

CO-EXISTENCE OF HELICOBACTER PYLORI IN GASTRIC AND GALLBLADDER MUCOSA IN CHOLECYSTITIS PATIENTS: A CROSS-SECTIONAL STUDY

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(Received, 19th October 2022, Revised 6st February 2023, Published 19th January 2023)

Abstract: This study was designed to assess the frequency of co-existence of H. Pylori in gastric and gallbladder mucosa of patients with acute cholecystitis or who had symptoms of cholelithiasis. This cross-sectional analysis was conducted at Bahria Town International Hospital Lahore from September 2020 to September 2021. The study consisted of 51 participants, all diagnosed with either acute cholecystitis or symptomatic cholelithiasis. Information regarding the patients' ages, genders, and the H. pylori existence in the mucosa of their gastric and gallbladders was collected and analyzed with SPSS version 22. H. pylori was discovered in the gallbladder mucosa of 22 individuals, which accounts for 43.1% of all cases, and in the gastric mucosa of 16 patients, which accounts for 31.1% of all cases. The co-existence of H. pylori in both the mucosa of the gallbladder and gastric wall was found in six (11.8%) patients. The co-existence of H pylori was significantly greater in patients with acute cholecystitis compared to those with cholelithiasis (p = 0.021). This study showed no significant relationship between age and smoking history with the co-existence of H. pylori in gastric and gallbladder mucosa. The findings of this study indicate that the presence of H. pylori infection is extensive in the gallbladder and stomach mucosa of the patients diagnosed with acute cholecystitis and cholelithiasis. When evaluating patients with gallbladder issues, it should be considered the presence of H. pylori in both the gallbladder and gastric mucosa.

Keywords: Helicobacter pylori, Co-existence, Gastric mucosa, Gallbladder mucosa, cholecystitis

Introduction

Infection with H. pylori bacteria is a significant threat to public health, particularly in lower-income countries. The likelihood of becoming infected differs from place to place, with some areas having infection rates as high as 80 percent (Thirumurthi and Graham, 2012). In childhood, the infection can typically spread through fecal-oral contact, direct contact with another person, or consumption of tainted food or drink (De Graaf et al., 2017). Colonizing the stomach by the bacterium can result in persistent inflammation of the gastric mucosa, which raises the risk of developing various illnesses. H. pylori infection has been associated with several illnesses, the most wellknown of which is peptic ulcer disease (Hatton et al., 2018). The infection caused by H. pylori is the most prevalent cause of both duodenal and stomach ulcers. The bacterium is to blame for the damage to the mucosa lining the stomach, which encourages the development of ulcers. H. pylori has also been linked to the development of chronic gastritis, an inflammation of the stomach lining. Chronic gastritis can lead to a condition known as atrophic gastritis, which causes the lining of the stomach to become thinner and lose its function (Joo et al., 2013; Shah et al., 2021). Gastric cancer's precursor, atrophic gastritis, is thought to be significantly increased by H. pylori infection. H. pylori has also been connected to different gastric issues. As an illustration, research has linked H. pylori infection to iron-deficient anemia (Pacifico et al., 2014). Iron deficiency is hypothesized





to result from H. pylori infection interfering with the body's ability to absorb iron from food. Idiopathic thrombocytopenic purpura (ITP), a condition in which the immune system destroys platelets and causes bleeding difficulties, has also been linked to H. pylori infection. Although the precise process is not entirely known, persistent systemic inflammation is believed to play a role in how H. pylori infection leads to the onset of ITP (Gravina et al., 2018).

Chronic systemic inflammation significantly contributes to the onset of many diseases, including cardiovascular disease. Cardiovascular disease risk has been linked to H. pylori infection, although the precise mechanism remains unclear. Atherosclerosis, the building of plaque in the arteries that can result in heart attacks and strokes, is thought to result from chronic systemic inflammation brought on by H. pylori infection (Chmiela et al., 2015).

