

**OMICS International** 

# Epidemiology of *Helicobacter pylori* Infection among Symptomatic Patients, Correlation with Endoscopic Findings and it's Association with Type II Diabetes Mellitus

Antoine Abi Abboud\*, Hassan Al Moussawi, Majd Rustom and Walid Abdel Khalek

Medicine Division of Gastroenterology, Faculty of Medical Sciences, Lebanese University, Lebanon

# Abstract

**Background:** Helicobacter pylori (H. pylori) is an important global pathogen infecting approximately 50% of the world's population. This study was undertaken in order to estimate the prevalence of Helicobacter pylori infections among symptomatic patients in Lebanese Hospital Geitaoui University Medical Center (HLG-CHU) and Middle East Institute of Health (MEIH), to investigate the associated risk factors, the endoscopic findings and its association with type 2 diabetes.

**Method:** This is an observational analytic case-control study, carried out over a period of 6 months, from January 2016 till June 2016, including all patients complaining of upper gastrointestinal (GI) symptoms. The patients (n=226) were enrolled following same protocol in both the hospitals. All subjects completed a validated questionnaire and underwent upper GI endoscopy. The histo-pathological diagnosis of H. pylori infection in biopsy specimen was done using the modified Giemsa stain. H. Pylori prevalence and associated factors were analysed by Students t-test, Chi-square test and Fisher exact test. Statistical analysis was performed using the statistical program SPSS version 22.

**Results:** The overall prevalence was 38.9% with no difference between sexes. There was no association with age, residential region, alcohol and caffeine use and smoking. Low level of education and non-steroidal anti-inflammatory (NSAIDs) use were the only significant factors. Bloating, nausea and early satiety were significant predictors of H. pylori infection. The most commonly identified endoscopic finding was gastritis (78.3%), only duodenitis and oesophagitis were significantly associated with H. pylori. Prevalence of H. pylori was 38.8% and 39%, respectively, in patients with diabetes and having no diabetes.

**Conclusion:** H. Pylori prevalence was found to be high. Individuals who had low educational level and NSAIDs consumers, were under higher risk of infection than others. H. pylori infection appears not to be associated with diabetes.

**Keywords:** *Helicobacter pylori* infection; Endoscopic findings; Type 2 diabetes mellitus

## Introduction

Helicobacter pylori (H. pylori) is a gram negative, non-spore forming spiral bacterium which colonizes the human stomach and is prevalent worldwide. Since its discovery in the 1980s, much has been learned about this bacterium and its associated disease states. In 1994, the National Institute of Health Consensus Conference, recognized H. pylori as a cause of gastric and duodenal ulcers. Later that year, the International Agency for Research on Cancer declared H. pylori to be a group I human carcinogen for gastric adenocarcinoma. There is also evidence that H. pylori infection is a risk factor for gastric mucosa-associated lymphomas (MALT lymphomas). Furthermore, the organism is thought to be involved in other human illnesses such as hematologic, autoimmune disorders, insulin resistance and the metabolic syndrome [1,2]. High rates of H. pylori infection are associated with low socioeconomic status and educational levels.

Evidence has recently been published suggesting that the prevalence of *H. pylori* infection might be increased in diabetic patients and in obese patients with an impaired glucose tolerance as opposed to normal population. It was hypothesized that alterations in glucose metabolism may have a role in promoting *H. pylori* colonization due to chemical changes in the gastric mucosa. Another explanation may be that the immune status in diabetic patients, which is strongly compromised, may lead to an increased susceptibility to *H. pylori* infection [3]. However, the link between *H. pylori* infection and diabetes remains

J Gastroint Dig Syst, an open access journal ISSN: 2161-069X

controversial, as some studies indicate a higher prevalence of infection in diabetic patients, [4-8], while others report no difference [9-12].

