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RESEARCH ARTICLE

Association of Helicobacter Pylori with Gall Stone Disease

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Abstract: ***Background***: Gallstonedisease(cholelithiasis)isacommongastrointestinalconditionwith multifactorial etiology, including genetic, metabolic, and microbial factors. Helicobacter pylori (H. pylori), a gram-negative bacterium linked to gastric pathologies, has been implicated in gallstone pathogenesis through mechanisms such as bile acid metabolism alterations, lipid profile changes, and chronic biliary inflammation. ***Objectives***: This study evaluatestheassociationbetweenH.pyloriinfectionandgallstone disease, examining demographic factors, clinical features, endoscopic findings, and histopathological outcomes. ***Methods***: A prospective observational study was conducted on 100 patients diagnosed with gallstone disease. H. pylori detectionemployed RapidUreaseTest (RUT) and Giemsa staining. Clinical, demographic, endoscopic, andhistopathological data were analyzed. ***Results***: Thecohortincluded78%femaleparticipants, withameanageof41.91± 14.96 years and peak prevalence in the 20–39 age group (48%). Abdominal pain was the most frequent symptom (55%), followed by heartburn (30%) and bloating (15%). Endoscopy revealed antral gastritis in 73% and erosive gastritis in 27% of cases. Histopathologyconfirmedchroniccholecystitiswith cholelithiasis in 89% and acute-on-chronic inflammation in 11%. H. pylori detection rates varied significantly between RUT (97%) and Giemsa staining (16%), with Giemsa positivity declining in older patients, highlighting diagnostic limitations. ***Conclusions***: The findings suggest a potentiallinkbetweenH.pyloriandgallstonedisease, emphasizing the need for advanced molecular diagnostics. Further studiesare warranted to elucidate H. pylori's role in gallstone pathogenesis and explore its preventive potential.

Keywords: Gallstone disease, Helicobacter pylori, cholelithiasis, Rapid Urease Test, chronic cholecystitis.

INTRODUCTION

Gallstone disease (cholelithiasis) is a common gastrointestinal condition caused by the formation of stones in the gallbladder, which stores bile essential for digestion. These stones can lead to a spectrum of clinical manifestations, from asymptomatic cases to severe complications such as cholecystitis, pancreatitis, and biliary obstruction. The disease has a multifactorial etiology, involving genetic predisposition, metabolic abnormalities, and microbial factors.[1]

Among potential microbial contributors, *Helicobacter pylori* (*H. pylori*), a gram-negative bacterium primarily associated with gastric pathologies, has garnered attention for its possible role in gallstone disease.[2] *H. pylori* is widely known for causing chronic gastritis, peptic ulcers, and gastric cancer, but its systemic effects—mediated through chronic inflammation, immune dysregulation, and metabolic changes—may extend to the biliary system.[2] Studies suggest that *H. pylori* may influence gallstone formation through mechanisms such as bile acid metabolism alterations, lipid profile changes, and chronic inflammation of the biliary epithelium.[3–4]

Epidemiological data regarding the association between *H. pylori* and gallstone disease are conflicting.[5] While some studies report a significant correlation,[6] others find no conclusive link,[7] possibly due to differences in study design, population demographics, and geographical variations.[5] Furthermore, the role of *H. pylori* in different gallstone types (cholesterol, pigment, and mixed stones) remains an area of active research, with hypotheses suggesting a stronger association with pigment stones due to bacterial involvement in bilirubin metabolism.[8]

Understanding the interplay between *H. pylori* and gallstone disease could have significant diagnostic and therapeutic implications.[9] If a causal relationship is established, routine screening and eradication of *H.pylori*may serve as a preventive strategy for high-risk populations.[5] This study aims to explore the potential association between *H. pylori* infection and gallstone disease, providing new insights into the microbial factors contributing to biliary pathology and addressing existing gaps in the literature.[5–7]

MATERIAL &METHODS

This prospective observational study will be conducted in the Department of General Surgery at CSSH Medical College to investigate the clinical correlation between *Helicobacter pylori* infection and gallstone disease.

StudyDuration

The research will be conducted over a two-year period, spanning from 2023 to 2025.

EthicalConsiderations
Approval for the study will be obtained from the Institutional Ethics Committee prior to its commencement. All participants will provide written informed consent, ensuring voluntary participation and adherence to ethical research principles.

SampleSizeandPopulation
The study will include 100 patients diagnosed with gallstone disease who meet the inclusion and exclusion criteria.

InclusionCriteria
Patientswithaclinicaldiagnosisofgallstonedisease.

Individualswhovoluntarilyconsenttoparticipateinthestudy.

ExclusionCriteria
- Immunocompromisedpatients.
- Patientsunwillingtoparticipate.

