

The Prevalence of *Helicobacter Pylori* In Upper Gastrointestinal Disorders



Fuad A. Baban and Mohammad O. Mohammad

College of Medicine, University of Sulaimani, Kurdistan Region/Iraq.

Abstract

Background: *Helicobacter pylori* are a gram negative spiral bacilli that cause one of the most chronic infection through out the world. *Helicobacter pylori* infection is associated with many important and serious gastrointestinal diseases like peptic ulcer, chronic gastritis, a known precursor of gastric carcinoma.

Method: we studied the prevalence of *Helicobacter pylori* infection in a group of patients who presented with various upper G.I discomfort between marches 1997 –November 1999. 400 patients were enrolled, they were examined clinically and by upper G.I endoscopy and 2 piece biopsy were taken from the antrum for rapid urease test for detection of *Helicobacter pylori* infection.

Results: *Helicobacter pylori* infection is a common infection and for any age group the incidence of infection was more than 60% , pain was the most common presenting symptom (93%) , patient harboring ulcer were 88% positive for urease (89% in DU , 86% in GU), 72% of patients with endoscopic findings other than ulcer were urease positive and 70% of patients with gastric carcinoma were positive while 64% of patients with negative endoscopy(non ulcer dyspepsia) were urease positive.

Conclusion: infection with *Helicobacter pylori* is common in this community and is strongly associated with an increase risk of peptic ulcer ,gastritis and gastric carcinoma .However many dyspeptic patients with *Helicobacter pylori* infection have no endoscopic abnormality (Functional, non ulcer dyspepsia) . We found that urease test is a rapid , cheap and practical test for the detection of *Helicobacter pylori* infection .

Keywords: *Helicobacter pylori*, urease test, peptic ulcer

Introduction

Helicobacter pylorus is a new organism for an old problem. It is first reported by Bizzozero in the stomach of dog in 1893[1] A comprehensive autopsy study by Doeuges showed a prevalent of 43% for spiral organism in the human stomach, but he was unable to find a relationship between the presence of organism and various gastric diseases [2].

In 1983 Warren and Marshall from Australia were able to isolate the curved organism from human stomach and to culture it and was first called *Campylobacter* like organisms (CLO) [3].

In 1989 the decision was taken by international agreement that this organism on the bases of bacteriological and ultra structure represents an entirely new genus,

hence called *Helicobacter pylori* [4,5].

Helicobacter pylorus a short, spiral , gram negative bacillus measure 0.2-0.5 micron in width with multiple (4-6) sheathed unipolar flagellae which prefers micro aerophilic environment, it reside in the mucus gel coating the epithelial cell of stomach and gastric mucosa other than stomach [5].

Helicobacter pylori is worldwide in distribution, it is one of the commonest and probably most chronic infection throughout the world[6].

Infection is acquired during childhood and increase with age, there is higher frequency of *Helicobacter pylori* infection in the low socioeconomic status and family members of infected individuals and spread occur via

person to person transmission[7-9]. *Helicobacter pylori* have a key role in the development of many important and serious disease of gastrointestinal tract from non-autoimmune gastritis through gastric and duodenal ulcer to gastric cancer and gastric lymphoma [10,11,12].

Helicobacter pylori are capable to produce many enzymes like catalyses, phospholipase, protease and urease [4,13]. There are many methods for diagnosing the presence of *Helicobacter pylori*, some of these tests are invasive and need biopsy to perform Gram's stain, culture or RAPID UREASE TEST (RUT) and histological examination and touch cytology (Imprint) [14].

There are several non-invasive methods for diagnosing *Helicobacter pylori* like C-urea breath test, radioactive C [14]-urea, ELISA [15,16].

The aim of this study is to determine the incidence of *Helicobacter pylori* in Sulaimani province in patients with various upper gastrointestinal disorders using Rapid Urease Test (RUT).

Patients and Methods

This is a prospective study conducted at Sulaimani general teaching hospital between March 1997 to Nov. 1999. Four hundred patients were studied consequentially using special forum questionnaire including age, chief complain and duration , aggravating and relieving factors , smoking ,alcohol , diet , drug history ...

All patients underwent complete upper GI endoscopy .Two biopsies from antrum were taken and they were crushed by sterile needle then put in Urea solution [17] .The solution observed for any change in color. Change of color from yellow to pink indicates the presence of *Helicobacter pylori*.

If the solution remained yellow for 24 hours, it was regarded as negative result.

Patient who received antibiotic for at least 3 days for the last four weeks and patients below 12 years of age were excluded from the study.

Results

Four hundred patients with various gastrointestinal discomforts were included in the study, 59% were male and 41 % were female.

