**University of Thi-Qar**

**College of Medicine**

** *Endoscopic and gastric histopathological view in dyspeptic patients and their relationship to presence of H.pylori infection***

***By***

**Wedad hussein Ali**

**Baraa Dakheel Faraj**

*Sixth stage, college of medicine*

***Supervised by***

***Assist. Prof Dr. Haider M. AL Yasiri***

**

***Dedication:***

*We humbly dedicate this study to enhancement of medical students in making a case study.*

*The student's aim to develop a study which is worthy of credits as a certificate reference in continuing development of medical practice for better services to the patients.*

*we would like to Express our appreciation to our supervisor, Dr. Haider Al. yassery for the patient guidance, encouragement and advice he has provided throughout our time as his student. I have been extremely lucky to have a supervisor who cared so much about my work, and who responded to my questions and queries so promptly. Also we are very gratefully to our friends and our family.*

*Finally, I dedicate this study to women who raise me , who's been a rock of stability throughout my life , whose loving as a spirit sustain me still..*

***My mother***

**Acknowledge**

The completion of this study could not have been possible without participant and assistant of many people where , their contributions sincerely appreciated and gratefully acknowledged.

However , The group we would like to Express their participation and indebtedness particularly the following:

**Assis.Prof. Hameed Naeem Mousa**

Department of pathology, college of medicine, University of Thi-Qar

And all member of histopathological unit .

**The endoscopist &gastroenterologist . Dr.Faez k. Al Surhan**

Department of endoscopy, college of medicine , University of Thi\_ Qar

**The endoscopist &gastroenterologist Dr. Sadek J. Yassen**

Department of endoscopy

**The endoscopist &gastroenterologist Dr. Ali Ajel**

Department of endoscopy

**Physician and family medicine , Dr. Muslim Nahi**

In college of medicine, Thi Qar University

To their endless support & kind and understanding spirit ,guidance and best counsel during case collection , slides reading and statistical analysis

To all relatives and friend, who in way or other shared their support either morally, financially or physically. Thank you.

Above all , to the Great the author of acknowledge and wisdom for his countless love .

***The content:***

*Abbreviation …………………………………………………………….page 6*

*Abstract …………………………………………………………………page 7*

***Chapter one***

*Introduction …………………………………………………….page 10*

*Aim of study …………………………………………………… page 12*

***Chapter two***

*Methodology ………………………………………………….page 14*

***Chapter three***

*The result …………………………………………………page 18*

***Chapter four***

*Discussion …………………………………………….page 25*

*Conclusion …………………………………………page 27*

*Recommendation ……………………………..page 27*

*Reference …………………………………………page 28*

***Abbreviation:***

|  |  |
| --- | --- |
| *H.pylori*  *NSAIDs*  *OGD*  *SPSS* | *Helicobacter pylori*  *Non steroidal anti inflammatory drugs*  *Esophagogastric\_*  *duodenoscope*  *Statistical package for social science* |

***Abstract:***

**Background:**

Helicobacter pylori related gastritis is a major health ailment in developing nations. There is high morbidity and mortality ranging from chronic gastritis to gastric malignancies. Prevalence of H. pylori infection varies markedly from country to country andin a country, region to region. Aim: To study the prevalence of H. pylori gastritis in patients undergoing endoscopy and its association with the development of gastrointestinal diseases.

**Subjects and Methods:**

The study was carried out in Al hussein teaching hospital from September 2018 to March 2019 . 40 Patients presenting with dyspeptic symptoms were subjected to upper gastrointestinal endoscopy , investigated for H. pylori infection through h.pylori antibody IGg test ,and histopathological examination done for antral biopsy. Data analysis was carried out using SPSS .

**Results:**

H. pylori infection was diagnosed in 57.5%of patients screened. There was no statistically significant difference in sex and age related distribution of H. pylori infection. However, a statistically significant association of H. pylori infection with sydney grading of gastritis .

**Conclusion:**

H.pylori infection relation statistically not significant with demographic characteristic of the patient and endoscopic finding .

But it's significant in relation to gastritis according to sydney grading where p value < 0.005 and < 0.001 in relation with presence of acute and chronic changes.

