

Original Research Article

Endoscopic assessment and *Helicobacter pylori* status evaluation in operated cases of peptic ulcer perforation

Prashant Rao*, Sarika Mayekar, Vishwajit Pawar, Mohan Achyut Joshi

Department of Surgery, Lokmanya Tilak Municipal Medical College and General Hospital, Mumbai, Maharashtra, India

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***Correspondence:**

Dr. Prashant Rao,

E-mail: prao009@gmail.com

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ABSTRACT

Background: *Helicobacter pylori*'s role in delaying ulcer healing after surgical repair for peptic ulcer perforation causing ulcer persistence hasn't been definitively established as it has been for uncomplicated ulcers.

Methods: Authors performed an endoscopy and *H. pylori* status evaluation in 30 patients at an average of 6.2 weeks after simple omental patch closure for perforated peptic ulcer.

Results: A positive *H. pylori* status was found in 12 patients (40%) of which 9 had active ulcers. None in the negative group had an active ulcer. *H. pylori* infection was the only factor found to be responsible for ulcer persistence after surgery.

Conclusions: A reasonable approach would thus be to perform an endoscopy 6 weeks after surgery to assess ulcer healing and *H. pylori* status. *H. pylori* eradication therapy should then be selectively initiated for patients with an active ulcer or positive *H. pylori* status.

Keywords: *Helicobacter pylori*, Peptic ulcer perforation, Ulcer persistence, Ulcer recurrence

INTRODUCTION

Duodenal or gastric perforation is one of the most catastrophic complications that could occur in patients with Peptic Ulcer Disease (PUD). Although the role of *Helicobacter pylori* infection in uncomplicated peptic ulcer disease has been definitively established through various studies since its identification by Marshall and Warren in 1984, the precise relationship between the organism and peptic ulcer perforation is yet to be established.¹ Its prevalence in perforated peptic ulcer is found to vary in different studies between 0-100% with mean being around 65-70%, in contrast to 90-100% prevalence found in uncomplicated disease.² It is also well known that *H. pylori* infection can cause delayed ulcer healing, refractory and recurrent ulcers despite acid suppression in uncomplicated cases. However, the same

is yet to be established for perforated peptic ulcers. This forms the basis of this study which aims at identifying the relation between *H. pylori* and peptic ulcer perforation.

METHODS

This study is a prospective observational study which was performed in the Department of Surgery at Lokmanya Tilak Municipal Medical College, Mumbai, India after obtaining institutional ethics committee approval. All patients who underwent laparotomy with simple omental patch closure for perforated peptic ulcer between June 2013 to June 2014 were eligible for inclusion into the study.

Following surgery patients were administered a combination of third generation cephalosporin and metronidazole. Ranitidine was used as the acid

suppressant. No patient was empirically started on *H. pylori* eradication therapy. They were asked to follow up at 6 weeks after discharge for endoscopic evaluation. Patients who received *H. pylori* eradication therapy postoperatively and/or PPIs, NSAIDs or antibiotics, during 4 weeks preceding the endoscopy, were excluded.

At follow-up patients were enquired for presence of symptoms suggestive of persistent or recurrent PUD following which the state of ulcer healing was noted at endoscopy. 7 mucosal biopsies were taken from within 3 cm of pylorus.

Three samples were sent for culture studies, three for histo-pathological examination and the seventh was tested immediately using Rapid Urease tests kits in the endoscopy suite itself Patient was considered to have *H. pylori* infection if both RUT and histology were positive or if culture study was positive irrespective of the other results. Chi square test with Yates correction and Fischer’s exact test were used to analyze the categorical data. Whereas student t test was used for continuous variables. A p value of <0.05 was taken as significant.

RESULTS

A total 30 patients were included into the study (M: F=9:1) (mean=33.43 years, SD=15.44, range=18 to 65 years) following up at an average of 6.2 weeks after surgery. Duodenal ulcer perforation was much more common compared to gastric perforation with size of perforation being less than 1 cm in all except 2 cases and history of NSAID consumption being present in only 16.16%. 11 patients had post-operative dyspeptic

symptoms with epigastric discomfort being the most common symptom followed by epigastric pain and bloating.

A positive *H. pylori* status was found in 12 patients (40%). Nine of these 12 cases had active ulcers, whereas none in the negative group had an active ulcer. Antral gastritis was noted in 18 cases (60%). Significantly more patients in the infected group continued to have dyspeptic symptoms.

Characteristics of patients with a positive H. Pylori status (Table 1)

All patients in *H. pylori* positive group were male with a mean age of 35.33, however neither was found to be significant. 9 of these 12 cases had active ulcers. Whereas none in the negative group had an active ulcer. This observation was statistically significant (p<0.001).

