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Research Article

Study of Some Affecting Factors on the Infection with Peptic Ulcer

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ABSTRACT

Background: *H. pylori infection invariably causes active chronic gastritis. In most people, this may be clinically silent throughout life, but in a substantial minority, it causes gastroduodenal diseases* ***Objective:*** *The study was designed to investigate the risk factors associated with perforated peptic ulcer.* ***Patients and materials:*** *In this study involved 51 consecutive patients (32 male and 19 femle) aged from (<20- >50) who underwent endoscopic examination by specialist doctors, all patients were interviewed regarding personal bio data (age, sex, job, marital status, and place of residence), selected risk factors (smoking status, NSAID use, soft drinks, stress, Caffeine, blood group, whether food was spicy or not, family history of peptic ulcer, and presence of chronic comorbidities* ***Results:*** *the result showed great variation between risk factors (32/62.7%) from samples was smoking ,(26/51%) of them drink carbonated drinks , (44/86.3) unusual drink of Caffeine, (43/84.3%) had an extremely stress , (30/58.8%) add spices to meals ,(41/80.4%) of them use NSAIDs, while (29/57%) of them had a family history of Infection of peptic ulcer anther wise the blood groups for all patients samples ,the blood group O was the highest incidence between them and record (25/49%) ,while the(RH^+) factors was high in studied samples(41/80%) in comparison with (RH^-) .* ***Conclusion:*** *These findings suggest that if gastric acid has a role in acid-peptic symptoms and/or healing, milk is not a suitable therapeutic option. The benefit of milk against an appropriate placebo on symptoms or healing in people with acid-peptic illnesses has not been carefully investigated.*

Keywords: *Peptic ulcer, H.pylori, perforated peptic ulcer, Gastric ulcer, Peptic illnesses.*

INTRODUCTION

Since about 40 years ago, Helicobacter pylori has been recognized as an important human pathogen. It is still the most widespread human bacterial pathogen, infecting about half of the world's population, despite the benefits of healing infected people and decreased infection transmission in places where socioeconomic living conditions have grown (Hooi et al., 2017 Aug). As a result, it continues to be a significant global cause of illness and mortality.

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Always Infection with H. pylori causes active chronic gastritis. A considerable minority develop gastroduodenal disorders, such as peptic ulcer disease, noncardiac gastric cancer, and gastric mucosa-associated lymphoid tissue (MALT) lymphoma. The majority of individuals remain clinically silent throughout their entire lifetimes. It also raises the risk of gastroduodenal ulcers and bleeding in people taking nonsteroidal anti-inflammatory medicines (NSAIDs) such as aspirin and is responsible for symptoms in a subset of patients with functional dyspepsia.

H. Pylori has been examined extensively. A literature search returns nearly 45,000 results. The epidemiology of Infection, biology, genetics, pathophysiology, illness manifestation, diagnosis, and treatment have been studied extensively. However, there are considerable information gaps. Despite several epidemiological studies identifying infection risk factors, the specific route of infection transmission remains unknown. Numerous aspects of the relationship between host and pathogen, as well as the factors of disease expression, remain unclear. This interaction's pathophysiology is complex and has been explored in depth elsewhere (Kusters et al., 2006 Jul, & Chmiela & Kupcinskas, 2019 Sep).

Helicobacter pylori is a spiral-shaped gram-negative bacteria that colonizes the mucoid lining of the human stomach (Smyk et al., 2014). This bacterium is responsible for the development of peptic ulcer disease. It is highly pathogenic and affects more than half of the world's population (Nevine et al., 2015, & Mamoun et al., 2015). It is characterized by the polymorphism phenomenon; it may appear in the form of a coccoid or bacillary form. It is the primary cause of stomach and duodenal ulcers; these diseases have become more common in recent times due to the spread of this type of bacteria. One of the most important virulence genes associated with stomach and bowel disease is cytotoxin-associated protein (cag A), which is carried by specific genetic patterns of this bacteria (Salimzadeh et al., 2015, & Wang et al., 2015). The prevalence of these bacteria is due to the virulence genes carried by specific genetic patterns of these bacteria .The bacteria have evolved an antibiotic-resistance to the stomach acid of the microbial through colonization in a very small area of gastric lactation and secretion of the urease that converts urea in the medium to ammonia, allowing them to live in the human stomach for the rest of their lives if untreated (Bakir et al., 2012).