***Chapter one***

***Introduction***

**Introduction:**

H. pylori is a Gram-negative bacterium whose presence in the stomach of infected individuals is linked to the development of several gastric diseases, such as chronic gastritis(1) . Although it is estimated that 50%of the world population is infected by H. pylori, only a small percentage of infected patients develop more severe pathologies, such as ulcers (10%-15%) and stomach adenocarcinomas (less than 1%)[2], the latter representing 15.4% of the cancers produced by infectious agents worldwide in 2012[3].These values suggest that while relevant to the development of severe diseases, including gastric cancer, this pathogen could also play other roles in the human host<4>.h. pylori infects approximately 50% of the human population worldwide and the infection could reach more than 70% in developing countries(1,2)

The chronic inﬂammation induced by H. pylori interferes with the normal physiology of the gastric acid secretion in different degrees, thus leading to chronic gastritis, which, in most individuals, remains asymptomatic and does not progress. However, in some cases, altered gastric secretion, associated with tissue injury, induces the development of peptic ulcer, whereas in other cases, gastritis progresses to atrophy, intestinal metaplasia, and eventually to gastric carcinoma or rarely to gastric lymphoma, due to the persistent immune stimulation of the lymphoid tissue [5] The consequences of infection have been associated with the development of different gastro-intestinal diseases, such as gastric ulcers, gastric cancer, mucosa-associated lymphoid tissue(MALT) lymphoma and biliary tract cancer(6).

Moreover, H. pylori infection has also been associated with extra gastric diseases, such us ischemic heart diseases(7)”, type 2 diabetes mellitus(8), anemia(9)adverse metabolic traits in obese subjects(10)] and insulin resistances(11), to mention but a few .Despite the existence of such associations, these diseases occur only in a small percentage of infected people, suggesting that the bacteria frequently persists in the human host without inducing any obvious signs of disease, and it has been suggested that H. pylori may also play a beneﬁcial role in human health(12-17)

A wide range of laboratory investigations are available for diagnosis of H. pylori. The tests belong to non- invasive group and invasive group. Non-invasive tests include, urea breath test, serological Immunoglobulin G (IgG) and ImmunoglobulinM (IgM) detection, saliva and urinary antibody test, and stool antigen test.[ 18] The invasive tests are endoscopy based tests, which include histopathological examination, rapid urease test (RUT)and polymerase chain reaction. Whereas, invasive tests carry high sensitivity and speciﬁcity of> 90%,(19)the role of non-invasive tests such as serology is limited in areas of high prevalence, because of non-distinction between previous and current infection(20).

Dyspepsia is derived from the Greek words dys and peps and literally means ”difﬁcult digestion(21)dyspepsia is generally deﬁned as chronic or frequently recurring epigastric pain/discomfort originating in gastro-duodenal region and may be accompanied with other gastrointestinal symptoms such as nausea, belching, vomiting, postprandial fullness and early satiety(22)Chronic dyspepsia symptoms can be unceasing, sporadic or recurrent(23). It is considered to be important to public health, because it is remarkably common, can be disabling, and can poses major social and economic burden(24)People with functional dyspepsia have a signiﬁcantly reduced quality of life when compared to the general population(25) .Annual incidence of dyspepsia is approximately 9-10% and 15%patients have chronic (>3 months in a year), frequent (>3episodes per week) and often very severe symptoms(26).Functional dyspepsia is considered to possess a wide spectrum of non speciﬁc upper gastrointestinal symptoms without any organic alteration(27)accounting for 60% of patient referrals to gastroenterology clinics(28)

***Aim of study :***

*To study the incidence of H.pylori in patients with epigastric pain*

*In endoscopic unit and to know the effect of H.pylori on endoscopic and histopathological finding*

***Chapter two***

***Methodology***

***Aim of study***

**Methodology :**

The study was carried out from September 2018 to March 2019 in AL Hussein teaching hospital a Secondary hospital in AL Nasiriya . Where it is only public hospital in Thi-Qar with functioning endoscopic facilities and received referrals from many hospitals and outpatient clinic.

The study was Cross -Section prospective study.