Since, in the current literature, there is no comprehensive review representing an evidence-based knowledge on the prevalence of *H. Pylori* infection and its potential associations with dyspeptic symptoms in the Middle Eastern countries [12-16] and in Lebanon [17-19]; and on the association of *H. pylori* and diabetes mellitus [19] we conducted this study aiming to determine the prevalence of *H. pylori* infection by using gastric biopsy detection in symptomatic patients, to examine potential risk factors that may influence the acquisition of *H. pylori* infection, and its possible association with diabetes.

# Methodological Approach

## Subjects

All patients during the study period complaining of upper

\*Corresponding author: Antoine Abi Abboud, Medicine Division of Gastroenterology, Faculty of Medical Sciences, Lebanese University, Lebanon, Tel: 9613220520; E-mail: draaaulfsm@hotmail.com

Received May 08, 2017; Accepted May 29, 2017; Published June 05, 2017

**Citation:** Abboud AA, Khalek WA, Abboud AA, Moussawi HAI, Rustom M, et al. (2017) Epidemiology of *Helicobacter pylori* Infection among Symptomatic Patients, Correlation with Endoscopic Findings and it's Association with Type II Diabetes Mellitus. J Gastroint Dig Syst 7: 503. doi: 10.4172/2161-069X.1000503

**Copyright:** © 2017 Abboud AA, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Page 2 of 5

gastrointestinal symptoms (dyspepsia, epigastric pain, heart burn, hematemesis dysphagia, nausea, vomiting, anorexia, early satiety, weight loss, melena, bloating) for more than one month presenting for upper GI endoscopy were recruited consecutively upon signing a written informed consent.

Exclusion criteria were:

a) Patients of type-1 diabetes

b) Non-cooperative patients who refuse to give consent or participate in the study

c) Patients on *H. pylori* eradication therapy.

d) Patients who had used antibiotics during the preceding 30 days.

e) Patients with a history of vagotomy or operations on the upper gastrointestinal tract.

f) Patients with endoscopic diagnosis of gastric cancer proven by histopathological examination.

# Methods

## Study design

This is a prospective observational analytic case-control study, including n=226 patients carried out in two hospitals; HLG-CHU & MEIH over a period of 6 months, from January 2016 till June 2016.

#### Data collection

The study protocol was reviewed and approved by the local ethics committee, and signed informed consents were obtained from each participant. All enrolled subjects were interviewed, by means of a structured questionnaire, to obtain general demographic details and to gather information regarding recent use of drugs and their medical history.

# Data analysis

Data was collected retrospectively from 226 patients who underwent gastric biopsy. Data collection was conducted through paper CRFs that was provided to each investigational site for the collection of all study data for enrolled patients. Data validation was performed by the execution of programmed and manual edit checks in order to control any erroneous, ambiguous or incomplete data.

Parameters			Biopsy	Res	sult	Chi Sauara	n Value	Test	
		Ne	Negative		ositive	Cni-Square	p-value	rest	
Cav	Male	60	60.0%	40	40.0%	0.095	0 771	Chi-Square	
Sex	Female	78	61.9%	48	38.1%	0.065	0.771	test	
Place of residence	Village	13	59.1%	9	40.9%	0.040	0.040	Chi-Squar	
	Town	125	61.3%	79	38.7%	0.040	0.042	test	

Table 1: Association between epidemiologic factors and H. pylori infection.

Statistical analysis was performed using the statistical program SPSS version 22. Descriptive analysis of qualitative variables comprised the sample size, the frequencies and the percentages. Descriptive analysis of quantitative variables included the number, the mean, and the standard deviation. A statistical significant relation exists if p-value is less than 5% using  $\alpha$  error equal to 5%. Tests used for the analytical analysis were Chi-square test, Fisher exact test, and Students *t*-test.

# Results

Among the 226 enrolled patients, 88 (38.9%) were positive for *H. pylori* infection: 40 males (54.54%) and 48 females (45.45%). The remaining 138 patients that were found negative for *H. pylori* infection. There were 60 males (43.47%) and 78 females (56.53%) among them as shown in (Table 1).