Risk factors:

NSAIDs (nonsteroidal anti-inflammatory drugs): Ingestion of NSAIDs following H. pylori infection is a common risk factor for PUD. Peptic ulcers affect approximately 25% of chronic NSAID users (15% gastric and 10% duodenal). PUD complications (specifically haemorrhage and perforation) are much more common in patients taking NSAIDs (Charles Brunicardi, 2010).

Smoking: Smoking increases both stomach acid output and duodenogastrointestinal reflux. Smoking affects both gastroduodenal prostaglandin and pancreaticoduodenal bicarbonate production, which can raise a person's likelihood of getting an ulcer, as well as slow the healing of existing ulcers and contribute to ulcer recurrence (Koivisto et al., 2008).

Stress: Emotional stress is no longer thought to be a cause of ulcers, but people who are stressed often say their ulcers hurt more when they are stressed (Bas et al., 2008).

Borén and coworkers recently discovered that people with blood group O have more H. pylori receptors (Kanbay et al., 2005).

Caffeine: Consuming beverages or foods that contain Caffeine might cause increased acid output in the stomach. A preexisting ulcer may become worse as a result of this, although Caffeine is not the only factor that contributes to the activation of stomach acid (Aldoori et al., 1997).

Alcohol: Despite the fact that alcohol is commonly listed as a risk factor for PUD, there is a dearth of data to corroborate this association. PUD is commonly associated with

excessive amounts of alcohol intake (Karp et al., 2004).

The objective was to investigate the risk factors associated with perforated peptic ulcers.

MATERIALS AND METHODS

This study was achieved at the endoscopic unit of Al-Hussein teaching hospital. The study population included patients who complained of abdominal symptoms suggestive of gastric–duodenal pathology who was referred by specialist doctors for endoscopic examination at Al- Hussein teaching hospital in Karbala province during the period between November 2019 to January 2020. This study involved 51 consecutive patients (32 male and 19 female) aged from (**<20- >50**) who underwent endoscopic examination by specialist doctors. all patients were interviewed regarding personal biodata (age, sex, job, marital status, and place of residence), selected risk factors (smoking status, NSAID use, soft drinks, stress, Caffeine, blood group, whether food was spicy or not, family history of peptic ulcer, and presence of chronic comorbidities), along with details of selected symptoms of their conditions (epigastric pain, vomiting, nausea, and burning sensation). The endoscopic results were recorded on the same datasheet as that used by the examining doctor. In addition, H. pylori infection and blood group was tested for in 51 patients studied. Data were entered into a computer program (specifically, the Statistical Package for Social Science, version 18) and analyzed for the results presented in this article.

RESULT

Table 1: demographic characteristics of the studied sample

Age		
Age	Frequency No	Percentage %
<20	3	5.8%
21-30	10	19.6%
31-40	4	7.8%
41-50	11	21.5%
>50	23	45%
Total	51	100%
Gender		
Gender	Frequency No	Percentage %
Male	32	62.7%
Female	19	37.3%
Total	51	100%
Residence		
Residence	Frequency No	Percentage %
Urban	38	74.5%
Rural	13	25.5%
Total	51	100%
Marital status		
Marital status	Frequency No	Percentage %
Married	43	84.3%
Single	8	15.7%
Total	51	100%
Occupation		
Occupation	Frequency No	Percentage %
Employed	13	25.5%
Workers	28	55%
Housewife	10	19.5%
Total	51	100%

Table 1 shows the demographic characteristic for the total sample enrolled in the study, such as (age, gender, residence, marital status, and occupation). The most common age group was (>50/45%), which accounted for 45%. Male was more common (32 /62.7%) compared to female (19/37.3%). The residence of studied cases was high in the urban (38/74.5%) versus rural (13/25.5%). Most of the examined cases were married (43/84.3%); otherwise, single (8/ 15.7%). Also, most of the patients were workers (28/55%).

Table 2: illustrate ABO blood groups

ABO		
ABO	Frequency No	Percentage %
A	11	21.3%
B	9	18%
AB	6	11.7%
O	25	49%
Total	**51**	**100%**
RH		
RH	Frequency No	Percentage %
RH+	41	80%
RH-	10	20%
Total	**51**	**100%**

Table 2 shows the blood groups for all patient samples; the blood group O was the highest incidence between them and record (25/49%), while the(RH^+) factors were high in studied samples(41/80%) in comparison with (RH^-) .