The study was conducted with 50 patients who referred for OGDcenter .The patients who consented to participate in the study were randomly recruited to meet the required sample size ,excluded from participating were patients on proton pump inhibitors ,any antibiotics or Bismuth in the preceding 1 mouth .The patients were selected on basis of chief complaints of Dyspepsia and age of patients range from 15 to 80 . informed consent was taken from all patients after explaining ton them the nature and purpose of the study. Ethical clearance was taken prior to study from ethical committee in the hospital.

Diagnosis of dyspepsia was based on clinical findings ,we defined the dyspepsia if the patient had one or more of these symptoms with duration of three months or longer; postprandial fullness , early satiation, epigastric pain ,epigastric burning ,bloating in upper abdomen ,nausea, vomiting[29] and After taken questionnaire (table 1) from the patients.

|  |  |
| --- | --- |
| Questionnaire |  |
| Name |  |
| Sex |  |
| Age |  |
| Occupation |  |
| Resident |  |
| Blood group |  |
| Presence of pain |  |
| Site |  |
| Aggravated factor |  |
| Relieved factor |  |
| Duration of pain |  |
| Associated symptom |  |
| Drugs used |  |
| NSAIDs use and duration |  |
| Steroids and duration |  |
| Spicy food |  |
| alcohol |  |
| smoking |  |
| Family history |  |

Patient`s blood samples were collected in tubes and centrifuged for 5 mint. 3drops of serum add into H.pylori antibody test kit (ABON Biopharm)(Specificity 94.1% , Sensitivity 95.1%)and the results (+ or -) taken after 10 minutes (depended on information taken from cassette within the kit )

After taken blood samples ,Endoscopy was carried out by specialist endoscopist using (Olympus EVIS GIT -Q24OZ OR SP 240) forward viewing esophagogastroduodenoscopy.

The process of the endoscopy procedure was explained to the patients, then 10% xylocaine pharyngeal spray was administered to the patient's pharynx to paralyze the gag reflex and diazepam 5mg I.V . The patient was then placed on his left lateral position on the endoscopy couch. The endoscopist categorized the patients based on the endoscopic finding into four groups:

1- normal, 2- abnormal nonulcerative (any evidence of mucosal lesion without ulcer, e.g. erosion, erythema, nodularity, atrophy, white plaque, and petechiae), 3- ulcerative, and 4-combination of 2 and 3

. One endoscopic biopsy fragment was obtained from each patient from antrum.this endoscopic biopsy was sent to histopathology department in formalin container.4 μ thickness section were cut from each block and mounted on a slide. The Slide was stained with normal H and E, stain and Giemsa stain. Histopathological assessment of gastric mucosa was carried out by a specialist pathologist. Biopsies were evaluated for the intensity of mononuclear inflammatory cellular infiltrates, inflammatory activity (neutrophilic infiltrations), glandular atrophy, metaplasia, reparative atypia, and dysplasia [[21](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6014253/#ref21)]. Additionally, the cases were graded according to the Houston-updated Sydney system [[20](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6014253/#ref20)], which was graded according to the intensity of mono-nuclear inflammatory cellular infiltrates within the lamina propria: absent inflammation (Grade 0), mild inflammation (Grade 1), moderate inflammation (Grade 2), and severe inflammation (Grade 3).

***Chapter three***

***The Result***

**Result:**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
|  | | | | | | |
| Table 2:Gender ,age , resident relation to h .pylori infection | | | | | | |
| Percentage of h.pylori + from total number | **P value** | **H.pylori antibody positive**  **N. of cases Percentage** | | **Percentage%** | **No. of cases** | **Data** |
|  | **0.146** |  |  |  |  | **Gender** |
| 30% | **70.5%** | **12** | **42.5%** | **17** | **Male** |
| 27.5% | **47.8%** | **11** | **57.5%** | **23** | **female** |
|  | **0.818** |  |  |  |  | **Age** |
| 17.5% | **63.6%** | **7** | **27.5%** | **11** | **15-30** |
| 27.5% | **57.8%** | **11** | **47.5%** | **19** | **31-45** |
| 12.5% | **50%** | **5** | **25%** | **10** | **>45** |
|  | **0.38** |  |  |  |  | **Employment** |
| 12.5% | **71.4%** | **5** | **17.5%** | **7** | **employed** |
| 10% | **80%** | **4** | **12.5%** | **5** | **student** |
| 7.5% | **37.5%** | **3** | **20%** | **8** | **not employed** |
| 27.5% | **55%** | **11** | **50%** | **20** | **house wife** |
|  | **0.822** |  |  |  |  | **Resident** |
| 32.5% | **59%** | **13** | **55%** | **22** | **From AL Nasiriya** |
| 25% | **55.5%** | **10** | **45%** | **18** | **Out side** |

