International Surgery Journal
John B et al. Int Surg J. 2017 Oct;4(10):3350-3353
http://www.ijsurgery.com
pISSN 2349-3305 | eISSN 2349-2902

DOI: http://dx.doi.org/10.18203/2349-2902.isj20174494

Original Research Article

Prevalence of *Helicobacter pylori* in peptic ulcer perforation

Binni John*, Bipin P. Mathew, Vipin Chandran C.

Department of General Surgery, Medical college, Kottayam, Kerala, India

Received: 22 August 2017
Accepted: 20 September 2017

***Correspondence:**
Dr. Binni John,
E-mail: drvipinchandran@gmail.com

ABSTRACT

Background: *Helicobacter pylori* have an important role in the pathogenesis of peptic ulcer disease. The aim of the present study was to observe the prevalence of *H. pylori* in peptic ulcer perforation cases and the rationale of *H. pylori* eradication therapy post operatively and to investigate factors associated with peptic ulcer.
Methods: This cross-sectional study was conducted at the Department of general surgery, Government Medical College, Kottayam for a period 20 month from March 2012 to October 2013 after getting approval from institutional ethics committee. A total of 113 patients were participated in the study after meeting inclusion criteria. After getting written consent from the patients with perforated peptic ulcer, resuscitation and laparotomy was performed in the emergency department. *H. pylori* infection was confirmed by histopathological examination by Giemsa staining. Based on the histopathological report, the prevalence of *H. pylori* infection in the patients was assessed and was given appropriate *H. pylori* eradication regimen.
Results: The mean age of presentation of the patients was 52.81±14.5 years. Male to female ratio was 4.14:1. Out of 113 cases, 67 cases (59.3%) had duodenal ulcer perforation while 46 cases (40.7%) had gastric ulcer perforation. Of them 53(46.9%) cases were positive *H. pylori* positive. No significant association was found between the incidence of *H. pylori* infection in peptic ulcers with smoking, hypertension, diet intake, NSAIDS intake. In our study association between *H. pylori* and diabetes mellitus (p=0.02) found to be significant which can be further investigated.
Conclusion: According to our study the prevalence of *H. pylori* infection in perforated peptic ulcer disease is 47% which must be considered as significant. Hence all the patients undergoing laparotomy for peptic ulcer perforation should be investigated for *H. pylori* infection and if positive we must start the anti *H. pylori* regimen for them, which is more cost effective.

Keywords: Diabetes mellitus, Helicobacter pylori, Histopathological examination, Perforated peptic ulcer

INTRODUCTION

Perforated peptic ulcer is one of the most common surgical emergencies worldwide with associated mortality upto 30-50%.[1] Prevalence of *Helicobacter pylori* was one of the risk factor for peptic ulcer disease. As many as 60-90% (gastric and duodenal) of ulcers are associated with *H. pylori*.[2] Several diagnostic methods can be employed for the detection of *H. pylori* such as non-invasive serological tests which measures specific anti *H. pylori* immunoglobulins IgG and or IgA and invasive tests such as bacterial culture, histopathological examination of biopsy specimen with different stains and assays for urease activity.[3]

This study is mainly intended to observe the prevalence of *H. pylori* in peptic ulcer perforation cases by histopathological examination and the rationale of *H. pylori* eradication therapy postoperatively. Along with this other factor like type and location of ulcer, and proportion of *H. pylori* infection in factors like smoking,

NSAID intake, diet, hypertension, diabetes mellitus etc. was also studied.

METHODS

This was a hospital based cross sectional study conducted at the Department of general surgery, Government Medical College, Kottayam for a period 20 month from March 2012 to October 2013 after getting approval from institutional ethics committee. All patients who present in surgery casualty undergoing surgery for peptic ulcer perforation were included in the study. Exclusion criteria were patients who are not giving consent, previously diagnosed cases of *Helicobacter pylori* infection and suspected cases of malignant perforation.

