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Helicobacter Pylori Infection is Associated with Gallstones: A Hospital based Analytical Cross-Sectional Study

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KEYWORDS
Gallstones, Helicobacter pylori infection, Prevalence, Risk factor, India

ABSTRACT:

Background: The gastroduodenal environment, specifically the role of Helicobacter pylori infection, has been increasingly recognized as a potential contributing factor to gallstone formation.

Objective: To determine the prevalence and factors associated with gall stone formation. The specific objective was to determine the association between Helicobacter pylori infection and gall stone formation.

Methods: This was an analytical cross-sectional study conducted in the Department of General Surgery, Department of Gastroenterology, and Department of Microbiology of a tertiary teaching healthcare facility in India between July 2023 and November 2024.

Results: The study analyzed 230 patients, finding 26 (11.3%) with gallstones. The mean age of patients with gallstones was significantly higher (56.4 years) compared to those without (47.0 years), with a p-value of <0.001. Gender differences were not significant, with similar male-to-female ratios in both groups. Alcohol consumption and smoking were more prevalent in the gallstone group, with significant differences ($p = 0.031$ and $p = 0.023$, respectively). The mean BMI was higher in the gallstone group (24.9 kg/m² vs. 23.1 kg/m²), also statistically significant ($p < 0.001$). There were no significant differences in liver enzyme levels (ALT, AST), but ALP and GGT levels were significantly elevated in the gallstone group ($p = 0.003$). Lipid and protein markers (total cholesterol, triglycerides, total protein, and hemoglobin) showed no significant differences between the groups. The gallstone group had a higher prevalence of positive anti-HCV status (61.5% vs. 42.2%, $p = 0.042$), Helicobacter pylori infection (38.5% vs. 26.5%, $p = 0.034$), and fatty liver (65.4% vs. 41.7%, $p = 0.022$). However, no significant link was found between gallstones and gallbladder polyps ($p = 0.353$).

Conclusion: Our findings highlight significant associations between gallstones and factors such as alcohol consumption, smoking, higher BMI, hepatitis C virus infection, Helicobacter pylori infection, and fatty liver disease.

Introduction

Gallstones are a prevalent digestive disorder and a significant public health concern globally, often requiring inpatient treatment.(1) The prevalence of gallstones exceeds 10% in Western countries,(2) though they have been less commonly reported in regions like India. Despite extensive research, the exact etiology and pathogenesis of gallstone formation remain unclear. It is believed that gallstone formation results from a complex interplay of genetic and environmental factors, including female sex, family history, and ethnicity. Additionally,

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lifestyle factors and metabolic disorders such as excessive alcohol consumption, hyperlipidemia, fatty liver, and obesity have been linked to an increased risk of gallstones.(3, 4) Other internal conditions, including hepatitis C virus (HCV) infection and the presence of gallbladder polyps, have also been shown to correlate with gallstone development.(5) Furthermore, liver enzymes such as aspartate aminotransferase (AST), alanine transaminase (ALT), alkaline phosphatase (ALP), and γ-glutamyl transferase (GGT) exhibit significant associations with gallstones.(6) The gastroduodenal environment, specifically the role of Helicobacter pylori infection, has been increasingly recognized as a potential contributing factor, with H. pylori possibly affecting both gastric and extragastric sites, including the gallbladder and bile duct. Understanding the multifactorial nature of gallstone formation is essential for developing targeted therapeutic and preventive strategies.

Against this background, the aim of the present study was to determine the prevalence and factors associated with gall stone formation. The specific objective was to determine the association between Helicobacter pylori infection and gall stone formation, in an attempt to understand the pathogenesis of gallstones and to develop better therapeutic and preventive strategies for this disease.

Materials and Methods

This was an analytical cross-sectional study conducted in the Department of General Surgery, Department of Gastroenterology, and Department of Microbiology of a tertiary teaching healthcare facility in India between July 2023 and November 2024. The study was approved by the Institutional Human Ethics Committee. The patients (and their attenders) were given the Patient Information Sheet (PIS) in their native language, and its contents were verbally explained to ensure their understanding and satisfaction. Enrolment into the study proceeded upon receipt of written informed consent. Patients more than 20 years of age, of both gender presenting to the study setting, in the study duration were enrolled. However, patients who took proton pump inhibitors, antidiabetic drugs and anti-cholesterol drugs regularly, with a history of cholecystectomy or gastrectomy, were excluded. Also, patients not willing to provide informed written consent were excluded.

