ISSN Print: 2394-7500
ISSN Online: 2394-5869
Impact Factor: 5.2
IJAR 2016; 2(4): 786-789
www.allresearchjournal.com
Received: 13-02-2016
Accepted: 21-03-2016

Dr. Chalana
Assistant Professor, Department of Pathology, Maharajah's Institute of Medical Sciences, Nellimarla, Vizianagaram, Andhra Pradesh, India

Dr. Karthik
Assistant Professor, Department of General Medicine, Maharajah's Institute of Medical Sciences, Nellimarla, Vizianagaram, Andhra Pradesh, India

Correspondence
Dr. Karthik
Assistant Professor, Department of General Medicine, Maharajah's Institute of Medical Sciences, Nellimarla, Vizianagaram, Andhra Pradesh, India

Helicobacter associated gastritis: A histomorphological profile of the gastric antral mucosa

Dr. Chalana and Dr. Karthik

Abstract
Background and Objectives: Fifty percent of people worldwide suffer from this bacterial infection of the stomach antrum. Hyperpepsia, peptic ulcers, gastric adenocarcinoma, gastric MALT-associated lymphomas, and both acute and chronic gastritis have all been tied to *Helicobacter pylori* (*H. pylori*) infection.
Material and Methods: This research study was carried out at the Department of General Medicine, Maharajah's Institute of Medical Sciences, Nellimarla, Vizianagaram, Andhra Pradesh, India, between Study periods February 2015 to January 2016. A total of fifty cases with GERD and acid reflux disease symptoms were examined.
Results: Fifty patients who visited a private and medical gastrointestinal clinic with symptoms of acid peptic disease were included in the current investigation. Fifty cases were selected for the endoscopic examination, and biopsies were taken from each as needed for the research.
Conclusion: We conclude that when evaluating patients exhibiting symptoms of acid reflux disease, a comprehensive clinical examination and history are necessary, in addition to endoscopic evaluation and methods to identify the presence of the *H. pylori* bacteria. The sensitivity of the organism's detection is increased by using a variety of distinct stains.

Keywords: Histomorphological, peptic disease, gastric antral mucosa, helicobacter, gastritis

Introduction
Throughout the 20th century, there have been numerous reports of spiral organism isolation from human stomachs and the possible role these organisms play in gastrointestinal illness in humans. Numerous novel insights into the pathology of the gastroduodenum and implications for the management of peptic ulcer disease were brought about by the discovery of *H. pylori* [1].
Prolonged active gastritis is linked to stomach *H. pylori* infection. Early detection and removal of *H. pylori* infection can result in the healing of gastric inflammatory lesions and the reversal of precursor lesions that lead to cancer, such as intestinal metaplasia and gastric atrophy [2].
The Sydney method of classifying gastritis prioritizes geographic, morphological, and etiological details. Based on the results of an endoscopic biopsy, which can identify *H. pylori*, peptic ulcer disease is treated. Histopathology has emerged as the gold standard for detection because the main objective of these investigations is to determine if the organisms and gastritis are present or not. The presence of *H. pylori* can be shown by regular H&E sections, which can identify epithelial changes in the stomach mucosa colonized by the bacteria [2, 3].
Since efficient targeted treatment for *H. pylori*-associated gastroduodenal illnesses has been widely accepted, the detection of *H. pylori* in antral biopsies is now normal practice. Early *H. pylori* infection detection and removal is critical because it not only promotes the healing of inflammatory gastric lesions but also aids in the reversal of precursor lesions that lead to cancer, including intestinal metaplasia and gastric atrophy. Examining the variety of histological alterations in the stomach mucosa in patients with active chronic gastritis using light microscopy was the aim of this study [3, 4].

Methodology

This research study was carried out at the Department of General Medicine, Maharajah's Institute of Medical Sciences, Nellimarla, Vizianagaram, Andhra Pradesh, India, between Study period February 2015 to January 2016. A total of fifty cases with GERD and acid reflux disease symptoms were examined.

Inclusion criteria

- Patients with gastritis.

Exclusion criteria

- Includes acute gastritis, hemorrhage, and gastric cancer patients.
- Esophageal cancer.