Sample size

According to the formula $4PQ/L^2$ the sample size was 315.As number of cases in the present study was less, we restricted our sample size only to the patients undergoing surgery for peptic ulcer perforation and who gave consent. Hence the sample size was 113.

Demographic data, medical history, past history of peptic ulcer, use of NSAIDS were recorded. After getting written consent from the patients with perforated peptic ulcer, resuscitation and laparotomy was performed in the emergency department. Biopsy will be taken from the peptic ulcer perforation site with intact mucosa and will be sent for histopathological examination to detect the organism by Giemsa staining. Perforation was closed and re-enforced with an omental patch. Based on the histopathological report, the prevalence of *H. pylori* infection in the patients was assessed and was given appropriate *H. pylori* eradication regimen.

Statistical analysis

Data was entered in Excel work sheet and analysed with SPSS software. Data analysis of the patient's characteristics was performed by descriptive statistics. Differences in proportions was analysed with the chi-square test. Fisher's exact test was used when the expected value in any cell was <5. The difference in age was compared with unpaired t test. The statistical significance was set at p=0.05.

RESULTS

The commonest age of presentation is between 45 and 60 years. There is no case with age <18 years and >85 years. The mean age of presentation was52.81±14.5years (Table 1). Among the 113 cases studied, 91(81%) were males and 22(19%) females. Male to female ratio was 4.14:1 (Figure 1).

Table 1: Age distribution among study participants.

Age group	*Helicobacter pylori*		Total
	Present (%)	Absent (%)	
<30	1(12.5%)	7(87.5%)	8(100%)
30-45	15(50%)	15(50%)	30(100%)
45-60	20(50%)	20(50%)	40(100%)
>60	17(48.6%)	18(51.4%)	35(100%)
Total	53(46.9%)	60(53.1%)	113(100%)

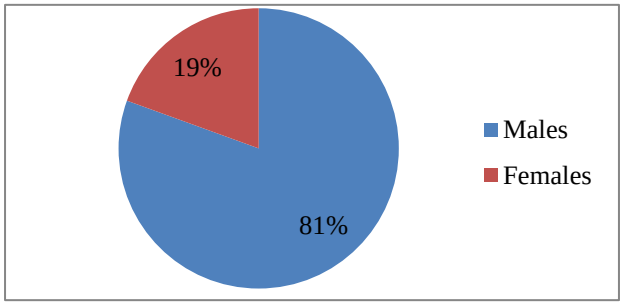

Figure 1: Distribution of gender among study participants.

Out of 113 cases, 67 cases (59.3%) had duodenal ulcer perforation while 46 cases (40.7%) had gastric ulcer perforation. Of 46 gastric ulcer cases, 37(80.4%) patients had pre-pyloric perforation. 53(46.9%) cases were positive *H. pylori* positive while 60(53.1%) cases were *H. pylori* negative (Table 2).

Many factors associated with *H. pylori* infection were studied and their association with prevalence of *H. pylori* was found to be statistically not significant except with diabetes as shown in Table 3. Out of 113 patients, 49 were smokers and 64 were non-smokers. Of them 19 and 34 cases were diagnosed with *H. pylori* respectively. This difference was not significant statistically (p=0.130). 53 patients had a history of NSAIDS intake while 60 were NSAID non-users. Among them 23 and 30 patients were *H. pylori* positive respectively. Patients with hypertension i.e. 19 out of 44 were had a prevalence of *H. pylori* while 34 patients out of 69 normotensives were *H. pylori* positive.

Table 2: Relation between type of perforation and *H. pylori* status.

Site of perforation	*H. pylori*		Total (%)	Statistical value
	Present (%)	Absent (%)		
Duodenal	28 (41.8)	39 (58.2)	67 (100.0)	X^2=1.727, df-1, p=0.189
Gastric	25 (54.3)	21 (45.7)	46 (100.0)	
Total	53 (46.9)	60 (53.1)	113 (100.0)	

Prevalence of *H. pylori* was seen in 27 and 26 cases out of 64 and 49 persons with regular and irregular diet respectively. Among the 34 pan users 14 and among the 79 nonusers, 39 were *H. pylori* positive. Out of 53 diabetics, 19 were *H. pylori* positive and among the 60 non-diabetics, 34 were *H. pylori* positive. Association between *H. pylori* and diabetes mellitus was found to be significant (p=0.02).