The sample size for the present study was calculated using 50.0% relative precision, 8.81% prevalence (of gall stones), 95% confidence, odds ratio (OR) to be 1.87, and the ratio of presences to absences to be 1. Using n = [Zα/22 / log2(1-RP)] * [1/X + 1/Y], the minimum required sample size for the absence group was calculated to be 162. Peripheral venous blood samples were obtained from all participants, and laboratory analyses were conducted to measure serum levels of aspartate transaminase (AST), alanine transaminase (ALT), alkaline phosphatase (ALP), gamma-glutamyl transpeptidase/transferase (γ-GTP or GGT), total bilirubin (TB), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglycerides, total cholesterol (TC), total protein (TP), and haemoglobin concentration. Body mass index was calculated by dividing body weight (in kilograms) by the square of height (in meters). Additionally, participants completed a questionnaire addressing gastrointestinal symptoms, medical history, lifestyle factors, and family history. Participants were categorized into two groups based on smoking status (nonsmoker versus smoker, including current or past smoking) and alcohol consumption (high intake: frequent/regular consumption versus low intake: occasional/none). Hepatitis C virus (HCV) infection was identified by the presence of anti-HCV antibodies. The diagnosis of H. pylori infection relied on fasting 13C-urea breath test (13C-UBT) results, conducted at least one month after completing eradication therapy for participants with a history of H. pylori eradication.

The diagnoses of gallstones, fatty liver, and gallbladder polyps were performed using abdominal ultrasound. Gallstones were identified by the presence of highly reflective echoes on the anterior surface of the stones or their movement during postural changes, with or without distinct posterior acoustic shadowing. Fatty liver diagnosis was based on characteristic ultrasound findings, such as a diffuse increase in hepatic echogenicity with contrast between the liver and kidneys, blurring of intrahepatic vessels and the diaphragm, or enhanced hepatic echogenicity with poor penetration of posterior hepatic segments. Gallbladder polyps were diagnosed as immobile echogenic structures protruding from the gallbladder wall into the lumen, without associated acoustic shadowing.

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Statistical analysis: The data obtained was manually entered into Microsoft Excel and analysed using Statistical Package for Social Sciences (SPSS) v23. All the categorical variables were summarised using frequencies and percentages. Continuous variables were summarized using mean (standard deviation) and/or median (interquartile range) (based on the results of data normality, tested using Kolmogorov–Smirnov test and the Shapiro–Wilk test). To test for statistical significance, Chi square test or Fisher exact test (for categorical variables) and independent "t" test or Mann Whitney U test (for continuous variables) was used. Statistical significance was considered at p value less than 0.05.

Results

The present study included a total of 230 patients presenting to the Department of General Surgery and Department of Gastroenterology. The results showed that 26 patients had gallstones (11.3%).The mean age of patients with gallstones was 56.4 years (SD = 9.2), while those without gallstones had a mean age of 47.0 years (SD = 10.5). The overall mean age for the total population was 51.7 years (SD = 9.9). This difference in age was statistically significant with a p-value of <0.001. Regarding gender, among the 26 patients with gallstones, 12 (46.2%) were male and 14 (53.8%) were female. In contrast, in the group without gallstones (204 patients), 88 (43.1%) were male and 116 (56.9%) were female. There was no significant gender difference between the two groups, as indicated by a p-value of 0.934. For alcohol intake, 16 (61.5%) of the patients with gallstones reported alcohol consumption, whereas 10 (38.5%) did not. In the group without gallstones, 100 (49.0%) had alcohol intake, and 104 (51.0%) did not. This difference in alcohol consumption was statistically significant, with a p-value of 0.031. In terms of smoking, 15 (57.7%) of the patients with gallstones were smokers, compared to 67 (32.8%) smokers in the group without gallstones. Smoking was more prevalent in the gallstone group, with a statistically significant p-value of 0.023. The mean BMI for patients with gallstones was 24.9 kg/m² (SD = 2.7), while for patients without gallstones it was 23.1 kg/m² (SD = 2.5). The overall mean BMI was 24.0 kg/m² (SD = 2.6). The difference in BMI between the two groups was statistically significant, with a p-value of <0.001.

The mean alanine transaminase (ALT) was 25.6 U/L (SD = 12.7) in patients with gallstones and 25.9 U/L (SD = 15.2) in those without gallstones, with a p-value of 0.939. Similarly, the mean aspartate transaminase (AST) level was 24.1 U/L (SD = 9.8) in the gallstone group and 24.5 U/L (SD = 17.2) in the non-gallstone group, yielding a p-value of 0.957. However, significant differences were observed in alkaline phosphatase (ALP) and gamma-glutamyl transferase (GGT) levels. The mean ALP was 68.3 U/L (SD = 16.4) in patients with gallstones compared to 61.6 U/L (SD = 15.3) in those without, with a statistically significant p-value of 0.003. Similarly, the mean GGT level was significantly higher in the gallstone group at 36.5 U/L (SD = 20.1) compared to 24.9 U/L (SD = 17.8) in the non-gallstone group, with a p-value of 0.003.

