Original article:

Prevalence of Helicobactor Pylori in Dental Plaque of Patients with Perforated Duodenal Ulcer

Gyanendra S. Mittal

Assistant Professor, Department of Surgery, Santosh Medical College, Ghaziabad, Uttar Pradesh, India.

Corresponding Author: Dr. Gyanendra S. Mittal, Assistant Professor, Department of Surgery, Santosh Medical College, Ghaziabad, Uttar Pradesh, India.

ABSTRACT

Background: Helicobactor pylori plays an important role in the pathophysiology of peptic ulcer disease. However, its role in duodenal ulcer perforation is not well established and results are conflicting. A dental plaque in tooth of patient of perforated duodenal ulcer may function as a reservoir for H. pylori especially in patient with poor oral hygiene.

Methods: A total of 59 patients of perforated duodenal ulcer and 30 normal healthy volunteers were included in the study. For detection of H. pylori in dental plaque, samples were collected from 2 molar teeth using a periodontal curettage. Gastric antrum biopsy was taken only in perforated duodenal ulcer patients at the time of laparotomy. Rapid urease test was used to identify bacteria.

Results: In 27 (45.76%) of patients H. pylori was present in dental plaque and out 21 (35.59%) harbour the bacteria in their gastric antrum. In NHV only 6 (20%) were positive for H. pylori in their dental plaque. That was statistically significant.

Conclusion: Prevalence of H. pylori was significantly more in dental plaque of duodenal ulcer patients as compare to NHV, but its role in the etiology of perforation still require more research.

Key words: Duodenal Ulcer Perforation, H. Pylori, Dental Plaque, Peritonitis.

INTRODUCTION

Helicobactor pylori (H. pylori) is a micro-aerophilic, gram negative, spiral and mobile bacteria.[1] It has been investigated intensely since 1982 when it was first discovered by Salmon. It's role has been established in peptic ulcer disease and now it is believed that most of the duodenal ulcer diseases are either caused by infection with H. pylori or ingestion of drugs (like NSAID's and steroids) or hyper secretion of gastrin as in ZE Syndrome.[2,3] But its role in the complications of peptic ulcer disease like haemorrhage or perforation has not been established. The exact role of H. pylori in functional dyspepsia is also not very clear.

Aveu et al. suggested that patients with poor oral hygiene are likely to have H. pylori in their dental plaque.[4] He also proposed that even after triple therapy, H. pylori infection may reoccur in patients with poor oral hygiene more frequently than in patients with good oral hygiene. Anderson et al. showed that dental plaque served as a reservoir for this pathogen.[5] Studies regarding the prevalence of H. pylori in dental plaque & its association with systemic complications are indecisive.

The present study was carried out to know the prevalence of H. pylori in dental plaque (DP) and gastric antrum of patients of duodenal ulcer perforation (DUP) patients and in dental plaque of normal healthy volunteer (NHV) and tried to established an association between infection of H. pylori in dental plaque and DUP patients.

METHODS

This present study was carried out in Dept. of Surgery with collaboration of Dept. of Pathology in UCMS & GTB hospital, Delhi. The study was approved from institute's ethical committee. The NHV were taken from OPD of Surgery department whereas DUP patients were those present in surgical emergency. Confirmation of DUP was done by laparotomy and then these cases were included in the study. NHV and DUP patients of either sex who gave the consent of inclusion in the study were enrolled.

A total of 30 NHV were included in the study. Volunteers with symptoms of active peptic ulcer, gallstones, pancreatitis and non-ulcer dyspepsia or treatment with amoxycillin and meterogyl, especially in the immediate past six months were exclude from study. DP was removed from gingival side of two molar teeth with the help of a sterile needle.

59 cases of DUP peritonitis were included during the period of 1 yr. After admission in emergency resuscitation started with IV fluids, antibiotics, nasogastric intubation and relevant investigations with pre-anaesthesia checkup were obtained. DP was removed from gingival side of two molar teeth with same done with NHV. On laparotomy, Graham's patch repair of duodenal perforation was done in all patients and after through lavage drain put and abdomen closed in layers as a standard procedure. Multiple mucosal biopsies were taken from antrum of stomach during laparotomy. An attempt was made to introduce flexible endoscopic biopsy forceps from the perforation site guided towards antrum of stomach. Once in the stomach the tip of forceps was opened for taking biopsy from the antrum. During biopsy the tip of the forceps was guided and supported by palm of the other hand on anterior surface of stomach. All the biopsies were taken by closed method, if we were unable to guide the tip of forceps into the stomach and such cases were excluded from the study.

All samples collected from NHV (dental plaque) and patients of DUP (dental plaque and gastric antrum) were sent separately for rapid urease test (RUT). Samples were initially put into urea broth with phenol red as an indicator and readings were taken after 4 hr. The test was taken as positive for H. pylori, if colour of medium turned pink.