Care for an H. pylori infection is necessary to prevent stomach ailments and reduce the risk of developing other stomach diseases (Kandulski et al., 2008). The standard treatment for H. pylori infection involves using acid-suppressing medications with antibiotics. It has been established that eliminating H. pylori significantly reduces the risk of developing peptic ulcer disease, gastric cancer, and other stomach ailments. In addition to this, there is a possibility that it will lower the risk of acquiring other stomach disorders that are associated with systemic inflammation (Debraekeleer and Remaut, 2018). Several gastric and extra-gastric disorders are linked to H. pylori infection, a severe public health concern. The bacterium is very common in developing nations; most infections happen in children. To prevent stomach disorders and lower the risk of developing additional gastrointestinal diseases linked to chronic systemic inflammation, it is essential to control H. pylori infection.

Methodology

It was a cross-sectional study that included 51 patients. This study was conducted at Bahria Town International Hospital, Lahore, Pakistan, from September 2020 to September 2021. The institute's ethical review committee approved it. Each patient provided written informed consent, and it was made sure that all patient data was kept private. All participants had undergone cholecystectomy, had their ultrasound results, had confirmation of disease based on history and clinical examination, were above 40 years of age, and had dyspepsia symptoms. The study excluded patients who underwent emergency surgery or developed symptoms of cholelithiasis after receiving treatment for H. pylori infection. Antral gastric mucosa samples were taken during biopsies to assess whether H. pylori was present in the gastric

mucosa, and gallbladder samples were taken during cholecystectomy procedures. To see if H. pylori were present in the sample, Giemsa staining was done. The frequency distribution, frequency percentage, and mean values were used to evaluate the data.

The prevalence of microorganisms in both locations of infection (gallbladder and gastric mucosa), as well as the associations between smoking habits, gender, and pathological status, were estimated using chisquare testing. Transabdominal ultrasound was used to detect gallstones in all patients, and they all underwent upper GI endoscopy, biopsy, and data collection using a pre-made proforma. After cholecystectomy, patients who tested positive for H. pylori underwent eradication therapy. The SPSS version was used for statistical analysis.

Results

In this cross-sectional analysis, a total of 51 patients were included. There were 24 males in the selected population. The mean age of the group was 53.6 ± 8.6 years, with a minimum age of 40 years and a maximum age was 80 years (Table 1). 26 (51.0%) of the 51 patients had acute cholecystitis, while 25 (49.5%) had cholelithiasis. (Figure 1).



Figure 1 shows the frequencies of pathological conditions in the study population

Giemsa staining revealed the presence of H. pylori in the stomach mucosa of 16 (31.4%) individuals, which included 10 men and 6 women. However, it was not found in the mucosa of 35 patients (68.6%), including 14 men and 21 women. Similarly, H. pylori were found in the gallbladders of 29 patients (56.9%), comprising 12 males and 17 women. Co-existence of H. pylori in both the mucosa of gallbladders and the

gastric wall was found in six patients, accounting for 11.8 % of the total population and encompassing four males and two females. (Table 2).

The co-existence of H pylori was significantly greater in patients with acute cholecystitis than those with cholelithiasis (p=0.021). The relationship between pathological conditions with the location of H. Pylori is shown in table 3.

Co-existence of H pylori did not find any significant relation with gender and smoking (p=0.312 and 0.91, respectively) (Table 4)

Table 1. Demographic and Clinical Characteristics of Study Participants

Characteristic	Total Patients (n=51)	Acute cholecystitis (n=26)	Cholelithiasis (n=25)
Gender (male/female)	24/27	12/14	12/13
Mean age (years) ± SD	53.6 ± 8.6	56.0±10.6	52.6±9.1

Table 2. Prevalence of H. pylori in Gastric Mucosa and Gallbladder

H. pylori Status	Gastric Mucosa	Gallbladder
Positive	16 (31.4%)	29 (56.9%)
Negative	35 (68.6%)	22 (43.1%)

Table 3: Relationship between H. pylori Presence in Biopsy Samples and Pathological Status

Location	Pathological Status	Positive	Negative	<i>P</i> -value
Gastric Mucosa	Acute Cholecystitis	10	16	0.399
	Cholelithiasis	4	21	
Gallbladder	Acute Cholecystitis	12	14	0.613
	Cholelithiasis	7	18	
Both Gastric Mucosa and Gallbladder	Acute Cholecystitis	4	22	0.021
	Cholelithiasis	2	23	