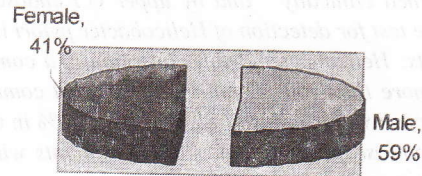
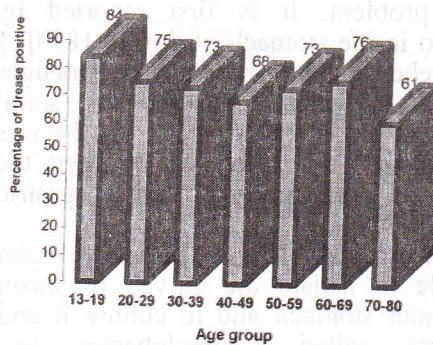


Fig.1 Gender Distribution

Urease test was positive in (84 %) in age group 13-19 years and (61%) in 70-80 years a



The main presenting symptoms was pain (93%) , Epigastric pain were the most common site (88%) hunger pain were found in 56% and nocturnal pain in60% , relapse were present in 63 % of patients . (Tab. 1).

Table 1. Presenting symptoms

SYMPTOMS	NUMBERS AND PERCENTAGE OF THE PATIENTS		MALE %	FEMALE %
	NUMBER	%		
Pain	368	93	57	43
Epigastric	354	88.5	51	38
Finger	228	57	35	22
Diffuse	147	37	45	55
Radiation	185	46		
Back	82	44	46	54
Rt. Hypoch.	16	9		
Chest	6	3		
Lt. Hypoch.	3	1.5		
Umbilical	2	1		
Hunger	225	56	59	41
Nocturnal	241	60	56	44
Relapse	247	62	57	43
Heart Burn	179	45	59	41
Nausea & Vomiting	136	34	53	47
Seasonal	88	22	68	32
Haem. & Melaena	16	4	57	43

Diet was the most common aggravating factor (59%), while psychological stress and smoking were related to the symptoms in 48% and 30 % respectively (Tab.2).

**Table 2
The Aggravating factors**

Aggravating factors (positive history)	Numbers and percentage of the patients		Male %	Female %
	Number	%		
Diet	237	59	60	40
Psyche. Stress	192	48	53	47
Smoking	121	30	87	13
Alcohol	23	5.7	96	4

Urease test was positive in 88% of patients with ulcer (89% in duodenal ulcer and 86% in gastric ulcer) .Patients with functional (non ulcer dyspepsia were 64%). (Tab.3 and 4).

**Table 3
Edoscopic finding and urease test**

Endoscopic findings	Number	Urease positive	%
Ulcer	102	90	88
Other than ulcer	148	107	72
Negative (NUD)	185	120	64

Table 4
Patients with Ulcer and Urease reaction

Ulcer	Number of positive urease	%	Male %	Female %
Duode	71/80	89	68	32
Gastric	19/22	86	74	26
Total	90/102	88	69	31

Smoking was more related to gastric ulcer (54%) while it was 37.5% in duodenal ulcer. (Tab.5).

Table 5
Relation of ulcer to smoking

Ulcer	Number of smokers	%
Duodenal	30/80	37.5
Gastric	12/22	54.5
Total	42/102	41

We found that duodenal ulcer were four times more common than gastric ulcer (Tab.6).

Table 6
The Patients with ulcer

Ulcer	Number and percentage of patients		Male %	Female%
	No.	%		
Duoden	80	20	67.5	32.5
Gastric	22	5.5	73	27
Total	102	25.5	69	31

Non ulcer dyspepsia NUD defined as chronic or recurrent pain or discomfort centered in the upper abdomen were endoscopy fails to identify a definite structural cause [18]. The urease test was positive in 80% of patients with duodenitis, 74 % in gastritis , 67 % in esophagitis and 70% in patients with carcinoma of stomach .(Tab.7)

Table 7
Patients with endoscopic findings other than ulcer with positive urease reaction

NUD	Patients (No.)	urease positive (No.)	%
Duodenitis	26	21	80
Gastritis	62	42	74
Esophagitis	15	10	67
Duodenitis & gastritis	3	3	100
Duodenitis & Esophagitis	2	2	100
Carcinoma	13	8	61
Stomach	10	7	70
Esophagus	3	1	33
Erosion	8	8	100
Polyp(G&D)	6	5	83
Total	148	107	72

