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Original Research Article

Presence of *Helicobacter pylori* infection in gastroduodenal perforations

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ABSTRACT

Background: Gastroduodenal perforations constitute one of the commonest surgical emergencies encountered. *Helicobacter pylori* is a gram negative bacterium that has infected more than half the world's population. The most commonly recognized manifestation of *H. pylori* infection in India is peptic ulcer disease. Although the relationship between *H. pylori* infection and peptic ulcer has been well defined, the relationship of *H. pylori* infection with gastroduodenal perforation is still controversial. The objective of the study was to determine the presence of *H. pylori* in gastroduodenal perforations.
Methods: We conducted a prospective study, noting the number of cases which turned out to be positive for *H. pylori* in cases of gastroduodenal perforations intraoperatively, using rapid urease test.
Results: Out of 100 cases of gastroduodenal perforations operated, 74% were positive for the test. Gastric perforations positive for the test were 81.4% and duodenal perforations positive for the test were 68.42%.
Conclusions: There is a positive attribution between *H. pylori* infection and gastroduodenal perforations.

Keywords: Gastroduodenal perforations, *Helicobacter pylori*

INTRODUCTION

Gastrointestinal perforations constitute one of the commonest surgical emergencies encountered. Management of these patients continues to be highly demanding despite the advances made in diagnosis and surgical therapy. The etiological spectrum of perforation peritonitis in India differs significantly from its western counter parts.[1] One of the leading causes is found to be peptic ulcer disease.

Peptic ulcer disease is a problem of the gastrointestinal tract characterized by mucosal damage secondary to pepsin and gastric acid secretion. It usually occurs in the stomach and proximal duodenum.[2] Various factors may contribute to the development of PUD. Among various factors it is seen that, in a number of developing countries, *H. pylori* infection affects more than 80% of middle-aged adults.[3]

It is clear from multiple randomized prospective studies that curing *H. pylori* infection dramatically alters the natural history of PUD, decreasing the recurrent ulcer rate from more than 70% in patients treated with a course of acid suppressive therapy alone (in whom *H. pylori* is not eradicated) to less than 20% in patients treated with a course of antibacterial therapy.

Although the relationship between *H. pylori* infection and peptic ulcer has been well defined, the relationship of *H. pylori* with perforated ulcer is still controversial.[4] Hence, a study of association between gastroduodenal perforations and *H. pylori* infection is undertaken, since eradication of *H. pylori* after simple closure of a perforated ulcer reduces the incidence of recurrent ulcer.[5]

Aim of the study was to determine the presence of *H. pylori* in gastroduodenal perforations.

METHODS

This was a prospective, observational study conducted in the hospitals attached to Bangalore Medical College and Research Institute, Bengaluru from April 2018 to December 2019. The study group included 100 patients diagnosed with gastric or duodenal perforations meeting the inclusion and exclusion criteria. They underwent a detailed history taking, physical examination and pre-operative investigations. Treatment with resuscitative measures was started preoperatively. The patients with clinical findings and radiological investigation suggestive of gastrointestinal perforation underwent emergency explorative laparotomy.

Inclusion criteria

Patients of either sex aged ≥18 years and all patients with a diagnosis of gastric or duodenal perforation were included.

Exclusion criteria

Traumatic perforations, patients not undergoing surgery, hollow viscus perforations other than gastric or duodenal perforation were excluded.

Under strict aseptic precautions, after general anesthesia, exploratory laparotomy was performed. The patients with intraoperative findings of gastric or duodenal perforations were included in the study. Biopsy was taken from the site of perforation. Primary closure of the perforation with omental patch carried out and abdomen closed in layers. Standard post-operative care was provided.

The biopsy taken was subjected to rapid urease test and observed. If the solution turned pink on exposure to the tissue, it was considered a positive test. If the solution remained the same colour, it was considered a negative test. The rapid urease test was observed for 1 hour, and to a maximum of 24 hours in doubtful cases to look for positivity of the test and details recorded.

RESULTS

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software and appropriate statistical tests were used.

Categorical data was represented in the form of Frequencies and proportions. Chi-square test or Fischer's exact test (for 2x2 tables only) was used as test of significance for qualitative data.

Continuous data was represented as mean and standard deviation. Independent t test was used as test of significance to identify the mean difference between two quantitative variables.

P value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.

Diagnosis

In the study 43% had Gastric perforation and 57% had duodenal perforation.