Table 3: Association of factors with *H. pylori* infection.

Factors		*H. pylori*		Total (%)	Statistical values
		Present (%)	Absent (%)		
NSAIDS	Present	23 (43.4)	30 (56.6)	53 (100.0)	X^2=0.493, df-1, p=0.483
	Absent	30 (50.0)	30 (50.0)	60 (100.0)	
Smoking	Present	19 (38.7)	30 (61.3)	49 (100.0)	X^2=2.295; p=0.130
	Absent	34 (53.2)	30 (46.8)	64 (100.0)	
Diabetes mellitus	Present	19 (35.8)	34 (64.2)	53 (100.0)	X^2=4.897, df-1, p=0.027
	Absent	34 (56.7)	26 (43.3)	60 (100.0)	
Hypertension	Present	19 (43.2)	25 (56.8)	44 (100.0)	X^2=0.401; p=0.527
	Absent	34 (49.3)	35 (50.7)	69 (100.0)	
Diet	Regular	27 (42.2)	37 (57.8)	64 (100.0)	X^2=1.318, df-1, p=0.251
	Irregular	26 (53.1)	23 (46.9)	49 (100.0)	
Pan users	Present	14 (41.4)	20 (58.6)	34 (100.0)	X^2= 0.640; p=0.424
	Absent	39 (49.4)	40 (50.6)	79 (100.0)	

Postoperative outcome

There was no death during immediate postoperative period. None had any complications that required re surgery/laparotomy during immediate postoperative period or during the same admission.

DISCUSSION

Peptic ulcer perforation is one of the major surgical emergencies.[4] The optimal surgical treatment for perforated duodenal ulcer has been controversial. Simple repair has been the most commonly performed procedure since its popularisation by Graham in 1937. However, long-term follow-up of patients who underwent simple repair reveals a high incidence of ulcer relapse.[4] Now it is advocated that simple repair followed by *H. pylori* eradication therapy for positive cases is the ideal management to be followed. Even though there is enough data regarding the relationship between uncomplicated peptic ulcer disease and *H. pylori* (90-100%), data regarding perforated peptic ulcer and *H. pylori* is limited. Hence, we tried to explore the prevalence of *H. pylori* infection in peptic ulcer perforation so that we can start the anti *H. pylori* regimen more judiciously.

In our study, the mean age of the study participants was 52.81±14.5 years. Males are more affected than females (M: F ratio was 4.14:1). This was similar with the findings of Dogra et al.[5]

In our study, the prevalence rate of *H. pylori* infection was 46.9%. This was comparable to the studies of Hussain et al in which the prevalence rate was 67%.[6]

In this study, patients having history of NSAIDS intake showed a less prevalence rate of infection in 43.4% cases compared to nonusers. This is similar with the observations of Ullah et al.[7] In this study, patients with smoking habit had a low infection rate of 38.7% compared to non-smokers 53.2% whereas in a study done by Pillay et al a significant association was found between smoking and incidence of *H. pylori*infection.[8]

From the results of the study, it was observed that the presence of *H. pylori* infection was higher in

hypertensive patients compared to normotensives. This was similar with the findings of Vinutha Shankar et al.[9] This association of incidence of *H. pylori* infection with hypertension might be due to known relation between salt intake and hypertension.[10,11]

Among 113 patients 53 were diabetics. Of them, 19 were *H. pylori* positive and among the 60 non-diabetics, 34 were *H. pylori* positive. The prevalence of *H. pylori* was more in non-diabetics which was in accordance with previous studies. Previous data suggest that prevalence, difficulty of eradication, incidence of complications and mortality was more for *H. pylori* associated peptic ulcer disease with diabetes.[12,13]

