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Original Research Article

Helicobacter pylori colonization of gallbladder in patients with symptomatic cholelithiasis

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ABSTRACT

Background: The relation of *Helicobacter pylori* and its presence in gallbladder mucosa in patients with symptomatic cholelithiasis has been a topic of various studies. In order to establish this relation, this study has been performed so as to bring to light the capability of the organism to harbor in the gallbladder.
Methods: The patients underwent gastroscopy for specimens of gastric mucosa followed by laparoscopic/open cholecystectomy and histopathological examination of gallbladder specimen.
Results: The results showed *H. pylori* in 16 (40%) in gastric mucosa specimens and 4 (10%) in gallbladder mucosa specimens.
Conclusions: This revealed a significant correlation of *H. pylori* colonization in gallbladder mucosa with respect to gastric mucosa colonization.

Keywords: Gallbladder mucosa, Gastric mucosa, *Helicobacter pylori*

INTRODUCTION

Helicobacter pylori has been in the limelight for ages together since their identification in 1982. When there were theories proposed that no bacteria would be able to survive the acid in the stomach, these organisms paved a way to a complete and different understanding about the abilities they contain. The bacterium has been associated as etiology for various conditions like peptic ulcer, carcinoma of the stomach, chronic/atrophic gastritis, etc. There have been studies that have found the existence of *H. pylori* in organs other than the stomach.[1,2] This study is conducted, to be able to assess and analyze the *H. pylori* colonization in the gallbladder.

METHODS

A group of 40 patients hailing from Tamil Nadu and Puducherry, who suffered from symptomatic cholelithiasis had been accepted into the study after due consent being taken. They were admitted in the surgical ward at Aarupadai Veedu Medical College and Hospital, Puducherry, India. The patients underwent gastroscopy for gastric mucosal biopsy and therapeutic laparoscopic cholecystectomy. Their gastric mucosa and gallbladder specimens were sent for histopathological examination. The 40 patients who were included in the study were selected within an inclusion and exclusion criteria. Patients both male and female between the ages of 18 to 60 with symptomatic cholelithiasis came under the inclusion criteria. The exclusion criteria contained patients with haemolytic diseases, acute cholecystitis, acalculous cholecystitis, gallbladder polyps, gallbladder carcinoma and also patients who have undergone H. pylori eradication in the last 6 months.

Informed written consent had been taken from the patients to proceed with the study. The patients in the

group were subjected to detailed history and complete physical examination. Radiological evidence proving cholelithiasis was received. Relevant investigations for preoperative assessment were taken. Gastroscopy was done for these patients after their consent. During gastroscopy 6 specimens of gastric mucosa were retrieved and sent for histopathological examination. Later, anaesthetic fitness was obtained and patient was posted for laparoscopic cholecystectomy. There were no post op complications for any of the patients and recovery operative was uneventful. The excised gallbladder specimen was sent for histopathological examination.

RESULTS

Out of the 40 patients considered in this study, only 4 patients' gallbladder histopathology revealed *H. pylori* in the gallbladder mucosa, 16 patients had gastric mucosa *H. pylori* positivity. 2 patients from the sample presented with positivity in both gastric and gallbladder mucosa for *H. pylori* (Table 1).

Table 1: Histopathological analysis.

	H. pylori positive	*H. pylori* negative
Gastric	16 (40%)	24 (60%)
Gallbladder	4 (10%)	36 (90%)
Both	2 (5%)	

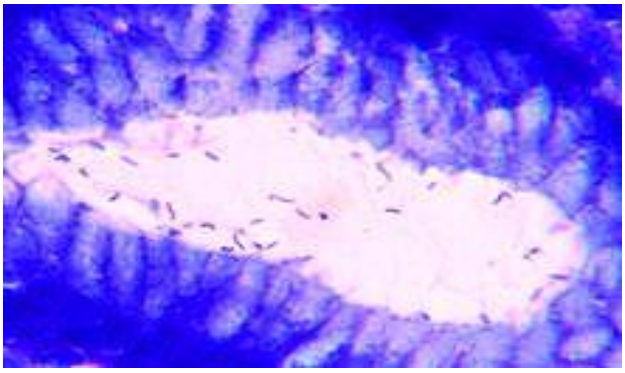

Figure 1: *H. pylori* seen in Giemsa stain.

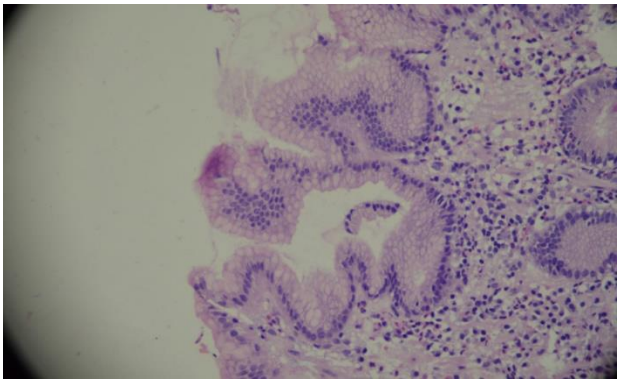

Figure 2: *H. pylori* seen in haematoxylin and eosin staining.