Results

Table 1: Age and gender of 50 gastritis patients

Age	Patient	Male patient	Female patient
11-20	4	3	2
21-30	10	7	4
31-40	14	5	0
41-50	6	3	9
51-60	5	3	3
61-70	5	4	3
>70	6	4	0
Total	50	29	21

There was a male-to-female ratio of 2.22:1 among the 50 stomach samples that were examined; 29 of the biopsies included male patients, and 42% involved female patients. Table 1 shows a wide range of ages for the 50 individuals that were evaluated. In the third and fourth decades, there were the greatest numbers of cases of stomach lesions - between twenty and twenty-six.

Table 2: Clinical signs of gastritis in a patient

Sr. No.	Clinical symptoms	Number of patient with symptoms
1.	Nausea	36
2.	Vomiting	45
3.	Abdominal pain	47
4.	Heart burn	41

Patients with chronic gastritis most commonly complained of heartburn, nausea, vomiting, and stomach pain. The majority of the participants in our study had a variety of symptoms, with heartburn being the most common, followed by nausea and abdominal discomfort.

Table 3: Status of *Helicobacter pylori* in different age groups

Age	Patient	*H. pylori* positive	*H. pylori* negative
11-20	2	1	4
21-30	10	2	9
31-40	12	3	5
41-50	9	1	5
51-60	5	2	2
61-70	7	2	7
>70	5	3	4
Total	50	14	36

The patients with the highest *H. pylori* prevalence were in their forties and fifties. The fourth decade of life is when *H. pylori* infections are most common. The patients who tested positive for *H. pylori* infection were mostly in their 40s. After that, as you age, your chance of contracting *H. pylori* lowers.

Table 4: The location of different esophageal lesions

Sr. No.	Distribution of esophageal lesion	Number of cases
1.	Chronic reflex esophagitis	17
2.	Chronic non-specific esophagitis	10
3.	Vascular ectopia	8
4.	Barrets esophagus	6
5.	Normal histology	5
	Total cases	46

Endoscopy inspection of the esophagus's external appearance was part of the evaluation process for GERD and acid peptic sickness. When doctors felt it was necessary, lesions were biopsied. In 46 out of the 50 instances, esophageal biopsies were performed. Six of the subjects had no discernible clinical changes in the esophagus. The most common esophageal lesions in our patient series were Chronic Non-Specific Esophagi (10 cases) and Chronic Reflux Esophagi (10 cases). Barrett's esophagus, which occurred in 8 of our cases, was the second most frequent esophageal pathology after vascular ectasia of the esophagus.

Discussion

This study included fifty patients who visited a private gastroenterology clinic and an outpatient medical gastroenterology clinic for treatment of acid peptic sickness. In the endoscopic investigation, biopsies were taken from each of the 50 cases, which is the minimal amount needed.

Sixty-four percent of subjects in our study had positive *H. pylori* tests. This is in close agreement with the results of studies conducted by Maitra *et al*. (61.4% positivity in her series), Yakoob *et al*. (62% positivity in a case control study conducted in Italy), and researchers studying the Pakistani population. But Akanda *et al*. discovered a greater positivity of 71% in his series of cases when the prevalence of *H. pylori* in stomach antral biopsies was investigated [5-7].

In the analysis, eleven to eighty-eight patients were included. The biggest number of cases occurred in the third decade (20 cases) and the fourth decade (30 cases). Yakoob *et al*. (mean age = 45) and (mean age = 43) found comparable results in their separate investigations. Plotting *H. pylori* positivity against age shows that the prevalence of positive *H. pylori* bacteria steadily rises with age. Optimism is 50% among those between the ages of 21 and 30; it rises to 69% among those between the ages of 31 and 40, and reaches 77.7% among those between the ages of 41 and 60. The percentage of those who test positive starts to slightly decrease around age 60. According to Maitra *et al*., the age group between 31 and 40 had the highest positive rate (67.2%), while the age group between 50 and 59 had the lowest (63.8%). These results are consistent with their findings. She also noticed that the percentage of positive people fell after the age of 60. The study's findings are in line with the observed decline in optimism. In her study, she did identify a 69.3% increase, but in those aged 20 to 29, we did not find a comparable high proportion. In just 50% of the cases were we able to reach a suitable conclusion [5, 7].