METHODOLOGY
Gallbladder specimens will be collected from participants undergoing cholecystectomy. Histopathological evaluation will be performed as follows:
Sectionswillbesubjectedtoroutinehematoxylinandeosin(H&E) staining for morphological analysis.

Additional sections will be processed using Giemsastaining to identify the presence of H. pylori.

Specimens will be fixedin10% buffered formalin, and Giemsastaining will be performed using a modified protocol to enhance bacterial visualization. Light microscopy will be utilized to identify *H. pylori-like* bacteria, characterizedas curved, spiral, bent, pole-like, or fusiform in morphology.

StatisticalAnalysis
Descriptive statistics will be used to summarize the data. Categorical variables will be comparedusingtheChi-squaretest, withap-valueof<0.05 considered statistically significant.

InformedConsent
All participants provided written informed consent after beingin formed of the study's objectives and procedures.

RESULT

The study enrolled 100 patients clinically diagnosed with gallstone disease, with a mean age of 41.91 ± 14.96 years. Themajorityofpartici pants (48%) were between 20 and 39 years of age, followed by 34% in the 40–59 years group, 14% aged 60 years or above, and 4% under the age of 20. Female patients formed the predominant proportion of the study population, constituting 78%, while males accounted for 22%. These findings highlight the predominance of gallstone disease in younger to middle-agedindividuals and its significant prevalence among females. (Table 1).

Table1: Demo graphic CharacteristicsofStudy Participants

Characteristic	**Subcategory**	**n(%)**
AgeRange(Years)	<20	4(4%)
	20–39	48(48%)
	40–59	34(34%)
	≥60	14(14%)
MeanAge	-	41.91±14.96
Sex	Female	78(78%)
	Male	22(22%)

Abdominal pain emerged asthemostfrequentlyreportedsymptom, affecting 55% of the patients, making it the leading cause for seeking medical attention. This was followed by heartburn in 30% and abdominal bloating in 15%. The prominence ofabdominalpainunderscoresitsdiagnosticrelevance in gallstone disease. (Table 2).

Table2: Clinical Presentation of Study Participants

ChiefComplaint	**Number of Patients(n)**	**Percentage(%)**
PainAbdomen	55	55%
Heartburn	30	30%
AbdominalBloating	15	15%

Diagnostic evaluation using upper gastrointestinal endoscopy revealed that antral gastritis was the most prevalent finding, observed in 73% of the patients, while 27% demonstrated erosive gastritis. Histopathological examination of gallbladder specimens further corroborated these findings, with 89% of patients diagnosed with chronic cholecystitis associated with cholelithiasis, and 11% diagnosed with acute cholecystitis withcholelithiasis. These findingsemphasizethepredominanceofchronicinflammatorychanges in the gallbladder among patients with gallstone disease. (Table 3).

Table3: Diagnostic Findings of Study Participants

DiagnosticMethod	Finding	n(%)
UpperGIEndoscopy	AntralGastritis	73(73%)
	ErosiveGastritis	27(27%)
Histopathology	ChronicCholecystitiswith Cholelithiasis	89(89%)
	AcuteCholecystitiswith Cholelithiasis	11(11%)

The study explored the presence of *Helicobacter pylori* using two diagnostic modalities: the Rapid Urease Test (RUT) and Giemsa staining. The RUT demonstrated an overwhelming positivity rate of 97%, suggesting a high prevalence of *H. pylori* infection among the cohort. In contrast, Giemsa staining detected *H. pylori* in only 16%ofthespecimens, indicatingpotential methodologicaldifferencesinsensitivityorvariability inbacterialload. Age-stratified analysis revealed consistent positivity rateswithRUTacrossall age groups, exceeding 94% in all categories. Conversely, Giemsa positivity declined with increasing age, being highest(25%)inpatientsunder20years and absent in individuals aged 60 years or above. This divergence underscores the necessity to evaluate the diagnostic accuracy of different methodologies and their implications in assessing *H. pylori* infection. (Table 4).

Table4: Detection of H. pyloriin Gall stone Patients

DiagnosticTest	Result	n(%)
RapidUreaseTest(RUT)	Positive	97(97%)
	Negative	3(3%)
GiemsaStain	Positive	16(16%)
	Negative	84(84%)

These results suggest a significant association between *H. pylori* and gallstone disease, as evidenced by the high detection rates with RUT. The age-dependent decline in Giemsa positivity further reflects potential differences in bacterial distribution or host-pathogen interactions, warranting further investigation into the roleof*H.pylori*inthepathogenesisofgallstone disease

DISCUSSION

The present study investigated the association between *Helicobacter pylori* (*H. pylori*) infection and gallstone disease in a cohort of 100 patients, examining demographic factors, clinical features, endoscopic findings, and histopathological outcomes. The findings provide a nuanced perspective on the role of *H. pylori* in gallstone pathogenesis while emphasizing diagnostic challenges. The results are discussed in the context of previous literature to elucidate their implications.