The infection occurred more frequently among male participants (40%) as compared to females (38.1%), however the difference was statistically not significant (p=0.771) as presented in (Table 1). The data shown in Table 2 predicted that exposure to *H. pylori* was higher among participants in the age group 28-39 years (57.58%), though age was not found to be associated with infection status.

The overall *H. pylori* infection rates were 40.9% in patients from villages, and 38.7% from towns; nonetheless, the difference was not statistically significant (P>0.05) as depicted in (Table 1).

Higher rates of *H. pylori* infection were found among smokers as compared to non-smokers (42.5% *vs.* 34.9%); however, the difference was found to be statistically non-significant (p=0.243).

All other variables; alcohol and caffeine consumption were not associated with *H. pylori* infection (p>0.05) (Table 3).

A significant (p=0.024) association of *H. pylori* infection rate was found with a decreased level of education. Individuals with lower educational levels had a higher risk than high school graduates and those with a higher education (Table 4).

No significant association (p>0.05) was found of *H. pylori* infection in patients with present medical history (Table 5). It is worth mentioning that in the diabetic group *H. pylori* was positive in 19/49 (38.8%) cases while in non-diabetics, *H. pylori* was positive in 69/177 (39%) cases (Table 5).

The most common presenting symptoms were epigastric pain (70.4%), bloating (41.6%) and heart burn (41.2%). The most commonly identified endoscopic findings were gastritis (78.3%), duodenitis (30.1%), and esophagitis (22.6%). Only duodenitis and oesophagitis (Table 6) were significantly associated with *H. pylori* (p=0.025 and 0.000 respectively).

### Discussion

*H. pylori* is the most common chronic bacterial infection in humans [1-2]. Its prevalence varies greatly among countries and even among

Age	Positive biopsy		Chi-Square	p-value	Test				
Age < 18	2	50.00%	0.21	0.644	Fisher's Exact Test				
Age 18 - 28	7	26.92%	1.784	0.206	Fisher's Exact Test				
Age 29 - 39	19	57.58%	0.017	5.646	Chi-Square test				
Age 40 - 50	20	45.45%	0.976	0.323	Chi-Square test				
Age 51 - 61	11	27.50%	0.267	0.102	Chi-Square test				
Age 62 - 72	10	31.25%	0.927	0.336	Chi-Square test				
Age ≥ 73	3	21.43%	1.924	0.258	Fisher's Exact Test				

 Table 2: Positivity of infection among different age groups.

Page 3 of 5

Parameters			Biopsy	Result		Chi Sauraa		<b>T</b> ==4	
		Negative		Positive		Cili-Square	p-value	Test	
Cmaker	No	69	65.1%	37	34.9%	1 265	0.042	Chi Squara teat	
Smoker Y	Yes	69	57.5%	51	42.5%	1.305	0.243	Chi-Square test	
Alashal	No	89	57.8%	65	42.2%	0.474	0.1.10	Chi Squara taat	
Alcohol	Yes	49	68.1%	23	31.9%	2.174	0.140	Chi-Square test	
Caffeine	No	81	57.4%	60	42.6%	2.061	0.151	Chi Squara toat	
	Yes	57	67.1%	28	32.9%			Chi-Square test	

Table 3: Association between lifestyle factors and H. pylori infection.

Level of Education			Biopsy	Result		Chi Saura		Test	
		Negative		Positive		Chi-Square	p-value	Test	
Formal education	primary	34	48.6%	36	51.4%	7.475	0.024	Chi-Square test	
	secondary	36	62.1%	22	37.9%				
	tertiary	68	69.4%	30	30.6%				

Table 4: Association between educational level and H. pylori infection.

Parameters			Biopsy	Result				<b>T</b> = =4	
		Negative		Positive		Cni-Square	P-value	Test	
HTN	No	96	61.1%	61	38.9%	0.000	0.969	Chi Squara taat	
	Yes	42	60.9%	27	39.1%	0.002		Chi-Square test	
DM	No	108	61.0%	69	39.0%	0.001	0.979	Chi Caucas tost	
	Yes	30	61.2%	19	38.8%	0.001		Chi-Square test	
	No	106	62.0%	65	38.0%				
Dyslipidemia	Yes	31	57.4%	23	42.6%	0.362	0.548	Chi-Square test	
CKD	No	134	60.4%	88	39.6%	2 507	0.450	Eicher Teet	
UND	Yes	4	100.0%	0	0.0%	2.397	0.159	FISHER TEST	

Table 5: Association between medical history and H. pylori infection.