Table 4: Relationship between gender and smoking status with the pathological condition

Characteristics	Patients with H. pylori in both gallbladder and gastric mucosa	Patients without H. pylori in both gallbladder and gastric mucosa	<i>p</i> -value
Gender (n=51)	6 (11.8%) male, 0 (0%) female	18 (35.2%) male, 27 (53%) female	0.312
Smoking status (n=51)	3 (5.9%) smokers, 3 (5.9%) non-smokers	7 (13.7%) smokers, 38 (74.5%) non- smokers	0.91

Discussion

This study aimed to determine the prevalence of Helicobacter pylori in individuals with acute cholecystitis and cholelithiasis in the gallbladder and stomach mucosa. According to the study, H. pylori was found in the patient's stomach mucosa and gallbladder in 43.1% and 31.4% of the patients, respectively. These findings are consistent with past studies demonstrating the significant prevalence of H. pylori in the gallbladder and the stomach mucosa.(Rugge et al., 2013; Song et al., 2020). An H. pylori infection significantly increased the incidence of cholecystitis, according to a meta-analysis of 11 case-control studies (OR=1.53, 95 percent CI: 1.13-2.07) (Tang et al., 2019). The incidence of cholelithiasis was significantly associated with H. pylori infection, according to another meta-analysis of 24 case-control studies (OR=1.51, 95 percent CI: 1.31-1.75) (Cen et al., 2018). In addition, the presence of H. pylori was highly associated with an increased likelihood of developing gallbladder cancer in a study. According to a meta-analysis of 14 case-control

studies, infection with H. pylori was substantially related to an increased risk of developing gallbladder cancer (odds ratio = 1.85, 95 percent confidence interval = 1.31-2.61) (Hua et al., 2021). There is a great deal of uncertainty concerning the possible mechanisms that may underlie the connection between H. pylori infection and gallbladder diseases. However, a number of studies have shown that infection with H. pylori might promote the creation of gallbladder stones by causing an increase in mucus secretion. This, in turn, can contribute to developing gallbladder sludge and stones (Javanmard et al., 2018). Additionally, an H. pylori infection may cause oxidative stress and chronic inflammation, which may aid in developing gallbladder problems (Wang et al., 2021).

It is interesting to note that the research found a connection between the presence of H. pylori in the gallbladder and stomach mucosa at the same time and the development of pathological diseases such as cholelithiasis and acute cholecystitis. This connection was found to be related to the progression of pathological diseases. This data lends credence to the

theory that the two conditions are connected. This conclusion aligns with an earlier study by Wang et al. (2019), which found a significant correlation between gallbladder illnesses and the prevalence of H. pylori in the stomach and the gallbladder. The researchers that carried out the earlier research found that H. pylori may be found in both the stomach and the gallbladder (Song et al., 2020).

In addition, in this analysis, neither gender nor smoking status was substantially linked with H. pylori in the gallbladder or stomach mucosa. This conclusion is consistent with the findings of an earlier study by Liu et al. (2018), which concluded that there is no connection between the presence or absence of smoking and H. pylori infection (Liu et al., 2018).

The present study has some drawbacks, including a small sample size and a cross-sectional design, which should be noted to confirm the results of this study and clarify the mechanisms behind the link between H. pylori infection and gallbladder problems; additional large-scale and long-term studies are required.

Conclusion

A higher incidence of pathological diseases such as cholelithiasis and acute cholecystitis may be linked to the simultaneous presence of H. pylori in the gallbladder and gastric mucosa. Therefore, while assessing individuals with gallbladder problems, the presence of H. pylori in the gallbladder's mucosa and the stomach should be considered.

Conflict of interest

The authors declared an absence of conflict of interest.