Table 3: risk factors affected by peptic ulcer

Risk factors		
smoking status	Frequency No	Percentage %
Smoking	32	62.7%
Non-smoking	19	37.3%
Total	51	100%
Carbonated drink		
Carbonated drink	Frequency No	Percentage %
Plenty drink	26	51%
drink normally	25	49%
Total	51	100%
Coffee &Tea		
Coffee &Tea	Frequency No	Percentage %
Plenty drink	44	86.3%
drink normally	7	13.7%
Total	51	100%
Stress		
Stress	Frequency No	Percentage %
Extremely	43	84.3%
Normally	8	15.7%
Total	51	100%
spices		
spices	Frequency No	Percentage %
Plenty eat	30	58.8%
Eat normally	21	41.2%
Total	51	100%
NSAIDs		
NSAIDs	Frequency No	Percentage %
Use	41	80.4%
Non - use	10	19.6%
Total	51	100%
Family history		
Family history	Frequency No	Percentage %
family history of Infection	29	57%
Non- family history of Infection	22	43%
Total	51	100%

Table 3 showed great variation between risk factors (32/62.7%) from samples smoking, (26/51%) of them drinking carbonated drinks, (44/86.3) unusual drinks of Caffeine, (43/84.3%) had extremely stressful, (30/58.8%) add spices to meals, (41/80.4%) of them use NSAIDs, while (29/57%) of them had a family history of Infection of peptic ulcer.

DISCUSSION

Peptic ulcer disease remains of global significance to human health. The incidence of peptic ulcers has decreased in many countries following the falling prevalence of H. pylori infection, but it has nonetheless remained an important clinical issue. *Helicobacter pylori* invade the stomach, persist in the acidic pH environment of the lumen, and burrow through mucus to reach the epithelial cell layer of the stomach (Ottemann & Lowenthal, 2002). The survival of H. pylori in an acidic stomach is incumbent on the urease enzyme, which mitigates gastric acidity and damages gastric mucosa by producing toxins such as Cag A and Vac A (Atherton, 2006 & Kuipers, 2007). Different types of media, including a high-salt diet, smoking, and low iron levels, are utilised by H. pylori for colonization of the host's tissues (Wroblewski & Peek, 2013).

The socio-demographic characteristics of the study population showed as shown in table 1. The highest detection rate of H. pylori infection was recorded in the age group over 50 years old (45%). The result of this study is coordinated with (Al-Mahdawi et al., 2017), which indicated that the age group most predisposed to H. pylori infection is the group (>50), and the action of the immune system in older individuals may be related to the increase of prevalence in this stage group .In our study become clear the distribution of patients with H. pylori infection according to gender; the highest infections were among males than females (62.7%) the result agrees with (Al-aajem, 2018). Due to their increased daily effort and other factors such as smoking and alcohol, males have a higher infection rate than females. The low infection rate in females compared to males may be due to antibiotics taken by females over the course of their lives (during pregnancy, after birth, in cases of abortion, and during urinary genital infections); these antibiotics inhibit the growth of H. pylori, which is the leading cause of gastrointestinal diseases (Al-Mahdawi et al., 2017). As shown in Table 1, a high percentage of patients were married (84.3%) and stayed in urban residences (74.5%). This incidence due to a study done in 2002 in Russia showed the examination of 68 patients with a peptic ulcer found correlations between relations in married couples and the majority of city's population because of lifestyle prefer to eat ready meals (Sharobaro, 2002). Job titles show up at a high rate with workers (55%) of studied samples. These findings were very similar to previous research conducted in both developing and developed countries, low socioeconomic class, and this is primarily due to poor compliance with medical treatment of known cases of peptic ulcer and a high rate of Infection with H. pylori, as well as stress in various forms (Courtney, 2008).

According to Table 2, the majority of studied samples had a high percentage of (O) blood group with a high rate in comparison with other blood groups. This result was similar to another study done in Mexico by Eduardo et al. noted that the predominant blood group type in PPU was O (70%) (Montalvo-Javé et al., 2011).

The present study showed the highest incidence rate of risk factors that affected peptic ulcer such as smoking status was found in (32/62.7%) studied samples, there is some study agree with our result Ivana et al. has been noted that smoking is harmful to the gastroduodenal mucosa, and H. pylori infiltration is denser in the gastric antrum of smokers (Ivana Đorđević et al., 2011).

(26/51%) and (44/86.35) out of patients consume plenty of carbonated drinks and Caffeine.These beverages are a potent stimulus of gastric acid secretion. Interestingly, 7-Up, which contains neither Caffeine nor protein and minimal amounts of calcium, is a potent stimulus of gastric acid secretion (Bunker & McWilliams, 1979). Cohen et al. have been recording that coffee is a stimulant of acid secretion regardless of its caffeine content. Caffeine content alone does not seem to be of importance since Coke, Tab, and tea, which all contain 50-100 mg of Caffeine, are not significantly greater stimulants of gastric acid secretion than 7-Up, which is free of Caffeine. This result deal with (Cohen & Booth, 1971).

