Original Research Article

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Prevalence of *Helicobacter pylori* infection in patients with gastric ulcer perforation admitted in a tertiary care centre

Muhammed Rashim P., S. Vineed*

Department of General Surgery, Government Medical College, Thiruvananthapuram, Kerala, India

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*Correspondence: Dr. S. Vineed,

Dr. 5. vinced,

E-mail: vineeds75@gmail.com

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ABSTRACT

Background: Gastric ulcers are one of the most prevalent gastro intestinal diseases. Perforation of gastric ulcer is most common and dreaded complication of a gastric ulcer. Causes of gastric ulcer include *Helicobacter Pylori*, the NSAID, smoking and alcohol. *Helicobacter Pylori* infection is a curable cause of gastric ulcer. As prevalence of *H. pylori* differ in populations, prevalence of *H. pylori* in our population need to be assessed for determining treatment strategy for gastric ulcer.

Methods: Cross sectional study conducted in patients operated for perforation peritonitis and diagnosed to have gastric ulcer perforation in a tertiary care centre. Full thickness biopsy was taken from gastric ulcer perforation edge during the surgery for perforation peritonitis. The biopsy was stained with Giemsa stain and looked for the presence of *H. pylori*.

Results: *Helicobacter pylori* prevalence was 48.8% in our study. 8 females (out of 16) and 51 males (out of 105) had *H. pylori* positivity. 43 had history of smoking. Among them 20 were diagnosed to be having *H. pylori* positive. 26 persons had history of pan chewing. Among pan chewers 17 were *H. pylori* positive. Among 50 patients with history of alcoholism, 24 got *H. pylori* positivity. Among 40 patients with history of NSAIDs, 14 were positive for *H. pylori*. **Conclusions:** Prevalence of *Helicobacter pylori* in gastric ulcer perforation in present study is 48.8%. *Helicobacter Pylori* eradication should be added in treatment protocols for perforated gastric ulcers.

Keywords: Helicobacter pylori, Gastric ulcer perforations, Perforation peritonitis, Risk factors for peptic ulcer

INTRODUCTION

Peptic ulcer disease is one of the most prevalent gastro intestinal diseases. Peptic ulcers are defined as erosions in gastric or duodenal mucosa that extend through the muscularis mucosa. Gastric ulcer are focal defects in gastric mucosa which extends into submucosa or deeper. They are caused by imbalance between peptic acid and mucosal defenses. Roughly 75% of gastric ulcers are associated with *Helicobacter pylori* infection. The mean prevalence of *H. pylori* infection in patients with perforated peptic ulcer (which includes both gastric and duodenal ulcers) is approximately 60% compared with

90–100% in uncomplicated ulcer disease.² Non-infected gastric ulcer patients tend to be NSAID users. Smoking and alcohol consumption are also associated with perforated peptic ulcer.

Perforated peptic ulcer is one of the most common surgical emergencies in South India. With the improvement in medical therapy for peptic ulcer, elective ulcer surgery declined significantly, the percentage of emergency operations for complicated ulcers has recently increased from 60 to 90%. Peptic ulcer perforation is one of the most common surgical emergencies. The optimal treatment for this is still unclear. Simple repair with

omental patch is most commonly performed surgery. But patients undergoing simple closure have high incidence of ulcer recurrence. So it is now advised to add *H. pylori* eradication to the treatment protocols for perforated peptic ulcer. There is 90-100% association of H. pylori with peptic ulcer disease but the prevalence of H. pylori in perforated peptic ulcer ranges from 0-92%. This difference is partly attributed to population demographics of study group. Gastric ulcer perforations are more in this part of the world, compared to Western population. Hence determining prevalence of *H. pylori* in perforated gastric ulcers is important so as to start H. pylori eradication therapy more judiciously. Eradication of H. pylori will be decreasing recurrence rates of perforated ulcers. In the last few decades several studies have evaluated the Helicobacter Pylori infection in gastric ulcer perforation. Only very few studies are available in Indian population especially in south Indian population.^{4,5} Kerala being a state with high incidence of peptic ulcer disease, need of such a study is obvious. If a statistically significant relationship between H. pylori and gastric ulcer perforation is established, anti H. pylori drugs can be administered to treat infection which also helps in reducing recurrence. This study aimed at evaluating the prevalence of H. pylori infection in patients with acute perforated gastric ulcers in patients admitted in a tertiary care centre. This study also aims to identify risk factors associated with gastric ulcer perforation.