**The clinical finding :**

The patients’ age ranged from 15years to 80 years with an average of years.17(42.5%) patients were male and 23(57.5%) were female.The proportion of male cases positive for H. pylori antibody test70.5% (12/17) was not statistically significant when compared to the proportion of females positive for H. pylori 47.8 % (11/27) with P value of 0.146 .Similarly, comparison of H. pylori positive patients wit age [15\_30 ]years 63.6% (7/11) and age [31\_45]years 57.9% (11/19) and age [>45] years 50% (5/10) did not yield any statistically significant result with P value of 0.81 .Regarding Resident 22 (45%) patients from Al Nasiriya and 18 (45%) out side of Al- Nasiriya from surrounding areas and circumstances which is statically not yield any significant were p value <0.822.Table2 .

Also relation of duration of dyspepsia (Table 3) and presence of risk factor to h.pylori positive not yield any statistical significant (table 4) Out of 40patients, all of them presented to our hospital with upper abdominal pain out of which 23 (57.5%)(23/40) patients had H. pylori infection.

Similarly, 9 (22.5%) patients presented with nausea or Vomiting(55.5 %)(5/9) had h.pylori infection ,1 (1/40)(2.5%) patient with hematemesis which h.pylori negative, 9 (9/40) (22.5%) with Melena ,55.5%(5/9) positive h. pylori , 18 (45.5%) had bloating ,(50%) (9/18) of them had h.pylori positive, and 2 (5%)patients with weight/appetite loss. Smoking not show statistical relation to h. Pylori infection where 10(25% ) patients are smoker , 8 (80%) (8\10) of them h. pylori positive.

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Table 4: relation of h. pylori to presence of risk factor | | | | | | |
| Percentage of h. pylori + from total number | P value | h. pylori antibody positive  N. of cases Percentage | | Percentage% | N. of cases | Data |
| 15% | 0.223 | 46.1% | 6 | 32.5% | 13 | NSAIDs |
| 10% | 0.37 | 44.4% | 4 | 22.5% | 9 | Steroids |
| 17.5% | 0.34 | 70% | 7 | 25% | 10 | Spicy food |

**Endoscopic finding:**

EGD revealed features of endoscopic gastritis in 37 (92.5%) (37/40) patients. 32 (68.5%) (32/37) of them had pangastropathy, 3 (8.1% )( 3/37) patients had ulcer , 1 (2.7%) (2/37) patient had mass and 1(2.7%)(1/37) had both ulcer and pangastropathy.6 (15%) (6/40) patients had duodenitis and 5 (12.5) (5/40) had flat duodenal mucosa (grossly celiac disease) . Regarding esophageal endoscopic finding ,6 (15%)(6/40 ) had GERD ,3 (7.5%) (3/40) patients had esophageal candidiasis and 4 (10%) (4/40) had hiatal hernia .table 5

All endoscopic finding regarding gastritis and other finding not show any statistical significant. Table 6

|  |  |  |
| --- | --- | --- |
| Table 5: prevalence of endoscopic finding | | |
| Percentage% | N. of cases | **Endoscopic finding** |
|  | 32 | **1.Endoscopic gastritis** |
|  | 3 | A .normal |
|  | 32 | B. pangastropathy |
|  | 4 | C. Ulcerations |
|  | 1 | D. mass |
|  | 6 | **3.Duodenitis** |
|  | 5 | Flat duodenal mucosa |
|  |  | **4.Esophagus** |
|  | 6 | GERD |
|  | 3 | Esophageal candidiasis |
|  | 4 | Hiatal hernia |

