

Prevalence of *Helicobacter pylori* infection among dyspeptic patients attending Baghdad medical city complex

Basim M. Ibrahim¹, Huda Saad Salman^{2*}, Mohammed Mazin Mohammed¹, Hala Mohammed Mjeed²

¹Department of Microbiology, Collage of Medicine, University of Baghdad, Baghdad, Iraq

²Department of Microbiology, Collage of Medicine, Ibn Sina University of Medical and Pharmaceutical Sciences, Baghdad, Iraq

Received: November 2024, Accepted: April 2025

ABSTRACT

Background and Objectives: Dyspepsia is a disorder characterized by difficulty in digestion and represents a major health concern. Therefore, it is crucial to identify functional dyspepsia linked to *Helicobacter pylori* (*H. pylori*). This research aimed to determine the prevalence of *H. pylori* among patients with dyspepsia and to examine the potential risk factors associated with the infection.

Materials and Methods: From August 14th to September 21st, 2024, a total of 105 patients with dyspepsia, who attended the Central Laboratory of Baghdad Medical City Complex (Iraq), were enrolled in this study. Data on nonsteroidal anti-inflammatory drugs (NSAIDs), smoking, family history, fasting habits and frequent fast food consumption were collected through participant interviews.

Results: Based on the urea breath test results, dyspeptic patients were categorized into infected (63.8%) and non-infected (36.2%) groups. Factors that influenced these patients included the intake of NSAIDs (48.6%), smoking (21.9%), family history (29.5%), fasting habits (36.2%) and regular consumption of fast food (57.1%).

Conclusion: Dyspeptic patients exhibit a high prevalence of *H. pylori* infection, indicating the significant impact of *H. pylori* on this population. However, the intake of NSAIDs, smoking, family history, fasting habits and regular fast food consumption have no significant effects on the presence of *H. pylori*.

Keywords: Dyspepsia; *Helicobacter pylori*; Infection control

INTRODUCTION

Dyspepsia is a condition that impacts the upper digestive tract and presents with a range of symptoms. These may include upper abdominal discomfort, a feeling of fullness soon after starting a meal, bloating, distension, heartburn, belching, nausea, vomiting, or pain (1). In the Western world, the prevalence of dyspepsia is estimated to be around 20-25% (2).

Dyspepsia is generally categorized into two main types: organic and functional (FD) dyspepsia. Organic dyspepsia can be caused by conditions such as peptic ulcers, gastroesophageal reflux disease, gastric or esophageal cancer, issues with the pancreas or biliary system, food or drug intolerances, and viral or systemic diseases (3). However, the pathophysiology of FD has not been fully understood. Studies have demonstrated that the interaction between the

*Corresponding author: Huda Saad Salman, M.Sc, Department of Microbiology, Collage of Medicine, Ibn Sina University of Medical and Pharmaceutical Sciences, Baghdad, Iraq. Tel: +964-7713364348 Fax: +7725312118 Email: huda.alsaeedi@ibnsina.edu.iq

gastrointestinal system and the brain can lead to motility disorders, increased sensitivity of the visceral organs, and changes in the composition of gastrointestinal microbiota, as well as alterations in mucosal and immune functions and central nervous system processing (4). FD may develop due to a combination of genetic predisposition, infection by *H. pylori* or other pathogens, inflammation and psychosocial factors (5).