Discussion

Helicobacter pylorus produces a variety of protein which appears to mediate its damaging effects on the gastric mucosa. The urease produced by Helicobacter pylori catalyses the hydrolysis of urea to yield ammonia and CO₂. This provides a more alkaline microenvironment that protects Helicobacter pylori from the effects of gastric acid, which prevents gastric colonization by other bacteria. The urease results in damage to gastric mucosa by the hydroxide ions generated by equilibration of water with ammonia. Helicobacter pylori produce surface protein

that are chemotactic for human neutrophil and monocyte and activate monocyte to produce super oxides, interleukin-1, and Tumor necrosis factor α . *Helicobacter pylori* are also capable of producing potentially toxic enzymes such as urease, mucinase, lipase, phospholipase. Approximately 50 % to 60% of *Helicobacter pylori* isolates produce a toxin that induces non lethal vacuolation in a variety of cell lines, this toxin is known as vacuolating cytotoxin.

Helicobacter pylori reduce somatostatin secretion which is a potent inhibitor of many GI secretory function which results in increased gastrin which cause increased acid secretion and duodenal ulceration [13,17,20,21,22,23].

We found a strong association between *Helicobacter pylori* and various GI lesions. 89% of our patients with duodenal ulceration were *Helicobacter pylori* positive by urease test, which is comparable with other studies, Richard G long Et al. reported 90% [19] and Zuhair A.Kassire Et al. 1994 reported 88.3 % [17] and Stephen Patchet in 1996 reported more than 95% [24].

Zuhair A.K. Et al.1994 reported the presence of bacteria in 60% of patients with gastric ulcer [17] While our study shows higher association of about 86% which is similar to what was reported by other studies [19,24,25,26] like Marshal B.J. study 1987 demonstrate the presence of bacteria in 70% of patients with gastric ulcer [25], Richard G long, Brain T Cooper in 1998 reported 75% [19] and Stephen Patchet in 1996 report 90% [24].

This can be explained by better exclusion criteria and excluding patients who receive

antibiotics for 7 days in the last 4 weeks. Also we found that smoking was related to dyspeptic symptoms in about 30% of patients, it was about six times more in male. 41% of ulcer patients were smokers, this evidence support the significance of smoking in the pathogenesis of peptic ulcer.

The prevalence of *Helicobacter pylori* in patients with endoscopic finding other than ulcer and patients with gastric carcinoma was 72% and 70% respectively. Patients with non ulcer dyspepsia were only 64% urease positive which is comparable with Al Kassir study (63%) [17], While Hermens DS [27] reported 24 % and George A [28] report 82%. This wide variation may be explained by various geographic distributions and the type of population studied and the method used for detection of *Helicobacter pylori* infection.

Conclusion

Helicobacter pylori are the main cause of duodenal and gastric ulcer .It is likely that *Helicobacter pylori* also play a pivotal role in non ulcer dyspepsia and gastric cancer[29]. Rapid urease test (RUT) is a simple cheap test to identify *Helicobacter pylori* infection [30].

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References

- [1]John G. Barllet, *Campylobacter pylori; fact or fancy* Gastroenterology 1988, **94**. 229-238.
- [2]Cornelius P. Dooly. Hartly Cohen. Annals of Internal Medicine 1988,**108**. 70-79.
- [3]Marshall, B.J Warren, J.R. *unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration*; Lancet 1984, 1311-1315
- [4]Richard V. Heathly. The *Helicobacter pylori* hand book 1995, Ch. 1 page 2.
- [5] Godwin CS, Armstrong JA, Chilvers T et al. *Transfer of Campylobacter pylori and Campylobacter mustelae to Helicobacter gen. nov. As Helicobacter pylori Comb nov.* Int J Syst. Bacteriol 1989, **39**; 397-405.
- [6]Blecker U, Lanciers S, Mahta D, et al *Familial cluster of Helicobacter pylori infection.* Clin Pediatr . 1994, **33**: 307- 308.