Endoscopio Findingo			Biopsy	Chi Causara	D Value		
Endoscopic Findings	Neg	ative	Po	sitive	Chi-Square	F-value	
Normal	No	116	58.0%	84	42.0%	6.955	0.010
Normai	Yes	22	84.6%	4	15.4%	0.000	
Gastric ulcer	No	125	60.4%	82	39.6%	0.470	0.625
	Yes	13	68.4%	6	31.6%	0.472	
Durdanalulaan	No	135	61.9%	83	38.1%	4.0078	0.267
Duodenai uicei	Yes	3	37.5%	5	62.5%	1.937-	
Costritio	No	33	67.3%	16	32.7%	1.020a	0.308
Gastillis	Yes	105	59.3%	72	40.7%	1.039	
Ducdonitio	No	104	65.8%	54	34.2%	5 006a	0.025
Duodennis	Yes	34	50.0%	34	50.0%	5.000-	
Econhagitia	No	124	67.4%	60	32.6%	16 692a	0.000
	Yes	14	33.3%	28	66.7%	10.002	0.000

Table 6: Analysis of endoscopic lesions and correlation with H. pylori infection.

population groups within the same country. The prevalence of *H. pylori* colonization is about 30% in the United States and other developed countries as opposed to >80% in many developing countries [16]. In general, the overall prevalence of *H. pylori* infection in the Middle East, irrespective of time and age groups, ranged from 22% to 87.6% [16].

In this prospective survey, the histologic prevalence of *H. pylori* among 226 patients who underwent biopsies during upper GI endoscopy was 38.9%. This number is in line with the three previously conducted studies in Lebanon that estimated the prevalence between 21% and 52% [13-15]. In our study we found no difference in *H. pylori* prevalence between the sexes. Regarding the influence of the lifestyle on the prevalence of *H. pylori* infection our data supports the hypothesis that there is no significant association between *H. pylori* and alcohol use or smoking which was shown in multiple studies [20-23]. Markers of low socioeconomic status such as a low family income [24] and low

educational level [25-28], had all a higher likelihood of carrying *H. pylori* infection. Likewise, our current results established that: lower the education of people the higher is the risk for *H. pylori* infection. In our studied population, the most common reason for referral was dyspepsia (87.2%).

The prevalence of dyspepsia ranges from about 20-30% worldwide [29]. Despite a high prevalence of *H. pylori* in dyspeptic patients (40.6%) no significant association was found. These findings are in agreement with the earlier investigations reporting no association between dyspepsia and *H. pylori* [30,31].

The endoscopy results during our present study well demonstrated normal appearance just in 11.5% of cases. Our findings are fully contradictory to the earlier reports where normal endoscopy demonstrated the highest prevalence among symptomatic patients

[32-34]. According to the literature, in 50-60% of patients, no endoscopic cause was apparent and the dyspepsia was considered to be idiopathic [34]. It is worth noteworthy that 15.4% of endoscopically normal looking mucosa revealed positive *H. pylori* histologically. Our results add support to earlier studies which highlighted the problem of disparity between normal endoscopic gastric mucosal appearance and histology in dyspeptic patients undergoing endoscopy [33]. This observation emphasizes the need for routine gastric mucosal biopsy in all symptomatic patients undergoing endoscopy. Gastritis was the commonest endoscopic pathology in this study (78.3%). It is well established in the literature that the commonest cause of gastritis is *H. pylori* infection [35]. However, in this study the relationship between *H. pylori* and endoscopic gastritis was statistically not significant.

