Original Research Article To find out the relationship of Helicobacter pylori infection with erosive and non-erosive gastritis.

Dr. Zaheen Gouri¹ (Post Graduate Student), Dr. Gajendra Singh Gaur² (Post Graduate Student), Dr. Shubendra Parmar³ (Post Graduate Student) & Dr. Abha Pandit⁴ (Professor Dept. of Internal Medicine)

Dept. of General Medicine, Index Medical College Hospital & Research Centre, Indore, M.P.^{1,2,3&4} Corresponding Author: Dr. Gajendra Singh Gaur

Abstract

Background & Methods: The aim of the study is to find out the relationship of Helicobacter pylori infection with erosive and non-erosive gastritis. Patients aged between fifteen to sixty years having dyspeptic symptoms and willing to undergo for upper gastrointestinal endoscopy and anti-Helicobacter pylori treatment were enrolled in this study. Every ethical issues which could arise were discussed with and informed written consent was taken from all patients.

Results: The chi-square statistic is 4.115. The *p*-value is .042505. The result is significant at p < .05 for Endoscopic finding. The chi-square statistic is 11.6629. The *p*-value is .020042. The result is significant at p < .05 for treatment. The chi-square statistic is 2.0318. The *p*-value is .154034. The result is *not* significant at p < .05 for follow. Pylori is associated more with erosive gastritis and treatment was slightly more beneficial in patients with erosive gastritis.

Conclusion: Prevalence of Helicobacter pylori infection among endoscopically proven gastritis patients in our study was nearly 89%. Our part of country is a high prevalence zone of Helicobacter pylori infection as proven with 89% positive cases among gastritis patients in our study. Also this increased prevalence could be attributed to the low socioeconomic status as majority of our study population were lower socioeconomic group.

Keywords: Helicobacter pylori infection with erosive and non-erosive gastritis. Study Design: Observational Study.

1. Introduction

Helicobacter pylori is a gram negative miniature aerophilic microorganisms found in the stomach which is one of the human contaminations with a worldwide inclusion. It has been accounted for that around half of the all-out human populace harbors this organism[1]. First found in 1982 by Australian researchers Barry Marshall and Robin Warren, after which the organic entity has been proposed to be a reason for a lot of illnesses connected with the gastrointestinal plot and has upset the field of gastroenterology[2]. Despite the fact that the commonness is overall the diseases because of Helicobacter pylori change in various nations

and, surprisingly, in various districts inside a country. Yet, a positive expanded commonness in the emerging nations is demonstrated[1].

Helicobacter pylori contamination is a demonstrated etiology for peptic ulcer illness and gastritis[3]. Infection on the off chance that persevering was viewed as a variable of positive gamble for adenocarcinoma of stomach and MALToma (mucosa related lymphoma). Studies have shown that ongoing gastritis and duodenal ulcer has a relationship of 100 percent contrasted with half in the controls who didn't have ulcer [4].

Helicobacter pylori was connected to persistent gastritis and gastric ulcer following its revelation 1982.Previously they were not accepted to be from a microbial reason. However, from that point forward a large number of the gastroenterological messes were hypothesized to be brought about by this life form. Indeed, even extra-gastrointestinal illnesses have been explored for Helicobacter pylori as their objective. The organic entity is believed to be a fundamental part of the regular environment of stomach. In any event, when the greater part of the populace harbors this living being just 20% of the people are causing illness or infection[5].

Helicobacter pylori however worldwide, its more in the creating than in the created nations. The sole wellspring of the creature is the human gastric mucosa. The specific component of transmission isn't clear yet it has been hypothesized to be or - oral or feco-oral .Destitution, congestion and unfortunate cleanliness favors transmission and subsequently makes sense of its expanded predominance in the creating countries[6].

2. Material and Methods

Study conducted at Index Medical College Hospital & Research Centre, Indore from Sep. 2022 to Nov. Sept 2023. Patients aged between fifteen to sixty years having dyspeptic symptoms and willing to undergo for upper gastrointestinal endoscopy and anti-Helicobacter pylori treatment were enrolled in this study. Every ethical issues which could arise were discussed with and informed written consent was taken from all patients. Treatment with H Pylori kit was given for 14 days.

After taking a detailed history and physical examination, patients were submitted to upper Gastrointestinal endoscopy and Rapid Urease Test (RUT) was done with one of the specimens taken from the predominant site of gastritis. Patients who were on Proton pump inhibitor or H2 blocker therapy were taken for endoscopy only after stopping these drugs for atleast 2 weeks.

INCLUSION CRITERIA:

1. Adults aged between 15 to 60 years having symptoms of dyspepsia.

EXCLUSION CRITERIA:

1. Patients who were regular users of NSAID and steroids, had peptic ulcer and its complications.

3. Result

| RUT | Endoscopic Findings | | | | | |
|----------|---------------------|------|-------------|------|--|--|
| | Erosive | | Non erosive | | | |
| | Ν | % | Ν | % | | |
| Positive | 62 | 89.8 | 23 | 74.1 | | |
| Negative | 07 | 10.2 | 08 | 25.9 | | |
| Total | 69 | 100 | 31 | 100 | | |

The chi-square statistic is 4.115. The *p*-value is .042505. The result is significant at p < .05.

| | | | After treatment | | | |
|----------------------|------------------|----|-----------------|--------------|--|--|
| Clinical features | Before treatment | | Resolved | Not resolved | | |
| | Ν | % | Ν | Ν | | |
| Abdominal pain | 37 | 37 | 26 | 11 | | |
| Bloating | 27 | 27 | 19 | 09 | | |
| Early Satiety | 13 | 13 | 08 | 05 | | |
| Nausea | 11 | 11 | 09 | 02 | | |
| Anorexia | 05 | 05 | 05 | 00 | | |
| Vomiting | 07 | 07 | 07 | 00 | | |