METHODS

It is cross sectional study. The Study carried out at Department of Surgery, Government Medical College Hospital, Thiruvananthapuram, India.

Study population

Patients admitted in surgical wards of Govt. Medical College Thiruvananthapuram who were operated for perforation peritonitis and diagnosed to have gastric ulcer perforation

Period of the Study was eighteen months from March 2016 to September 2017.

Sample size

Calculated sample size was 64. The actual sample size taken for the study was 121.

Sample size calculation

According to a study conducted by Sharma AK, Mittal S, Malvi SK named "Association of *H. pylori* with peptic perforation in Chattisgarh region of India" (TropGastroenterol2000;21:42–3) prevalence of *H.pylori* in perforated peptic ulcer was found to be 61%. Based on the above study sample size is calculated as below

N=4 pq/d2

P=prevalence 61/100=0.61 q=1-p= 1-0.61= 0.39 d=20% of p= 20/100 x 61/100= 0.122 N=(4 x 0.61 x 0.39)/ (0.122)2 = 64

Inclusion criteria

All cases operated for perforation peritonitis diagnosed to have gastric ulcer perforation and underwent operative procedure

Exclusion criteria

All patients who are not willing to give written consent.

Study variables

Helicobacter pylori

Gram negative bacilli residing in stomach believed to be causative for gastric and duodenal ulcers, gastritis and carcinoma stomach

Gastric ulcer

Focal defects in gastric mucosa which extends into submucosa or deeper caused by imbalance between peptic acid and mucosal defences

Gastric perforation

A hole in the wall of stomach leading on to spillage of its content into abdominal cavity

Data collection tools

Data was collected from patients and bystanders using a semi-stuctured questionnaire based interview, clinical examination, lab investigations and intraoperative findings. *Helicobacter pylori* detection was done by Giemsa staining of wedge biopsy specimen taken from the perforation site. Giemsa staining was done from Dept. of Pathology, Govt. medical college, Thiruvananthapuram

Data analysis

Data was entered into excel sheet.

Categorical variables are expressed as proportions and quantitative variables are expressed as mean and standard deviation

Statistical test of significance-chi square test for categorical variables and student t test for quantitative variables.

Analysis of data was done using appropriate statistical software.

It is a cross sectional study conducted in tertiary care teaching hospital during March 2016 to September 2017. The patients admitted in surgical wards during this period operated for perforation peritonitis and diagnosed to have gastric ulcer perforation are included in the study.121 cases were included in the study.

Data was collected from patients and bystanders using a semi-stuctured questionnaire based interview, clinical examination, lab investigations and intraoperative findings. *Helicobacter Pylori* detection was done by giemsa staining of wedge biopsy specimen taken from the perforation site. Analysis of data was done using appropriate statistical software.

In 121 cases studied of gastric perforation, 89 were in prepyloric region, 24 was in antrum in lesser curvature and 8 were in proximal stomach in body (Figure 1).

Helicobacter pylori was isolated from 43 (48.3%) patients with prepyloric perforation, 12 (50%) patients with perforation in antrum and 4 (50%) patients with perforation in body (Table 1). Most perforation was 1cm or less in diameter. There was 4 perforations with >2cm diameter. H. pylori prevalence was not associated with size of perforation.

