Potential Role of *Helicobacter pylori* Infection in Hepatocellular Carcinoma: A Clinical and Laboratory-Based Study

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Abstract

Background: *Helicobacter pylori* (*H. pylori*) infection has been a pertinent global health concern with possible extra pulmonary related problems including liver concerns. This research required to determine the prevalence of *H. pylori* infections and its relationship with liver fibrosis and cirrhosis among patients attending the hospital while accounting a range of relevant demographics and clinical factors. Methodology: The total population sample in this study was 215, enrolled as participants for a cross-sectional observational study. Evaluation of *H. pylori* was conducted through serological and Chemiluminescence Immunoassay (CLIA) techniques. Other parameters that needed to be noted included demographic information, co-existing health disorders, lifestyle habits such as smoking or drinking alcohol, and biochemical indicators of liver health like ALT, AST, ALP, bilirubin, albumin etc. For comparative analysis between the populations, frequency distribution was done and later a comparative assessment through P-value relational definitional assessment for significant associations was computed. Results: The respondents were 51.6% women, and mostly in the 26-45 age bracket. *H. Pylori* contamination was revealed in 67.5% of the cases with 43.26% positive by Chemiluminescence Immunoassay (CLIA) and 24.19% by serology. Considerable associations were noted with *H. Pylori* for infection and elevated hepatic transaminases ALT, AST and ALP, and increased bilirubin levels ($p < 0.05$). Conclusion: It does appear from these results that *H. Pylori* infection has a considerable association with liver fibrosis or cirrhosis, demonstrating a need to control *H. Pylori* infection among patients with chronic liver disease or those who are prone to this disease.

Keywords: *Helicobacter pylori*, Hepatocellular carcinoma, Liver fibrosis, Cirrhosis, Liver function.

1. Introduction

In simple terms, Hepatocellular Carcinoma, also known as HCC, refers to liver cancer for which there is no treatment. HCC is known to be the deadliest type of cancer the primary form of liver cancer,

Significance | The study is to determine the pervasiveness of H. pylori infection among patients with liver illness of HCC.

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with HCC as the leading cause of death of more than 800,000 people (Ferenci et al., 2010). Liver cancer cases worldwide rose to 900,000 in 2020, with liver cirrhosis further worsening the mortality rate from HCC (Huang et al., 2024). Asian countries and sub-Saharan Africa have the maximum prevalence of HCC due to the high rates of chronic viral HBV and HCV infections (Mak and Kramvis, 2021). The being of chronic liver disease, especially with cirrhosis greatly increases the likelihood of emerging HCC (Tsukuma et al., 1993). In comparison with other countries, many evolving countries still face persistent public health challenges and low socioeconomic status exacerbates these challenges leading to amplified incidence rate of HCC along with high mortality owing to insufficient access to early diagnosis and treatment facilities, which is a global concern (Lodato, 2006). Furthermore, the chronic infection of HBV and HCV virus, alcohol abuse, non-alcoholic fatty liver illness (NAFLD), and even some regulated substances like aflatoxins serve as potent environmental risks for HCC. However, what is considered the leading contributor to HCC in other countries is chronic HCV infection, especially in nations where there is widespread access to hepatitis B vaccinations (Thrift et al., 2016). HCV is a potent causative element for chronic liver diseases, further exacerbated by cirrhosis, greatly raising chances of hepatocellular carcinoma. It inflicts chronic inflammation of liver tissues which increases the progression of fibrosis to cirrhosis faster and can lead to HCC (Czaja, 2014).

The association of viral infections with liver pathology remains critical for studying the development pathways of HCC from cirrhosis (D'souza et al., 2020While the link between viral hepatitis and liver cancer is well established, recent studies indicate that some other infectious agents such as *Helicobacter pylori* (*H. pylori*) may be factors in HCC and cirrhosis progress. *H. pylori*'s notoriety stems from its peptic ulcer and gastric cancer causative role due to being a gram-negative gastritis organism (Okushin et al., 2018). Despite that, there is growing evidence that this bacterium could extend its impact to the liver. Some surveys have indicated that *H. pylori* may raise liver enzyme levels and the risk of liver fibrosis (Kim et al., 2017). Some have gone so far as to say that chronic *H. pylori* infection could foul the progress of liver diseases like cirrhosis and even HCC due to systematic inflammation, immune system alteration, and disruption of the gut microbiome (Tufael et al., 2024). All factors considered, the relationship between *H. pylori* and liver pathologies like liver fibrosis and hepatocellular carcinoma is still severely lacking understanding and not developed substantially in medical literature. *H. pylori*'s role in liver disease is still under active study with respect to liver fibrosis and HCC along with other potential risk factors like HCV (Waluga, 2015). The absence of these core aspects in the available literature further emphasizes the need towards examining the potential gastric impacts of *H. pylori* and its relations with other factors that contribute toward liver disease.