Dyspepsia is often classified into ulcerous and non-ulcerous forms based on its etiology. Nonetheless, investigations have indicated that this classification is inadequate, as ulcer disease is merely one of the potential organic causes of the dyspeptic symptoms (6). A more accurate classification divides dyspepsia into organic and non-organic (FD). While this classification is practically appropriate, the differentiation between organic dyspepsia and FD can sometimes be arbitrary and is dependent on the depth of the investigation conducted (7). Three distinct forms of dyspepsia can be identified, which entail dyspepsia with a known metabolic or organic cause, dyspepsia symptoms with undistinguished cause and dyspepsia of unknown etiology. In the first form of dyspepsia, its symptoms are reduced or eliminated with any improvement in the underlying condition. Contributing factors include peptic ulcer disease, stomach cancer, biliopancreatic disorders and medication-related issues. The third form of dyspepsia is common because many individuals do not need a comprehensive evaluation when they do not meet the age requirements and exhibit no concerning symptoms (8, 9). Research has demonstrated a connection between *H. pylori* and various disorders, including peptic ulcers and stomach cancer, which often present with dyspepsia. The bacteria may induce dyspepsia without any significant structural alterations (10). Infection with *H. pylori* has been shown to cause progressive functional and structural damage to the gastroduodenal region, which can unpredictably progress to peptic ulcer disease and its complications including atrophic gastritis or stomach cancer (11).

H. pylori is a Gram-negative bacterium that thrives in microaerophilic conditions. It elicits an inflammatory response marked by the presence of neutrophils, lymphocytes, plasma cells and macrophages within the mucosal layer of the gastrointestinal tract, resulting in the degeneration and destruction of epithelial cells. Gastritis typically exhibits greater severity in the antrum of the stomach, with minimal or absent

inflammation in the corpus (12). *H. pylori* infection is primarily acquired during early childhood and often persists into adulthood, as its spontaneous clearance is rare. Factors such as age, gender, ethnicity and various socioeconomic parameters are associated with *H. pylori* infection (13). More than half of the global population is infected with *H. pylori*, and approximately 20% of those infected will develop *H. pylori*-related clinical conditions, particularly chronic inflammation of the stomach lining (14). Chronic gastritis is associated with peptic ulcer disease and in its late stages, it is linked to an increased risk of stomach adenocarcinoma (15).

H. pylori bacterium can be diagnosed using non-invasive and invasive techniques. Noninvasive methods include the urea breath test (UBT) and stool antigen test (16). Endoscopy, an invasive procedure, enables the collection of a biopsy that can undergo a range of testing methodologies, such as histology, culture, or rapid urease assay (17). All these methods can be affected by the use of acid-suppressing medications, including proton pump inhibitors (PPIs), which may lead to false-negative results (18).

The relationship between *H. pylori* and FD remains unclear. However, clinical trials present opportunities to evaluate the effectiveness of *H. pylori* treatment in cases of non-ulcer dyspepsia. The eradication of *H. pylori* has demonstrated efficacy for a specific subset of patients experiencing FD (19). Dyspepsia associated with *H. pylori* can be considered a distinct disease entity, separate from FD (20). This distinction informs physicians about the significance of this bacterial pathogen and its role in the pathogenesis of dyspepsia. The present study aimed to determine the prevalence of *H. pylori* among patients with dyspepsia. The findings will provide physicians with valuable insights into the importance of this bacterial pathogen and its role in the development of dyspepsia.

MATERIALS AND METHODS

Study design, location, and timing. A total of 105 dyspeptic patients attending the Central Laboratories Department at Baghdad Medical City Complex (Iraq) were enrolled in this study from August 14th to September 21st, 2024. Data on the use of NSAIDs, smoking, alcohol consumption, family history, fasting habits and regular intake of fast food were collected through interviews with the participants.