RESULTS

Commonest age of presentation is 45-60 years. Lowest age reported was 14years and highest age was 85 years. The mean age of presentation was 49.56±15.31. Among 121 cases studied 105(86.8%) were males and 16(13.2%) were females. Male to female ratio 6.56:1. *H. pylori* was present in 8(50%) of females and 51 (48.6%) of males, though association was not statistically significant.

Site of perfoartion **Frequency** Percentage H. pylori present H. pylori Absent Pre Pyloric 89 76.6% 43 46 12 Antrum 24 19.8% 12 8 4 4 Body 6.6%

Table 1: H. pylori and site of perforation.

Risk factors		H. pylori present (%)	H. pylori present (%)	Total	Statistical value
Smoking	Present	20 (46.5)	23 (53.5)	43	χ2 =0.135 df=1 p=0.713
	Absent	39 (50)	39 (50)	78	
Pan chewing	Present	17 (65.4)	9 (34.6)	26	$\chi 2 = 3.663 \text{ df} = 1$ p=0.05
	Absent	42 (44.2)	53 (55.8)	95	
Alcohol	Present	24 (48)	26 (52)	50	$\chi 2 = 0.961 \text{ df} = 2$
	Absent	35 (49.3)	36 (50.7)	71	p=0.619
NSAID	Present	14 (35)	26 (65)	40	$\chi 2 = 4.528 \text{ df} = 1$
	Absent	45 (55.6)	36 (44.4)	81	p=0.033

Out of 121 patients 43 had history of smoking. Among them 20 were diagnosed to be having *H. pylori* positive. Among 78 non smokers, 39 were having H. pylori positivity. This difference was not statistically significant (p=0.713). 26 persons had history of pan chewing. Among pan chewers 17 were H. pylori positive and among non pan chewers 42 were H. pylori positive, the difference is statistically significant (p=0.05). Among 50 patients with history of alcoholism, 24 had H. pylori positivity and among 71 non alcoholics 35 had H. pylori positivity. The difference was not statistically significant (p=0.619). Among 40 patients with history of NSAIDs, 14 were positive for *H. pylori* and among 81 patients with no history of NSAIDs 45 had H. pylori positivity. Association between H. pylori and NSAIDs were found to be statistically significant (Table 2).

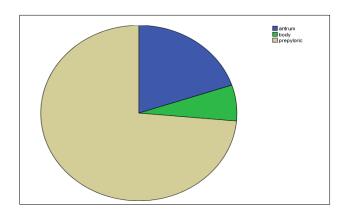


Figure 1: Site of perforation.

Prevalence of *H. pylori* according to this study was 48.8%. Of the 121 patients studied, 59 were positive for *H. pylori*.

DISCUSSION

In this study of prevalence of *H. pylori* in gastric ulcer perforations, 121 cases were included.

Prevalence of *H. pylori* according to this study was 48.8%. Of the 121 patients studied, 59 were positive for *H. pylori*. This is similar to study by John et al, with a prevalence rate of 46.9%. The study by Hussain et al, showed a prevalence rate of 67%. The prevalence according to study by Dogra et al, was 92%.

Commonest age group of presentation is 45-60years. 46 patients out of 121 belong to this category (38%) This is in accordance with the data in literature which shows that peptic ulcer perforation is frequently seen in 4th and 5th decade of life.^{9,10} Lowest age reported was 14years and highest age was 85 years. The mean age of presentation was 49.6±15.3 years. In a similar study by Arveen S et al, the mean age was 43.4±14.4 years.⁵ In another study from by Surapaneni et al, the mean age was 53 years. 11 In study by Dogra et al, mean age was 49.2 years. 8 In study by John et al, commonest age of presentation is 45-60yrs with mean age of 52.81± 14.5 yrs.6 Tas et al, study showed a mean age of 51.7±20 yrs. 12 That study also showed that age above 60 years was a statistically significant factor affecting mortality. In our study there was 32 patients (26.4%) with age above 60 years.