This study comes from an increasing understanding of the involvement of *H. pylori* in non-gastric diseases, particularly its possible role in liver pathology (Mohammadi et al., 2023). Considerable effort has been made regarding the issue of chronic viral infections such as HCV, but the role of bacterial infection, like *H. Pylori*, has not been given due attention (Amieva & El–Omar, 2008). This gap in literature is degenerated by conflicting evidence; some posits a relation between *H. pylori* and liver fibrosis or cirrhosis, while others contest this claim (Mantovani et al., 2019). In this context, we aim to provide insights regarding the relationship of *H. pylori* infection with liver fibrosis, and cirrhosis, predominantly in the location of HCV infection and hepatocellular carcinoma (HCC). The study seeks to understand the extra-gastric consequences of *H. pylori* infection by looking at its prevalence in a population mid ailment with liver disease as well evaluating its potential impact on liver-colored fibrosis and cirrhosis. This study is significant because it may reveal another dimension on the progression of liver disease and add to the understanding of HCC prevention and control. Knowing whether or not *H. pylori* infection worsens liver disease in people already predisposed to liver disease, especially those with chronic viral hepatitis, can change the method to treatment. Furthermore, this may stimulate more research into the involvement of *H. pylori* and other microbial organisms in the pathology of liver complications. Hence, the aim of the study is to determine the pervasiveness of *H. pylori* infection among patients with liver illness and its possible association with liver fibrosis and cirrhosis as well as its possible role in the development of HCC among continuing hepatitis C patients.

2. Materials and Methods

2.1 Samples

This research was carried out as a cross-sectional experimental study for the resolution of evaluating suggestion of *Helicobacter pylori* (*H. pylori*) infection with liver disease with special emphasis on liver fibrosis, cirrhosis of liver and hepatocellular carcinoma (HCC). Study sample included 215 participants who came from the herpetology department of Tertiary Medical Center, Dhaka from April 2023 to November 2023. Members were included on the basis of their clinical judgment of liver diseases which included chronic hepatitis, liver fibrosis, cirrhosis or HCC. This study included: people within the age of 18 to 70 years with confirmed liver disease based on clinical evaluation and imaging, as well voluntary informed consent. Participants were uninvolved from this study if they had ever had a liver transplantation, other forms of cancer that were not liver cancer prior, or were known to have any active illnesses of the stomach tract other than *H. Pylori* (Kim et al, 2022). Every participant was described from a demographic position with

respect to age, sex, height, as well as weight (which was converted into body mass index BMI) as relevant smoking and alcohol habits, and their occupation. Besides the demographic information, clinical information concerning the presence of co-existing conditions such as high blood pressure and diabetes mellitus, along with other comorbidities, was also gathered through standardized conferences and existing medical documents. The study sample was split into two groups dependent on whether or not they had *H. Pylori* infection: *H. Pylori* positive group and *H. Pylori* negative collection.

2.2 Laboratory Tests

2.2.1 H. pylori Detection

A mixture of serologic samples established the judgement for *H. pylori* infection. For the serologic examination, antibodies targeting *H. pylori* were recognized Chemiluminescence Immunoassay (CLIA) testing which measures IgG antibodies towards *H. pylori*.