Men outweigh women in our series, which is in line with observations made in the literature on the gender distribution of patients. Thirteen girls and sixty-nine males received diagnoses. The results are in line with the findings

of Niv *et al*., who found a ratio of 1.6:1 in their series regarding the male-to-female proportion, or M: *H. pylori* positivity was found in 63.7% of men and 64.5% of women, with a mean positive of 64.5% throughout the total study population. This result is consistent with Maitra *et al*.'s [5] findings, which showed a positive of 63.1% in men and 56.5% in women [8, 9].

We graded all the antral biopsies that had discernible inflammation after counting the number of them using the Modified Sydney grading system. From mild (27 cases), to moderate (47 cases), to severe (15 cases), the degree of inflammation varied. The percentage of patients with mild inflammation (33.3%), moderate inflammation (87.23%), and severe inflammation (93.3%) who tested positive for *H. pylori*. This study, by Vijaya VA *et al*. loc. cit, concurs well with this, reporting a positive rate of 57% in mild inflammation cases, 88.4% in moderate inflammation instances, and 100% in severe inflammation cases [10]. This helps to explain the robust association found between *H. pylori* positive and inflammation. The proportion of positive cases with severe inflammation was lower in our analysis than in the previously reported study. A greater percentage of favorable results was linked in our study to mild inflammation. This study's key conclusion is that higher graded inflammation was also associated with a greater density of organisms. Whereas inflammatory cells with severe inflammation infiltrated deep into the muscularis mucosae, in mild inflammation they remained close to the mucosa's surface, in the pits, the lamina propria, and the neck of the pyloric glands [11-15].

There were 65 instances with active disease, based on the Sydney activity score. Of these patients, 84.6% had positive *H. pylori* test findings. By contrast, the success rates reported by Vijaya VA *et al*. [10] and Charanjeet *et al*. [16] were 91.4% and 90.4%, respectively (see citations in footnote). The association between metabolic activity and *H. pylori* was statistically significant. We discovered 22 instances of intestinal metaplasia in our collection of biopsies. 72.7% of this sample tested positive for *H. pylori*. The results of Vijaya VA *et al*. [10] are consistent with the positive status of 78.2%. This is not the case with Charanjeet *et al*. [16], who found a modest 25% *H. pylori* positive in his series of 50 cases. We found that the incidence of intestinal metaplasia was significantly greater in samples that tested positive for *H. pylori*. The organisms, however, were not seen in the metaplastic epithelium but rather in the nearby stomach mucosa [17].

We suggest utilizing Light Green Carbol Fuchsin stain as a supplementary staining method for the detection, or as a substitute for the more costly and time-consuming Warthin Starry silver stain. In our patient series, Chronic Reflux Esophagi was the most common esophageal lesion, followed by Chronic Non Specific Esophagi and others. Cases of persistent reflux esophagitis have been associated with both vascular papillae elongation and basal zone expansion. This result is in line with the findings of Calabrese *et al*., who identified this alteration as a marker for the reflux condition in individuals with chronic reflux esophagi. In 10 of the cases where epithelial cell destruction was a factor, we found that balloon degeneration of the epithelial cells was a feature [17]. It has been suggested that the presence of these balloon cells indicates injury to the epithelium. The intraepithelial Eosinophils are another sign of reflux illness. Scientists Tummala V. and colleagues. Only six of our patients had intraepithelial eosinophils found, but [18].

We saw Barrett's esophagus in 14 of our cases, which is the second most frequent esophageal pathology. Often, columnar cells resembling stomach mucosal cells made up the metaplastic epithelium. In two cases with Barrett's esophagus, we found ulceration, but the inflammation in the other four cases was nonspecific and not very bad. That Barrett's esophagus ulcers and inflammation are not diagnostic signs is in line with Zhang *et al*.'s findings. There were two further histological changes in the esophagus: vascular ectasia and chronic nonspecific esophagitis [19].

Conclusion

There is compelling evidence that the development of chronic gastritis and related gastroduodenal diseases such as peptic ulcers, adenocarcinoma, and lymphoma is mostly caused by *Helicobacter pylori* infection. Barrett's esophagus, vascular ectasia, and chronic nonspecific esophagitis were among the other frequently diagnosed conditions. Very few people's esophaguses were in perfect condition. When Barrett's esophagus is diagnosed, patients are closely watched. In conclusion, a thorough clinical examination and history, an endoscopic examination, and methods to identify the presence of the *H. pylori* bacterium are now necessary for the diagnosis of a case exhibiting signs of acid peptic disease. The sensitivity of the detection procedure is increased by combining various kinds of distinctive stains.