Among 121 cases studied 105(86.8%) were males and 16(13.2%) were females. Male to female ratio 6.56:1. *H. pylori* was present in 8(50%) of females and 51(48.6%) of males, though association was not statistically significant (p=0.915). In study by Dogra et al, male to female ratio was 3:1.8 The study by Surapaneni had a male female ratio of 9:1.¹¹ In study by John et al male to female ratio was 4.1:1.6

In 121 cases studied of gastric perforation, 89 (71.8%) were in prepyloric region, 24 (19.8%) was in antrum in lesser curvature and 8 (6.6%) were in proximal stomach in body. In study by Tas et al site of perforation was prepyloric region in 68.2%. In study by John B et al 80.4% patients had prepyloric perforation. Helicobacter Pylori was isolated from 43(48.3%) patients with prepyloric perforation, 12(50%) patients with perforation in antrum and 4 (50%) patients with perforation in body.

Most perforation was 1cm or less in diameter (99). There were 4 perforations with >2cm diameter. *H. pylori* prevalence was not associated with size of perforation. The study by Anbalakan et al, gives an ulcer size mean of 9.5 mm and median of 5mm.¹³

Out of 121 patients 43 had history of smoking. Among them 20 (46.5%) were diagnosed to be having *H. pylori*

positive. Among 78 non smokers, 39 (50%) were having *H. pylori* positivity. This difference was not statistically significant (p=0.713). A study by Ogihara et al, showed that smoking was negatively associated with *H. pylori* infection and risk of *H. pylori* seropositivity decreased linearly with smoking. In study by John et al, prevalence of *H. pylori* was not associated with smoking. In a study by Pillay et al, significant association was found between smoking and prevalence of *H. pylori*. The prevalence of *H. pylori* in smokers is reported as low in study by Ogihara et al, normal in study by Brenner et al, and high in study by Parasher and Eastwood. In 1,16,17

Among 50 patients with history of alcoholism, 24 got *H. pylori* positivity and among 71 non alcoholics 35 had *H. pylori* positivity. The difference was not statistically significant (p=0.619). The study by Murray and collaborators showed inverse correlation between alcohol ingestion and presence of active *H. pylori* infection.¹⁸

Among 40 patients with history of NSAIDs, 14 (35%) were positive for *H. pylori* and among 81 patients with no history of NSAIDs 45 (55%) had *H. pylori* positivity. Association between *H. pylori* and NSAIDs were found to be statistically significant. This is similar to studies conducted by John B et al, and Ullah et al.^{6,19} Incidence of NSAID users in this study is 33%. In study by Wadaani et al., 48.9% of patients had history of overconsumption of NSAIDs.²⁰ In study by Ambalakan et al, only <2% of patients had history of NSAID use.¹³

CONCLUSION

Prevalence of *Helicobacter Pylori* in gastric ulcer perforation in my study is 48.8. Among risk factors for gastric ulcer perforation, NSAID use and Panchewing has got a statistically significant association with *Helicobacter Pylori*. Mean age of presentation was 49.56. Among sites of gastric perforation, Prepyloric is commonest (73.6%), Antrum comprises 19.8%, and Body 6.6%.

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Ethical approval: The study was approved by the

Institutional Ethics Committee

REFERENCES

- Rauws EA, Langenberg W, Houthoff HJ, Zanen HC, Tygat GN. Campylobacter pyloridis-associated chronic active antral gastritis. A prospective study of its prevalence and the effects of antibacterial and antiulcer treatment. Gastroenterol. 1988;94:33-40.
- 2. Gisbert JP, Legido J, García-Sanz I, Pajares JM. *Helicobacter Pylori* and perforated peptic ulcer prevalence of the infection and role of nonsteroidal and anti-inflammatory drugs. Dig Liver Dis. 2004;36:116-20.