2.2.2 Liver Function and Biochemical Markers

Liver function was determined based on vaccine ALT, AST, ALP, and total bilirubin, as well as albumin and creatinine in the context of Lala et al.'s (2023) work. In aggregate, these indicators give vital information regarding the magnitude of liver injury and necrosis in the liver cells. Transaminases, comprising ALT and AST, serve as deputations for hepatocyte injury, whereas, ALP and bilirubin are indicators of bile elimination (Rana et al., 2024). Albumin states the synthetic function performed by the liver while creatinine level indicates renal function, which is routinely affected in late liver disease. Moreover, a complete blood count (CBC) test was performed to measure hemoglobin, total white blood cell count, platelet count, and hematocrit (Hellgren et al., 1986). Focus in this study was embattled toward thrombocytopenia which is common in chronic liver pathology such as cirrhosis since it can be very useful to determine hepatic portal hypertension or cirrhosis (Salam et al., 2024).

2.2.3 Assessment of Liver Disease Severity

The evaluation for the presence of liver disease was done in detail with the help of a physical assessment, laboratory investigations, imaging studies, and other medical imaging techniques. When available, electrography measures liver stiffness or other non-invasive markers like APRI and FIB-4 scores are used to estimate the level of fibrosis. Ultrasound, CT scan, and MRI are done to evaluate other organs and tissues in patients with HCC. In other cases, a liver biopsy may be needed to determine the stage of cirrhosis or the presence of HCC with tissue diagnosis.

2.3 Statistical Analysis

Statistical data was analyzed using SPSS Statistics version (IBM). Descriptive statistics was performed alongside demographic and clinical data, as well as reputed laboratory examinations. Age, LFT and platelet count as continuous variables were calculated per mean ± SD. Frequencies and percentages were provided for variables such as gender, smoking, alcohol use, and *H. pylori* infection status. Pertinent statistical systems were used to study the relationship between *H. pylori* infection and the parameters of liver disease that include liver enzymes, fibrosis, and cirrhosis. For participants grouped as *H. pylori* positive and *H. pylori* harmful, the categorical variables of sex, smoking and alcohol drinking were analyzed by means of chi square tests, while liver function tests and age were measured as unceasing variables and analyzed by independent t tests. In assessing the impact of *H. pylori* infection on liver disease progression, a multivariate logistic regression model was applied. This model controlled for covariate confounders of interest which were age, sex, alcohol, smoking, and other comorbid conditions like diabetes and hypertension for estimating odds ratios (OR) and 95% confidence interval (CI) of liver fibrosis, cirrhosis and HCC.

2.3.1 Multivariate Analysis and Adjustments

Various independent risk factors with potential of progression to liver disease such as *H. pylori* infection, Hepatitis C virus infection, alcohol use, smoking and other chronic illnesses were assessed using multivariate logistic regression. All variables were examined for interaction effects, and the presence of multicollinearity on independent variables was checked. In a stepwise approach, significant predictors were included in the final model.

2.3.2 Statistical Significance

All tests were set to $p < 0.05$ for statistical significance. Strength of association was estimated with odd ratios (OR) and 95% confidence intervals (CI). For groups with varying severity of liver disease, the non-parametric Kruskal-Wallis test was applied to compare liver function parameters like bilirubin levels or ALT and AST indicating non-normal distribution.

2.3.3 Ethical Considerations:

Ethical principles of the Helsinki Declaration were followed throughout this study. Ethics approval was granted by the Institutional Review Board (IRB). Informed consent was gathered ensuring confidentiality of the data shared by participants. Participants were free to exit the study anytime without consequence.

3. Results

3.1 Demographics of the Study Population

The research included 215 participants. Out of them 104 were males which is 48.4% and 111 were females which is 51.6% which shows a composed gender quantity. Participants averaged 37.6 ± 10.1 years old as shown in **Table 1.** The modal age collection was 26-45 years (63.5%) which contains 23.3% of members from 26 to 30 years and 20.9% of members from 31 to 35 years. Members also were distributed by age as follows: 15-20 years (4.7%), 21-25 years (9.3%), 26-30 years (23.3%), 31-35 years (20.9%), 36-40 years (18.6%), 41-45 years (16.3%), and > 45 years (7.0%). The popular of the cohort were employed as cleaners 30.2% and nurses 25.6%. The remaining

Table 1. Demographic Features of Respondents (N = 215)

Variable	Category	Frequency (n)	Percent (%)
Sex	Male	104	48.4
	Female	111	51.6
Age Group	15–20 years	10	4.7
	21–25 years	20	9.3
	26–30 years	50	23.3
	31–35 years	45	20.9
	36–40 years	40	18.6
	41–45 years	35	16.3
Occupation	>45 years	15	7.0
	Doctor	15	7.0
	Nurse	55	25.6
	Ward master	18	8.4
	Staff	20	9.3
	Lab technician	5	2.3
	Trainee	12	5.6
	Student	10	4.7
	Cleaner	65	30.2
	Manager	3	1.4
	Director	2	0.9