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Table 6: Relation of endoscopic gastritis to h .pylori infection | | | | | | |
| Percentage of h. pylori + from total number | P. value | **H. pylori antibody positive**  **Percentage% N. of cases** | | **Percentage** | **N. of cases** | **Endoscopic finding** | |
| 5% |  | **8.7%** | **2** | 7.5% | 3 | **A. Normal** | |
| 45% | 0.549 | **78.3%** | **18** | %80 | 32 | B. pan gastropathy | |
| 5% | **8.7%** | **2** | 7.5% | 3 | C. Ulcerations | |
| 2.5% | **4.3%** | **1** | 2.5% | 1 | D. both B and C | |
| 2.5% | **0** | **0** | 2.5% | 1 | **E .mass** | |
| 57% |  | **100%** | **23** | 100% | 40 | **Total** | |

**Histopathological finding :**

In gastric biopsy, where 40 samples collected from Antrim show 9 (22.5%) (9/40) patients had grade 0 according to modified sydney grading for gastritis no on from grade 0 had h.pylori antibody positive. 19 (47.5%) (19/40) patients had grade 1 from them 15 (65.2%)(15/23) had h.pylori positive from total h.pylori positive samples. 8 (20%) (8/40) patients had grade 2 and 4 (10%)(4/40) patients had grade 3 ,among those 4 (17.4%) and (17.4%) had h.pylori antibody positive respectively. Sydney grading gastritis show statistically significant result where p value <0.005 table 7 Lastly, histopathological changes such presence of acute or chronic changes were statistically significant while other changes such as presence of atrophic changes or lymphoid aggregation are not significant.

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| **Table 7: relation of histopathological gastritis to h. pylori infection according to Sydney grading** | | | | | | | |
| **sydney grading of gastritis for antral biopsy** | | **N. of cases** | **Percentage%** | **H .pylori antibody positive** | | **P.value** | **Percentage of h. pylori + from total number** |
|  | **N. of cases** | **Percentage%** |
|  | **grade 0** | **9** | **22.5%** | **0** | **0** | **o.oo5** | **0** |
| **grade 1** | **19** | **47.5%** | **15** | **65.2%** | **37.5%** |
| **grade 2** | **8** | **20%** | **4** | **17.4%** | **10%** |
| **grade 3** | **4** | **10%** | **4** | **17.4%** | **10%** |
| **Total** | | **40** | **100%** | **23** | **100%** | **57.5%** |

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Table 8: histopathological changes relation to h. pylori infection | | | | | | |
| Data | N. of cases | Percentage % | H. pylori positive  N. of cases percentage | | P value | Percentage of h. pylori + from total number |
| acute changes | 9 | 22.5% | 4 | 44.4% | 000. | 10% |
| Chronic changes | 22 | 55% | 19 | 86.4% | 47.5% |
| no changes | 9 | 22.5% | 0 | 0 | 0 |
| atrophic changes in antral biopsy | 15 | 37.5% | 10 | 66.7% | 0.361 | 25% |
| Lymphoid aggregation | 2 | 5% | 1 | 50% | 0.827 | 5% |
| Regenerative changes | 3 | 7.5% | 0 | 0 | 0.01 | 0 |

***Chapter four***

***Discussion***

***Conclusion***

***Reference***

**Discussion :**

Gastritis, gastric ulceration, and gastric malignancies have many etiological factors, among which H. pylori infection is the principal cause. H. pylori infection is dependent upon many

variables such as age, sex, socioeconomic status, dietary habits, genetic, and immunological factors. In the present study, we did not get a significant difference in H. Pylori prevalence according to gender. This is in concordance with the study results of Tarkhashvili et al.,[29] and Shokrzadeh et al.,[30] In contrast a study by Kaore et al.,[31] showed higher prevalence in male gender .Age distribution of H. pylori infection did not show any trend towards increase or decrease in infection with the advancing age. Though maximum percent of H. pylori positivity 63.6%(7/11)) was seen in the age group of 15\_30 years. There was no statistically significant difference in prevalence of H. pylori in the age

. This is similar to the observations laid byShokrzadeh et al.,[30] and Kaore, et al.,[31] reported increased H. pylori infection in age groups of 20‑40 years than the older age group.In the present study.

