patients, but was not agreement with **Naumovski-**

Mihalie et al., (2000), who reported that H pylori infection was higher in Child's class C group. On the other side, **Schmulson et al., (1997),** found that the prevalence of H pylori was inversely related to the Child-Pugh grading. Relating H pylori prevalence to the oesophageal varices grades revealed no significant relation, these data were supported by **Mostafa et al., (1993) Shaheen (1993) and Mc Cormick et al., (1991),**

Cytokines are suspected to play a crucial role in pathogenesis of H pylori associated gastric disease, **Takahashi et al., (1998).**

In the present study, gastric IL-8 level was significantly higher in patients with gastric dyspepsia than patients with CLD or those subjects with non gastric dyspepsia. Mucosal level of IL-8 in gastric tissue supernatants in subjects with H pylori infection either with or without CLD was significantly higher in comparison with H pylori negative patients. The same result was also obtained by **Daniel et al., (2001),** who added that exposure of gastric epithelial cells to H pylori infection stimulate the induction of IL-8 secretion as an inflammatory mediator. These findings were consistent with the possibility that IL – 8 is playing an important roles in H pylori associated gastric inflammation **(Yamaoka et., al 1998)**.It is of interest that the mucosal level of IL 8 correlated significantly with H pylori density as well the cellular infiltration. **Ando et al., (1996)** stated that H pylori density may be one of the pathogenic factors causing infiltration of inflammatory cells through elevated IL 8 secretion in gastroduodenal diseases. According to **Harris et al.,(1996)**H pylori urease and other proteins are capable of activating mononuclear phagocytes and stimul - ating them to produce many cytokines

including IL 8,at the level of gene transcription.

In this study there was a significant correlation between IL-8, MNCs and PMNs infiltration in gastric mucosa suggesting that IL-8 causes severe inflammation. This was also supported by **(Yamaoka 1998, Gad allah 2001),** They stated that IL-8 produced by H pylori induce migration to PMNs and MNCs and then subjected to activation by locally secreted IL-8 yielding degranutation and respiratory burst that may be involved in mucosal injury in the stomach.

The present study showed no significant difference in IL-8 level as regard to gastric histopathology in CLD group. This agrees with **Yamaoka et al., (1998)** as they stated that H pylori infection increases expression and production of chemokine IL-8 irrespective of gastric pathology.

Also it was found that mucosal level of IL-8 was significantly higher in patients with deudenal ulcer compared with other endoscopic findings in CLD group. This was in accordance to **Yamaoka et al., (1998)**. H pylori damages the mucosa both directly through the urease catalyzed synthesis of ammonia and production of cytotoxins and bacterial enzymes that deplete mucus and damage epithelial cells, and indirectly through the stimulation of local and systemic immune responses *(Dixon, et al., 1996)*.

In conclusion, it appeared from the results of this study that the aetiology or the severity of liver disease could not be related to H pylori infection. The liver status does not play a role in the prevalence of H pylori. IL-8 showed significant increase in H pylori positive vs H pylori negative patients and positive correlation was found between its gastric level and H pylori density and cellular infiltration.

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دراسة العلاقة بين الإصابة بالميكروب الحلزونى ومادة الانترلوكين (8) فى معدة المرضى المصابين بأمراض الكبد المزمنة

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تزايد الاهتمام بالميكروب الحلزونى خلال الأعوام القليلة الماضية ويرجع ذلك إلى توحد العلاقة بينها وبين أمراض الجهاز الهضمى وخاصة التهاب المعدة وقرحة الاثنى عشر وسرطان المعدة.

ولقد اختلف العلماء فى أن مرضى الالتهابات المعدية المزمنة المصاحبة بارتفاع ضغط الدم بالدورة البابية هم معرضون لوجود وتكاثر الميكروب الحلزونى بنسبة اقل أو اكثر من المرضى الغير مصابين بأمراض الكبد المزمنة. ولقد عنيت هذه الدراسة بتوضيح العلاقة بين الإصابة بالميكروب الحلزونى المعدى وبين إفراز مادة الإنترلوكين (8) فى مرضى الكبد المزمن.

تشتمل هذه الدراسة على (80) مريض حيث تم تقسيمهم إلى: مجموعة المرضى المصابين بأمراض الكبد المزمنة (50) مريضا. ومجموعة المرضى الغير مصابين بأمراض الكبد المزمنة

(30) مريضا مجموعة عسر الهضم (المجموعة الضابطة). وقد تم تقسيمهم عن طريق المنظار إلى مرضى عسر الهضم ناتج عن المعدة ومرضى عسر الهضم ناتج عن أسباب أخرى. وقد تم اخذ التاريخ المرضى لجميع المرضى مع فحص اكلينيكى كامل بالإضافة إلى: اخذ عينات كبد من المجموعة الأولى لتحديد النوع المرضى المزمن للكبد.ومنظار علوى على المعدة والمرئ مع اخذ عينات من الغشاء المخاطى المبطن للمعدة لفحص التغيرات المرضية فى الأنسجة والبحث عن البكتريا وقياس نسبة الإنترلوكين (8). وقد أثبتت الدراسة ارتفاع نسبه المصابين بالميكروب الحلزونى مع زيادة السن وكان الفارق ذو دلاله إحصائية. ولم توجد أى دلاله إحصائية بين المصابين بالميكروب الحلزونى بالنسبة لسبب المرض الكبدى أو شدته أو عند تحليل الخلايا المعدية أو درجه وجود دوالى المرىء. وثبت تقارب نسبة الإصابة بالميكروب الحلزونى بين مجموعة المرضى المصابين بأمراض الكبد المزمنة ومجموعة مرضى عسر الهضم غير المعدى وارتقاعها فى مجموعة عسر الهضم المعدى. كما لوحظ ارتفاع نسبة الإنترلوكين (8) فى حالات الإصابة بالميكروب الحلزونى فى المجموعات الثلاثة وزيادة انتشار خلايا الالتهاب فى خلايا النسيج المعدى ولوحظ وجود علاقة وثيقة بين كثافة الإصابة بالميكروب الحلزونى وارتفاع نسبة الإنترلوكين (8) وخلايا الالتهاب فى الأنسجة المعدية. كما لوحظ ارتفاع نسبة الإنترلوكين (8) فى حالات الإصابة بالميكروب الحلزونى فى حالات قرحه الإثنى عشر بين مجموعة المرضى المصابين بأمراض الكبد المزمنة