Table 2. Liver Function Test Results by *H. Pylori* Status

iver Function Test	H. pylori Negative (n = 83)	H. pylori Positive (n = 132)	p-value
ALT (U/L)	38.9 + 14.3	58.4 + 20.1	< 0.001
AST (U/L)	40.3 + 19.8	60.1 + 28.5	< 0.001
ALP (U/L)	101.2 + 44.7	145.6 + 65.2	< 0.001
Bilirubin (mg/dL)	1.02 I 0.48	1.58 + 0.75	< 0.001
Albumin (g/dL)	4.15 + 0.70	3.95 + 0.88	0.05
Creatinine (mg/dL)	0.85 + 0.20	0.90 + 0.32	0.43

Table 3. Association Between *H. Pylori* Infection and Liver Disease Severity

Liver Disease Severity	H. pylori Negative (n = 83)	H. pylori Positive (n - 132)	p-value
Liver Fibrosis	32 (38.5%)	58 (43.9%)	0.23
Cirrhosis	10 (12.0%)	28 (21.2%)	0.04*
HCC	5 (6.0%)	15 (11.4%)	0.03*

Note: *p < 0.05 indicates statistical significance.

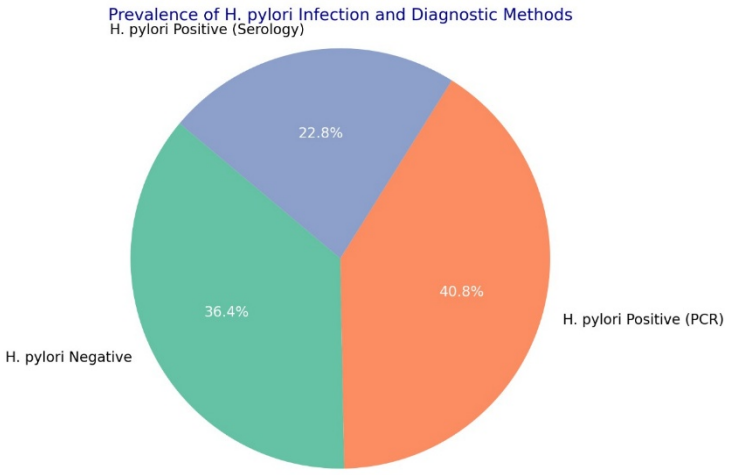

Figure 1. Prevalence of *H. Pylori* Infection and Diagnostic Methods

were ward masters 8.4%, some staff 9.3%, unimportant number of therapeutic doctors 7.0% and lab technicians 2.3%.

The information is meaningful as it illustrates relatively different exposures to risk factors which could lead to liver disease, having been derived from *H. Pylori* infection or Hepatitis virus. Concerning lifestyle factors, 44.2% claimed to be alcohol consumers while 48.8% labeled themselves as smokers. The described alcohol and smoking habits are risk factors associated with the disease around the liver, especially when talking about the advance of the disease known as cirrhosis or Hepatocellular Carcinoma (HCC). Other co-morbid diseases or conditions which were reported in the study were: Hypertension and diabetes. Participants with Hypertension represented 28.4% of the sample while Diabetic participants made up 27.9%. There's a striking 14.9% of the sample that suffers from both Hypertension and Diabetes, marking these conditions as overlapping. The aforementioned co-morbidities are known to worsen the damage to the liver as well as change the rate of progression into the disease, especially when chronic infections like HCV are present.

3.2 Prevalence of Helicobacter Pylori Infection

According to this study, the commonness of *H. Pylori* infection is 61.4%. This indicates that 132 participants were found positive for the organism. Active *H. Pylori* infection through Chemiluminescence Immunoassay (CLIA) based biological testing was established in 93 participants (43.3%). Positive serology figure-hugging past or present exposure to the animal was noted in 52 (24.2%). These **Figure 1** suggest an increased burden of *H. Pylori* exposure in the population and some planning for gastric and liver associated diseases. *H. Pylori* is a widely recognized gastric pathogen, the level of its influence to liver disease fibrosis and cirrhosis is still open to debate. Noting that deposition of over sixty percent of the participants from this study was infected with *H. Pylori* highlights the value in appreciating the role of this microbe in the liver disease in the population under study.