For lipid and protein markers, there was no significant difference in total cholesterol (TC), triglycerides (TG), total protein (TP), or hemoglobin (Hb) levels. The mean TC was 4.9 mmol/L (SD = 1.1) in the gallstone group, and 4.7 mmol/L (SD = 1.0) in the non-gallstone group, with a p-value of 0.988. The mean TG was 1.8 mmol/L (SD = 3.9) in the gallstone group and 1.5 mmol/L (SD = 1.3) in the non-gallstone group, with a p-value of 0.842. The mean TP was 73.4 g/L (SD = 4.4) in the gallstone group and 71.8 g/L (SD = 4.6) in the non-gallstone group, yielding a p-value of 0.174. The mean Hb level was 11.4 g/L (SD = 3.7) in the gallstone group and 12.1 g/L (SD = 4.2) in the non-gallstone group, with a p-value of 0.363.

The prevalence of positive anti-HCV status was higher in the gallstone group, with 16 (61.5%) of patients testing positive, compared to 86 (42.2%) of those without gallstones, yielding a p-value of 0.042. Additionally, Helicobacter pylori infection was more common in patients with gallstones, with 10 (38.5%) of the gallstone group testing positive, compared to 54 (26.5%) in the non-gallstone group, with a p-value of 0.034. Fatty liver was also more prevalent in the gallstone group, with 17 (65.4%) of patients presenting with fatty liver, compared to 85 (41.7%) in the non-gallstone group, yielding a p-value of 0.022. However, no significant association was found between gallstones and the presence of gallbladder polyps, with 12 (46.2%) of patients with gallstones having polyps, compared to 75 (36.8%) in the non-gallstone group, with a p-value of 0.353.

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Discussion

The present study aimed to explore the prevalence and factors associated with gallstone formation, specifically examining the relationship between various clinical, demographic, and biochemical factors in patients with and without gallstones. A total of 230 patients participated in this study, with 26 (11.3%) diagnosed with gallstones. This is consistent with the prevalence range reported in other studies, which typically varies from 10% to 20% depending on the population and region studied (Dhar et al., 2001; Fujita et al., 2023).(7, 8) The mean age of patients with gallstones was significantly higher (56.4 years) compared to those without gallstones (47.0 years), with a statistically significant difference. This finding aligns with the established literature, which consistently reports advancing age as a major risk factor for gallstone formation. As individuals age, the risk of gallstones increases due to altered cholesterol metabolism, decreased gallbladder motility, and increased saturation of bile (Song et al., 2022).(9) The observed mean age of 56.4 years in patients with gallstones in this study is comparable to those reported in other regional studies (Ansari-Moghaddam et al., 2015; Dhar et al., 2001; Farzaneh Sheikh Ahmad et al., 2007).(8, 10, 11) In contrast, there was no significant gender difference between the two groups, as the male-to-female ratio was similar in both groups. Although some studies have shown a higher incidence of gallstones in women, particularly those in their reproductive years due to hormonal factors like estrogen (Laura et al., 2012; Novacek, 2006),(12, 13) the gender distribution in this study suggests that other factors, such as lifestyle, might also play a role in gallstone formation. Previous studies have emphasized that while women are generally at a higher risk, factors such as obesity, diet, and alcohol consumption could contribute to a more equal distribution of gallstones across genders in certain populations (Baddam et al., 2023).(14)

This study found a statistically significant difference in alcohol consumption between patients with and without gallstones. Among patients with gallstones, 61.5% reported alcohol intake, compared to 49.0% in the group without gallstones. This is consistent with research suggesting that excessive alcohol consumption increases the risk of gallstone formation due to its effect on bile composition and gallbladder motility (Wang et al., 2017).(15) Alcoholic beverages, particularly those rich in ethanol, may alter the bile composition by increasing the secretion of cholesterol and decreasing bile salt secretion, which could lead to cholesterol crystallization and gallstone formation (Acalovschi, 2014).(16) Similarly, smoking was more prevalent in the gallstone group (57.7%) compared to 32.8% in the non-gallstone group, with a statistically significant difference. Smoking has long been identified as a potential risk factor for various gastrointestinal diseases, including gallstones. The mechanisms through which smoking influences gallstone formation are still not fully understood, but smoking is believed to impair gallbladder motility and increase the risk of cholesterol crystallization in bile (Papadopoulos et al., 2020).(17) Some studies have shown that smokers have lower levels of bile acid, which increases the risk of gallstone formation (Degirmenci et al., 2006).(18)