A (43/84.3%) persons found suffering from stress and (30/58.8%) plenty eat spices foods. Our result agreed with Danish research found a correlation between psychological stress and the incidence of peptic ulcer (Levenstein et al., 2015). Although there is no clear demonstration for the relationship of spices and seasoning with peptic ulcers, and reactions and tolerance differ from one person to another, the person who has peptic ulcer is advised to keep out foods rich in spices, especially black pepper, curry, and nutmeg and mustard seed, as they will make the symptoms worse.

For patients who had a family history of peptic ulcer and used NSAIDs medication found (29/57%) and (41/80.4) respectively. The study refers to a high incidence of NSAID use. This study goes with a study done in Pakistan. This may be explained by the fact that the majority of peptic ulcer cases were persistent users of NSAIDs either as part of their medical treatment (joint and back pain) or as a preventative antiplatelet therapy (Rahman, 2002). It is unclear why these obvious genetic strong correlations. There exists a rare genetic link between familial hyper pepsin anaemia type I (a hereditary trait associated with increased pepsin production) and peptic ulcers. H pylori can enhance pepsin secretion, and a retrospective examination of the sera of a family evaluated prior to the discovery of H pylori found that their elevated pepsin levels were more likely due to H pylori infection.

CONCLUSION

These findings suggest that if gastric acid has a role in acid-peptic symptoms and/or healing, milk is not a suitable therapeutic option. The benefit of milk against an appropriate placebo on symptoms or healing in people with acid-peptic illnesses has not been carefully investigated.

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Conflict of interest:
The author declares that there is no conflict of interest.

Authors' contributions:
The Authors' contribution to the preparation of the research paper consists of personal and individual efforts to simplify the concept and the problem, as well as to find solutions. All aspects of the research were written by the researcher, including the introduction, materials and methods, results, discussion, and statistical analysis.

REFERENCES

Al-Mahdawi, M. A. S., Ahmed, A. K., & Ali, G. H. (2017). Detection of cagA and vacA Helicobacter pylori Virulence Genes in Gastric Biopsies of patients with Gastroduodenal disease using Polymerase chain reaction (PCR) technique. *Diyala Journal for pure science*, *13*(4).

Al-aajem, B. M. R. (2018). Seroprevalence of *H. Pylori* in Patients with Gastric Upset in Baquba City. *International Journal of Science and Research (IJSR)*, *7*(2).

Al-Mahdawi, M. A. S., Ahmed, A. K., & Ali, G. H. (2017). Detection of cagA and vacA Helicobacter pylori Virulence Genes in Gastric Biopsies of patients with Gastroduodenal disease using Polymerase chain reaction (PCR) technique. *Diyala Journal for pure science*, *13*(4).

Aldoori, W. H., Giovannucci, E. L., Stampfer, M. J., Rimm, E. B., Wing, A. L., & Willett, W. C. (1997). A prospective study of alcohol, smoking, Caffeine and the risk of duodenal ulcer in men. *Epidemiology; 8*, 420-4.

Atherton, J. C. (2006). The pathogenesis of *Helicobacter pylori*-induced gastro-duodenal diseases. Annual review of

pathology: *Mechanisms of disease, 1*, 63-96.

Bas, G., Eryilmaz, R., Okan, I., & Sahin, M. (2008).Risk factors of morbidity and mortality in patients with perforated peptic ulcer: *Acta Chir Belg: 108*, 424-7.

Bakir, W., Al-kawaz, H., Hasoon, H., & Majeed, A. (2012). Detection of DNA Helicobacter pylori, and distribution of CagA genotype in cancerous and precancerous tissue. *Iraqi J. of cancer and medical genetics, 5*(2), 127- 132,

Bunker, M. .L, & McWilliams, M. (1979). The caffeine content of common beverages. *J Am Diet Assoc; 79*, 28-31.

Chmiela, M., & Kupcinskas, J. (2019 Sep). Review: pathogenesis of Helicobacter pylori infection. *Helicobacter.; 24Suppl 1*, e12638.

Charles Brunicardi, F. (2010). Schwartz's Principles of Surgery Stomach. The McGraw-Hill Companies, *USA, 9th Edition.; 26*, 907-22.

Cohen, S., & Booth, G. H. (1971). Gastric acid secretion and lower esophageal sphincter pressure in response to coffee and Caffeine. *N Engl J Med; 293*, 897-901.