- [7] Malaty HM, Graham DY, Klein PD et al; *transmission of Helicobacter pylori infection, studies in families of healthy Individuals*. Scand J. gastroenterol 1991, **26**, 927-932.
- [8] Drumm B, Perez -Perez GI, Blaser MJ, et al *Intra-familial clustering of Helicobacter pylori infection*. N Engl. J Med. 1990, **322**, 359-363
- [9] Graham DY, Malaty HM, Evan DG et al -economic status; *Epidemiology of Helicobacter pylori in an asymptomatic population in united state, effect of age race and socio*. Gastroenterology 1991, **100**, 1495-1501.
- [10] Monitor weekly, *new focus*, 1994, **13**, 15-16.
- Shao Lin, John L, Mark S et al *intra-family transmission of Helicobacter pylori infection*.
- [11] International J. Gastroenterol 1997.12-15.
- [12] M Burstein, E Monge, R Leon et al. *Low peptic ulcer and high gastric cancer prevalence in a developing country with high prevalence of infection by Helicobacter pylori*. J.Clin. gastroenterol 1991; **13**(2): 154-156.
- [13] Dixon MF *Pathophysiology of Helicobacter pylori infection*. Scand. J. Gastroenterol 1994, **29**(Supp 201) 7-10.
- [14] David A. Johnson; *new dimensions in Helicobacter pylori infection*. Emergency medicine 1996, 74- 86.
- [15] Rathbon BJ, Healthy RV: *Helicobacter pylori and GI disease*. Boston black well scientific 1992.
- [16] Andrew Soll, John Isenberg; *Peptic ulcer disease, epidemiology and pathophysiology and clinical manifestation*. Cecil text book of medicine 21 edition, 2000, 126,671-676.
- [17] Zuhair A. Kassir, Aras A. Abdulla, *Helicobacter pylori in various gastrointestinal disease*. J.Fac Med Baghdad 1994, **36**(3) 341-349.
- [18] Nicholas J. Talley, *Functional gastrointestinal disorder: Cecil text book of Medicine*, 21st edition, 2000, **131**, 687-688.
- [19] Richard G long, Brain T Cooper. *Gastrointestinal disease*. RL Souhami and J Moxham, Text book of medicine 3rd edition 1998, 634-640
- [20] Alam K., Schubert TT., Bologna SD. *Increased density of H.pylori on antral biopsy is associated with severity of acute and chronic inflammation and likelihood of D.U*. Am. J. Gastroenterol, 1992, **87**; 424-28.
- [21] Goggin PM, Marreo JM., Jazrawi RP et al, *Effect of H.pylori on gastric mucosal hydrophobicity in man*, Gastroenterol. 1990, **98**, A49.
- [22] Odurul, Peterson HD, Anderson IB et al; *Gasrin and somatostatin- H.pylori infected antral mucosa*, Gut, 1994, **35**, 615-618.
- [23] Schubert TT, Schnell GA, *Campylobacter pylori prevalence in patients undergoing upper endoscopy*. Gastroenterol. 1989, **96**, A455.
- [24] Stephen Patchet. *Helicobacter pylori in Gastroduodenal diseases*. International journal of gastroenterology, 1996, 10-13.
- [25] Marshal BJ, Warren JR., Francis GJ, et al. *Rapid urease test in the management of campylobacter pylori is -associated gastritis*. Am. J. Gastroenterol. 1987, **82**, 200-10.
- [26] Charasz N, Roucayrol AM, Chaplain C, et al. *Prevalence of Campylobacter pylori in fundic atrophic gastritis with or without achlorhydria*. Gastroenterol. 1989, **96**, A83.
- [27] Hermens DS, Mephee MS, Quiason SG, et al. *Detection of Campylobacter pylori in patients with non ulcer dyspepsia; Experience in a community hospital*. Gastroenterol. 1989; **96**: A 206
- [28] George AM, Dooly CP, Dehesa M et al. *Evidence against a role of Campylobacter pylori infection in the pathogenesis of non ulcer dyspepsia*. Gastroenterol. 1989; **96**: A170
- [29] FREDE-silverstein; *Atlas of gastrointestinal endoscopy*, 2nd edition, Mosby Wolfe, 1995, 307.
- [30] John Greenaway, Mike Bramble, *methods of testing for H.pylori* international journal of gastroenterology, 1996, 13-16.

ئاستى تووشبوون بە بەكتىراي (هيليكوپه كتهر) له ناساغى به كانى بهشى سەرەودى كۆنە ندامى ھەرسدا

فؤاد بابان و محمد عمر محمد

كۆنلجى پزىشكى / زانكۆى سلیمانى / ھەرلەي كوردستان- عىراق

پوختە

بنەماي لىكۆلئىنەو ھەكە : ھىلىكۆبەكتەر پايلورى (ھ. پايلورى) كە بەكتىرايەكى لورلپىچى گرام نىگەتقىقە ئىبىتە ھۆى سووتانەو ھەكە كۆنلەي بەرلۆو لە سەراپاي جىھاندا . نەخۆشەكان ھەر لە مندالىيەو توش ئىبن . تووشبوونەكە بە زۆر شىو ھەكەوئىت ھەك ھەوى گەدەو دوانزەگىرئ و گەدەسۆى كۆنلەي ، كەبەھۆكارىكى ناسراوى شىرپەنجەي گەدە دانئەنرئت

پىگەي كار : لەنىوان(مارتى ۱۹۹۷) و (تشرىنى دووھى ۱۹۹۹) ، لە نەخۆشخانەي گشتى فىركارى لە سلیمانى لە چوارسەد نەخۆش كۆلرايەو ھەكە سكالآ و نىشانەي ھەرسى جىاجىايان ھەبوو پاش پشكىنى سەر جۆيى ، ھەناوبىنى گەدەو دوانزەگىرئ يان بۆ كراوھ دوو نمونە لە دالانى گەدە ھەرگىرا بۆ پشكىنى (يورىيىز) خىراي بۆ سەلماندنى بوون و نەبوونى (ھ. پايلورى) .