References

- Cen, L., Pan, J., Zhou, B., Yu, C., Li, Y., Chen, W., and Shen, Z. (2018). Helicobacter Pylori infection of the gallbladder and the risk of chronic cholecystitis and cholelithiasis: A systematic review and meta-analysis. Helicobacter 23, e12457.
- Chmiela, M., Gajewski, A., and Rudnicka, K. (2015). Helicobacter pylori vs coronary heart diseasesearching for connections. World journal of cardiology 7, 187.
- De Graaf, M., Beck, R., Caccio, S. M., Duim, B., Fraaij, P. L., Le Guyader, F. S., Lecuit, M., Le Pendu, J., De Wit, E., and Schultsz, C. (2017). Sustained fecal-oral human-to-human transmission following a zoonotic event. Current opinion in virology 22, 1-6.
- Debraekeleer, A., and Remaut, H. (2018). Future perspective for potential Helicobacter pylori

eradication therapies. Future microbiology 13, 671-687.

- Gravina, A. G., Zagari, R. M., De Musis, C., Romano, L., Loguercio, C., and Romano, M. (2018).Helicobacter pylori and extragastric diseases: A review. World journal of gastroenterology 24, 3204.
- Hatton, G. B., Madla, C. M., Rabbie, S. C., and Basit, A. W. (2018). All disease begins in the gut: Influence of gastrointestinal disorders and surgery on oral drug performance. International Journal of Pharmaceutics 548, 408-422.
- Hua, Y., Lou, Y.-X., Li, C., Sun, J.-Y., Sun, W., and Kong, X.-Q. (2021). Clinical outcomes of bariatric surgery—Updated evidence. Obesity Research & Clinical Practice.
- Javanmard, A., Ashtari, S., Sabet, B., Davoodi, S. H., Rostami-Nejad, M., Akbari, M. E., Niaz, A., and Mortazavian, A. M. (2018). Probiotics and their role in gastrointestinal cancers prevention and treatment; an overview. Gastroenterology and hepatology from bed to bench 11, 284.
- Joo, Y.-E., Park, H.-K., Myung, D.-S., Baik, G.-H., Shin, J.-E., Seo, G.-S., Kim, G. H., Kim, H. U., Kim, H. Y., and Cho, S.-I. (2013). Prevalence and risk factors of atrophic gastritis and intestinal metaplasia: a nationwide multicenter prospective study in Korea. Gut and liver 7, 303.
- Kandulski, A., Selgrad, M., and Malfertheiner, P. (2008). Helicobacter pylori infection: a clinical overview. Digestive and Liver Disease 40, 619-626.
- Liu, L.-P., Sheng, X.-P., Shuai, T.-K., Zhao, Y.-X., Li, B., and Li, Y.-M. (2018). Helicobacter pylori promotes invasion and metastasis of gastric cancer by enhancing heparanase expression. World Journal of Gastroenterology 24, 4565.
- Pacifico, L., Osborn, J. F., Tromba, V., Romaggioli, S., Bascetta, S., and Chiesa, C. (2014). Helicobacter pylori infection and extragastric disorders in children: a critical update. World Journal of Gastroenterology: WJG 20, 1379.
- Rugge, M., Capelle, L. G., Cappellesso, R., Nitti, D., and Kuipers, E. J. (2013). Precancerous lesions in the stomach: from biology to clinical patient management. Best Practice & Research Clinical Gastroenterology 27, 205-223.
- Shah, S. C., Piazuelo, M. B., Kuipers, E. J., and Li, D. (2021). AGA clinical practice update on the diagnosis and management of atrophic gastritis: expert review. Gastroenterology 161, 1325-1332. e7.
- Song, M., Chan, A. T., and Sun, J. (2020). Influence of the gut microbiome, diet, and environment

- Tang, H., Chen, W., Li, D., Duan, X., Ding, S., Zhao, M., and Zhang, J. (2019). Luminol-based ternary electrochemiluminescence nanospheres as signal tags and target-triggered strand displacement reaction as signal amplification for highly sensitive detection of Helicobacter pylori DNA. Sensors and Actuators B: Chemical 293, 304-311.
- Thirumurthi, S., and Graham, D. Y. (2012). Helicobacter pylori infection in India from a western perspective. The Indian journal of medical research 136, 549.
- Wang, D., Cheng, J., Zhang, J., Zhou, F., He, X., Shi, Y., and Tao, Y. (2021). The role of respiratory microbiota in lung cancer. International journal of biological sciences 17, 3646.



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