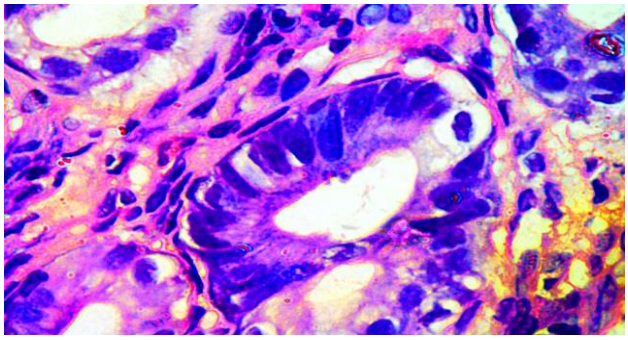

Figure 3: *H. pylori* seen in haematoxylin and eosin stain.

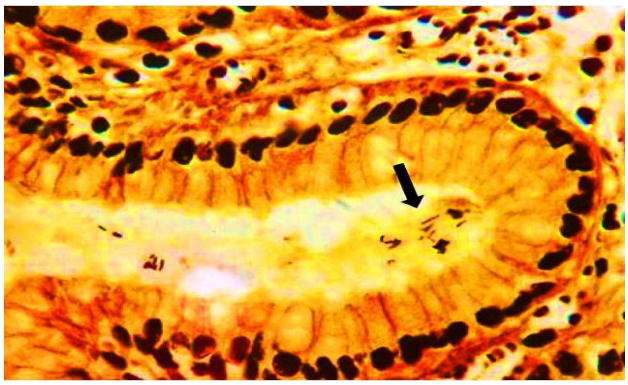

Figure 4: *H. pylori* seen in Warthin- starry silver stain.

This clearly reveals from the sample of the study that there can be presence of *H. pylori* in the gallbladder mucosa in 10% of patients with cholelithiasis. Incidentally 40% of the patients show positivity for *H. pylori* in the gastric mucosa. On finding a relationship between values of both criteria, significance is found on data analysis. The chi-square statistic of Table 1 is 9.6. The p-value is 0.001946. This result is significant at $p < 0.05$. The analysis gives a 5% chance of *H. pylori* bacteria colonization of both gastric and gallbladder mucosa in patients with symptomatic cholelithiasis.

DISCUSSION

The fact that *H. pylori* has been around the research tables for about 3 to 4 decades implies the much-needed information yet to be retrieved. There have been numerous studies done to be able to ascertain the relation between the gallbladder and *H. pylori*.[3,4]

Gallstones are cholesterol or pigmented stones. The brown pigmented amongst those gallstones have an inclination toward formation due to secondary bacterial infection probably due to bile stasis. Calcium bilirubinate which is precipitated and bacterial deposits conceive a primary part of the stone.[5] Bacterial colonization among the gallbladder mucosa and the bile ducts produce b-glucoronidase, phospholipase A, and bile acid hydrolase causing large amounts of unconjugated bilirubin, stearic

acids, and unconjugated bile acids, which can accompany calcium, resulting in gallstone precipitation.[6] Duodenal content becomes infected with age and associated intestinal motor disorders or gastric pathology, the bile too gets contaminated and, if bile stasis is associated, due to a stricture post sphincterotomy, brown stones can take shape.[7] The infection into the biliary tract is possible by the ascension through the duodenal papillary sphincter or descending through the portal system.[8] There have been developed numerous ways to ascertain the presence of *H. pylori* bacteria, the most common of which are rapid urease test, staining of mucosal biopsy specimens and stool for antigens.[9]

Histopathological examination plays the key role in the outcome of this study. Methods used in pathology to ascertain the presence of *H. pylori* are by staining. The ones that can be used are Hematoxylin and Eosin, Giemsa, Warthin-Starry silver stain, Immunohistochemistry stain. Specimens stained by hematoxylin and eosin reveal about 69-93% specificity and 87-90% sensitivity, 90-100% specificity to the other special stains.[9] Giemsa stain is widely popular in use because it is reasonably low-priced and vastly available for laboratory purposes. Giemsa stain is able to help highlight the inflammatory cells as well as the *H. pylori* cells. It is also exemplary in utility to be able to identify even miniscule number of bacteria in the sample.[10] Gastrointestinal infection with *H. pylori* may increase the risk for biliary colonization of the same (Figure 1, 2, 3, and 4).[11,12]

CONCLUSION

The ability of *H. pylori* colonization in the gallbladder to give rise to cholelithiasis is possible so far, as studied from the analysis above. The presence of the bacterium in the gastric mucosa is also a factor that has to be undertaken into account as an etiological factor for cholelithiasis due to its significant value on statistical estimation.

It is highly recommended to pursue research in this field to be able to guarantee the confirmation of the relation between cholelithiasis and gallbladder mucosa with *H. pylori*. The need of the hour would be to ascertain the role of *H. pylori* eradication to reduce the incidence of cholelithiasis.

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