Original Article

Comparison of demographic, clinical and endoscopic characteristics of peptic ulcer due to Helicobacter Pylori and NSAIDs

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Background: *Helicobacter Pylori (H.P) and Non Steroidal Anti inflammatory Drugs (NSAID) are the most common cause of peptic ulcer Disease (PUD). This study was designed to identify endoscopic, clinical and demographic features of patients with H.P positive in comparison with NSAID related PUD.*

Methods: *From December 2004 to March 2008, 5885 patients underwent upper GI Tract endoscopy in a large referral Teaching Hospital of Babol Medical University. All patients with endoscopic diagnosis of PUD were enrolled in the study. H.P positive patients were assigned as group A and NSAID user as group B. Then their clinical, demographic, endoscopic characteristics were compared.*

Results: *A total of 749 (12.3%) patients diagnosed as Duodenal Ulcer (DU), among them 600 subjects were eligible for analysis. Three hundred thirty one (55.2%) were H.P positive, 80 (13.3%) were NSAID user and 189 (31.5%) had non H.P non NSAID related ulcer. Two hundred ninety seven patients diagnosed having Gastric Ulcer (G.U), 143 (48.2%) were H.P positive, 46 (15.5%) were NSAID user and 108 (36.3%) had non H.Pylori, non NSAID related ulcer. Mean age of patients with G.U was 54.2±17 and D.U was 46.9±17.5 years ($p<0.04$).*

Anterior wall of bulb and antrum were the most common site of patients with D.U and G.U respectively.

Conclusion: *This study shows non H.Pylori non NSAID related ulcers comprise a significant portion of peptic ulcer disease in north of Iran.*

Key words: *Peptic ulcer, H.Pylori, NSAID, Endoscopy.*

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The main causes of peptic ulcer disease (PUD) are helicobacter pylori (HP) infection and the use of Non Steroidal Anti Inflammatory Drugs (NSAIDs)(1), but the factors that make patients prone to ulcer have not been completely recognized yet. HP infection as an important cause of PUD is approximately always associated with active chronic gastritis, but only 10- 15% of infected patients have obvious peptic ulcer and the main cause of this difference is unrecognized (1). In recent years many studies suggested a significant proportion of PUD was not related to these two risk factors (2) there are variations in the reported rate of non H pylori non NSAID related ulcer (3). Differences in demographic and endoscopic characteristics of the ulcers including ulcer location, its size and associated findings such as simultaneous bleeding have not been assessed completely in these patients (4). NSAIDs are one of the most used drugs in clinics and its common important complications are dyspepsia and peptic ulcers. Approximately, 30 million people in the world use NSAIDs daily (5). Recent studies show some difference but some research showed no differences in clinical, endoscopic and demographic features between ulcers due to HP and NSAIDs. Definitely, because of the difference in leading mechanism of ulcers, there may be some differences in endoscopic features, ulcer place and their demographic and clinical manifestations which can be helpful in progression of treatment options and even in the prevention of complications of these ulcers. A study in 2006 revealed that NSAIDs-related ulcers have more bleeding compared with those not related to NSAIDs.

This study from Japan assessed the endoscopic features of gastric ulcer (GU) in HP positive patients and NSAIDs users and confirmed that multiple ulcers are more common among the NSAIDs users. Hemorrhagic ulcer was reported to be higher in this group (6). Another study from Australia in 2000, investigated endoscopic and demographic data in ulcers with and without HP infection. In this study, the average size and number of ulcers did not differ statistically between the two groups (7). Our study was designed to investigate the endoscopic, demographic and clinical features in two groups of patients with HP infection and NSAIDs consumption.

Methods

In this case-control study, between december 2004 through march 2008 all consecutive patients with gastrointestinal complaints underwent endoscopy in Beheshti General Hospital of Babol Medical University. Endoscopic features of ulcers such as location and number, demographic data including age, sex and history of smoking were studied then these data were compared among patients with histologically documented HP infection and patients with the history of preceding 4 weeks NSAIDs consumption before endoscopy. HP infection was confirmed with 2 sample biopsies from the antrum and gastric body then H&E staining was performed for histology study. Patients with history of gastritis but without HP infection and those whose pathological results were compatible with malignancy were excluded. Patients who were current smokers and smoked for at least 6 months were considered as smokers. NSAIDs user is defined as one who had a permanent use it NSAIDs or aspirin in preceding one month. GUs with a history of NSAIDs consumption and without considering HP infection status was defined as NSAIDs-related GU (7). In general, the patients were divided into two groups, group A, positive for HP infection and group B, NSAIDs-related PUD.

The groups were compared using the Student's t- test for continuous variables and the chi-square test (or fisher's exact test if required) for categorical variables. The statistical significance was based on two-sided design-based tests evaluated at the 0.05 level of significance. All the statistical analyses were performed using SPSS version 13 (SPSS Inc, Chicago, IL, USA).