3.3 Association Between H. Pylori Infection and Liver Disease Parameters

Test of liver functions which included evaluation of ALT, AST, ALP, bilirubin, albumin and even creatine pertaining diagnosis of *H. pylori* infection showed that strangely the infection impacted the liver and its functioning. Furthermore, it was noted that *H. pylori* infected individuals did tend to have higher level of ALT, AST, ALP and bilirubin than those who were negative as illustrated in **Table 2**. It may be proposed that, the impact of *H. pylori* infection does affect liver function by, the infection causes damage to the liver and so increases the burden and workload which is why it is associated with liver dysfunction.

The differences in mean values of ALT, AST, ALP, and other markers divided the sampled participants into *H. pylori* infected and non-infected groups implies that *H. pylori* infection worsens liver damage. Increased ALT and AST are sure signs of liver damage; on the other hand, significantly high ALP implies the possibility of cholestatic or other biliary diseases. Rise in the amount of bilirubin seems plausible because the infection with *H. pylori* might cause an obstruction to normal liver function, thereby constricting the required amount of liver function needed to keep the body healthy. Although the *H. pylori* and liver enzyme connection are notable, the reasoning given flying through the 'reasons given' circuit justifies itself. Albumin levels, as a proxy for liver synthetic activity of the liver, demonstrated no major changes between groups (p=0.05). This indicates that while *H. pylori* infection might influence liver enzymes, it does not cause significant disruption in hepatic protein synthesis during early stages. For this cohort, creatinine levels, suggesting renal function, also did not vary significantly (p=0.43). This shows that *H. pylori* infection did not have an impact on renal function in this group.

3.4 Severity of Liver Disease and H. Pylori Infection

The positive *H. pylori* group showed more severe liver disease, particularly liver fibrosis, cirrhosis, and hepatocellular carcinoma (HCC). In the same table two, the presence of cirrhosis and HCC in *H. pylori* infected patients was significantly higher in comparison to *H. pylori* negative patients **Table 3**. The data from the table exhibited that the cirrhosis and HCC prevalence were meaningfully higher in patients diagnosed with *H. pylori* infection. These indications reinforce that such infections worsen liver pathology, particularly in the context of the preexisting liver damage or other HCV-alcohol risk factors, thus establishment the assumption that *H. pylori* could be conducive to further liver damage.

3.5 Multivariate Regression Analysis

To estimate the independent relationship of *H. pylori* infection with liver disease severity, a multivariate logistic regression analysis was passed out while adjusting for age, sex, alcohol intake, smoking, and other comorbid conditions such as hypertension and diabetes. From the regression models, it was revealed that the presence of *H. pylori* contamination was indeed associated with advanced liver disease; *H. pylori* infected patients had 1.7-fold increased risk of developing cirrhosis (OR = 1.72, 95% CI: 1.01–2.92, p = 0.04) and 2.0 fold increased risk of developing HCC (OR = 2.05, 95% CI: 1.12–3.75, p = 0.02) when compared to non *H. pylori* infected patients **Figure 2.** The interpretation of these results is that *H. pylori* infection self-reliantly increases the odds of liver fibrosis, cirrhosis, and HCC, irrespective of other factors.

4. Discussion

4.1 Overview of the Key Findings

The current investigation reveals a noteworthy link between *Helicobacter pylori* (*H. pylori*) infection and liver dysfunction in sufferers with antecedent liver damage or chronic conditions like