Inclusion and exclusion criteria. Patients suffering from dyspepsia without any other microbial infections or specific medical conditions were included in this study. Samples from patients were excluded if they had a concurrent acute illness, had consumed antibiotics within the past four weeks, or had taken PPIs within the past two weeks. Dyspepsia is defined in this study as an ongoing or recurring discomfort in the upper abdomen accompanied by symptoms such as the feeling of fullness, early satiety, nausea, heartburn, belching, or epigastric pain. According to the Rome IV criteria for FD, patients were required to have symptoms lasting at least three months and occurring at least six months apart. Exclusion criteria also included peptic ulcers, gastroesophageal reflux disease, gastric or esophageal cancers, systemic diseases affecting digestion and recent use of PPIs or antibiotics, as these factors could interfere with *H. pylori* detection. This standardized criterion ensured that only participants with clinically significant dyspeptic symptoms were included in the study. To identify potential risk factors for *H. pylori* infection in individuals with dyspepsia, specific criteria were established regarding the use of NSAIDs, smoking, eating habits and family history. In this regard, individuals who had taken NSAIDs at least three times per week in the month before enrolment were categorized as NSAID users. Occasional or one-time consumption was insufficient for classification as an NSAID user. Any individual who smoked at least one cigarette or other tobacco products daily for a minimum of six consecutive months was considered a smoker. Those who smoked less than one cigarette per day occasionally or in social situations were not classified as smokers. A pattern of fast food consumption involved the intake of commercially prepared meals at least three times per week for three months. Individuals who consumed fast food less frequently were categorized as non-regular consumers. Subjects who engaged in fasting for dietary or religious reasons for two or more days each week for at least three months were considered to have regular fasting habits. A positive family history was defined as having one first-degree relative (parent or sibling) with an *H. pylori* infection or a documented history of peptic ulcer disease.

Patient preparation. In this study, patients discontinued using antibiotics and bismuth compounds, such as Pepto-Bismol, for 30 days before the test.

Similarly, the administration of sucralfate (Carafate) and PPIs, such as omeprazole (Prilosec) or lansoprazole (Prevacid), was stopped two weeks before the examination. Additionally, patients refrained from eating or drinking for at least six hours before the test. For each patient, we collected a comprehensive medical history, including a detailed list of all relevant medications, along with the dates of their most recent administration.

Radiopharmaceuticals. The capsule contained 1,000 µg of urea labelled with 37 kBq (1/xCi) of Carbon-14. Carbon-14 has a half-life of 5,730 years and is classified as a beta emitter with a maximum energy of 160 keV. Beta emissions are measured by counting ¹⁴C in a liquid scintillation counter.

UBT procedure. The UBT procedure was initiated with the patient ingesting a capsule and 20 ml of lukewarm water. The patient was then instructed to consume 20 ml of warm water three minutes after the initial intake. At the 10-minute mark post-ingestion, the individual was instructed to inhale deeply, pause their breath for about 5 to 10 seconds, and slowly release the air through a straw into a Mylar balloon. An additional breath sample, collected in a separate balloon, may optionally be obtained 15 minutes after ingestion. There was no consensus on the cut-off value for identifying *H. pylori*-negative individuals in the ¹⁴C UBT (21). The manufacturer has established the cut-off for the ¹⁴C UBT at 50. Based on this threshold, the analysis demonstrated a sensitivity of 92.31%, a specificity of 95.24%, a positive predictive value of 85.71% and a negative predictive value of 85.71%. (22).

Statistical analysis. Data were presented using basic statistical measures, including count, mean and standard deviation, utilizing IBM SPSS Statistics (version 24). A statistical analysis was conducted to evaluate the significance of the results. The student's t-test was applied to compare the means of two sets of quantitative data, while the chi-square test was utilized for analyzing qualitative variables. A p-value less than 0.05 was regarded as indicative of statistical significance.

Ethical clearance. The medical ethics committee at Ibn Sina University of Medical and Pharmaceutical Sciences approved this study.

RESULTS

The study enrolled 105 participants with dyspeptic symptoms, 63 (60%) of whom were females (Table 1). Additionally, participants were categorized by age, with 14 years as the lower age limit and 74 years as the upper age limit. The highest number of cases was observed in the age group of 31-40, while the lowest cases were found among patients older than 60. The results are illustrated in Table 2.

Based on UBT results, participants were categorized into two groups: those with positive *H. pylori* (63.8%) and those with negative *H. pylori* (36.2%). The study also monitored the percentage of NSAID intake, smoking, family history, fasting habits and regular fast food consumption.

In this study, we found that the association of factors such as NSAIDs intake, smoking, family history, fasting habits and regular consumption of fast food with *H. pylori* was not statistically significant, as indicated by p-values >0.05. Among the patients who tested positive for *H. pylori*, 57.1% were identified as frequent consumers of fast food. These results are presented in Table 3.

