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Evaluation of *Helicobacter pylori* infection and other risk factors in patients with benign peptic ulcer disease

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ABSTRACT

Objective: To assess and compare the risk factors in patients with benign gastric and duodenal ulcers and to correlate the prevalence of *Helicobacter pylori* (*H. pylori*) infection in benign peptic ulcer disease. **Methods:** A total of 30 consecutive patients with peptic ulcer disease were included in this study after upper gastrointestinal endoscopy. Their clinical profile and endoscopic findings were noted. Antral biopsies were subjected to histopathological examination and urease test for detection of *H. pylori*. Results were correlated. The study was cleared by the Institute Research Council and the Ethics committee. **Results:** The male: female ratio was 11:4. Overall, *H. pylori* infection was prevalent in 93.3% of the patients. Patients who took spicy food had a significantly higher rate of *H. pylori* positivity ($P=0.04$). Smoking, alcohol intake and NSAIDs did not affect *H. pylori* status in patients. There was no significant association between the site of the ulcer and *H. pylori* infection. **Conclusions:** Based on our observations we conclude that prevalence of *H. pylori* infection is similar in duodenal and gastric ulcers and intake of spicy food is a significant risk factor.

1. Introduction

Infection with *Helicobacter pylori* (*H. pylori*), a veritable pandemic, has emerged as one of the most prevalent infections all over the world. Although *H. pylori* infection is invariably associated with an inflammatory response, peptic ulcers and gastric malignancy are seen only in a subset of individuals with chronic infection. Presumably, both bacterial and host factors contribute to this differential response. The association of peptic ulcer with factors like smoking, NSAID usage and alcohol intake has been widely investigated.

According to a recent study in Verona, Italy, NSAID usage was significantly associated with an increased risk of duodenal ulcer^[1]. Researchers in Turkey have found that the presence of *H. pylori*, aspirin, alcohol and NSAIDs act as independent risk factors for duodenal ulcer^[2]. Singh *et al.* from New Delhi investigated factors influencing *H. pylori* eradication and ulcer healing^[3]. Higher body mass index and per-capita income were positive factors, while diet had no role in eradication of infection or ulcer healing^[3]. Thus, continued studies and research enable us to understand the role of *H. pylori* in peptic ulcer disease better.

This study was conducted with an aim to assess and compare the risk factors for benign gastric and duodenal ulcers, and to correlate the prevalence of *H. pylori* in benign gastric and duodenal ulcer disease.

2. Materials and methods

Consecutive patients diagnosed to have peptic ulcer disease during upper gastrointestinal endoscopy (UGIE) over a period of 6 months, were included in this prospective study. After obtaining an informed consent, clinical profile including the dietary habits was noted. Site and number of ulcers were also noted. For the diagnosis of *H. pylori* infection, four antral biopsies were taken for all patients. Two samples were sent for histopathological examination by Giemsa stain and the other two were subjected for urease test. Urease test was done with a solution prepared and standardized in our institute^[4]. Urease test result was noted at one hour and 24 hours for change in colour. A colour change from yellow to pink was considered as positive. A patient was declared positive for *H. pylori* if one or both of the tests were positive and, negative if both tests were negative.

The software used for statistical analysis was Graphpad Instat version 3.0. Contingency tables were made to test for differences in proportion and *Chi Square* test and Fisher's Exact test were done to test for significance. A "*P*" value of 0.05 was taken to be statistically significant.

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The study was cleared by the Institute Research Council and the Ethics committee.

3. Results

A total of 30 consecutive patients with peptic ulcer disease as diagnosed on endoscopy were included in the study. Out of these, 25 were between 20–60 years of age. 3 patients were younger than 20 years and 2 patients were older than 60 years of age, with the median age of the patients being 36 years old. The male: female ratio was 11:4. *H. pylori* status was similar in all the age groups, ranging from 90% to 100%. Although patients with gastric ulcer had a high rate of positivity for *H. pylori* (100%) as compared with patients with duodenal ulcer (87.7%), the difference was not significant ($P=0.57$). The overall prevalence of *H. pylori* in patients with peptic ulcer was 93.8%.

Smoking, alcohol intake and intake of non-steroidal anti-inflammatory drugs (NSAIDs) did not affect *H. pylori* status in patients with peptic ulcer (Table 1). However, when *H. pylori* status in peptic ulcer was correlated with dietary habits, it was found that patients who took spicy food had a significantly higher *H. pylori* positivity compared with those who took non-spicy food ($P=0.04$, Table 1). *H. pylori* status did not correlate with the site of ulcer (Table 2).

Table 1

Correlation of *H. pylori* status and risk factors for patients with peptic ulcer.

Risk factors	n (%)	<i>H. pylori</i> ⁺ [n (%)]	P value (CI)	RR
Smokers	16 (53.4)	15 (93.75)	1.00 (0.83 – 1.22)	1.01
Nonsmokers	14 (46.6)	13 (92.85)		
Alcohol intake +	14 (46.6)	13 (92.85)	1.00 (0.81–1.20)	0.99
Alcohol intake –	16 (53.4)	15 (93.75)		
Spicy diet	23 (76.6)	23 (100.00)	0.04 (0.88 – 2.23)	1.40
Nonspicy diet	07 (23.4)	05 (71.00)		
NSAID intake +	13 (43.4)	12 (92.30)	1.00 (0.80 – 1.18)	0.98
NSAID intake –	17 (56.6)	16 (94.10)		

CI: Confidence interval; RR: Risk ratio.