The correlation of smoking and H.pylori infection not show significant ,this similar to study carried by This study of patients in a general practice found no significant relation between smoking and active H pylori infection Hermann Brenner, at al.[32 ] .But against study done by Ahmed jumbo et al .. [33] .where show there is a strong association between cigarette smoking (P< 0.0001) and increased prevalence of H. pylori infection among smoker .

Regarding use of NSAIDs and H.pylori infection didn't get statistical significant, This is similar to observation laid by al assi MT et al .. [34] , NSAID use may now be responsible for most bleeding complications of ulcer disease, regardless of H. pylori status.

The commonest identifiable lesion at endoscopy was gastritis(80%). This is similar to observation laid by Jemilohun et al.,[35] in which the

correlation was not statistically significant. In contrast to our study the observation showed by Adlekha S et al .. [ 36] this is because we randomly select the patients and due to multiple drugs used by the patients which affect gastric mucosa grossly.

The correlation of endoscopic abnormality with H. pylori infection was statistically highly significant with a P < 0.01, proving endoscopic changes to be

a sensitive indicator of H. pylori infection. Histopathological gastritis present in 77.5% which statistically significant where p value <0.005 This is similar to observation laid by Taha M.M. et al [37] . and Zapata-Colindres et al.,[38] and

Ahmad et al.,[39] documenting H. pylori prevalence in gastric

ulceration patients to be 80% and 84% respectively. Cotran

et al.,[19] reported the international association of H. pylori with

gastric ulceration

**Conclusion:**

1. H.pylori infection not related to the sex , age and resident (rural or urban) as making for examined patients.
2. No relationship in our study shows between h.pylori infection and endoscopic finding included gastritis ,duodenitis, GERD .
3. Histopathological finding greatly related to H.pylori infection while acute or chronic
4. Grading of gastritis according to sydney grading had relationship to H.pylori infection.

***The recommendations:***

1. *Not use antibiotics unless we have a document for its presence*
2. *H.pylori is very important and you must check all patients with dyspepsia*
3. *All patients with H.pylori ,its not important to send to endoscopy*
4. *If we send for endoscopy, Its important to do biopsy for histopathological grading.*
5. *It's important to eradicate h.pylori in patients with histopathological changes*
6. *Inform the medical institution.*

**Reference:**

1 de Martel C, FerIay J, Franceschi S, Vignat J, Bray F, Forman D,Plummer M. Global burden of cancers attributable to infectionsin 2008: a review and synthetic analysis. Lancet Oncol 2012; 13:607-615 [PMID: 22575588 DOI: 10.1016/51470—2045(12)70137-7]

2 Suerbaum S, Micheth’ P. Helicobacter pylori infection. N EnglJ Med 2002; 347: 1175-1186 [PMID: 12374879 DOI: 10.1056/NEJMra020542]

3 Plummer M, de Martel C, Vignat J, FerIay J, Bray F, Franceschi5. Global burden of cancers attributable to infections in 2012: asynthetic analysis. Lancet Glob Health 2016; 4: e609-e616 [PMID:27470177 DOI: 10.1016/52214—109X(16)30143-7]

4- Helicobacter pylori in human health and disease: Mechanismsfor local gastric and systemic effectsDenisse Bravo, AniIei Hoare, Cristopher Soto, Manuel A Valenzuela,Andrew FG Quest

5-Robinson K, Argent RH, Atherton JC. The inﬂammatoryand immune response to Helicobacter pylori infection.Best Pract Res ClinGastroenterol. 2007;21( 2):237-25 9.

6- Shmuely H, Wattad M, Solodky A, Yahav J, Samra Z, Zafrir N.Association of Helicobacter pylori with coronary artery disease andmyocardial infarction assessed by myocardial perfusion imaging. IsrMed Assoc J 2014; 16: 341-346 [PMID: 25058994]

7- Liu J, Wang F, Shi S. Helicobacter pylori Infection Increase the Riskof Myocardial Infarction: A Meta-AnaIysis of 26 Studies Involvingmore than 20,000 Participants. Helicobacter 2015; 20: 176-183[PMID: 25382293 DOI: 10.1 11 1/heI.12188]