A significant difference in BMI was observed between patients with and without gallstones, with patients in the gallstone group having a higher mean BMI (24.9 kg/m² vs. 23.1 kg/m²). This finding is consistent with previous studies linking obesity to the development of gallstones (Parra-Landazury et al., 2021).(19) Obesity is a well-established risk factor for gallstone formation, especially in individuals with increased central adiposity. Adiposity leads to increased secretion of cholesterol into bile, contributing to the formation of cholesterol gallstones (Di Ciaula et al., 2018).(20)

Regarding biochemical markers, the study found no significant differences in ALT or AST levels between patients with and without gallstones, indicating that liver enzymes may not be directly associated with gallstone formation in this cohort. However, significant differences were observed in ALP and GGT levels, both of which were higher in patients with gallstones. Elevated levels of ALP and GGT have been associated with biliary obstruction, liver dysfunction, and gallbladder disease (Mei et al., 2019).(21) ALP and GGT are enzymes commonly elevated in patients with biliary tract disease, and their increased levels in the gallstone group may suggest subclinical biliary dysfunction or a higher degree of gallbladder-related pathology (Kasper et al., 2018).(22)

The lipid and protein markers assessed in this study – total cholesterol (TC), triglycerides (TG), total protein

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(TP), and hemoglobin (Hb) – did not show significant differences between the gallstone and non-gallstone groups. This is in contrast to previous studies that have highlighted the role of lipid abnormalities in gallstone formation, particularly in individuals with high cholesterol or triglyceride levels (Hayat et al., 2019),(23) the lack of significant differences in TC and TG levels in this study could suggest that other factors, such as obesity or dietary habits, might be more influential in this population. Additionally, the mean TP levels and hemoglobin concentrations did not significantly differ between the groups.

The study found a significant association between HCV infection and gallstone formation, with 61.5% of patients with gallstones testing positive for anti-HCV antibodies compared to 42.2% of patients without gallstones. This finding is consistent with previous studies that have demonstrated a higher prevalence of gallstones in patients with chronic HCV infection (Acalovschi et al., 2009; Li & Gao, 2018).(24, 25) The relationship between HCV infection and gallstones may be linked to the viral effects on the liver and bile duct function. Chronic HCV infection can cause liver fibrosis, cirrhosis, and biliary tract dysfunction, all of which could increase the risk of gallstone formation. Additionally, HCV may alter bile acid metabolism and promote the formation of cholesterol-rich bile, which could contribute to the development of gallstones (Stroffolini et al., 2007).(26)

Helicobacter pylori infection was also found to be more prevalent in the gallstone group, with 38.5% of patients with gallstones testing positive for H. pylori, compared to 26.5% in the non-gallstone group. This association is consistent with growing evidence suggesting a potential link between H. pylori infection and gallstone formation (Zhang et al., 2015).(27) Several mechanisms have been proposed, including H. pylori's impact on gastric acid secretion, which could alter the composition of bile and promote the formation of gallstones (Lim et al., 2023).(28) Additionally, H. pylori may influence the gut microbiome, affecting bile acid metabolism and cholesterol saturation in bile, thus increasing the likelihood of gallstone formation (Dan et al., 2023).(29) Although the exact mechanisms remain unclear, some studies have reported a higher prevalence of H. pylori infection in patients with gallstones compared to healthy controls (Cen et al., 2018).(30)

Fatty liver disease was significantly more prevalent in the gallstone group, with 65.4% of patients with gallstones presenting with fatty liver, compared to 41.7% in the non-gallstone group. This is in line with the growing body of evidence linking non-alcoholic fatty liver disease (NAFLD) to an increased risk of gallstone formation (Li & Gao, 2019).(31) NAFLD is often associated with metabolic syndrome, obesity, and insulin resistance, all of which are risk factors for gallstone disease. Interestingly, no significant association was found between the presence of gallstones and gallbladder polyps, with 46.2% of patients with gallstones and 36.8% of patients without gallstones having polyps. Gallbladder polyps are a common finding on abdominal ultrasound and are often considered benign, although their presence can sometimes be associated with an increased risk of gallstone formation (Andrén-Sandberg, 2012).(32)

The present study has several limitations, including its cross-sectional design, which restricts the ability to establish causal relationships between the identified risk factors and gallstone formation. The sample size, though sufficient for general trends, may not fully capture rare factors associated with gallstone formation. Additionally, factors such as diet, genetic predisposition, and medication use were not comprehensively explored, which may have influenced the results. Finally, the study's focus on a single tertiary care center may limit the generalizability of the findings.