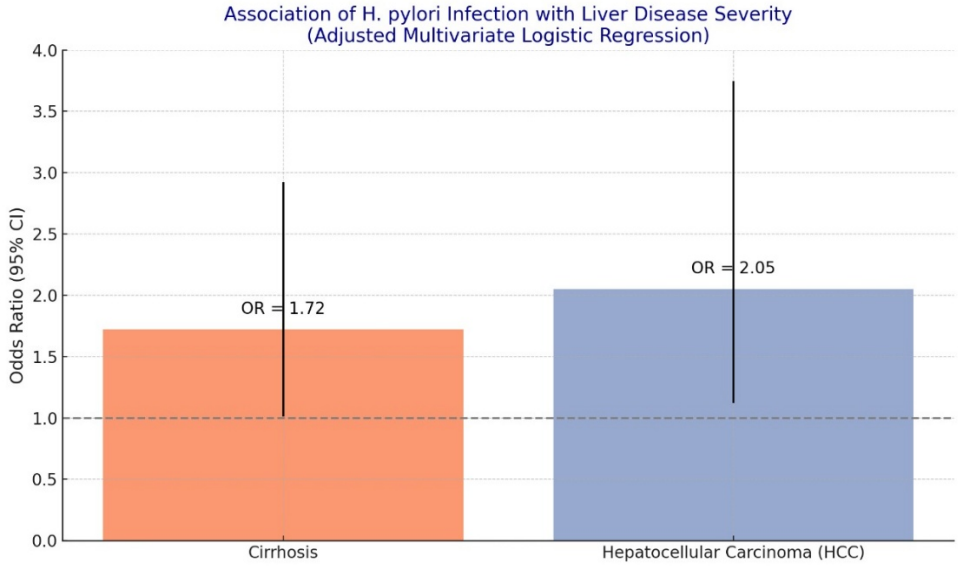

Figure 2. Association of *H. Pylori* Infection with Liver Disease Severity (Adjusted Multivariate Logistic Regression)

hepatitis and cirrhosis (Mantovani et al., 2024). We noticed that *H. Pylori* positive participants had significantly elevated liver enzymes, namely, ALT, AST, ALP, and bilirubin when compared to negative participants. These enzymes indicate cell death and diminished liver capacity; therefore, it is reasonable to assume that *H. Pylori* infection worsens liver damage. The investigation also discovered that the *H. Pylori* positive subgroup had greater rates of liver cirrhosis and hepatocellular carcinoma (HCC) revealing the possible contribution of this microorganism in the advancement of liver illnesses. In *H. Pylori* infected participants, regression analysis showed persistent dependence for both cirrhosis and HCC raising the assumption of *H. Pylori* infection as a liver disease progression factor, whether primary or secondary, in patients who harbor liver damage.

4.2 The Role of H. pylori in Liver Diseases

There has been increased research into how *H. pylori* may cause liver pathologies such as liver fibrosis, cirrhosis, and liver cancer (Tufael et al., 2024). For now, it has been shown that *H. pylori* infection becomes more potent with co-factors like infection with the hepatitis C virus (HCV), alcohol consumption, and other metabolic disorders like diabetes and hypertension. *H. pylori* has been shown to trigger chronic inflammation and immune response as well as other extra-gastric manifestations like in the liver (Rabelo-Gonçalves, 2015). The actual ability of the pathogen to stimulate the gut-liver axis by overproducing pro-inflammatory cytokines and altering the composition of the gut flora could further liver damage (Zheng & Wang, 2021). Further, *H. pylori* infection also induces oxidative stress and fibrosis in the liver which accelerates liver cancer. The liver pathways affected in *H. pylori* positive individuals as noted in this study supports the argument that something may be wrong with *H. Pylori* positive patients' liver. Altered levels of these enzymes are frequently confronted in clinical liver pathologies. The increase of bilirubin in *H. pylori* positive patients indicates that these patients may also have problems with liver function associated with bile secretion and discharge (Rees & Sinha, 1960).

4.3 H. pylori and Liver Fibrosis/Cirrhosis

The correlation between the *H. pylori* positive group and the prevalence of cirrhosis and its complications was evident in prior studies suggesting that *H. Pylori* has some role in liver fibrosis and cirrhosis (Tufael et al., 2024). Excessive immune response associated with chronic *H. pylori* might also facilitate oxidative stress which would lead to inflammation and subsequently increase the rate of fibrosis in the liver (Akter et al., 2024). It has also formulated the hypothesis that the infection could further elevate the liver's fibro genic reaction by increasing collagen restriction and the activation of hepatic stellate cells. In our study, the rate of cirrhosis was significantly higher in patients infected with *H. pylori* compared to those who were not (21.2% vs. 12.0%, p=0.04) (Feng et al., 2014). This indicates that chronic infection of *H. pylori* may influence the transition phase of liver inflammation by fibrosing the liver's tissues. Nevertheless, it should be highlighted that cirrhosis of the liver is a complex condition and *H. Pylori* infection could be one of the factors in addition to alcohol consumption, some metabolic conditions, and viral hepatitis (Abadi, 2017).