Courtney, M. (2008). Townsend: SABISTON Textbook of Surgery. Stomach. Elsevier, USA. *18th ed; 47*, 1236-56.

Hooi, J. K. Y., Lai, W. Y., Ng, W. K., Suen, M. M. Y., Underwood, F. E., & Tanyingoh, D. (2017 Aug). Global prevalence of Helicobacter pylori infection: systematic review and meta-analysis. Gastroenterology. 1;*153*(2), 420–9.

Ivana Đorđević, AleksandarZlatić, & Irena Janković (2011). Treatment of Perforative Peptic Ulcer. *Scientific Journal of the Faculty of Medicine in Niš; 28*, 95-107.

Ivana Đorđević, AleksandarZlatić, & Irena Janković (2011). Treatment of Perforative Peptic Ulcer. *Scientific Journal of the Faculty of Medicine in Niš; 28*, 95-107.

Kanbay, M., Gür, G., Arslan, H., Yilmaz, U., & Boyacioglu, S. (2005). The relationship of ABO blood group, age, gender, smoking, and Helicobacter pylori Infection. *Dig Dis Sci; 50*, 1214-7.

Karp, Seth, J., Morris, & James, P. G. (2004). Blueprints Surgery.Stomach and duodenum. Lippincott Williams & Wilkins, Philadelphia. *5th ed. 3*, 21-25.

Kusters, J. G., van Vliet, A. H. M., & Kuipers, E. J. (2006 Jul). Pathogenesis of Helicobacter pylori infection. *Clin Microbiol Rev.; 19*(3), 449–90.

Koivisto, T. T., Voutilainen, M. E., & Färkkilä, M. A. (2008). Effect of smoking on gastric histology in Helicobacter pylori-positive gastritis. *Scand J Gastroenterol; 43*, 1177-83.

Kuipers, E. J. (2007). Acid peptic disease: Epidemiology and Pathobiology. In: Goldman L, Ausiello D, eds. Cecil Medicine. 23rd ed. Philadelphia, Elsevier, 1009- 1013.

Levenstein, S., Rosenstock, S., Jacobsen, R. K., & Jorgensen, T. (2015). Psychological stress increases risk for peptic ulcer, regardless of Helicobacter pylori infection or use of nonsteroidal anti-inflammatory drugs. *Clin. Gastroenterol. Hepatol.*, *13*, 498–506.e1. [CrossRef] [PubMed]

Mamoun, M., Elsanousi, S., Khalid, A., Abdelmounem, E., & Mohamed, A. (2015). Molecular Identification 0f 16s Ribosomal RNA Gene of Helicobacter pylori Isolated from Gastric Biopsies in Sudan. *American Journal of Microbiological Research, 3*(2), 50-54.

Montalvo-Javé, E. E., Corres-Sillas, O., & Athié-Gutiérrez, C. (2011). Factors associated with postoperative complications and mortality in perforated peptic ulcer. *Cir; 79*, 141-8.

Nevine, M., El Deeba, A., & Amany, Y. (2015). An ultrastructural study of the association between Helicobacter pylori and the gastric mucous. *Egyptian Journal of Pathology, 35*, 1-13.

Ottemann, K. M., & Lowenthal, A. C. (2002). *Helicobacter pylori* use motility for initial colonization and to attain robust Infection. Infect. *Immun, 70*(4), 1984-90.

Rahman, M. A. (2002). Risk factors associated with peptic ulcer disease. *J Postgrad Med Inst; 16*, 161-5.

Salimzadeh, L., Bagheri, N., & Zamanzad, U. (2015). Frequency of virulence factors in Helicobacter pylori-infected patients with gastritis. *Microbial Pathogen, 3*.

Sharobaro, V. I. (2002). Correlations between clinical features, personality disorders and the antioxidant system in patients with peptic ulcer. *Klin Med (Mosk).; 80*(9), 35-7.

Smyk, D., Koutsoumpas, A., Mytilinaiou, M., Rigopoulou, E., Sakkas, L., & Bogdanos, D. (2014). Helicobacter pylori, and autoimmune disease: Cause or bystander. *World J Gastroenterol, 20*, 613-629.

Wang, Y., Chen, K., Chung, L., Meng, W., & Hsiang S. (2015). Diagnosis of Helicobacter pylori infection. *World J Gastroenterol, 21*(40), 11221-1123.

Wroblewski, L. E., & Peek, R. M. (2013). *Helicobacter pylori* in gastric carcinogenesis: mechanisms. *Gastroenterol Clin North Am, 42*, 285-298.