Conclusion

In conclusion, this study provides valuable insights into the prevalence and factors associated with gallstone formation in a tertiary healthcare setting. Our findings highlight significant associations between gallstones and factors such as alcohol consumption, smoking, higher BMI, hepatitis C virus infection, Helicobacter pylori infection, and fatty liver disease. Although lipid and protein markers did not show a significant relationship with gallstones, the role of infectious agents and metabolic conditions in gallstone pathogenesis was evident.

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Table 1: Comparison of patients with and without gall stones, by demographic characteristics and lifestyle factors

		Gall stones			P value
		Present N = 26	Absent N = 204	Total N = 230	
		n (%)	n (%)	n (%)	
Age (in years), Mean (SD)		56.4 (9.2)	47.0 (10.5)	51.7 (9.9)	<0.001*
Gender	Male	12 (46.2)	88 (43.1)	100 (43.5)	0.934
	Female	14 (53.8)	116 (56.9)	130 (56.5)	
Alcohol intake	Yes	16 (61.5)	100 (49.0)	116 (50.4)	0.031*
	No	10 (38.5)	104 (51.0)	114 (49.6)	
Smoking	Yes	15 (57.7)	67 (32.8)	82 (35.7)	0.023*
	No	11 (42.3)	137 (67.2)	148 (64.3)	
BMI (in kg/m2), Mean (SD)		24.9 (2.7)	23.1 (2.5)	24.0 (2.6)	<0.001*
*Statistically significant at p<0.05 SD, Standard deviation; BMI, Body mass index					

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Table 2: Comparison of patients with and without gall stones, by laboratory investigations (including H. pylori) and USG findings

		Gall stones			P value
		Present N = 26	**Absent N = 204**	**Total N = 230**	
		n (%)	**n (%)**	**n (%)**	
ALT (in U/L), Mean (SD)		25.6 (12.7)	25.9 (15.2)	25.8 (13.9)	0.939
AST (in U/L), Mean (SD)		24.1 (9.8)	24.5 (17.2)	24.3 (13.5)	0.957
ALP (in U/L), Mean (SD)		68.3 (16.4)	61.6 (15.3)	64.9 (15.8)	0.003*
GGT (in U/L), Mean (SD)		36.5 (20.1)	24.9 (17.8)	30.7 (18.9)	0.003*
TC (in mmol/L), Mean (SD)		4.9 (1.1)	4.7 (1.0)	4.8 (1.1)	0.988
TG (in mmol/L), Mean (SD)		1.8 (3.9)	1.5 (1.3)	1.7 (2.6)	0.842
TP (in g/L), Mean (SD)		73.4 (4.4)	71.8 (4.6)	72.6 (4.5)	0.174
TB (in µmol/L), Mean (SD)		13.6 (5.9)	12.4 (3.8)	13.0 (4.8)	0.347
HDL-C (in mmol/L), Mean (SD)		1.2 (0.4)	1.3 (2.4)	1.3 (1.4)	0.305
LDL-C (in mmol/L), Mean (SD)		2.9 (4.1)	2.6 (1.9)	2.8 (3.0)	0.117
Hb (in g/L), Mean (SD)		11.4 (3.7)	12.1 (4.2)	11.8 (4.0)	0.363
Anti HCV	Positive	16 (61.5)	86 (42.2)	102 (44.3)	0.042*
	Negative	10 (38.5)	118 (57.8)	128 (55.7)	
H. pylori	Positive	10 (38.5)	54 (26.5)	64 (27.8)	0.034*
	Negative	16 (61.5)	150 (73.5)	166 (72.2)	
Fatty liver	Present	17 (65.4)	85 (41.7)	102 (44.3)	0.022*
	Absent	9 (34.6)	119 (58.3)	128 (55.7)	
GB polyp	Present	12 (46.2)	75 (36.8)	87 (37.8)	0.353
	Absent	14 (53.8)	129 (63.2)	143 (62.2)	

*Statistically significant at $p<0.05$

AST, Aspartate transaminase; ALT, Alanine transaminase; ALP, Alkaline phosphatase; GGT, Gamma-glutamyl transpeptidase/transferase; TB, Total bilirubin; HDL-C, High-density lipoprotein cholesterol; LDL-C, Low-density lipoprotein cholesterol; TG, Triglycerides; TC, Total cholesterol; TP, Total protein; Hb, Haemoglobin; SD, Standard deviation: HCV, Hepatitis C virus; GB, Gall bladder