TABLE 2: TABLE SHOWING CLINICAL SYMPTOMS POST TREATMENT

The chi-square statistic is 11.6629. The *p*-value is .020042. The result is significant at p < .05.

| Endoscopic findings | Follow-up RUT | | | | | Total | |
|---------------------|-------------------|------|--------|------|-----|-------|--|
| | Positive Negative | | gative | | | | |
| | Ν | % | Ν | % | Ν | % | |
| Erosive | 14 | 19.1 | 59 | 80.8 | 73 | 100.0 | |
| Non erosive | 02 | 7.4 | 25 | 92.6 | 27 | 100.0 | |
| Total | 16 | 16 | 84 | 84 | 100 | 100.0 | |

TABLE 3: SHOWING H.PYLORI ERADICATION RATE & GASTRITIS TYPES

The chi-square statistic is 2.0318. The *p*-value is .154034. The result is *not* significant at p < .05.

Pylori is associated more with erosive gastritis and treatment was slightly more beneficial in patients with erosive gastritis.

4. Discussion

The greater part of the review populace were from the lower financial layers (54.3%). Socio monetary status is critical on the grounds that there is expanded colonization in the poor financial status gathering and those with lesser training separated from thinking about the hereditary variables in the event of creating and immature countries[7].

The outcomes from our review are in concurring with the demonstrated aftereffects of different examinations done all over the planet not entirely settled by the spot of the review, financial status as well as the method of transmission which makes the spread contamination from individual or by oro-oral or the feco-oral routes[8].

The Fast urease test being straightforward, savvy, and speedy in giving outcomes makes it a pragmatic and monetary method for testing for H. pylori diseases in patients not taking antimicrobials or proton siphon inhibitors who need an upper Gastrointestinal endoscopy. Thus Groove was utilized as single best test for finding of H. pylori gastritis in our review. As there is goal of disease and as the circulation of H. pylori disease becomes inconsistent after anti-toxins or proton siphon inhibitors, biopsy for the Fast urease test ought to be taken from two destinations, the body and the antrum at area of more noteworthy curve [9]. Because of restriction in offices, we took biopsy from just prevalent site of gastritis before treatment and just from one site after treatment for finding and appraisal of annihilation.

In this review, Helicobacter pylori contamination status was viewed as certain by a positive Trench test result. In view of this measure, out of 100 endoscopically demonstrated gastritis patients, 84% had H. pylori gastritis. The excess 16% patients were negative for Quick urease test[10].

Painless tests can be utilized for affirming the annihilation of Helicobacter pylori like urea breath test or stool antigen test besides in patients where rehash endoscopy is shown, as in the event of patients with gastric ulcer. As post treatment endoscopy was performed to recognize the progressions of gastric mucosa after triple treatment, Fast urease tests were finished for affirmation of the destruction of microorganisms.

5. Conclusion

Prevalence of Helicobacter pylori infection among endoscopically proven gastritis patients in our study was nearly 89%. Our part of country is a high prevalence zone of Helicobacter pylori infection as proven with 89% positive cases among gastritis patients in our study. Also this increased prevalence could be attributed to the low socioeconomic status as majority of our study population were lower socioeconomic group.

6. References

- 1. Omunakwe HE, Madubuike OC, Nwosu SO, Pughikumo CO, Nwauche CA. Gastric mucosa-associated lymphoid tissue: The need for prompt histologic diagnosis. Ann Trop Med Public Health 2011;4:113-5.
- 2. Makola D, Peura DA, Crowe SE. Helicobacter pylori infection and related gastrointestinal diseases. J Clin Gastroenterol 2007;41:548-58.

- 3. Duck WM, Sobel J, Pruckler JM, Song Q, Swerdlow D, Friedman C, et al. Antimicrobial resistance incidence and risk factors among Helicobacter pylori-infected persons, United States. Emerg Infect Dis 2004;10:1088-94.
- 4. Das JC, Paul N. Epidemiology and pathophysiology of Helicobacter pylori infection in children. Indian J Pediatr 2007;74:287-90.
- 5. Parkin DM, Pisani P, Ferlay J. Global cancer statistics. CA Cancer J Clin 1999;49:33-64, 1.
- 6. Malfertheiner P, Megraud F, O'Morain C, Bazzoli F, El-Omar E, Graham D, et al. Current concepts in the management of Helicobacter pylori infection: The Maastricht III Consensus Report. Gut 2007;56:772-81.
- 7. Graham DY, Sung JY. Helicobacter pylori. In: Feldman M, Friedman LS, Brandt LJ, editors. Sleisenger and Fordtran's Gastrointestinal and Liver Disease. Pathophysiology, Diagnosis, Management. 7th ed. Philadelphia: WB Saunders Co; 2006. p. 1049-66.
- 8. Dixon MF, Genta RM, Yardley JH, Correa P. Classification and grading of gastritis. The updated Sydney system. International workshop on the histopathology of gastritis, Houston 1994. Am J Surg Pathol 1996;20:1161-81.
- 9. Tarkhashvili N, Beriashvili R, Chakvetadze N, Moistsrapishvili M, Chokheli M, Sikharulidze M, et al. Helicobacter pylori infection in patients undergoing upper endoscopy, Republic of Georgia. Emerg Infect Dis 2009;15:504-5.
- Shokrzadeh L, Baghaei K, Yamaoka Y, Shiota S, Mirsattari D, Porhoseingholi A, et al. Prevalence of Helicobacter pylori infection in dyspeptic patients in Iran. Gastroenterol Insights 2012;4:24-7.