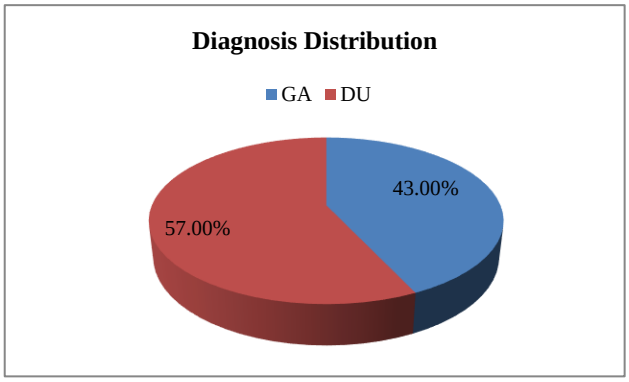


Figure 1: Pie diagram showing diagnosis distribution among subjects.

Age distribution

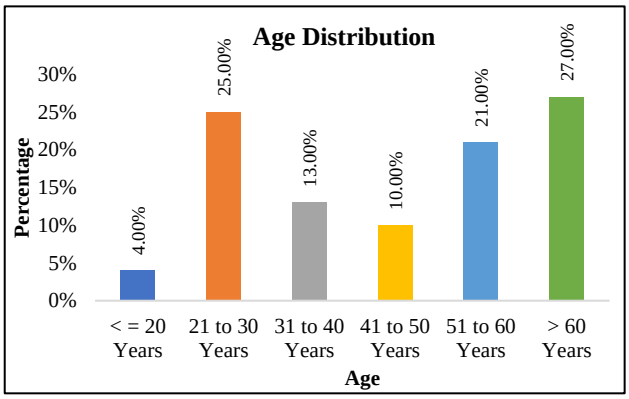


Figure 2: Bar diagram showing Age distribution of subjects.

Association between age and diagnosis

Majority of gastric perforations were in the age group >60 years (39.53%), whereas majority of the duodenal perforations were in the age group 21 to 30 years (22.81%).

There was no significant difference in age distribution between GA and DU groups (χ^2=8.216, df=5, p=0.145).

Mean age of patients- 45.29±16.49 years.

Mean age of patients with GA was 49.02±16.28 years

Mean age of patients with DU was 42.47±16.22 years.

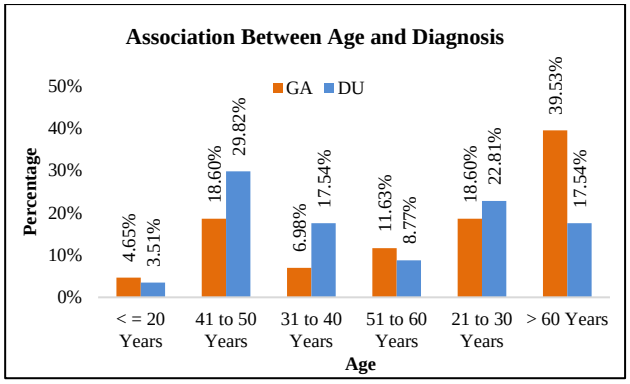


Figure 3: Bar diagram showing association between age and diagnosis among subjects.

Gender distribution

In the study 80% were males and 20% were females.

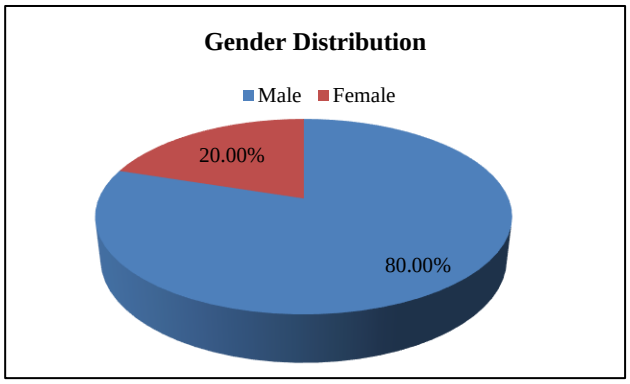


Figure 4: Pie diagram showing gender distribution of subjects.

Association between gender and diagnosis

GA- 76.74% were males and 23.26% were females.

DU- 82.46% were males and 17.54% were females.