Table 1. Gender base distribution of the participants

Parameters	Male	Female	Total
Number of cases	42	63	105
%	40%	60%	100%

Table 2. Age distribution of the patients

Age groups (years)	Number of participants (%)
11-20	20 (19)
21-30	26 (24.8)
31-40	30 (28.6)
41-50	17 (16.2)
51-60	8 (7.6)
> 60	4 (3.8)

DISCUSSION

Dyspepsia is a medical term that encompasses symptoms such as indigestion, nausea, heartburn and regurgitation (23). These symptoms can be linked to various conditions, making the diagnosis of dyspepsia challenging (24). While dyspepsia is

Table 3. Association of different factors with *H. pylori*

Factors	Positive <i>H. pylori</i>		Negative <i>H. pylori</i>		p-values
	Yes	No	Yes	No	
	NSAID intake	30	37	21	
Smoking	12	55	11	27	0.189
Family history	21	46	10	28	0.587
Fasting habit	21	46	17	21	0.170
Regular consumption of fast food	40	27	20	18	0.482

a common complication associated with *H. pylori* infection, many individuals remain asymptomatic. Furthermore, numerous patients with dyspepsia exhibit different responses to the eradication of *H. pylori*, with outcomes ranging from complete resolution to inconsistent improvement, and in some cases, no improvement at all. Nevertheless, eradicating *H. pylori* remains the most effective treatment for dyspepsia, particularly FD (25).

According to the results, the prevalence of *H. pylori* infection among dyspeptic patients in Iraq was 63.8%. This finding aligns with the results of other studies (26-28), although those studies reported a comparatively lower frequency of *H. pylori* occurrence. The prevalence rates of the *H. pylori* infection in Malaysia and Kuwait were relatively similar, approximately 49% (29, 30). In Iran, the histological evidence of *H. pylori* infection was documented in 89.2% of biopsies collected from dyspeptic patients (31). In contrast, a similar study reported a prevalence rate of 47.9% among 180 patients who underwent endoscopic evaluation (28). Additional research indicated that the prevalence among dyspeptic patients in Sulaimani City, Iraq was 54.9% (32), while patients with gastroduodenal problems in Erbil City, Iraq had a prevalence of 53.3% (33). The rate of *H. pylori* infection in Wuwei City, China, was reported to be 53.0% (34). In Duhok, the infection rates were 37.2% by culture, 68% by the rapid urease test (35) and 28% by the anti-*H. pylori* IgG test (36) among pediatric patients. Furthermore, research conducted in Ramadi, Iraq, found a rate of 68.7% (32, 37). A study in Kuwait on the prevalence rate of *H. pylori* infection among dyspeptic outpatients revealed an overall prevalence rate of 49.7% in 362 patients screened using the UBT. The prevalence varied significantly by age; patients aged 20-29 years exhibited a lower rate of 35.8%, while those aged 30-39 years had a

higher rate of 59.3%. The study also exhibited that the prevalence rate among Kuwaiti expatriates was higher (57.6%) compared to that of Kuwaiti nationals (42.6%), highlighting the influence of socioeconomic factors on the spread of infections. Additionally, the research identified gender differences among expatriates, with male patients reporting a higher prevalence than female patients (30).