Table 2

Correlation of *H. pylori* status and site of ulcer.

Site	Number*	<i>H. pylori</i> ⁺ [n (%)]
Stomach	8	8 (100.0)
Pylorus	16	6 (100.0)
Duodenum 1	16	14 (87.5)
Duodenum 2	9	8 (88.0)

* Some patients had ulcer at more than one site.

4. Discussion

Reports in literature have shown that peptic ulcer is a multi-factorial disease. Studies in the last few years have suggested an important role for *H. pylori* infection in the pathogenesis of peptic ulcer disease[4]. Influence of other factors might explain the discrepancy between the high prevalence of *H. pylori* infection of more than 50% in the general population and a low overall life-time occurrence of duodenal ulcer disease of about 10%. Prevalence of peptic ulcer disease was markedly higher in dyspeptic patients who were *H. pylori* positive[5]. Researchers in England

demonstrated that aspirin thromboprophylaxis increased the risk of peptic ulcer and gastro-intestinal bleed in hospitals[6]. Hollenz *et al.* found that primary care patients frequently develop ulcers while on NSAIDs[7].

In the present study, the majority of patients were smokers. History of alcohol and NSAID intake was present only in few patients. However, no correlation was seen with *H. pylori* status and smoking, alcohol and NSAID intake. On the contrary, Konturek *et al.* reported that NSAID use and smoking played a major role in the pathogenesis of peptic ulcer[8]. They reported a negative interaction between *H. pylori* and NSAID use on duodenal ulcers suggesting that *H. pylori* reduces the development of these ulcers in NSAID users.

In a recent report from Poland, male sex, smoking and presence of *H. pylori* were found to be significant risk factors for the development of duodenal ulcer[9]. According to a study from Mysore, India, chilli and its active principle, capsaicin, is not the cause of peptic ulceration, but a benefactor. It stimulates alkaline mucus secretion and gastric mucosal flow[10]. In the present study we found that patients who took spicy food had a significantly higher *H. pylori* status compared to patients who took non-spicy food.

Based on our observations we conclude that prevalence of *H. pylori* infection is similar in duodenal and gastric ulcers and intake of spicy food is a significant risk factor.

Conflict of interest statement

We declare that we have no conflict of interest.

References

- [1] Talamini G, Tommasi M, Amadei V, Pajusco B, Fratucello A, Lechi A, et al. Risk factors of peptic ulcer in 4 943 inpatients. *J Clin Gastroenterol* 2008; **42**: 373–380.
- [2] Salih BA, Abasiyanik MF, Bayyurt N, Sander E. *H. pylori* infection and other risk factors associated with peptic ulcers in Turkish patients: a retrospective study. *World J Gastroenterol* 2007; **13**: 3245–3248.
- [3] Singh N, Deb R, Kashyap PC, Bhatia V, Ahuja V, Sharma MP. Body mass index and per capita income influence duodenal ulcer healing and *H. pylori* eradication whilst dietary factors play no part. *Trop Gastroenterol* 2008; **29**: 26–31.
- [4] Jones VS, Kate V, Ananthakrishnan N, Badrinath S, Amarnath SK, Ratnakar C. Standardization of urease test for detection of *Helicobacter pylori*. *Indian J Med Microbiol* 1997; **15**: 181–183.
- [5] Wu HC, Tao BG, Wu WM, Gao Y, Xu QQ, Zhao K. Prevalence of peptic ulcer in dyspeptic patients and the influence of age, sex, and helicobacter pylori infection. *Dig Dis Sci* 2008; **53**: 2650–2656.
- [6] Madhusudhan TR, Rangan A, Gregg PJ. Gastric protection and gastrointestinal bleeding with aspirin thromboprophylaxis in hip and knee joint replacements. *Ann R Coll Surg Engl* 2008; **90**: 332–335.
- [7] Hollenz M, Stolte M, Leodolter A, Labenz J. NSAID-associated dyspepsia and ulcers: a prospective cohort study in primary care. *Dig Dis* 2006; **24**: 189–194.
- [8] Konturek SI, Bielanski W, Plonka M, Pawlik T, Pepera J, Konturek PC, et al. *Helicobacter pylori*, non-steroidal anti-inflammatory drugs and smoking in risk pattern of gastroduodenal ulcers. *Scand J Gastroenterol* 2003; **38**: 923–930.
- [9] Regula J, Hennig E, Burzyłowski T, Orłowska J, Przytułski K, Polkowski M, et al. Multivariate analysis of risk factors for development of duodenal ulcer in *Helicobacter pylori*-infected patients. *Digestion* 2003; **67**: 25–31.
- [10] Satyanarayana MN. Capsaicin and gastric ulcers. *Crit Rev Food Sci Nutr* 2006; **46**: 275–328.