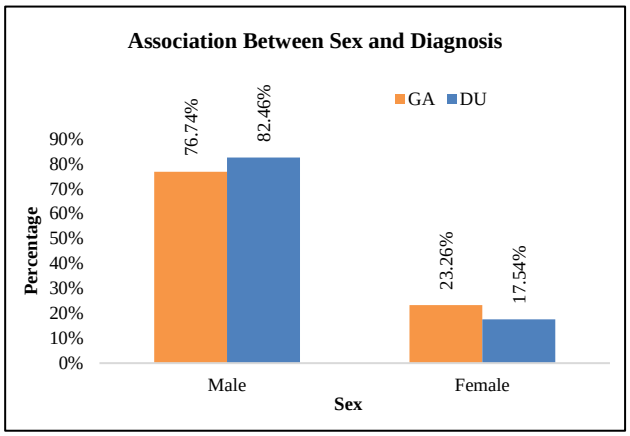


Figure 5: Bar diagram showing association between gender and diagnosis.

There was no significant difference in sex distribution between GA and DU diagnosis (χ^2=0.500, df=1, p=0.480).

Test distribution

Among the total number of patients 74% were positive for *H. pylori* infection.

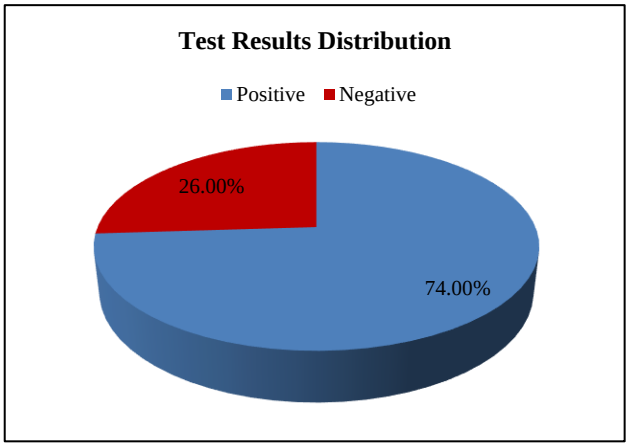
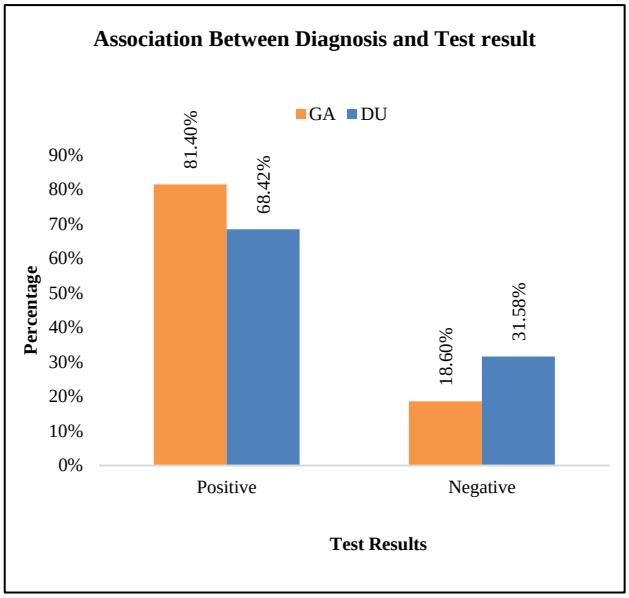


Figure 6: Pie diagram showing test result distribution among subjects.

GA- 81.4% were positive for test.

DU- 68.42% were positive for test.



However there was no significant difference in test results between GA and DU (χ^2=2.144, df=1, p=0.143).

Other collateral observations noted during the course of the study

Dietary factors and *H. pylori* infection: 78%- non vegetarians.

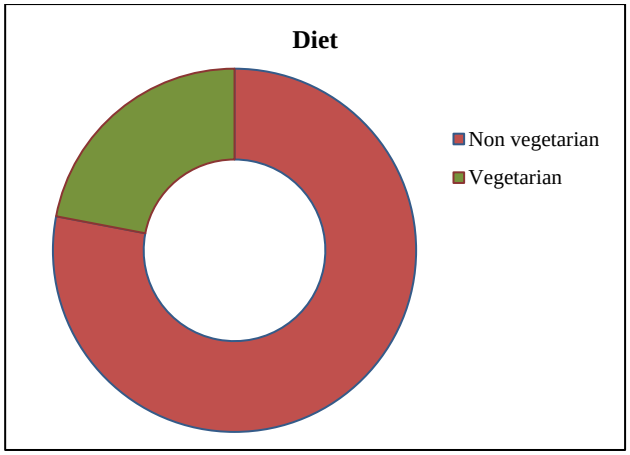


Figure 8: Diet distribution in patients with *H. pylori* infection.

