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Original Research Article

The prevalence of *Helicobacter pylori* in perforated peptic ulcer disease

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ABSTRACT

Background: Although the role of *Helicobacter pylori* infection in noncomplicated peptic ulcer disease has been definitively established, the precise relationship between the organism and ulcer complications is doubtful. 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.
Methods: This observational study was conducted in the Department of General Surgery, Himalayan Institute of Medical Sciences (HIMS), Swami Ram Nagar, Dehradun, over a period of January 2013 to December 2013 and included 75 subjects who underwent exploratory laparotomy for peptic ulcer perforation. The tests used for the diagnosis of *H. pylori* are mucosal biopsy at the time of surgery, Rapid urease test (RUT), Stool antigen test.
Results: In this study, *H. pylori* infection was found to be present in 61% of these patients as detected by biopsy. There were 66 males and 9 females. Infection with *H. pylori* is almost universal in patients aged more than 70 years.
Conclusions: In the Indian context patients presenting with perforation should be tested for infection with *H. pylori* utilizing a gastric antral mucosal biopsy taken at the time of operation for histological analysis and eradication therapy should be advised to all those who are found positive.

Keywords: *H. pylori*, Perforated peptic ulcer

INTRODUCTION

Although *H. pylori* eradication is now the standard treatment of uncomplicated and bleeding peptic ulcers, its role in perforation remains controversial and these brackets of opinions pompously affect the decision making of the surgeon.

In 1983, the whole thinking regarding pathophysiology and management of this disease was revolutionized when Warren and Marshall reported *Helicobacter pylori*. They are recognized as a major causative factor of peptic ulcer disease and their eradication leads to cure of disease, i.e. better symptom control and fewer relapses. *H. pylori* infection is almost always present in the setting of active chronic gastritis and is present in most duodenal (>90%) and gastric 60 % to 90% in ulcer patients.[1]

H. pylori can be diagnosed by mucosal biopsy, but noninvasive tests offer an effective screening tool and do not require an endoscopic procedure. It is now accepted that histologically confirmed chronic gastritis and duodenal ulceration is caused by *H. pylori* infection in over 90% of cases and *H. pylori* infection is responsible for 50% of gastric ulcer.[1]

Complications of peptic ulcer disease such as bleeding, perforation, and stenosis occur in 3% of ulcer patient a year, and unless maintenance treatment is given, 82% of ulcer treated with H_2 receptor antagonist relapse within one year.[2]

Although the role of *H. pylori* in causation of duodenal and gastric ulcer is beyond doubt according to various studies, the role of *H. pylori* in various complications of PUD is not well understood. Various authors have reported an association of *H. pylori* infection with perforated peptic ulcer disease and opinion is still divided on this issue.

In this study the prevalence of *H. pylori* infection in patients presenting with perforated peptic ulcer disease in the emergency will be assessed. This will help the surgeon in deciding whether *H. pylori* eradication therapy should be offered to all patients with perforated PUD or it should be applied selectively. This study will also assess the sensitivity of stool antigen test for *H. pylori* detection in patients with perforated peptic ulcer.

METHODS

This observational study was conducted in the Department of General Surgery, Himalayan Institute of Medical Sciences (HIMS), Swami Ram Nagar, Dehradun, over a period of January 2013 to December 2013 and included 75 subjects who underwent exploratory laparotomy for peptic ulcer perforation.

The exclusion criteria were patients who were haemodynamically unstable at time of laparotomy or refused for *H. pylori* test. Subjects were recruited from patients presenting with a primary diagnosis of perforated peptic ulcer after obtaining written informed consent. Ethical clearance for the study was granted by ethics committee of the university.

The study tools included were Biopsy forceps, Rapid urease test (RUT) kit, Stool antigen kit. RUT was performed at the time of exploratory laparotomy. A biopsy of mucosa was taken from the antrum of the stomach and was placed into a medium containing urea and an indicator such as phenol red. Sterile biopsy forceps were introduced at site of ulcer perforation and mucosal biopsies were taken within 3cm of pylorus. Biopsy specimens were put in 10% formalin for histopathological examination. Stool antigen test was done by using standard kit available (standard diagnostics).

Diluent to be added to stool sample and with the help of the dropper 3 drops were added to the test device. This test was carried out only in patients with perforation in the postoperative period. Interpretation of test result was done 10-15 minutes for the presence of two color bands i.e. test band and control band. In this study out of the above-mentioned laboratory tests histological examination was considered gold standard.

The result data obtained was evaluated and studied by analytical method Pearson Chi square test to ascertain statistical significance and with software SPSS, version 20.

RESULTS

In this study, *H. pylori* infection was found to be present in 61% of these patients as detected by biopsy. The result of diagnostic tests for *H. pylori* in patients with perforated peptic ulcer disease is shown in Table 1.

Table 1: Results of diagnostic tests for *H. pylori* infection in perforated peptic ulcer disease (n=75).

Diagnosis	RUT (%)	Biopsy (%)
Positive	62 (82.67)	46 (61.33)
Negative	13 (17.13)	29 (38.67)

In-patient with perforation those who had biopsy proven *H. pylori* infection all had positive rapid urease test. The false positive rate of RUT considering biopsy to be gold standard is 21% and sensitivity is 79%. Presence of *H. pylori* in patients with perforated peptic ulcer disease does not reach statistical significance (Pearson Chi square test $p<0.05$) and there is substantial number of patients who have perforations without any associated H. *pylori* infections.