All the 12 patients had antral gastritis (p=0.0003). 8 out of 12 patients (66.66%) had post-operative symptoms of PUD compared to just 16.66% in *H pylori* negative group (p value=0.0086).

Factors associated with active ulcer (n=9) (Table 2)

H. pylori infection was found to be universally present in cases with active ulcer (p<0.001). It was also associated with antral gastritis in all cases (p=0.004). Of them, 7 had post-operative symptoms of PUD (p=0.0042). Age, sex and NSAID intake were not found to be significantly related to healing of ulcer.

Table 1: Characteristics based on H. pylori status.

	<i>H. pylori</i> status positive		<i>H. pylori</i> status negative		P value
	Cases (n=12)	%	Cases (n=18)	%	
Active ulcer	9	75	0	0	<0.001
Antral gastritis	12	100	6	33.33	0.0003
Post-op symptoms present	8	66.66	3	16.66	0.0086
NSAID use	2	16.66	3	16.66	1
Sex (M: F)	12:0		5:1		0.255
Median age in yrs. (SD)	35.33(16.2)		32.16(15.2)		0.718

Table 2: Factors associated with ulcer status.

Characteristics.	Active ulcer present		Active ulcer absent		P values
	Cases (n=9)	%	Cases (n=21)	%	
Positive <i>H. Pylori</i> status	9	100	3	14.28	0.001
Antral gastritis	9	100	9	42.85	0.004
Post-op symptoms present	7	77.77	4	19	0.0042
H/O NSAID	2	22.22	3	14.28	0.622
Sex (M: F)	9:0		6:1		0.532
Median age in years (SD)	32.11(14.46)		34(15.76)		0.9605

DISCUSSION

The best therapeutic strategy following surgery for peptic ulcer perforation remains a matter of debate. Controversy exists regarding the prevalence of *H. pylori* infection in cases of peptic ulcer perforation, and its role in the recurrence or persistence of ulcer after surgery.

The prevalence of *H. pylori* infection in operated cases of perforated peptic ulcer in previously performed studies is found to vary between 0% to 100%.² Chowdhary et al in his study from India including 15 patients found *H. pylori* infection to be universally absent.³ On the contrary Matsukura et al found serum *H. pylori* IgG antibody to be positive in 95% of perforated duodenal ulcers and in 100% of perforated gastric ulcer patients.⁴ Kumar et al found a prevalence of 56.6%, whereas Chu et al and Reinbach et al both found a prevalence of 47%.⁵⁻⁷ Gisbert et al in his review article including data from 19 studies calculated the mean prevalence of *H. pylori* infection in perforated peptic ulcers to be 68.1%.² The *H. pylori* positivity rate in this study was found to be 40%. The low frequency noted in this study may be due to the fact that all patients received intravenous antibiotics which might have had an eradictory role. Another factor is that both culture and histology are not fool proof in detecting *H. pylori*.

9 out of 12 (75%) patients with positive *H. pylori* status, while none from negative group had an active ulcer. That means all patients with unhealed duodenal ulcer had *H. pylori* infection and *H. pylori* infection was the only factor found to be causally related to active ulcer in this study. This finding is similar to results in study conducted by Kumar et al and Chu et al.^{5,6} Apart from active ulcer *H. pylori* infection was also significantly found to be related to antral gastritis.

Post-operative dyspeptic symptoms were found to be significantly related to a positive *H. pylori* status and to the presence of an active peptic ulcer. Epigastric discomfort was the most common symptom noticed followed by epigastric pain and bloating. The effect of *H. pylori* eradication on the natural history of perforated peptic ulcer has been addressed in a few studies. Ng et al, conducted a randomized control trail wherein they demonstrated that *H. pylori* eradication reduced the ulcer relapse rate to 4.8% at 1 year compared to 38.1% in patients receiving omeprazole alone.⁸ This remission rate seems to be similar to that previously reported for uncomplicated ulcers after *H. pylori* eradication.⁹

CONCLUSION

H. pylori infection seems to cause ulcer persistence or recurrence in cases of perforated peptic ulcer. Eradication of the organism by appropriate therapy in diagnosed cases of *H. pylori* infection may promote ulcer healing

and prevent ulcer relapse. A reasonable approach would be to perform an endoscopic examination 6 weeks after surgery once he/ she has recuperated from the acute illness to check for status of ulcer healing and to determine the *H. pylori* status. *H. pylori* eradication therapy should be initiated only for patients with a positive *H. pylori* status or an active ulcer.

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