4.4 H. pylori and Hepatocellular Carcinoma (HCC)

The association between HCC and *H. pylori* positivity (>11.4% in *H. Pylori* Positive individuals vs. 6% in Negative Individuals, p=0.03) stands out in our results given that HCC is the second most prevalent cancer by morbidity rate (Prudente et al., 2025). While

the primary association of *H. pylori* is with gastric cancer, there is increasing evidence that it might also influence HCC development, especially in the setting of chronic liver diseases. The inflammation catalyzed by *H. pylori*, specifically in conjunction with other liver damage causatives like chronic viral hepatitis and/or alcoholic liver disease, could enhance the likelihood for liver cancer (S. Kumar et al., 2021). The ways in which *H. pylori* affects HCC are not well understood, but multiple pathological avenues have been discussed. For example, *H. pylori* might induce the secretion of pro-inflammatory cytokines TNF-α and IL-6, resulting in hepatic inflammation and fibrosis, which are instrumental in HCC progression (Dincă et al., 2022). Moreover, *H. pylori* is known to cause oxidative stress and DNA damage which are pivotal in the start of oncogenesis. This study's finding of significantly higher rates of HCC in individuals infected with *H. pylori* seem to strengthen the potential contribution of the pathogen to the malignancy's premalignant progression, especially in the context of other risk factors (Shin et al., 2023).

4.5 Implications of Co-Morbidities

Most of the study population had co-morbidities of hypertension (28.4%) and diabetes (27.9%) which are both considered to exacerbate the progression of liver disease (Singh et al., 2021). These co-morbidities may worsen the impact of *H. pylori* infection in liver disease. Diabetes, for example, is linked to greater fat infiltration of the liver, greater insulin resistance, and greater hepatic fibrosis. Hypertension, on the other hand, is a well-established risk determinant for cardiovascular diseases which, in turn, may impair liver functions. Additionally, the combination of *H. pylori* infection and these co-morbidities could pose an increased risk for developing cirrhotic liver and HCC. The study pointed out that people with *H. pylori* infection and co-morbid hypertension or diabetes had significantly worse liver function than those without these conditions, underscoring the need to address these factors in chronic liver disease patients (Belete et al., 2024).

4.6 Study Limitations

This study certainly provides useful information, but it also comes with certain limitations. First, containing the study within a specific time frame makes this research unable to assess true causation between *H. pylori* infection and liver disease progression. This would require more research to verify the connection between *H. pylori* infection and the liver's development of fibrosis, cirrhosis, oncogenic HCC. Moreover, the research focused on diagnosing *H. Pylori* using serological means and Chemiluminescence Immunoassay (CLIA) which do not differentiate between active and former infections. Alongside this, the research evaluates progression of liver disease with liver function tests in addition to clinical examination rather than using biopsy or electrography which contains more accurate information on the level of liver fibrosis.

5. Conclusion

This research conclusively illustrates the link between *H. pylori* infection and the liver's dysfunction fibrosis, cirrhosis, and HCC. The evidence hints that *H. pylori* may influence the modulating factor in the advancement of liver disease along with other risk factors like hepatitis, alcohol consumption, and metabolic disorders. Considering the wide occurrence of *H. pylori* infection in people, additional research is required to understand how exactly *H. pylori* infection links with liver disease and to study the possible treatment options for the infection for those applied to liver complications.

Author contributions

M.H and M.S.B. conceptualized, conducted lab and field works, analyzed data, wrote the original draft, reviewed, and edited; M.M.H. and A.S. conducted research design, validated methodology, analyzed, visualized the data, reviewed, and edited; S.S.D. P.P. and M.S validated the methodology, analyzed data, investigated, visualized, reviewed, and proof-read; M.M.R. M.A.S. and M.K.M.U. conceptualization, conducted research design, validated methodology, conducted analysis, investigated, visualized the data, reviewed, supervised and edited the paper. All authors read and approved the paper for publication.

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Competing financial interests

The authors have no conflict of interest.

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