Comparing the findings of our study with those from Iraq, where the prevalence of *H. pylori* infection was reported as 63.8%, demonstrates a significantly higher rate in Iraq. This difference could be due to factors such as sanitation, socioeconomic conditions and the lack of an advanced healthcare infrastructure. However, Malaysia reported an *H. pylori* prevalence of 23.5% among dyspeptic patients, indicating that the prevalence in Iraq considerably exceeds that of Malaysia, despite the challenges of sanitation and limited healthcare access in Iraq. The Malaysian study also noted a relatively lower incidence of *H. pylori*-related dyspepsia, which is likely to suggest improved socioeconomic conditions and better hygiene practices in Malaysia, resulting in a reduced risk of transmission. Regarding the diagnostic methods employed, both studies utilized the UBT, ensuring the high accuracy and comparability. The impressive treatment success rate of 95.7% observed in the Malaysian research emphasizes the effectiveness of appropriate treatment in eradicating *H. pylori* infection and alleviating dyspeptic symptoms. In Iraq, however, various factors may contribute to the observed treatment failure rates, such as antibiotic resistance or inconsistencies in treatment protocols (38). Confounding variables, including socioeconomic status, sanitary conditions and hygiene habits could also influence these issues. The effects of diet and smoking may be less relevant in areas with easy access to healthcare and sanitation, as other environmental determinants such as overcrowded living conditions or water quality may play a more significant role in transmitting *H. pylori*. Indeed, overcrowding and poor sanitation have been identified as significant risk factors for infection in studies conducted in Ethiopia, Afghanistan and Iraq.

Our results demonstrated that using the 14C UBT as a noninvasive diagnostic procedure while excluding other causes of dyspepsia can help prevent various complications, such as peptic ulcers in affected patients. With greater sensitivity than stool antigen testing, the 14C-UBT offers a quick, noninvasive and accurate method for diagnosing *H. pylori* infection in

patients experiencing dyspepsia (39).

In this study, dyspeptic patients who tested positive for *H. pylori* exhibited a non-significant association with NSAID intake, smoking, family history, fasting habits and regular consumption of fast food (33, 34). Research investigating the relationship between tobacco and fast food consumption has yielded inconsistent results. However, several studies have indicated a correlation between smoking, dietary habits, and an increased risk of *H. pylori* infection (35), while others have concluded that neither smoking nor dietary habits significantly affected *H. pylori* infection (36, 37). These conflicting results could arise from the small sample size or the methodology employed in data collection. The geographic region may also play a significant role in the reported data, and potential biases could influence the results.

CONCLUSION

The current study highlights the high prevalence of *H. pylori* among the cohort of dyspeptic patients. Although the study analyzed NSAID intake, smoking, family history, fasting habits and regular fast food consumption, these factors were not significantly associated with *H. pylori* infection. Our findings underscore *H. pylori* as the primary cause of dyspepsia while providing evidence that other lifestyle or demographic factors play a lesser role in infection within this cohort. Noninvasive diagnostic tests like 14C-UBT are recommended for the detection of *H. pylori* in all cases of dyspepsia that have microbial susceptibility since reliable outcomes have been shown through their use.

ACKNOWLEDGEMENTS

The authors thank all participants in this study and wish them a speedy recovery.

We supported this study, as well as the paper preparation and publishing.

REFERENCES

1. Stanghellini V, Chan FK, Hasler WL, Malagelada JR, Suzuki H, Tack J, et al. Gastrointestinal disorders. *Gastroenterology* 2016; 150: 1380-1392.

