Original Research Paper

Volume - 15 | Issue - 06 | June - 2025 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

General Surgery

A COMPARATIVE PROSPECTIVE STUDY ON HELICOBACTER PYLORI AS A CAUSATIVE IN CHOLELITHIASIS AND ITS SEVERITY OF PRESENTATION WHEN IT ASSOCIATES WITH H PYLORI INDUCED GASTRITIS

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ABSTRACT "Marshall and Warren revolutionized our understanding by conclusively identifying H. pylori as the main cause of gastritis and peptic ulcers. Their research aimed to investigate whether H. pylori also plays a role in the development of cholelithiasis."

KEYWORDS :

INTRODUCTION

Marshall and Warren's groundbreaking discovery of Helicobacter pylori (H. pylori) as the primary causative agent of gastritis and peptic ulcer disease has significantly advanced our understanding of gastric pathophysiology. Subsequent research has expanded this knowledge, linking H. pylori to various gastric disorders, including gastric adenocarcinoma and MALT lymphoma. Notably, recent studies have identified Helicobacter DNA, including that of H. pylori, in bile, liver, and biliary epithelium of individuals with hepatobiliary disorders, suggesting that Helicobacter species may extend beyond the stomach.

In a significant development, researchers isolated a strain of H. pylori from the liver of a patient with cirrhosis, marking the first instance of demonstrating the viability of Helicobacter bacteria in the human liver, akin to observations in animals. This finding underscores the potential for H. pylori to colonize extra-gastric sites, challenging previous assumptions about its localization.

Earlier studies on biliary diseases were limited in scope, often lacking control groups and adjustments for confounding factors. However, recent investigations have detected H. pylori DNA in gallbladder tissue. For instance, a study involving 94 patients with symptomatic gallstones found that 37% had H. pylori in their gallbladder mucosa, as determined by various diagnostic tests, including Giemsa staining, immunohistochemistry, and rapid urease tests.

Despite these significant findings, investigations into the potential role of Helicobacter in the biliary tree as a risk factor for cholelithiasis have been somewhat limited in their scope. Earlier studies lacked adjustments for confounding factors. To address this research gap, we are poised to conduct a comprehensive and comparative prospective study on H. pylori as a potential causative factor in cholelithiasis. Our study will also delve into the severity of cholelithiasis presentations when associated with H. pylori-induced gastritis. This research aims to contribute valuable insights into the complex interplay between H. pylori and biliary diseases, shedding light on their potential associations and implications for disease severity.

AIMS AND OBJECTIVES

Aims:

The purpose of the study was to ascertain whether H. pylori is a causal agent of cholelithiasis.

Objectives:

A prospective investigation pertaining to immunohistochemistry, Giemsa staining, and PCR-based cholelithiasis :

1. Research on gallstones
2. To determine if H. pylori is associated
3. To investigate the degree of presentation severity in relation to the H. Pylori study."

METHODOLOGY

This case-control study focuses on patients who underwent cholecystectomy and aims to investigate the association between Helicobacter pylori (H. pylori) infection and Cholelithiasis. The study is conducted at ASRAM Medical college and Hospital in Eluru, and the enrollment period spans from January 2024 to January 2025.

Inclution Criteria

1. Patients more than 25 years and up to 60 years of age groups in both sexes presenting with cholelithiasis in CHRI
2. Patient with BMI between 20 to 27
3. Patients consented for inclusion in the study according to designated proforma

Exclusion Criteria

1. Patients less than 25 years of age
2. Patients more than 60 years of age.
3. Macroscopic malignancy and perforation.
4. Patient with severe co morbidities.
5. Patients with BMI >27.
6. Patient not consented for inclusion in the study

RESULTS

The average age of participants in the study was 38.97±9.96 years for cases and 41.30±8.84 years for controls, showing comparable age distributions between the two groups. Gender distribution was evenly split among cases (50% females, 50% males) and more skewed in controls (60% females, 40% males). Stone composition among cases showed 46.7% with multiple stones and 53.3% with single stones, while controls had 63.3% with multiple stones and 36.7% with single stones. Gastritis severity differed, with cases having 53.3% moderate and 46.7% severe gastritis, and controls having 53.3% mild, 33.3% moderate, and 13.3% severe gastritis.

DISCUSSION

The findings of this study provide a comprehensive insight into the demographic and clinical characteristics associated with gallstones, contributing to our understanding of the complex factors influencing gallstone development. The following discussion elaborates on key aspects revealed by the results.

Age and Gender

Distribution The comparable mean age between cases and controls suggests that age may not be a standalone factor influencing gallstone formation in this population. However, the gender distribution reveals an interesting pattern. While cases showed an equal distribution between males and females, controls exhibited a higher proportion of females. This discrepancy may indicate a potential gender-specific susceptibility to gallstones, warranting further investigation into hormonal, genetic, or lifestyle factors that could contribute to this difference.