Infection with *H. pylori* is almost universal in patients aged more than 70 years and maximum infection rate excluding this group was seen in 51-60 years age group as shown in Table 2.

Table 2: Prevalence of *H. pylori* infection according to age.

Age group (years)	Patient with perforated peptic ulcer (n=75)	
	No. of patient	***H. pylori* positive (%)**
18-30	9	5(55.55)
31-40	20	12(60%)
41-50	22	13(59%)
51-60	12	9(75%)
61-70	9	5(55.55%)
>70	**3**	3(100%)

Among the 75 patients presenting with perforation there were 66 males and 9 females amongst which 60% of male patients and 66% of females demonstrated infection with *H. pylori*. 34 patients had gastric perforation and 41 had duodenal perforation. *H. pylori* infection was noted in 18 out of 34 gastric perforation (52.94%) and in 28 out of 41 duodenal perforation patients (68.29%).

Only 15 had positive history of NSAIDS. Amongst these 15, 8 were positive for *H. pylori* infection (53.34%). 60 patients did not have any history of NSAIDs out of these 38-tested positive for *H. pylori* 63.33%. Similarly, a history of intake of steroids was present in only 10 out of 75 patients with peptic ulcer perforation. *H. pylori* infection was seen in 6 out of these 10 patients (60%). The rate of *H. pylori* infection was same amongst the smokers and nonsmokers.

34.66% patient who use to take alcohol and 26.66% who did not take alcohol were positive for *H. pylori* infection respectively. Stool antigen test was done in the first 20 cases in the post-operative period once the patient passed stools and it was found to be uniformly negative in all the cases.

DISCUSSION

The question arises weather the *H. pylori* infection is an important factor in patients with perforated peptic ulcer. If this association can be established, then it would mean that all patients with perforation should receive *H. pylori* eradication therapy in postoperative period as an adjunctive treatment.

In this study, *H. pylori* infection was documented in 46 patients (61.33%) by histopathology and in 62 patients (82.67%) by the rapid urease test. It shows that substantial number of patients with perforation do not harbor the H. pylori bacilli and so not all of these patients would be candidate of eradication therapy. Therefore, it would be prudent for the surgeon to gather gastric antral mucosal biopsy at the time of operation to document the presence of *H. pylori* and only those who test positive should be offered eradication therapy.

Few studies have shown lower prevalence rate for *H. pylori* infection in patients with perforation as compared to patients with simple ulcer were published by Kumar et al (33.32%), Chaudhary et al (0%), Reinbach et al (47%) and Sakaguchi et al (42.1%), where the *H. pylori* positivity rate was approximately 33 to 47%.[3-6]

An intermediate association with *H. pylori* as seen in this study has also been reported by the following authors Debongni et al (56%), Ng et al (69.8%), Aman et al (68%), Sharma et al (61.4%), Gisbert et al (60%), showed that *H. pylori* infection in perforated peptic ulcer is in range of 55-70% as as has been reported in this study.[7-11] The study by Debongni 1995 reported *H. pylori* infection in normal population as 36% in perforated ulcer 56% and 86% in patients with ulcer without perforations. They concluded patients with perforation are a heterogeneous group and recurrent ulcer mainly seen in *H. pylori* infection.

Rapid urease test for *H. pylori* detection his more positive result as compare to histological examination as seen in this study and also been demonstrated by other authors namely Kumar et al who evaluated 86 patients with perforated peptic ulcer disease and 43 (50%)were found positive on rapid urease test and 29 (33.72%) patients on histology.[12]

Bateson at al reported strong association between *H. pylori* infection and current smoking while in this study the association of *H. pylori* infection with smoking in perforated peptic ulcer disease patients is not found to be significant.[13]

Overall alcoholics have higher prevalence of peptic ulcer disease as reported by Parlet et al but as seen in this study history of alcohol intake doesn't impact infection with *H. pylori*.[14] Shipnen et al 1992, Grahm et al 1991 reported that *H. pylori* infection more common in lower socioeconomic status and author have reported similar results in this study amongst patients with perforation *H. pylori* infection is the same across the socioeconomic status (50% for lower socioeconomic group, 56% in the rest).[15,16]

The stool antigen test for detection of *H. pylori* was carried out in 20 out of 75 patients with perforated peptic ulcer and it was found to be negative for *H. pylori* for all the 20 patients. The sensitivity of stool antigen test in simple ulcer as reported in literature is in range of 88-100%.[17] However, no report is available on utilization of stool antigen test for detection *H. pylori* in complicated ulcer disease specially in postoperative setting.

Demonstration of *H. pylori* in peptic ulcer perforation using stool antigen test was found to be negative. The probable reason being that patients with perforated peptic ulcer pass stools approximately 5 days after surgery, having received antibiotics and proton pump inhibitors during this period which might render the stool sample negative for antigen. Secondly most patients after surgery from perforated peptic ulcer perforation pass loose stools in postoperative period or require enema which reduces the bacterial load and hinders positivity of tests. Based on the results of this study stool antigen test does not seem to be a feasible test in the surgical patient.

CONCLUSION

The prevalence rate of *H. pylori* infection in patients with perforated peptic ulcer is approximately 61%. In the Indian context patients presenting with perforation should be tested for infection with *H. pylori* utilizing a gastric antral mucosal biopsy taken at the time of operation for histological analysis and eradication therapy should be advised to all those who are found positive. Stool antigen test is not a good tool to asses *H. pylori* infection in patients with perforated peptic ulcer in the perioperative setting.

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