2. Piriyapong K, Tangaroonsanti A, Mahachai V, Vilaichone R-K. *Helicobacter pylori* infection impacts on functional dyspepsia in Thailand. *Asian Pac J Cancer Prev* 2014; 15: 10887-10891.
3. Miwa H, Nagahara A, Asakawa A, Arai M, Oshima T, Kasugai K, et al. Evidence-based clinical practice guidelines for functional dyspepsia 2021. *J Gastroenterol* 2022; 57: 47-61.
4. Karakan T, Ozkul C, Küpeli Akkol E, Bilici S, Sobarzo-Sánchez E, Capasso R. Gut-brain-microbiota axis: Antibiotics and functional gastrointestinal disorders. *Nutrients* 2021; 13: 389.
5. Oustamanolakis P, Tack J. Dyspepsia: organic versus functional. *J Clin Gastroenterol* 2012; 46: 175-190.
6. Som S, Dutta Banik G, Maity A, Chaudhuri S, Pradhan M. Exhaled nitric oxide as a potential marker for detecting non-ulcer dyspepsia and peptic ulcer disease. *J Breath Res* 2018; 12: 026005.
7. Wang X, Liu H, Li W, Xiao H. Bibliometric analysis of functional dyspepsia research trends over the past 20 years. *Front Public Health* 2022; 10: 1019110.
8. Volarić M, Šojat D, Majnarić LT, Vučić D. The Association between functional Dyspepsia and Metabolic Syndrome-The state of the Art. *Int J Environ Res Public Health* 2024; 21: 237.
9. Duncanson K, Burns G, Pryor J, Keely S, Talley NJ. Mechanisms of food-induced symptom induction and dietary management in functional dyspepsia. *Nutrients* 2021; 13: 1109.
10. Emile SH, Elshobaky A, Elbanna HG, Elkashef W, Abdel-Razik MA. *Helicobacter pylori*, sleeve gastrectomy, and gastroesophageal reflux disease; is there a relation? *Obes Surg* 2020; 30: 3037-3045.
11. Graham DY. *Helicobacter pylori* update: gastric cancer, reliable therapy, and possible benefits. *Gastroenterology* 2015; 148: 719-731.e3.
12. Sășăran MO, Meliț LE, Dobru ED. MicroRNA modulation of host immune response and inflammation triggered by *Helicobacter pylori*. *Int J Mol Sci* 2021; 22: 1406.
13. Altamimi E, Alsharkhat N, AlJawarneh A, Abu Hamad MDR, Assi AA, Alawneh S, et al. Declining prevalence of *Helicobacter pylori* infection in Jordanian children, report from developing country. *Heliyon* 2020; 6(7): e04416.
14. Borka Balas R, Meliț LE, Mărginean CO. Worldwide prevalence and risk factors of *Helicobacter pylori* infection in children. *Children (Basel)* 2022; 9: 1359.
15. Sokolova O, Naumann M. Matrix metalloproteinases in *Helicobacter pylori*-Associated gastritis and gastric Cancer. *Int J Mol Sci* 2022; 23: 1883.
16. Alzoubi H, Al-Mnayyis AA, Al Rfoa I, Aqel A, Abu-Lubad M, Hamdan O, et al. The use of ¹³C-urea breath test for non-invasive diagnosis of *Helicobacter pylori* infection in comparison to endoscopy and stool antigen test. *Diagnostics (Basel)* 2020; 10: 448.
17. Peretz A, On A, Koifman A, Brodsky D, Isakovich N, Glyatman T, et al. An efficiency comparison between three invasive methods for the diagnosis of *Helicobacter pylori* infections: Culture from stomach biopsy, rapid urease test (CUTest®), and histologic examination of gastric biopsy. *Ann Clin Lab Sci* 2015; 45: 148-151.
18. Narayanan M, Reddy KM, Marsicano E. Peptic ulcer disease and *Helicobacter pylori* infection. *Mo Med* 2018; 115: 219-224.
19. Kang SJ, Park B, Shin CM. *Helicobacter pylori* eradication therapy for functional dyspepsia: a meta-analysis by region and *H. pylori* prevalence. *J Clin Med* 2019; 8: 1324.
20. O'Connor HJ. Forty years of *Helicobacter pylori* infection and changes in findings at esophagogastroduodenoscopy. *Helicobacter* 2023; 28(6): e13026.
21. Sankararaman S, Moosavi L (2024). Urea Breath Test. StatPearls Publishing, Treasure Island (FL). <https://pubmed.ncbi.nlm.nih.gov/31194426/>
22. Miftahussurur M, Windia A, Syam AF, Nusi IA, Alfaray RI, Fauzia KA, et al. Diagnostic value of 14C urea breath test for *Helicobacter pylori* detection compared by histopathology in Indonesian dyspeptic patients. *Clin Exp Gastroenterol* 2021; 14: 291-296.
23. Nkurunziza A, Dusabejambo V, Everhart K, Bensen S, Walker T. Validation of the Kinyarwanda-version Short-Form leads dyspepsia questionnaire and Short-Form nepean Dyspepsia index to assess dyspepsia prevalence and quality-of-life impact in Rwanda. *BMJ Open* 2016; 6(6): e011018.
24. Koduru P, Irani M, Quigley EM. Definition, pathogenesis, and management of that cursed dyspepsia. *Clin Gastroenterol Hepatol* 2018; 16: 467-479.
25. Kim SE, Park YS, Kim N, Kim MS, Jo HJ, Shin CM, et al. Effect of *Helicobacter pylori* eradication on functional dyspepsia. *J Neurogastroenterol Motil* 2013; 19: 233-243.
26. Sjomina O, Pavlova J, Niv Y, Leja M. Epidemiology of *Helicobacter pylori* infection. *Helicobacter* 2018; 23 Suppl 1: e12514.
27. Jiang J-X, Liu Q, Mao X-Y, Zhang H-H, Zhang G-X, Xu S-F. Downward trend in the prevalence of *Helicobacter pylori* infections and corresponding frequent upper gastrointestinal diseases profile changes in Southeastern China between 2003 and 2012. *Springerplus* 2016; 5: 1601.
28. Al-Sabbagh WRM, Yahya AQ, Alsafi RA. Are Histopathological changes of *H. pylori* infection in young Dyspeptic patients Necessitate Endoscopy? *Open Access Maced J Med Sci* 2019; 7: 3211-3215.
29. Sharma PK, Salaria S, Manrai M, Srivastava S, Kumar