8- Li JZ, Li JY, Wu TF, Xu JH, Huang CZ, Cheng D, Chen QK, Yu T.Helicobacter pylori Infection Is Associated with Type 2 Diabetes, NotType 1 Diabetes: An Updated Meta-AnaIysis. Gastroenterol Res Pract2017; 2017: 5 715403 [ PMID: 28883831 DOI: 10.1155/2017/5715403]

9- Xu MY, Cao B, Yuan BS, Yin J, Liu L, Lu QB.Associat1'onof anaemia with Helicobacter pylori infection: a retrospectivestudy. Sci Rep 2017; 7: 13434 [PMID: 29044219 DOI: 10.1038/s41598-01 7-13955-3]

10- Chen CX, Mao YS, Foster P, Zhu ZW, Du J, Guo CY. Possibleassociation between Helicobacter pylori infection and nonalcoholicfatty liver disease. ApplPhysiolNutrMetab 2017; 42: 295-301[PMID: 28177748 DOI: 10.1139/apnm-2016-0499]

11- Upala S, Sanguankeo A, Saleem SA, Jaruvongvanich V. Effectsof Helicobacter pylori eradication on insulin resistance andmetabolic parameters: a systematic review and meta-anaIysis. Eur JGastroenterologyHepatol 2017; 29: 153-159 [PMID: 27832037 DOI:10. 1097/ M E G. 0000000000000774]

12- Bach JF. The effect of infections on susceptibility to autoimmuneand allergic diseases. N Engl J Med 2002; 347: 911-920 [PMID:12239261 DOI: 10.1056/NEJMra020100]

13- Chen Y, BIaser MJ. Inverse associations of Helicobacter pylori withasthma and allergy. Arch Intern Med 2007; 167: 821-827 [PMID:17452546 DOI: 10.1001/archinte.167.8.821]

14- Cohen D, Shoham O, Orr N, Muhsen K. An inverse and independentassociation between Helicobacter pylori infection and the incidenceof shigeIIosis and other diarrheaI diseases. Clin Infect Dis 2012; 54:e35-e42 [PMID: 22157171 DOI: 10.1093/cid/cir916]

15- Islami F, Kamangar F. Helicobacter pylori and esophageal cancerrisk: a meta-anaIysis. Cancer Prev Res (Phila) 2008; 1: 329-338[PMID: 19138977 DOI: 10.1 158/1 940-6207.CAPR-08-0109]

16- Perry 5, de Jong BC, Solnick JV, de la Luz Sanchez M, Yang 5, LinPL, Hansen LM, TaIat N, HiII PC, Hussain R, Adegbola RA, FlynnJ, Canﬁeld D, Parsonnet J. Infection with Helicobacter pylori isassociated with protection against tuberculosis. PLoS One 2010; 5:e8804 [PMID: 20098711 DOI:10.1371/journal.pone.0008804]

17- Sonnenberg A, DeIIon ES, Turner KO, Genta RM. The inﬂuenceof Helicobacter pylori on the ethnic distribution of esophagealeosinophilia. Helicobacter 2017; 22: e12370 [PMID: 28029200DOI: 10.1111/heI. 12370

18- Malfertheiner P, Megraud F, O’Morain C, BazzoIi F,El- Omar E, Graham D, et aI. Current concepts in themanagement of Helicobacter pylori infection: The MaastrichtIII Consensus Report. Gut 2007;56:772-81.

19- Graham DY, Sung JY. Helicobacter pylori. In: Feldman M,Friedman LS, Brandt LJ, editors. SIeisenger and Fordtran’sGastrointestinal and Liver Disease. Pathophysiology,Diagnosis, Management. 7th ed. Philadelphia: WB SaundersCo; 2006. p. 1049- 66.

20-PrevaIence of Helicobacter Pylori Infection AmongPatients Undergoing Upper GastrointestinalEndoscopy in a Medical College Hospital in Kerala,IndiaAdlekha S, Chadha T1, Krishnan P2, Sumangala BDepartments of Pathology, 1Microbiology, and 2Surgery, Sree Narayana Institute of Medical Sciences, Chalakka,Ernakulam,Kerala, India

21-PrevaIence and impact of Helicobacter pylori in dyspepsiaY. Srinivas1, P. Kameshwari Prasad2, N. Divya Sai3

22- AI-Humayed SM, Mohammed-Elbagir AK, AI- WabeIAA, Argobi YA. The changing pattern of uppergastrointestinal lesions in Southern Saudi Arabia: anendoscopic study. Saudi J Gastroenterology 2010;16(1):35-7.