In our present study, duodenitis was found in 30.1% cases and oesophagitis in 18.6% cases, being the next most prevalent endoscopic findings after gastritis. In fact, significant relationships were found between duodenitis, oesophagitis and *H. pylori* with p-values of 0.025 and 0.000 respectively. Nevertheless, gastric ulcer (GU) (8.4%) and duodenal ulcer (DU) (3.5%) were the least prevalent findings. Although *H. pylori* infection was identified as the main cause of peptic ulcer disease (PUD) [36] no statistical significant correlation with *H. pylori* was found in our study.

The prevalence of *H. pylori* infection is estimated at over 90% for DU and 70% for GU [37,38]. However in our study, 31.6% of GU patients and 62.5% of DU patients were *H. pylori* positive. This decrease in the prevalence of PUD related to *H. pylori* was reported in several studies in Australia and the United States of America [20-37]. Several factors may have influenced the decreased rate of PUD associated with *H. pylori* such as the use NSAIDs. The association between *H. pylori* infection and use of NSAIDs in ulcer disease is controversial. *H. pylori* and NSAIDs are independent and synergistic risk factors for peptic ulcer disease and bleeding ulcer [36]. Our current results do not indicate any significant prevalence of PUD (12.5%) in patients positive for *H. pylori* and users of NSAIDs. The use of NSAIDs was not associated with *H. pylori* infection in patients with PUD. This result is also supported by a recent meta-analysis of Tang et al. [39].

Concerning diabetes, no association was found with H. pylori. In fact, the link between H. pylori infection and diabetes remained controversial, it was studied for the first time by Simon [26]. Simon reported a higher prevalence of *H. pylori* infection in diabetic patients compared with controls (62% vs. 21%) [26]. These data were further confirmed in 1996 by a case-control study that examined 143 diabetics [25]. Zelenkova had reported a prevalence of *H. pylori* to be lower in diabetics compared to non-diabetic controls (27% vs. 51%) [27,28]. In other studies, the prevalence of *H. pylori* infection was comparable between diabetic and control [10-17]. In our study we found no significant association between H. pylori and diabetes (p=0.979). This is in line with a study done by Naja et al. on the association of H. pylori infection with insulin resistance and metabolic syndrome (MetS) among Lebanese adults that found no association of H. pylori infection with IR or MetS, and concluded that the eradication of H. pylori infection to prevent IR or MetS is not warranted [15].

## Conclusion

The analysis of the research results for *H. pylori* on 226 patients showed that our study led to data comparable to those reported in the literature, particularly to the overall prevalence which was 38.9%. This infection is associated with low level of education and does not depend on gender, age, and area of residence. Gastritis is the most common endoscopic finding. NSAID use was suggested as playing critical role as

co-factor for *H. pylori* infection. Regarding the prevalence of *H. pylori* in ulcer disease, our results show a lower percentage compared with literature data. The present study suggests that *H. pylori* infection is not increased in diabetes mellitus.