ئەنجامەكان : نازار بلۆتەرىن سكالآي نەخۆشەكان بوو ، (ھ. پايلورى) لەو نەخۆشەكانەي (ھەوى دوانزەگىرئ و ھەوى گەدە ، گەدە سۆ و شىرپەنجەي گەدەيان) ھەبوو بە پىژەي (۸۹٪ ، ۸۶٪ ، ۷۴٪ ، ۷۰٪) بوون يەك لەدوايەك . بەلام ئەي نەخۆشەكانەي تەنبا گىرقتى ھەرس (بەد ھەرس) يان ھەبوو ، (ھ. پايلورى) لە (۶۴٪) يان دا دۆزرايەو .

دەرەنجام : تووشبوون بە (ھ. پايلورى) لە ناوچەكەماندا ، لە ھەموو تەمەنىكدا بلۆو و پەيوەندى يەكى تونود و پىخۆشكەرە بۆ تووشبوون بە ھەوى گەدەو دوانزەگىرئ و سووتانەو ھەي كوانلەي گەدەو شىرپەنجەي گەدە . ھەرەھا پشكىنى (يورىيىز) بە پىگەيەكى خىراو ھەرزان و بەردەست دا ئەنرئت بۆ دۆزىنەو (ناسىنەو) ي ئەم بەكتىرايە

هدى انتشار جرثومة هاليكوباكتر في اضطرابات الجهاز الهضمي العلوي

فؤاد بياجان و محمد عمر محمد

كلية العليا / جامعة السليمانية / اقليم كردستان - العراق

الخلاصة

اساس الدراسة : هليكوباكتر بايلوري هي بكتريا حلزونية ذات صبغة غرام السالبة التي تسبب احدى الالتهابات المزمنة الشائعة في جميع انحاء العالم . تكتسب الاصابة بهذه البكتريا منذ الطفولة ، ويرافق الاصابة بهذه البكتريا العديد من امراض الجهاز الهضمي المهمة والخطيرة كقرحة المعدة و الاثنى عشر و التهاب المعدة المزمن الذي يعتبر احد العوامل المعروفة للاصابة بسرطان المعدة .

طريقة العمل : قمنا بدراسة نسبة الاصابة بهاليكوباكتر في المرضى الذين كانوا يعانون من العوارض الهضمية المختلفة وقد تم دراسة (٤٠٠) مرضى ، ما بين (اذار ١٩٩٧ و تشرين ثاني ١٩٩٩) في مستشفى العام التعليمي في السليمانية . وتم فحص كافة المرضى سريريا مع تنظير للجهاز الهضمي العلوي مع اخذ خزعتين من المعدة (قرب الفتحة البوابية) لعمل فحص اليورينز السريع للكشف عن وجود هيلكوباكتر .

النتائج : تبين ان الالم هو اكثر العوارض التي كان يشكو منها المرضى و وجد ان نسبة الاصابة ب (هليكوباكتر بايلوري) في المرضى الذين كانوا يعانون من قرحة الاثنى عشر ٨٩٪ وفي المرضى الذين كانوا يعانون من قرحة المعدة ٨٦٪ وكانت نسبة الاصابة في المرضى الذين كانوا يعانون من التهاب المعدة المزمن ٧٤٪ و كانت نسبة الاصابة في المرضى الذين كانوا يعانون من سرطان المعدة ٧٠٪ وبينما كانت نسبة الاصابة في المرضى الذين كانوا يعانون من عسر الهضم الوظيفي ٦٤٪ فقط .

الاستنتاج : تعتبر الاصابة ب (ها ليكوباكتر بايلوري) في منطقتنا شائعة في كافة الاعمار و هناك علاقة وثيقة بين الاصابة بهذه البكتريا مع ازدياد خطورة الاصابة بالقرحة الهضمية و التهاب المعدة المزمن و سرطان المعدة ، و يعتبر فحص اليورينز طريقة سريعة و غير مكلفة و عملية للكشف عن هذه البكتريا .