Results

Between December 2004 and March 2008, 5885 cases of endoscopy were performed in the Endoscopy Center of Beheshti Hospital of Babol medical University Among them, 749(12.7%) cases had DU, 149 cases did not have pathological documents for HP status, but 600 cases had documented their pathological evidences for existence or inexistence of HP infection. Among the 600 patients, 331 (55.2%) were positive for HP, 80 (13.3%) had a history of NSAIDs consumption. While 189 (31.5%) did not have either HP infection or NSAIDs consumption.

Patients with duodenal ulcer: The demographic, clinical and endoscopic data of patients with DU are summarized in table 1. About 22% of those patients who were both NSAIDs users and HP positive smoked which was significantly more than H.P patients ($p<0.04$). Frequency of gastrointestinal bleeding (GIB) did not show a significant difference between these two groups.

Table 1. Demographic, clinical and endoscopic features of patients with duodenal ulcer in each group.

Variables	Group A (HP positive) (n=331)	Group B (NSAIDs users) (n=80)
Age (year)	45.11±16.66	46.39±17.12
Male gender (%)	59.9	55.6
Smoking (%)	12.3	10.0
Number of ulcers		
I	79.4	77.6
II	9.2	15.8
III	11.4	6.6
Place of ulcer		
Anterior	45.7	48.7
Posterior	7.5	10.3
Upper	16.2	15.4
Lower	16.8	17.9
Different	13.9	7.7

p= Nonsignificant

HP= helicobacter pillory, NSAID= non-steroids anti-inflammatory drugs

Patients with gastric ulcer: 297 patients with the diagnosis of GU were enrolled and assessed in this study. Among them, 143 (48.1%) were in group A, 46 (15.5%) patients were in group B and the other 108 subjects (36.4) were in non-NSAIDs non-HP group. Table 2 shows the

demographic, clinical and endoscopic data of patients with GU in each group. GU patients who were both HP positive and NSAIDs user had less number of ulcers (9.8%) compared to the Nasaid induced ulcer (37.9%) ($p<0.015$). In general the data of all patients with PUD (DU+GU) 897 patients were investigated. About 60% and 52.3% of patients in groups A and B were males, respectively. 228 patients (25.4%) were presented with upper GIB. The prevalence of DU in group A (HP positive) was higher than group B ($p<0.03$).

Table 2. Demographic, clinical and endoscopic data of patients with GU in each group.

Variables	Group A (HP positive) (n=143)	Group B (NSAIDs users) (n=46)
Age (years)	57.7±16.18	53.23±15.94
Male gender (%)	60.3	47
Smoking (%)	6.4	13.0
GIB (%)	17	16
Number of ulcers (%)		
I	76.7	80.4
II	9.5	6.5
III	13.8	13
Place of ulcer (%)		
Proximal	11.2	8.7
Body	28.4	23.9
Antrum	54.3	56.5
multiple	6.0	10.9

p= Nonsignificant

GIB= Gastrointestinal bleeding

Discussion

In this study, the mean age of patients with GU was more than those with DU (54.18±16.83 vs 46.97±17.56, respectively, $p<0.04$). Moreover, the prevalence of HP and NSAIDs users were 55.2% and 13.3% respectively in patients with DU, while 48.1% and 15.5% were among those with GU. A study in Australia revealed that the prevalence of HP infection and NSAIDs consumption is about 56% and 22% (8) and another study in Hong Kong reported these values for HP and NSAIDs to be 66% and 8.5% respectively (9). Our study showed GU was more prevalent in men ($p<0.001$). In this study, a significant difference was not seen between groups A and B considering the number of patients presented with upper GIB, but in a study in the USA in 1996, the prevalence of upper GIB was significantly more among the NSAIDs users (10). About 40% of patients with NSAIDs-related ulcer did not report their use (11) On the higher rate of non HP, non NSAID ulcer. Regarding the prevalence of smoking in our study, there was not a significant difference between the two groups, but a study among the Japanese Americans showed that smoking raised the risk of ulcer (12). Most of the GU in HP positive group (54.3%) and group B (%56.6) were placed in the antrum. A study in the USA in 1996 also demonstrated that most of the ulcers in HP positive group are in lesser curvature (85%) but it was mentioned if they mean it in antrum (10). In a Japanese study in 2006, 56% of these ulcers were also located in antrum (6).

Most of the DUs in both groups A and B were placed in anterior wall of bulb and there was no difference between the two groups. In other mentioned studies, the place of DUs was not clearly defined. In our study, the number of multiple GUs in HP positive and NSAIDs user patients (9.8%) were statistically less than the NSAID user group (37.9%) $p<0.05$. This was in consistence with the Japanese study (48% vs. 96%, $p<0.001$). This implies the protective effects of HP infection in NSAID-related ulcers (9). Our study showed a protective effect of H.P infection for NSAID induced peptic ulcer.

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