- 3. Tokunaga Y, Hata K, Ryo J, Kitaoka A, Tokuka A, Ohsumi K, et al. Density of H.Pylori infection in patients with peptic ulcer perforation. J Am Coll Surg. 1998;186:659-63.
- 4. Sharma AK, Mittal S, Malvi SK:Association of H.pylori with peptic perforation in Chattisgarh region of India. Trop. Gastroenterol. 2000;21:42-3.
- 5. Arveen S, Jagdish S, Kadmbari D:Perforated peptic ulcer in South India:an institutional perspective. World J Surg. 2009;33(8):1600-4.
- 6. John B, Mathew BP, Vipin Chandran C. Prevalence of *Helicobacter Pylori* in peptic ulcer perforation. Int Surg J. 2017;4(10):3350-3.
- 7. Hussain AA, Abro AH, Siddiqui FG, Memon AA. Prevalence of *Helicobacter Pylori* infection in Patients with Perforated Peptic Ulcer. JLUMHS. 2012;11(3):172.
- 8. Dogra BB, Panchabhai S, Rejinthal S, Kalyan S, Priyadarshi S, Kandari A. *Helicobacter Pylori* in gastroduodenal perforation. Med J DY Patil Univ 2014;7:170-2.
- 9. Kamani F, Moghimi M, Marashi SA, Peyrovi H, Sheikhvatan M. Perforated peptic ulcer disease:midterm outcome among Iranian population. Turk J Gastroenterol. 2010;21(2):125-8.
- Kocer B, Surmeli S, Solak C, Unal B, Bozkurt B, Yildirim O, et al. Factors affecting mortality and morbidity in patients with peptic ulcer perforation. J Gastroenterol Hepatol. 2007;22(4):565-70.
- 11. Surapaneni S, Rajkumar S, Reddy VBA. Perforation-Operation time interval; an Important Mortality Indicator in Peptic Ulcer Perforation. Journal of Clinical and Diagnostice Research, 2013;7(5):880-2.
- 12. Tas I, Ulger BV, Onder A, Kapan M, Bozdag Z. Risk factors influencing morbidity and mortality in perforated peptic ulcer disease. Ulusal Cerrahi Derg. 2014;31(1):20-5.
- 13. Anbalakan K, Chua D, Pandya GJ, Shelat VG. Five year experience in management of perforated peptic ulcer and validation of common mortality risk

- prediction models. Are existing models sufficient? A retrospective cohort study. Int J Surg. 2015;14:38-44.
- 14. Ogihara A, Kikuchi S, Hasegawa A, Kurosawa M, Miki K, Kaneko E, Mizukoshi H. Relationship between *Helicobacter Pylori* infection and smoking and drinking habits. J Gastroenterol Hepatolol. 2000;15(3):271-6.
- 15. Pillay KV, Htun M, Naing NN, Norsa'adah B. *Helicobacter Pylori* infection in peptic ulcer disease: the importance of smoking and ethnicity. Southeast Asian J Trop Med Public Health. 2007;38(6):1102-10
- Brenner H, Rothenbacher D, Bode G, Adler G. Relation of smoking and alcohol and coffee consumption to active *Helicobacter Pylori* infection:cross sectional study. BMJ. 315:1489-92.
- 17. Parasher G, Eastwood G.L. Smoking and peptic ulcer in the *Helicobacter Pylori* era. Eur. J. Gatroenter. Hepatol. 2000;12:843-53.
- Murray LJ, Lane AJ, Harvey IM. Inverse relationship between alcohol consumption and active *Helicobacter Pylori* infection. The Bristol Helicobacter project. Am J Gastroenterol. 2002;97:2750-5
- 19. Ullah A, Ullah S, Ullah A, Muzzafar-Ud-Din S, Khan M. Frequency of *Helicobacter Pylori* in patients presented with perforated peptic ulcer. JPMI. 2007:21(1):25-8.
- Wadaani HA. Emergent laparoscopy in treatment of perforated peptic ulcer:a local experience from a tertiary centre in Saudi Arabia. World J. Emerg. Surg. 2013;8(10):1749-7922-8-10.

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