Residence and Socioeconomic Status

The even distribution between rural and urban areas in both cases and controls implies that geographical location might not be a significant determinant in gallstone development in this study. However, the socioeconomic status disparities are noteworthy. Cases predominantly belonged to the upper middle class, while controls had a higher representation in the upper class. This suggests a possible association between higher socioeconomic status and gallstone risk, which could be attributed to lifestyle factors, dietary habits, or access to healthcare.

Stone Composition and Gastritis Severity

The observed differences in stone composition between cases and controls, with a higher prevalence of multiple stones in controls, suggest a potential link between the number of stones and the

likelihood of being classified as a case or control. Additionally, the variation in gastritis severity indicates a possible association between the inflammatory condition of the gallbladder and the risk of developing gallstones.

A study by Memon et al. found that 70.1% of patients with symptomatic cholelithiasis had H. pylori infection. However, the intensity of gastritis did not show a significant association with H. pylori gastritis. The research also established a correlation between a family history of gallstones and H. pylori gastritis.

Another study indicated that individuals with symptomatic cholelithiasis had a 37% higher likelihood of having H. pylori gastritis compared to non-cholelithiasis patients, corroborating the findings in our study.

Zhang et al. conducted a study revealing a 9.47% prevalence of cholelithiasis among those with H. pylori gastritis and 8.46% in non-H. pylori gastritis cases. They reported a 9.02% prevalence of gallstones in patients who underwent H. pylori eradication therapy.

In contrast, Sabbaghian et al. found a 33.3% positivity rate of H. pylori infection in patients undergoing cholecystectomy, exceeding the rates observed in our research.

Another study on 58 cholelithiasis patients reported 17 cases with H. pylori gastritis, contrasting with our findings.

Monstein et al. discovered a 55% positive rate of H. pylori infection in patients with cholesterol cholelithiasis, which was lower than our observed rate.

Takahashi et al. reported a 3.81% incidence rate of cholelithiasis among patients with negative H. pylori status, compared to a 6.08% incidence rate among positive cases. Patients on H. pylori eradication therapy had a 4.37% incidence rate.

Family History

The significant difference in family history reporting between cases and controls underscores the importance of genetic factors in gallstone formation. The higher percentage of cases with a positive family history suggests a hereditary predisposition to gallstones, aligning with existing literature. Understanding the specific genetic markers involved could provide valuable insights into risk assessment and targeted interventions.

Staining Techniques

The differences in positivity rates between Giemsa staining and 16S rRNA PCR highlight the significance of selecting appropriate diagnostic tools for detecting Helicobacter pylori (H. pylori) in gallstones. Giemsa staining demonstrated a higher positivity rate, suggesting its increased sensitivity in identifying H. pylori, especially in cases with active inflammation. Clinicians should consider these findings when choosing diagnostic methods for gallstone assessment in clinical practice.

A study indicated a contributing rate of 63% for H. pylori gastritis, surpassing our obtained results. Overall, the incidence of H. pylori among symptomatic cholelithiasis patients was found to be high. The statistically significant relationship between a familial history of cholelithiasis and H. pylori gastritis underscores the importance of genetic factors in the development of gallstones.

Radiological investigations, including ultrasonography (USG) and computed tomography (CT), play pivotal roles in diagnosing and assessing the severity of cholelithiasis, particularly when considering its association with H. pylori-induced gastritis. USG stands as the primary imaging modality for evaluating cholelithiasis due to its non-invasiveness, wide availability, and ability to detect gallstones and associated complications. Furthermore, CT scans provide detailed anatomical information and can reveal additional findings such as thickening of the gallbladder wall or evidence of pancreatitis, which may further elucidate the relationship between H. pylori infection, gastritis, and cholelithiasis severity.

Integrating these radiological investigations into the prospective study can offer comprehensive insights into the interplay between H. pylori infection, gastritis, and cholelithiasis, potentially guiding more targeted management strategies and improving patient outcomes.

CONCLUSIONS

In conclusion, this study significantly advances our comprehension of gallstone etiology by unveiling complex interrelations among demographic, clinical, and genetic variables. The discerned associations not only contribute to our current knowledge but also pave the way for future investigations focused on unraveling the underlying pathophysiological mechanisms. Moreover, these findings hold promise for the development of personalized strategies in gallstone prevention and management, thereby potentially improving clinical outcomes and enhancing patient care. Moving forward, continued research efforts are warranted to delve deeper into the intricacies of these associations and to translate these insights into innovative interventions that address the multifaceted nature of gallstone disease.

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