23-Ramin N, Mehrdad S, Mohammad RF, Amirreza D,LaIeh M. Prevalence of Helicobacter pylori in patientswith dyspepsia. Jundishapur J Microbiol.2014;7(9):12676.

24- Suzuki H, Matsuzaki J, Hibi T. What is thedifference between Helicobacter pylori associateddyspepsia and functional Dyspepsia? JNeurogastroenterologyMotil. 2011;17(2):124-30.

25- Chang L. Review article: epidemiology and qualityof life in functional gastrointestinal disorders .Aliment PharmacolTher. 2004;20(7):31-9.

26- Kumar A, Pate J, Sawant P. Epidemiology offunctional dyspepsia. J Assoc Physicians India2012;60:9-12.

27-Lan L, Yu J, Chen YL, Zhong YL, Zhang H, JiaCH. Symptom- based tendencies of Helicobacterpylori eradication in patients with FunctionalDyspepsia. World J Gastroenterology 2011;17(27):3242-7.

28-Oshima T, Miwa H. Treatment of functionaldyspepsia: where to go and what to do. JGastroenterol. 2006;41 (7): 718- 9

29. Tarkhashvili N, Beriashvili R, Chakvetadze N, Moistsrapishvili M, Chokheli M, Sikharulidze M, et al. Helicobacter pylori infection in patients undergoing upper endoscopy, Republic of Georgia. Emerg Infect Dis 2009;15:504‑5.

30.Shokrzadeh L, Baghaei K, Yamaoka Y, Shiota S, Mirsattari D, Porhoseingholi A, et al. Prevalence of Helicobacter pyloriinfection in dyspeptic patients in Iran. Gastroenterol Insights 2012;4:24‑7.

31.Kaore NM, Nagdeo NV, Thombare VR. Comparative evaluation of the diagnostic tests for Helicobacter pylori and dietary influence for its acquisition in dyspeptic patients:A rural hospital based study in central India. JCDR 2012;6:636‑41

32. Hermann Brenner, Dietrich Rothenbacher, Günter Bode, Guido Adler‏، relation of smoking and alcohol and coffee consumption to active Helicobacter pylori infection: cross sectional study

33. Ahmad Kumo Bello1, Ali Bala Umar2, Musa Muhammad Borodo3 ،

Prevalence and risk factors for helicobacter pylori infection in gastroduodenal diseases in Kano, Nigeria

## 34.relation to Helicobacter pylori infection and NSAID use.

al-Assi MT, et al. Endoscopy. 1996.Prevalence of Helicobacter Pylori Infection Among Patients Undergoing Upper Gastrointestinal

35.Jemilohun AC, Otegbayo JA, Ola SO, Oluwasola OA, Akere A. Prevalence of Helicobacter pylori among Nigerian patients with dyspepsia in Ibadan. Pan Afr Med J 2010;6:18.

36.Endoscopy in a Medical College Hospital in Kerala,

India Adlekha S, Chadha T1, Krishnan P2, Sumangala B Departments of Pathology, 1

Microbiology, and 2 Surgery, Sree Narayana Institute of Medical Sciences, Chalakka, Ernakulam,

Kerala, India

# 37.Helicobacter pylori chronic gastritis updated Sydney grading in relation to endoscopic findings and H. pylori IgG antibody: diagnostic methods

Taha M.M. Hassan, Samia I. Al-Najjar, [...], and Malek G. Alotibi

38. Zapata‑Colindres JC, Zepeda‑Gómez S, Montaño‑Loza A, Vázquez‑Ballesteros E, de Jesús Villalobos J, Valdovinos-Andraca F. The association of Helicobacter pylori infection and nonsteroidal anti‑inflammatory drugs in peptic ulcer disease.Can J Gastroenterol 2006;20:277‑80.

39. Ahmad FF, Jaffar R, Khan I. Helicobacter Pylori detection in chronic gastritis: A comparison of staining methods. J Ayub Med Coll Abbottabad 2011;23:112‑14

***Thank you***