



Research

The Effect and the Histopathological Changes of Helicobacter Pylori Infection on the Duodenal Mucosa **Among Dyspeptic Patients**

Dispeptik Hastalarda Helicobacter Pylori Enfeksiyonunun Duodenum Mukozası Üzerindeki Etkisi ve Histopatolojik Değişiklikleri

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ABSTRACT

Objective: Helicobacter pylori (H. pylori) infection is the main cause of dyspepsia, as it leads to duodenitis and subsequently duodenal ulcers. Early diagnosis of these bacteria by an invasive gastroscopic method combined with a histopathological study is important for eradication of these bacteria to prevent further consequences.

Aim of the study to explore the prevalence of H. pylori infection and duodenal histological changes in dyspeptic patients.

Methods: This cross-sectional study consisted of 125 patients who complained of dyspepsia and underwent endoscopy with biopsy taken at Al-Kindy Teaching Hospital in Baghdad, Iraq January 2015 to January 2023.

Results: The age of the patients ranged from 16-70 years (33.16±12.39). Among all dyspeptic patients, 75 (60%) were males and 50 (40%) were females. The histological results showed that the overall prevalence of H. pylori was 16% (20 cases) of all cases of duodenitis, and the rest, 84% (105 cases), were negative. Histopathological examination of duodenal mucosa showed a significant increase in villous broadening and shortening by 49.52% in duodenitis without H. pylori infection, while in duodenitis with H. pylori infection, it was 25% p=0.043.

Conclusion: These results showed a lower prevalence of H. pylori among dyspeptic patients with duodenitis, along with a significant histopathologic feature regarding duodenal villi.

Keywords: Helicobacter pylori, duodenitis, histopathology

ÖZ

Amaç: Helicobacter pylori (H. pylori) enfeksiyonu, daha sonra duodenit ve duodenum ülserlerine bağlı dispepsinin başlıca nedenidir. Bu bakterilerin invaziv gastroskop yöntemi ile histopatoloji çalışmasıyla erken teşhisi, bu bakterilerin daha fazla sonuç doğurmasını önlemek için erken eradikasyonu açısından önemlidir.

Dispeptik hastalarda H. pylori enfeksiyonunun ve duodenum histolojik değişikliklerinin yaygınlığını araştırmak.

Gereç ve Yöntem: Bu kesitsel çalışma, Ocak 2015 ile Ocak 2023 arasında Irak-Bağdat'taki Al-Kindy Eğitim Hastanesi'nde dispepsi şikayetiyle endoskopi yapılan ve biyopsi alınan 125 hastadan oluşuyordu.

Bulgular: Hastaların yaşları 16-70 yıl (33,16±12,39) arasında değişiyordu. Tüm dispeptik hastalar arasında 75 (%60) erkek ve 50 (%40) kadındı. Histolojik sonuçlar, H. pylori'nin genel yaygınlığının tüm duodenit vakalarının (20) %16'sı olduğunu ve geri kalanının (105) (%84) negatif olduğunu gösterdi. Duodenum mukozasının histopatolojik incelemesi, H. pylori enfeksiyonu olmayan duodenitlerde villöz genişleme ve kısalmada önemli bir artış (%49,52) gösterirken, H. pylori enfeksiyonu olan duodenitlerde bu artış (%25) idi (p=0,043).

Sonuç: Bu sonuçlar, duodenitli dispeptik hastalarda H. pylori'nin daha düşük yaygınlığını ve duodenum villusları ile ilgili önemli bir histopatolojik özelliği gösterdi.