Table 1: H. pylori in Dental plaque of DUP patients and NHV

	DUP (n = 59)	NHV (n = 30)	P value
Age range **Mean ±SD**	03-75 yrs 38.5 ±15.5	16-66 yrs 36.0 ± 13.9	0.290
H. pylori (+) in Dental Plaque	27 (45.76%)	6 (20%)	< 0.05
H. pylori (-) in Dental Plaque	32 (54.24%)	24 (805)	

Table 2: Results of H. pylori detection in DUP patients

	Dental Plaque	Gastric Antrum	P value
H. pylori (+)	27	21	Not significant
H. pylori (-)	32	38	

RESULTS

Initially a total of 65 patients were included in the study. Out of these 5 patients had diagnosis other than duodenal perforation and one patient died, so finally 59 patients were taken in the study. The mean age of DUP patients was 38.5 ± 15.1 years (mean ± SD) and of NHV was 36.0 ± 13.9 years (mean ± SD) was comparable (Table-1). Age range in DUP was 03-75 years and NHV was 16-66 years. Out of 59 DUP patients H. pylori was detected in dental plaque of 27 (45.76%) and gastric antrum of 21 (35.59%) patients (Table-2). In NHV, 6 (20%) out of 30 persons had H. pylori in their dental plaque. Level of significance was calculated by Chi-square test and this difference of presence of H. pylori in dental plaque between DUP patients and NHV was significant $p < 0.05$.

DISCUSSION

H. pylori remains an interest of research for last few decades concerning duodenal ulcer and its complications. It is found in the gastric antrum primarily in the deep portion of mucus gel layer that coats the gastric mucosa between the mucus gel layer and the apical surface of gastric mucosal epithelial cells.[6] H. pylori produces a variety of proteins that appears to mediate or facilitate its damaging effects on the gastric mucosa. Urease produced by H. pylori catalyzes the hydrolysis of urea to ammonia and CO2.[7] Production of urease is required for gastric colonization by H. pylori and may protect it from virulent effects of gastric acid, which normally prevents gastric colonization by other bacteria. Hydroxide ions generated by the equilibrium of water with ammonia may contribute to gastric mucosal damage.[8]

For the diagnosis of H. pylori infection various tests are there like RUT, radioactive carbon-13 and carbon-14 breath test, serological studies, polymerase chain reaction (PCR), culture and histology. We used RUT, as it is fastest method (results can be obtained within 4 hours) but false negatives are common. PCR is the most sensitive method but cannot differentiate between living and dead bacteria.

Many studies had been conducted regarding its role in association with duodenal ulcer[9] and its complications. H. pylori has been isolated from many patients high of duodenal ulcer and therefore it is recommended now that these patients should receive H. Pylori eradication therapy as it decreases relapse rate. There are only few reports about the effect of H. pylori in ulcer complications.[10,11] Rainback et al.[12] investigated the presence of H. pylori in perforated duodenal ulcer with the anti H. pylori IgG and C14 urease test and found it to be present in 50% of the patients. They saw that the results were same in the control group. Hosking et al. found the presence of H. pylori in 93 % of the gastric antrum biopsy of patients of chronic ulcer pain and 71% in case of hemorrhage.[13] Both these studies concluded that H. pylori didn't play any role in the complications of peptic ulcer. In contrast to these studies, Sebastain et al. found H. pylori in 83%[14] and Mihmanli et al. found in 88% patients of DUP.[15] They concluded that there was a significant relation between H. pylori infection and perforation of duodenal ulcer disease.

The optimal surgical treatment for perforated duodenal ulcer also has been debatable. Simple repair has been the most common procedure since its popularization by Graham in 1937.[16] However, long term follow-up of patients who underwent simple repair reveals a high incidence of ulcer relapse. In the 1980 prospective randomized study reported significantly fewer ulcer recurrences by adding immediate proximal gastric vagotomy to patch repair of ulcer perforation.[17] Although the procedure has shown to be safe without increasing the rate of perioperative

complications, it is technically demanding and requires prolonged operation time. With the recent rapid decline in the number of elective peptic ulcer operations, immediate proximal gastric vagotomy is unlikely to be a practical procedure for most surgical residents, the front-line personal managing patients with duodenal ulcer perforation.

Dental plaque is the reservoir of H. pylori infection.[4] Desai et al conducted a study of H. pylori in dental plaque and stomach, they concluded that dental plaque is a reservoir of H. pylori and its numbers are far greater in the dental plaque than in the stomach.[18] H. pylori in the DP was unaffected by medical therapy, and hence DP may be responsible for recrudescence of infection after cessation of therapy. They detected H. pylori in DP and gastric antrum in 98% and 67% in patients of dyspepsia respectively. However, incidence of H. pylori in India is different. Two studies conducted in Bombay told different incidence of H. pylori in DP. Majumdar et al showed 100% incidence of H. Pylori in 40 healthy volunteers.[19] Another study conducted by Kamat et al showed 22% incidence of H. pylori in healthy volunteers.[20]

Poor oral hygiene potentially affects the gastro-intestinal flora and may be involved in the development of chronic gastro-intestinal disease.[21] Studies also showed the presence of H. pylori in saliva indicating it is an important reservoir of bacteria as a route of transmission. Riggo & Lemnon found the H. pylori in 38 % of subjects with deep periodontal pockets.[22] Dye et al observed 41 % of patients with periodontitis were seropositive for H. pylori in a national survey of 4,504 subjects.[23] It is found in stomach of >50% of these persons. In subjects who were positive for H. Pylori in their stomach, 74% had chronic periodontitis.[24]