Prevalence of *H. pylori* infection with ulcer disease was more in patients with irregular diet habits (53.1%). Similar observations were made by Lim et al in his studies.[14] They found a significant association between these two factors and analysed that irregular meal timings changes the mucosal membrane and increases the susceptibility of bacterial penetration which may worsen the ulcer condition.[15]

CONCLUSION

Recurrent ulcer disease after peptic ulcer perforation mainly occurs in patients with *H. pylori* infection, which suggests that the microorganism plays an important role in this complication. All patients with perforated peptic ulcer should be treated by simple closure of the perforation and with therapy aimed at healing of the ulcer and eradicating the *H. pylori* infection, as disappearance of the organism prevents, or at least decreases, ulcer recurrence and ulcer perforation in patients with *H. pylori*-associated perforated ulcers after simple closure.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the institutional ethics committee

REFERENCES

1. Møller MH, Adamsen S, Thomsen RW, Møller AM. Multicentre trial of a perioperative protocol to reduce mortality in patients with peptic ulcer perforation. Br J Surg. 2011;98(6):802-10.
2. Peptic ulcer. Available at: https://www.parasitetesting.com/Peptic-ulcer.cfm. Accessed on 20 August 2016.
3. Gossens H, Glupezynski Y, Burett A, Van Den Borrec, Butzler JP. Evaluation of commercially available second-generation immunoglobulin G enzymes immunoassay for detection of H. pylori infection. J Clin Microbiol. 1992;30:176-80.
4. Søreide K, Thorsen K, Harrison EM, Bingener J, Møller MH, Ohene-Yeboah M, et al. Perforated peptic ulcer. Lancet. 2015;386(10000):1288-98.
5. Dogra BB, Panchabhai S, Rejinthal S, Kalyan S, Priyadarshi S, Kandari A. Helicobacter pylori in gastroduodenal perforation. 2014;7(2):170-2.
6. Hussain AA, Abro AH, Siddiqui FG, Memon AA. Prevalence of Helicobacter Pylori Infection in Patients with Perforated Peptic Ulcer. JLUMHS. 2012;11(3):172.
7. 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.
8. 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.
9. Shankar V, Kutty AVM, Annamalai N. Helicobacter pylori infection and hypertension: Is there an association? Biomed Res- India. 2012;23(4):537-9.
10. De Koster E, Buset M, Fernandez E, Deltenre M. Helicobacter pylori: link with gastric cancer. Eur JCancer Prevent. 1994;3:247-57.
11. Tsugane S, Tei Y, Takahashi T, Watanabe S, Sugano K. Salty food intake and the risk of Helicobacter pylori infection. Jpn J Cancer Res. 1994;85:474-8.
12. Townsend C, Daniel Beauchamp R, Mark Evers B, Mattox K. Sabiston Textbook of surgery. In: The Biological Basis of Modern Surgical Practice. Peptic ulcer disease. 20th edition. Elsevier; 2016:1197-1211.
13. Williams NS, Bulstrode CJK, O'connel PR. Bailey and Love's Short practice of surgery. In: Helicobacter pylori, Peptic ulcer. 26th edition. CRC press; 2013: 1030-6.
14. Lim SL, Canavarro C, Zaw MH, Zhu F, Loke WC, Chan YH, et al. Irregular Meal Timing Is Associated with Helicobacter pylori Infection and Gastritis. ISRN Nutrition. 2013;(2013):1-7.
15. Mukojima K, Mishima S, Oda J, Homma H, Sasaki H, Ohta S, et al., Protective effects of free radical scavenger edaravone against xanthine oxidase-mediated permeability increases in human intestinal epithelial cell monolayer. J Burn Care Res. 2009;30(2):335-40.

Cite this article as: John B, Mathew BP, Chandran VC. Singh K. Prevalence of Helicobacter pylori in peptic ulcer perforation. Int Surg J 2017;4:3350-3.