Anahtar Kelimeler: Helicobacter pylori, duodenit, histopatoloji

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INTRODUCTION

Dyspepsia is a term originating from two Greek words: the first is "Dus", meaning bad, and the second is "Peptien", which indicates digestion of the upper gastrointestinal tract (1). Globally, 10-37.9% of the population complain of dyspepsia depending on the geographical area (2). In Iraq, it affects about 26-41% of the population (3). The main cause of dyspepsia is pathologies of the upper gastrointestinal tract, such as gastric ulcers, duodenal ulcers, esophagitis, and tumors of the digestive tract (4). Other causes include and bacterial growth of gastrointestinal tract like Helicobacter pylori (H. pylori) infection which is the most common bacterial infection of the stomach and duodenum (5,6). Duodenum is the first part of small intestine that receives partially digested food from the stomach and more liable to be colonized with *H. pylori* in the duodenal bulb causing duodenitis and kissing ulcers especially in patients who had unusual anatomy of the bulb (7,8). The main mechanism of this disease is the hypersecretion of acid and gastrin, which leads to the development of gastric metaplasia in the proximal part of the duodenum, which is colonized by H. pylori, and a decrease in the secretion of bicarbonate (9). This infection will stimulate the immune system, causing infiltration of neutrophils, resulting in mucosal tissue damage due to the release of proteolytic enzymes and initiation of reactive oxygen metabolites in the duodenal epithelial cells (10). Other inflammatory cells will infiltrate the mucosal tissue are lymphocytes especially in patients infected with cytotoxin-associated gene A positive H. pylori in the bulb of the duodenum with expression of toll-like receptor-2 (TLR) and TLR 10 genes in the histopathological tissues in gastroduodenal disorders with H. pylori infection (11). There is also villous obliteration and intraepithelial lymphocytosis with interleukin 37 and the chemokine C-X-C motif chemokine ligand 9 secretions which is the common histopathological features of H. pylori infection causing duodenitis (12).

This study will explore the prevalence of *H. pylori* infection and duodenal histopathology in dyspeptic patients.

METHODS

This cross-sectional retrospective study consisted of 125 patients who complained from dyspepsia according to the Rome III criteria (presence of early satiation, postprandial fullness, epigastric pain or burning, in the absence of an organic, systemic or metabolic disease) that referred from their physicians for endoscopy at Al-Kindy Teaching Hosppital-Gastroscope Unitfrom January 2015 to January 2023. The study was approved by the Ethical and Scientific

Committee of Al-Kindy College of Medicine and the Ethical and Scientific Committee of Scientific Unit Medical Ethics Committee (decision number: 8, date:14.12.2023). The informed consent was not applicable. The inclusion criteria were patients aged more than15 years of age who complained from dyspepsia and upper abdominal pain while the exclusion criteria were subjects who had evidence or history of gastroduodenal malignancies, duodenal ulcer, gastric ulcer, hepatobiliary or pancreatic diseases, history of drugs intake like immunosuppressive therapy, proton pump inhibitors, antibiotics, non-steroidal anti-inflammatory drugs, pregnancy, hepatic or renal failure. Demographic data, including age and gender, were collected from the patients. All patients were fasting for liquids and foods and exposed to local anesthesia (about 6-9 puffs of 10% Lidocaine spray in their oropharynx) then endoscopy was performed using a flexible gastroscope GIF-H260; Olympus, Tokyo, Japan and display screen; Olympus optical endoscopic visualization-261H liquid crystal display monitor; Olympus, Tokyo, Japan. Four duodenal biopsies were taken from the bulb of the duodenum and others from the antrum and the corpus of the stomach for the detection of H. pylori and histopathological examination.

Histopathological analysis: Biopsies were fixed in 10% formalin, then embedded in paraffin blocks and cut in consecutive 3 μ m sections. Slides were stained with Hematoxylin and eosin stain and modified Giemsa stain, then were examined blindly by a pathologist.

Statistical Analysis

Data were evaluated using software Statistical Package for the Social Sciences (SPSS) version 26. Descriptive statistics such as frequencies and percentages were calculated. Unpaired Student's t-test was used for comparing the mean values of two groups. The independent-sample chi-square test was used to analyze related categorical variables. A p-value equal to or less than 0.05 was considered statistically significant.

RESULTS

The age of the patients ranged from 16-70 years (33.16 ± 12.39) years. Among all dyspeptic patients, 75 (60%) were males and 50 (40%) were females. The histological results, showed that the overall prevalence of *H. pylori* was 16% (20) of all cases of duodenitis, and the rest, 84% (105), were negative (Figure 1). Macroscopically, the biopsies were tiny, consisting of about two to three soft gray-whitish pieces, each one about 0.2-0.8 cm. Histopathological examination of duodenal mucosa (Figure 2) showed a significant increase in villous broadening and shortening

(49.52%) in duodenitis without H. pylori infection, while in duodenitis with H. pylori infection the percentage was 25% (p=0.043). Other histologic features like lymphoplasmacytic cells infiltrating the lamina propria, neutrophil infiltration, increased intra-epithelial lymphocytes, crypt hyperplasia, atypical lymphocytes, and gastric metaplasia demonstrated no significant differences between presence or absence of H. pylori infection (Table 1).