Page 4 of 5

#### References

- Goh KL, Chan WK, Shiota S, Yamaoka Y (2011) Epidemiology of Helicobacter pylori infection and public health implications. Helicobacter 16: 1-9.
- Parsonnet J (2011) Helicobacter pylori and gastric cancer. Gastroenterol Clin North Am 22: 89-104.
- Donath MY, Shoelson SE (2011) Type 2 diabetes as an inflammatory disease. Nat Rev Immunol 11: 98-107.
- Devrajani BR, Shah SZ, Soomro AA, Devrajani T (2010) Type 2 diabetes mellitus: A risk factor for Helicobacter pylori infection: Hospital based casecontrol study. Int J Diabetes Dev Ctries 30: 22-26.
- Bener A, Micallef R, Afifi M, Derbala M, Al-Mulla HM, et al. (2007) Association between type 2 diabetes mellitus and Helicobacter pylori infection. Turk J Gastroenterol 18: 225-229.
- Gulcelik NE, Kaya E, Demirbas B, Culha C, Koc G, et al. (2005) Helicobacter pylori prevalence in diabetic patients and its relationship with dyspepsia and autonomic neuropathy. J Endocrinol Invest 28: 214-217.
- Anastasios R, Goritsas C, Papamihail C, Trigidou R, Garzonis P, et al. (2002) Helicobacter pylori infection in diabetic patients: Prevalence and endoscopic findings. Eur J Intern Med 13: 376.
- Ko GT, Chan FK, Chan WB, Sung JJ, Tsoi CL, et al. (2001) Helicobacter pylori infection in chinese subjects with type 2 diabetes. Endocr Res 27: 171-177.
- Stanciu OG, Trifan A, Sfarti C, Cojocariu C, Stanciu C (2003) Helicobacter pylori infection in patients with diabetes mellitus. Rev Med Chir Soc Med Nat lasi 107: 59-65.
- Xia HH, Talley NJ, Kam EP, Young LJ, Hammer J, et al. (2001) Helicobacter pylori infection is not associated with diabetes mellitus, nor with upper gastrointestinal symptoms in diabetes mellitus. Am J Gastroenterol 96: 1039-1046.
- Khedmat H, Karbasi-Afshar R, Agah S, Taheri S (2013) Helicobacter pylori infection in the general population: A middle eastern perspective. Caspian J Intern Med 4: 745-753.
- Novis BH, Gabay G, Naftali T (1998) Helicobacter pylori: The middle east scenario. Yale J Biol Med 71: 135-141.
- Naous A, Al-Tannir M, Naja Z, Ziade F, El-Rajab M (2007) Fecoprevalence and determinants of Helicobacter pylori infection among asymptomatic children in Lebanon. J Med Liban 55: 138-144.
- Kalaajieh WK, Chbani-Rima A, Kassab TF, Baghdadi FM (2000) Helicobacter pylori infection in North Lebanon. Sante 10: 31-35.
- Naja F, Nasreddine L, Hwalla N, Moghames P, Shoaib H, et al. (2012) Association of H. pylori infection with insulin resistance and metabolic syndrome among Lebanese adults. Helicobacter 17: 444-451.
- 16. Eshraghian A (2014) Epidemiology of Helicobacter pylori infection among the healthy population in Iran and countries of the Eastern Mediterranean Region: A systematic review of prevalence and risk factors. World J Gastroenterol 20: 17618-17625.
- Demir M, Gokturk HS, Ozturk NA, Kulaksizoglu M, Serin E, et al. (2008) Helicobacter pylori prevalence in diabetes mellitus patients with dyspeptic symptoms and its relationship to glycemic control and late complications. Dig Dis Sci 53: 2646-2649.
- Upala S, Jaruvongvanich V, Riangwiwat T, Jaruvongvanich S, Sanguankeo A (2016) Association between Helicobacter pylori infection and metabolic syndrome: A systematic review and meta-analysis. J Dig Dis 17: 433-440.
- Zhou X, Liu W, Gu M, Zhou H, Zhang G (2015) Helicobacter pylori infection causes hepatic insulin resistance by the c-Jun/ miR-203/SOCS3 signaling pathway. J Gastroenterol 50: 1027-1040.
- Gasbarrini G, Pretolani S, Bonvicini F, Gatto MR, Tonelli E, et al. (1995) A population based study of Helicobacter pylori in infection in a European country: The San Marino study. Relations with gastrointestinal diseases. Gut 36: 838-844.