We have conducted this study to know the prevalence of H. pylori in DUP patients and in NHV. In our study 45% of DUP patients had H. pylori in their DP as compare to only 20 % in NHV, that is statistically significant with $p < 0.05$. We found 27.78 % of patients of DUP have H. pylori in their gastric antrum. As poor oral hygiene and presence of H. pylori is more common in DP of patients of DUP then NHV there may be some correlation between the two, but further studies are required to establish its role in pathophysiology of duodenal perforation. Mere presence of H. pylori does not mean it causes perforation but if it did, gastric antrum perforation would be more common than duodenal perforation?

REFERENCES

1. Dunn BE, Cohen H, Blaser MJ. Helicobacter pylori. Clin Microbiol Rev 1997;10:720-41.
2. Grahams DY. Treatment of peptic ulcer caused by H. pylori (Editoeial). N Eng J Med 1993; 328: 349-50.
3. Grahams DY. H.pylori: its epidemiology and its role in duodenal ulcer disease. J Gastrol Hepatol 1991;6:105-13.
4. Avcu N, Avcu F, Beyan C, et al. The relationship between gastricoral Helicobacter pylori and oral hygiene in patients with vitamin B12-deficiency anemia. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;92:166-9.
5. Andersen RN, Ganeshkumar N, Kolenbrander PE. Helicobacter pylori adheres selectively to Fusobacterium spp. Oral Microbiol Immunol 1998;13:51-4.
6. Marshal BJ, Barrett L, Prakash C. Survival of Camphylobactor pyloridis at acid pH. Gastroenterology 1987;92;1517.

7. Brady CE, Hadfield TL, Hyatt JR. Acid secretion serum gastrin levels in individuals with Camphylobactor pyloridis. Gastroenterology 1998;94:923-7.

8. Goodwin CS, McCulloch RK, Armstrong JA. Unusual cellular fatty acids and distinctive ultrastructure in a new spiral bacterium (Camphylobactor pyloridis) from the human gastric mucosa. J Med Microbiol 1985;19:257-67.

9. Tytgat GNU, Lee A, Graham DJ. The role of infectious agents in peptic ulcer disease. Gastroenterol Int 1993; 6:127-39.

10. Lind T, van Zanteen SV. Eradication of Helicobactor pylori using one-week triple therapies combining omeprazolewith two antimicrobials: The MACH study. Helicobactor 1996;1:138-44.

11. Mannes GA, Bayerdorffer E, Hochter W. Decreased relapse rate after antibacterial treatment of H. pylori associated duodenal ulcer. Munich duodenal ulcer trial. Eur J Gasterenterol Hepatol 1993;5:145-53.

12. Reinbach DH, Cruckshank G, McColl KEL. Acute perforated duodenal ulcer is not associated with H. pylori infection. Gut 1993;34:1344-7.

13. Hosking DW, Yung MY, Chung SC. Differing prevalence oh H. pylori in bleeding and nonbleeding ulcer. Gastroenterology 1992;102:A85.

14. Sebastian M, Chandram VP, Elashaal IH. H. pylori infection in perforated peptic ulcer disease. Br J Surg 1995;82:360-2.

15. Mihmanli M, Isgor A, Kabukcuoglu F. The effect of H. pylori in perforation of duodenal ulcer. Hepatogastroenterol 1998;45:1610-2.

16. Graham RR. The treatment of perforated duodenal ulcer. Surg Gynecol Obstet 1937;64:235.

17. Hay JM. Mediate definitive surgery for perforated duodenal ulcer does not increase operative mortality: A prospective control trial. World J Surg 1998;12:705-9.

18. Desai HG. Dental plaque:A permanent reservoir of H. pylori.Scand J Gastroenterol 1991;26:1205-8.

19. Majumdar P, Shah SM, Dhujibhoy KR et al. Isolation of H. pylori from dental plaque in healthy volunteer. Indian J Gastroenterol 1990;9:271-2.

20. Kamat AH, Mehta PR, Natu AA et al. Dental plaque: an unlikely reservoir of Helicobater pylori. Indian J Gastroenterol 1998;17:138-9.

21. Dowsett SA, Kowolik MJ. Oral Helicobacter pylori: Can we stomach it? Crit Rev Oral Biol Med 2003;14:226-33.

22. Riggio MP, Lennon A. Identification by PCR of Helicobacter pylori in subgingival plaque of adult periodontitis patients. J Med Microbiol 1999;48:317-22.

23. Dye BA, Kruszon-Moran D, McQuillan G. The relationship between periodontal disease attributes and Helicobacter pylori infection among adults in the United States. Am J Public Health 2002;92:1809-15.

24. Umeda M, Kobayashi H, Takeuchi Y et al. High prevalence of Helicobacter pylori detected by PCR in the oral cavities of periodontitis patients. J Periodontol 2003;74:129-34.