DISCUSSION

Duodenitis is an inflammation in the duodenal mucosal lining that occurs either alone or with gastritis. In this study, H. pylori constitutes about 16% of the causative agent of duodenitis, while another study showed that 90.5% were H. pylori positive (13). There is an association between the density of *H. pylori* infection, the inflammatory response, and neutrophil infiltration of the mucosa (14). In the United Kingdom, H. pylori was common with increasing age, causing gastritis and duodenitis, which is the opposite of the results of this study, which showed it to be common in the younger age group (33.16 ± 12.39) (15). This may be due to the sample size, differences in the clinical presentation of the patients, environmental factors, dietary habits, smoking, alcohol consumption, and history of other diseases. Another study illustrated that H. pylori prevalence was 82.6% in dyspeptic patients and proximal duodenitis was 37.7%, while distal



Figure 1. Prevalence of H pylori in duodenitis patients

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duodenitis was 16.9% (16). Eradication of H. pylori in dyspeptic patients with microscopic duodenitis leads to more improvement in their symptoms to a greater extent than in those without microscopic changes of duodenitis (17). Mild inflammation of mucosa of the duodenum is very common in asymptomatic cases, and infiltration with polymorphonuclear cells like neutrophils indicates the activity of the inflammatory process in symptomatic patients, that may progress to duodenal ulcer (18). Even children who are positive for *H. pylori* have symptomatic dyspepsia (19). Thus, duodenal biopsy is very important in diagnosis or monitoring many diseases (20). The gastric metaplasia in this study was 9.52% with H. pylori infection, while another study showed that the amount of H. pylori in the duodenal bulb was associated with the pathogenesis of duodenal ulcer and the extent of gastric metaplasia in the duodenal bulb (21). Moreover, gastric metaplasia in the duodenum was 87.7% in patients with non-complicated duodenal ulcer and 9.8% in patients with complicated duodenal ulcer, with a sensitivity of 83.6%, specificity at 92.8%, predictive accuracy value of 88.7%, relative risk of the predicted outcome at 7.5, relative risk of a different outcome at 0.11, odds ratio at



Figure 2. Duodenal villous with Neutrophils infiltration in the core (Arrow). (H&E stain , 40×HPF)

Table 1. Histopathologic features of duodenitis in dyspeptic patients regarding H. pylori					
Histological Features	<i>H. pylori</i> neg No.=105 No.%		<i>H. pylori</i> positive No.=20 No. %		p-value
Lymphoplasmacytic cells infiltrating of lamina propria	104	99.04	19	95	0.186
Villous broadening and shortening of mild degree	52	49.52	5	25	*0.043
Increase intra-epithelial lymphocytes	80	64	18	90	0.169
Crypts hyperplasia	40	38.09	6	30	0.491
Atypical lymphocytes.	4	3.80	0	0	NA
Gastric metaplasia	10	9.52	0	0	NA
*Significant, NA: Not applicable					

65.4. As a result, the predictive value of gastric metaplasia in the duodenum may be used as a marker of the noncomplicated clinical course of duodenal ulcer in *H. pylori* patients (22).

CONCLUSION

These results showed a lower prevalence of *H. pylori* among dyspeptic patients with duodenitis, along with a significant histopathologic feature regarding duodenal villi.

ETHICS

Ethics Committee Approval: The study was approved by the Scientific Unit Medical Ethics Committee of Al-Kindy College of Medicine (decision no: 8, date: 14.12.2023).

Informed Consent: The informed consent was not applicable.

FOOTNOTES

Authorship Contributions

Surgical and Medical Practices: M.N., B.M.M., J.A., Consept: M.N., B.M.M., J.A., Design: M.N., B.M.M., J.A., Data Collection or Processing M.N., B.M.M., J.A., Analysis or Interpretation: M.N., B.M.M., J.A., Literature Search: M.N., B.M.M., J.A., Writing: M.N., B.M.M., J.A.

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REFERENCES

- Barbara L, Camilleri M, Corinaldesi R, Crean GP, Heading RC, Johnson AG, et al. Definition and investigation of dyspepsia. Consensus of an international ad hoc working party. Dig Dis Sci. 1989;34:1272-6.
- Arnaout AY, Alhejazi TJ, Nerabani Y, Hamdan O, Arnaout K, Arnaout I, et al. The prevalence and risk factors of functional dyspepsia among adults in low- and middle-income countries: An international cross-sectional study. Medicine (Baltimore). 2023;102:e35437.
- Al-Khazraji JKA. The speed of eating and functional dyspepsia. JFacMedBagdad. 2016;58:348-53.
- Costa MB, Azeredo IL Jr, Marciano RD, Caldeira LM, Bafutto M. Evaluation of small intestine bacterial overgrowth in patients with functional dyspepsia through H2 breath test. Arq Gastroenterol. 2012;49:279-83.
- Omer SF, Ali MK. The isolation and identification of H. pylori among Iraq patients with chronic gastric inflammation. J Fac Med Baghdad. 2022;64:102-8.