Page 5 of 5

- Ozaydin N, Turkyilmaz SA, Cali S (2013) Prevalence and risk factors of Helicobacter pylori in Turkey: A nationally-representative, cross-sectional, screening with the (1)(3)C-Urea breath test. BMC Public Health 13: 1215.
- Murray LJ, McCrum EE, Evans AE, Bamford KB (1997) Epidemiology of Helicobacter pylori infection among 4742 randomly selected subjects from Northern Ireland. Int J Epidemiol 13: 880-887.
- Santos IS, Boccio J, Santos AS, Valle NCJ, Halal CS, et al. (2005) Prevalence of Helicobacter pylori infection and associated factors among adults in Southern Brazil: A population-based cross-sectional study. BMC Public Health 13: 118.
- Lim SH, Kwon JW, Kim N, Kim GH, Kang JM, et al. (2013) Prevalence and risk factors of Helicobacter pylori infection in Korea: Nationwide multicenter study over 13 years. BMC Gastroenterol 13: 104.
- 25. den Hollander WJ, Holster IL, den Hoed CM, van Deurzen F, van Vuuren AJ, et al. (2013) Ethnicity is a strong predictor for Helicobacter pylori infection in young women in a multi-ethnic European city. J Gastroenterol Hepatol 28: 1705-1711.
- Simon L, Tornoczky J, Toth M, Jámbor M, Sudár Z (1989) The significance of Campylobacter pylori infection in gastroenterologic and diabetic practice. Orv Hetil 130: 1325-1329.
- Oldenburg B, Diepersloot RJ, Hoekstra JB (1996) High seroprevalence of Helicobacter pylori in diabetes mellitus patients. Dig Dis Sci 41: 458-461.
- Zelenkova J, Souckova A, Kvapil M, Soucek A, Vejvalka J, et al. (2002) Helicobacter pylori and diabetes mellitus. Cas Lek Cesk 141: 575-577.
- Tack J, Talley NJ, Camilleri M, Holtmann G, Hu P, et al. (2006) Functional gastroduodenal disorders. Gastroenterology 130: 1466-1479.
- 30. López Gastón A, Andrusch A, Catuogno P (2003) Functional dyspepsia

and infection by Helicobacter pylori: A causal relation does not exist. Acta Gastroenterol Latinoam 33: 13-21.

- Boixeda D, Gisbert JP, Martín de Argila C, Cantón R, Bermejo F, et al. (1995) Is there a relationship between digestive symptoms and H. pylori infection? Rev Esp Enferm Dig 87: 8-14.
- Aduful HK, Naaeder S, Darko R, Baako B, Clegg-Lamptey J, et al. (2007) Upper gastrointestinal endoscopy at the Korle Bu Teaching Hospital, Accra, Ghana. Ghana Med J 41: 12-16.
- Ndububa DA, Agbakwuru AE, Adebayo RA, Olasode BJ, Olaomi OO, et al. (2001) Upper gastrointestinal findings and incidence of Helicobacter pylori infection among Nigerian patients with dyspepsia. West Afr J Med 20: 140-145.
- Fisher RS, Parkman HP (2002) Management of no-nulcer dyspepsia. N Engl J Med 339: 1376-1381.
- Clark, MI, Silk DB (2002) Gastrointestinal disease: Gastropathy and gastritis. Clinical Medicine. WB Saunders pp: 276-277.
- 36. Papatheodoridis GV, Sougioultzis S, Archimandritis AJ (2006) Effects of Helicobacter pylori and nonsteroidal anti-inflammatory drugs on peptic ulcer disease: A systematic review. Clin Gastroenterol Hepatol 4: 130-142.
- Kalaghchi B, Mekasha G, Jack MA, Smoot DT (2004) Ideology of Helicobacter pylori prevalence in peptic ulcer disease in an inner-city minority population. J Clin Gastroenterol 38: 248-251.
- Suerbaum S, Michetti P (2002) Helicobacter pylori infection. N Engl J Med 347: 1175-1186.
- Tang CL, Ye F, Liu W, Pan XL, Qian J, et al. (2012) Eradication of Helicobacter pylori infection reduces the incidence of peptic ulcer disease in patients using nonsteroidal anti-inflammatory drugs: A meta-analysis. Helicobacter 17: 286-296.

**Citation:** Abboud AA, Khalek WA, Abboud AA, Moussawi HAI, Rustom M, et al. (2017) Epidemiology of *Helicobacter pylori* Infection among Symptomatic Patients, Correlation with Endoscopic Findings and it's Association with Type II Diabetes Mellitus. J Gastroint Dig Syst 7: 503. doi: 10.4172/2161-069X.1000503