The demographic analysis revealed a predominance of female participants (78%), consistent with prior studies reporting a higher prevalence of gallstonediseaseinwomenduetohormonalfactors,includin g estrogen-mediated cholesterol saturation in bile and progesterone-induced gallbladder hypomotility.[1–3] The mean age of 41.91 ± 14.96 years, with a peak prevalence in the 20–39 age group (48%), differs from findings in Western populations, where gallstone incidence typically peaks in the 40–60 age range.[4] This younger age distribution aligns with regional variations in dietary habits, genetic predispositions, and infection patterns.[5]

Abdominal pain was the most commonly reported symptom (55%), consistent with the classical presentation of biliary colic described in numerous studies.[5–6] The relatively high prevalence of heartburn

(30%) and bloating (15%) in this cohort is notable and suggests an overlap with functional dyspepsia, a condition often linked to *H. pylori* infection.[7] Unlike Western populations, where gallstone disease often coexists with metabolic syndrome components such as obesity and diabetes,[1–3] the absence of significant comorbidities in this study suggests a potentially different pathogenic mechanism in this population.

Endoscopic findings revealed a high prevalence of antral gastritis (73%) and erosive gastritis (27%), consistent with previous studies linking *H. pylori* infection to gastroduodenal inflammation.[8] Histopathological analysis confirmed chronic cholecystitis with cholelithiasis in 89% of cases, closely aligning with classic descriptions of gallstone pathology.[9] Acute-on-chronic inflammation was observed in 11% of cases, slightly lowerthanthe20–30% reported bypreviousstudies,[10] possiblyreflectingearlierclinicalinterventions or differences in disease progression in this cohort.
The detection of *H. pylori* revealed significant discrepancies between diagnostic methods, withapositivityrateof97%usingtheRapidUreaseTest (RUT) and only 16% using Giemsa staining. Similar inconsistencies havebeen noted in earlier research, such as studies reporting higher detection rates of *H. pylori* DNA in gallbladder tissue using molecular techniques compared to traditional histologicalmethods.[11]Thishighlightsthelimitations of conventional diagnostic tools in identifying *H. pylori* in extra-gastric sites and underscores the need for more sensitive molecular diagnostics, such as PCR or immunohistochemistry.

Age-specific trends in *H. pylori* positivity revealed that RUT maintained consistently highdetectionrates(>94%)acrossallagegroups,whileGie msa staining positivity declined with age, reaching 0% in patients aged 60 years or older. This decline could be due to age-related changes in gallbladder mucosa, reducing bacterial colonization.[12] This finding contrasts withstudies documenting increasing prevalence of gastric *H. pylori* infection with age,[13] highlighting a possible divergence in the epidemiology of *H. pylori* in gastric and biliary environments.

This study adds to the growing body of evidence suggesting a potential link between *H. pylori* infection and gallstone disease, though the exact role remains contentious.[14] While molecular studies have identified*H.pylori*DNA in gallbladder tissue, the inconsistencies in detection rates using different diagnostic methods underscore the need for further research. Future studies employing advanced molecular diagnostics and larger sample sizes are warranted to confirm the role of *H. pylori* in gallstone pathogenesis and determine whether it acts as a causative agent or a bystander in a multifactorial disease process.

CONCLUSION

This study highlights a significant association between *Helicobacter pylori* infection and gallstone disease, with a high detection rate of 97% via Rapid Urease Test (RUT). However, the low Giemsa stain positivity (16%) underscores diagnostic discrepancies, likely due to variations in test sensitivity, bacterial load, or potential false positives with RUT. Chronic cholecystitis with cholelithiasis predominated (89%), reflecting established gallstone-related pathology.

The demographic analysis revealed a marked female predominance (78%) and a declining Giemsa positivity with age, suggesting possible age-related changesinallbladdermucosalintegrityorimmuneresponse . Whilepotential mechanisms, including bile composition alterations and gallbladder motility effects, support a role for *H. pylori* in gallstone pathogenesis, the findings also raise questions about whether thisrepresentstruebiliarycolonizationor gastric contamination.
Standardized diagnostic protocols and advanced molecular techniques such as PCR are necessary for definitive *H. pylori* detection in biliary tissue. Further research is required to clarify the clinical relevance of *H. pylori* in gallstone disease, its pathogenic mechanisms, and the impact of eradication therapy, ensuring management remains aligned with established therapeutic practices.

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