- AL-Hadithi HS. Association between Helicobacter pylori infection & atrophic gastritis. J Fac Med Baghdad. 2007;48:461-2.
- Avramenko AA. influence of pecularities of anatomical structure (shape) of duodenal bulb on the mechanism of formation of "kissing" ulcers of patients with chronic non-atrophic gastritis. Wiad Lek. 2020;73(12 cz 1):2568-71.
- Alzerwi N. Post-bulbar duodenal ulcer with anterior perforation with kissing ulcer and duodenocaval fistula: A case report and review of literature. World J Clin Cases. 2022;10:9071-7.
- Olbe L, Fandriks L, Hamlet A, Svennerholm AM, Thoreson AC. Mechanisms involved in Helicobacter pylori induced duodenal ulcer disease:an overview. World J Gastroenterol. 2000;6:619-23.
- 10. Weiss SJ. Tissue destruction by neutrophils. N Engl J Med. 1989;320:365-76.
- Mohammed SK, Rasheed MN, Asker BA, Adhiah AH, Gabber Z, Abbood SA. Circulating interleukin 37 and the chemokine CXCL9: Studies on gastroduodenal disorders with H. pylori infection. Iraqi J Biotechnol. 2024;23:128-36.
- Mohammed SK, Rasheed MN, Asker BA. The expression of genes TLR2 and TLR10 in the gastric tissue of patients with gastroduodenal disorders caused by Helicobacter pylori. Baghdad Sci. J. 2024;21:2500-11.
- Phull PS, Price AB, Stephens J, Rathbone BJ, Jacyna MR. Histology of chronic gastritis with and without duodenitis in patients with Helicobacter pylori infection. J Clin Pathol. 1996;49:377-80.
- 14. Saha DR, Datta S, Chattopadhyay S, Patra R, De R, Rajendran K, et al. Indistinguishable cellular changes in gastric mucosa between Helicobacter pylori infected asymptomatic tribal and duodenal ulcer patients. World J Gastroenterol. 2009;15:1105-12.
- McNulty CAM. The first 5 years of Helicobacter pylori research-with an emphasis on the United Kingdom. Helicobacter. 2023;28:e12982.
- Caballero-Plasencia MR, Caballero-Mateos AM, Caballero-Plasencia AM. Inflammatory map of the gastroduodenal mucosa in patients with upper gastrointestinal symptoms. The role of H. pylori infection. Rev Gastroenterol Mex (Engl Ed). 2023;88:238-45.
- Mirbagheri SS, Mirbagheri SA, Nabavizadeh B, Entezari P, Ostovaneh MR, Hosseini SM, et al. Impact of microscopic duodenitis on symptomatic response to Helicobacter pylori eradication in functional dyspepsia. Dig Dis Sci. 2015;60:163-7.
- Serra S, Jani PA. An approach to duodenal biopsies. J Clin Pathol. 2006;59:1133-50.
- Hossain MS, Das S, Begum SMKN, Rahman MM, Mazumder RN, Gazi MA, et al. Asymptomatic duodenitis and Helicobacter pylori associated dyspepsia in 2-year-old chronic malnourished Bangladeshi slum-dwelling children: A cross-sectional study. J Trop Pediatr. 2021;67:fmaa079.
- Friedel D, Sharma J. Duodenal Biopsy. StatPearls [Internet]. 2023 Jul 24. Treasure Island (FL): StatPearls Publishing; 2023.
- Futami H, Takashima M, Furuta T, Hanai H, Kaneko E. Relationship between Helicobacter pylori infection and gastric metaplasia in the duodenal bulb in the pathogenesis of duodenal ulcer. J Gastroenterol Hepatol. 1999;14:114-9.
- Marshalko OV, Konorev MR. The prognostic value of gastric metaplasia in the duodenal mucosa in patients with Helicobacter pylori positive duodenal bulb ulcer. Klin Med (Mosk). 2008;86:43-8.