Funding source

None.

Conflict of Interest

None.

References

1. Dooley CP. Background and historical considerations of *Helicobacter pylori*. Gastroenterol Clin. North Am. 1993;22:1-2.
2. D'Elios MM. *Helicobacter pylori*, the story so far. Med Secoli. 2007;19:6415.
3. Jain AK. Should we eradicate *Helicobacter pylori* to improve gastric histology? Indian J Gastroenterol. 2002;21:2-3.
4. Ozturk S, Serinsoz E, Kuzu I, *et al*. The Sydney System in the assessment of gastritis: Inter-observer agreement. Turk. J Gastroenterol. 2001;12:36-9.
5. Maitra TN, Ghosh S. Gatritis and Helicobacter (Camylobacter) Pylori- Merely one more piece in the Jigsaw Puzzle or the final answer? Indian J Pathol. Microbiol. 1991;34(1):67-79.
6. Yakoob MY, Hussainy AS. Chronic gastritis and *Helicobacter pylori*: A histopathological study of gastric mucosal biopsies. J Coll. Physicians Surg. Pak. 2010;20(11):773-75.
7. Akanda MR, Rahman AN. Comparative study of different methods for detection of *Helicobacter pylori* in gastric biopsies. Dinajpur Med. Col J. 2011;4(1):1-6.
8. Dandin AS, Pawale J, Athanikar S. *Helicobacter pylori* associated gastritis. J Clin. Diagn. Res. 2012;6(2):211-14.
9. Liu C. Bacterial outer membrane proteins and host mucins involved in colonization of the gastric mucosa by the zoonotic pathogen *Helicobacter heilmannii* [Dissertation]. Ghent University.

10. Vijaya L, George R, Arvind H, *et al*. Prevalence of primary angle-closure disease in an urban south Indian population and comparison with a rural population: The Chennai Glaucoma Study. Ophthalmology. 2008;115(4):655-60.
11. Misra V, Hatwal D, *et al*. *Helicobacter pylori* and associated histological changes in gastric biopsies of patients with leprosy. Indian J Pathol. Microbiol. 2001;44(3):271-275.
12. Okumura T, Ericksen RE, Takaishi S, *et al*. K-ras mutation targeted to gastric tissue progenitor cells results in chronic inflammation, an altered microenvironment, and progression to intraepithelial neoplasia. Cancer Res. 2010;70(21):8435-45.
13. Regev A, Fraser GM, Braun M, *et al*. Sero-prevalence of *Helicobacter pylori* and length of stay in a nursing home. Helicobacter. 1999;4:89–93.
DOI: 10.1046/j.1523- 5378.1999.98640.x.
14. Watanabe K, Nagata N, Nakashima R, *et al*. Predictive findings for *Helicobacter pylori*-uninfected, infected and -eradicated gastric mucosa: Validation study. World J Gastroenterol. 2013;19(27):4374.
15. Tongtawee T, Kaewpitoon S, Kaewpitoon N, *et al*. Correlation between gastric mucosal morphologic patterns and histopathological severity of *Helicobacter pylori*-associated gastritis using conventional narrow band imaging gastroscopy. Bio Med. Res. Int.; c2015.
16. Ahluwalia C, *et al*. Comparison of Endoscopic Brush Cytology with Biopsy for Detection of *Helicobacter pylori* in Patients with Gastroduodenal Diseases. Indian J Pathol. 2001;44(3):283-288.
17. Calabrese C, Di Febo G, Brandi G, *et al*. Correlation between endoscopic features of gastric antrum, histology and *Helicobacter pylori* infection in adults. Ital. J Gastroenterol Hepatol. 1999;31(5):359-65.
18. Tummala V, Barwick KW, Sontag SJ, *et al*. The significance of intraepithelial eosinophils in the histologic diagnosis of gastroesophageal reflux. Am J Clin. Pathol. 1987;87:43-47.
19. Zhang C, Yamada N, Wu YL, *et al*. Comparison of *Helicobacter pylori* infection and gastric mucosal histological features of gastric ulcer patients with chronic gastritis patients. World J Gastroenterol. 2005;11(7):976.