DISCUSSION

The discovery of *H. pylori* by Warren and Marshall in 1983, paved way to several studies highlighting the association between *H. pylori* and peptic ulcer diseases and carcinoma stomach.[6]

It is now believed that 80% to 95% of duodenal ulcers and approximately 75% of gastric ulcers are associated with *H. pylori* infection. Infection with *H. pylori* has been shown to temporally precede ulcer formation, and when this organism is eradicated as part of ulcer treatment, ulcer recurrence is extremely rare.[7] These observations have secured the place of *H. pylori* as the primary causative factor in the pathogenesis of PUD.

Though this has been fairly established, there is still a relative doubt and controversy between the presence of *H. pylori* and gastroduodenal perforations.[4]

In present study, 80% of our subjects were male and 20% were female, showing predominance of gastric and duodenal perforations in the male gender. More than half the patients, i.e., 57% were diagnosed to have duodenal perforation. The maximum number of patients, i.e., 27% were elderly, followed by 25% of them being in the third decade of life, thus suggesting an almost bimodal presentation.

In present study, 74% of the gastroduodenal perforations, were positive for test result. Among gastric and duodenal perforations, 81.4% of gastric perforations were positive for the test, where as 68.42 percent of duodenal perforations were positive for the test, indicative of the presumed incidence of *Helicobacter pylori* infection in our study group. In a study conducted by Gisbert et al, 62% of the patients with perforated peptic ulcer were infected by *H. pylori*, while the microorganism was detected in 87% of the patients without this complication.[8] Similarly, in an Indian study conducted by Dogra et al, from Pune conducted a study and found out that out of 50 cases of gastro duodenal perforation who underwent exploratory laparotomy, as many as 46 cases (92%) turned out to be positive for *H. pylori* and only four cases (8%) were negative for this infection.

Limitations

Study was not designed to determine cause to effect phenomenon. There was no comparison group.

CONCLUSION

Therefore, according to the results of our study, there is a positive attribution between *H. pylori* infection and gastroduodenal perforations.

Hence, detection of *H. pylori* with a simple test, intra-operatively in gastroduodenal perforations necessitates early treatment and eradication of the infection, thus eventually reducing recurrence of the disease and its associated complications.

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REFERENCES

1. Bali RS, Verma S, Agarwal PN, Singh R, Talwar N. Perforation peritonitis and the developing world. ISRN Surg. 2014;2014:1-4.
2. Ramakrishnan K, Salinas RC. Peptic ulcer disease. Am Fam Phys. 2007;76(7).
3. Soriano IS, Dempsey DT. Benign gastric disorders. In: Zinner MJ, Ashley SW, eds. Maingot's abdominal operations. McGraw Hill Professional; 2018;461-465.
4. Gisbert JP, Legido J, Garcıa-Sanz I, Pajares JM. *Helicobacter pylori* and perforated peptic ulcer. Prevalence of the infection and role of non-steroidal anti-inflammatory drugs. Digest Liver Dis. 2004;36(2):116-20.
5. El-Nakeeb A, Fikry A, El-Hamed TM, El Awady S, Youssef T, Sherief D, et al. Effect of *Helicobacter pylori* eradication on ulcer recurrence after simple

closure of perforated duodenal ulcer. Int J Surg. 2009;7(2):126-9.
6. Pincock S. Nobel Prize Winners Robin Warren and Barry Marshall. Lancet. 2005;366(9495):1429.
7. Kusters JG, van Vliet AH, Kuipers EJ. Pathogenesis of *Helicobacter pylori* infection. Clin Microbiol Rev. 2006;19(3):449-90.
8. Gisbert JP, Khorrami S, Carballo F, Calvet X, Gené E, Dominguez-Muñoz E. *H. pylori* eradication therapy vs. antisecretory non-eradication therapy (with or without long-term maintenance antisecretory therapy) for the prevention of recurrent bleeding from peptic ulcer. Cochrane Database Syst Rev. 2004;2:CD004062.
9. Dogra BB, Panchabhai S, Rejinthal S, Kalyan S, Priyadarshi S, Kandari A. *Helicobacter pylori* in gastroduodenal perforation. Med J Dr. DY Patil Univ. 2014;7(2):170.

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