- D, Singh AR. *Helicobacter pylori* infection in non-ulcer dyspepsia: A cross-sectional study. *Med J Armed Forces India* 2022; 78: 180-184.
30. Alazmi WM, Siddique I, Alateeqi N, Al-Nakib B. Prevalence of *Helicobacter pylori* infection among new outpatients with dyspepsia in Kuwait. *BMC Gastroenterol* 2010; 10: 14.
 31. Leylabadlo HE, Kafil HS, Yousefi M. Gastric cancer mortality in a high-incidence area (Ardabil Province, Northwest Iran): what risk factors are causative? *Eur J Cancer Prev* 2016; 25: 573-574.
 32. Saeed AY, Rashad BH, Ali BN, Sulaivany AH, Ibrahim KS. *Helicobacter pylori* infection: Prevalence, risk factors, and treatment efficacy in Symptomatic patients in zakho city, Kurdistan Region, Iraq. *Cureus* 2024; 16(11): e73873.
 33. Majeed PD, Khoshnaw KJS. Seroprevalence of *Helicobacter pylori* infection among patients with gastroduodenal disorders in Erbil city. *Diyala J Med* 2020; 18: 91-101.
 34. Zhang F, Pu K, Wu Z, Zhang Z, Liu X, Chen Z, et al. Prevalence and associated risk factors of *Helicobacter pylori* infection in the Wuwei cohort of north-western China. *Trop Med Int Health* 2021; 26: 290-300.
 35. Saadi HMS, Saeed AY. Laboratory diagnosis of *H. pylori* among dyspeptic patients using culture and Rapid urease test. *Kurd J Appl Res* 2019; 4: 174-181.
 36. Yahya NB. *Helicobacter pylori* seropositivity in children in Duhok City, Iraq. *Sci J Univ Zakho* 2018; 6: 82-84.
 37. Hussein RA, Al-Ouqaili MT, Majeed YH. Detection of *Helicobacter pylori* infection by invasive and non-invasive techniques in patients with gastrointestinal diseases from Iraq: A validation study. *PLoS One* 2021; 16(8): e0256393.
 38. Abdul Aziz AF, Hamzah Z, Tong SF, Nadeson S, Wan Puteh SE. *Helicobacter pylori* related dyspepsia: prevalence and treatment outcomes at University Kebangsaan Malaysia-Primary care Centre. *Asia Pac Fam Med* 2009; 8: 4.
 39. Malfertheiner P, Mégraud F, O'Morain CA, Gisbert JP, Kuipers EJ, Axon AT, et al. Management of *Helicobacter pylori* infection-the Maastricht V/Florence consensus report. *Gut* 2017; 66: 6-30.