Peptic ulcers and oral health status

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Abstract

Purpose: *Helicobacter pylori* infection plays a crucial role in pathogenesis of peptic ulcers; however, among infected individuals only a small percentage will develop peptic ulcers at any time during their life. This low virulence suggests that many additional factors beside *H. pylori* are implicated in pathogenesis of the disease. The aim of the study was to determine whether there is a relationship between the prevalence of peptic ulcers and oral health status.

Material and methods: The evaluation of dental status was performed in *H. pylori* infected population. The study involved 93 peptic ulcer patients (77 duodenal ulcer, 16 gastric ulcer) with ulcer niche not related to non-steroidal anti-inflammatory drugs (NSAIDs) consumption and 93 gender and age matched dyspeptic controls. *H. pylori* infection was determined in endoscopically taken slices from gastric mucosa with two methods (CLO-test and histology).

Results: Both in duodenal and gastric ulcer patients, the number of filled teeth was lower and debris index was higher than in controls, the number of decayed teeth was also higher in gastric ulcer patients. The number of natural teeth, number and type of prosthetic restorations, as well as the periodontal index, did not differ between the ulcer and control groups. Poor oral health in patients with peptic ulcers corresponded with education level, smoking habit, and visits to the dentist.

Conclusions: Poor oral health is associated with the prevalence of peptic ulcers not related to NSAIDs consumption, but it appears doubtful that it is a significant pathogenetic factor in ulcer disease.

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Introduction

Half of the world population is infected with *H. pylori*; however, a strong pathogenetic association of *H. pylori* infection was only found with peptic ulcer disease. Since only a small part of the infected population develops peptic ulcers [1], the presence of *H. pylori* infection alone is inadequate as the sole etiological agent to duodenal and gastric ulcer development. This means that in the pathogenesis of this disease, additional factors must be involved.

Former studies indicated that *H. pylori* can colonize not only the stomach, but also the oral cavity; a strain has been isolated from dental plaque and its implication in stomach infection has been proven [2-4]. Poor oral health predisposes patients to more frequent infection of the stomach and makes elimination more difficult during eradication therapy [5-7]. Whether other factors than oral infection with *H. pylori* implicate the oral cavity to peptic ulcer development remains to be established. Although significant consideration has been given to the link between oral health and peptic ulcer disease, this association has been suggested but never confirmed. Therefore, the aim of the study was to determine whether there is association between the prevalence of peptic ulcers and oral health status in the *H. pylori*-infected population.

Material and methods

The study was performed in 186 *H. pylori* infected subjects. Among them were 77 duodenal ulcer and 16 gastric ulcer patients with ulcer niche not related to consumption of non-steroidal anti-inflammatory drugs (NSAIDs) and 93 gender and age matched dyspeptic controls without ulcer history and normal upper GI endoscopy (*Tab. 1* and *2*). All participants

Table 1. **Data of non-NSAIDs-related duodenal ulcer patients and controls**

	Duodenal ulcer	Control	
N	77	77	
Age	48.0±14.5	48.2±14.2	
Gender (M/F)	58/19	58/19	
Education (E,J/ S,U)	43/34	31/46	
Smokers	49	32	p=0.0096
Dentist visits			
(regular/ non-regular)	11/66	31/46	p=0.0005
Number of teeth	16.6±10.0	18.8±8.7	
Number of decayed teeth	4.4±4.0	3.1±3.0	
Number of filled teeth	3.2±4.0	5.7±4.2	p=0.0000
Plaque index	1.5±0.8	1.1±0.8	p=0.0076
Periodontal index	1.7±2.0	1.2±1.3	
Denture wearers	36	44	
Fixed	7	15	
Removable	22	19	
Fixed and removable	7	10	
Total time of daily toothbrushing			
0 min	23	14	
less than 2 min	12	12	
2 min or more	37	47	

Education level consists of elementary school (E), junior high school (J), senior high school (S), and university (U)

Table 2. **Data of non-NSAIDs-related gastric ulcer patients and controls**

	Gastric ulcer	Control	
N	16	16	
Age	57.8±14.1	57.9±13.8	
Gender (M/F)	8/8	8/8	
Education (E,J/ S,U)	11/5	6/10	
Smokers	9	4	
Dentist visits			
(regular/ non-regular)	1/15	5/11	
Number of teeth	12.5±9.3	13.7±9.4	
Number of decayed teeth	6.2±4.8	2.0±1.4	p=0.027
Number of filled teeth	1.3±4.8	4.9±4.4	p=0.032
Plaque index	1.9±0.9	0.8±0.7	p=0.003
Periodontal index	2.5±2.4	1.4±2.0	
Denture wearers	6/16	9/16	
Fixed	1	3	
Removable	4	6	
Fixed and removable	1	0	
Total time of daily toothbrushing			
0 min	5	4	
less than 2 min	3	0	
2 min or more	5	10	

underwent endoscopy with GIF V2 gastroscope (Olympus) during which slices of gastric mucosa from prepyloric and corpus regions were taken; two slices from each side for histological examination, one for CLO-test. *Helicobacter pylori* infection in the stomach was determined using two methods, rapid urease test (CLO-test) and histological examination. Urease test was prepared in the Department of Physiology Medical University of Białystok according to the method of Marshall et al. [8]. The result of the test was considered positive if during incubation at room temperature it changed color from orange to pink; positive and negative controls were performed each day. Specimens for histological examination were placed in formalin, submitted to standard processing, and then stained with hematoxylin-eosin and Giemsa. One experienced pathomorphologist made microscopic assessment. Only subjects with two positive tests for *H. pylori* were included.

Demographic data, oral hygiene practices, and dentist visits were based on questionnaire fulfilled by all subjects before oral examination. The dentist visits were classified as regular if taken at least once a year.

Oral examination involved the evaluation of natural dentition (number of natural teeth, carious teeth, filled teeth, plaque index, periodontal index) [9,10], and specification of dental prosthesis (fixed, removable).

The results (means ±S.D.) were evaluated statistically with Mann-Whitney U-test, χ^2 test, and Fisher exact test, as appropriate. A p value of less than 0.05 was accepted as statistically significant.

The study was approved by the Local Ethical Committee and informed consent was obtained from all participants before study began.

Results

Both in the gastric and duodenal ulcer patients the number of filled teeth was lower and debris index was higher than in controls (*Tab. 1*); additionally, in gastric ulcer patients, the number of decayed teeth was higher (*Tab. 2*). If the two ulcer groups were pooled, they exhibited lower educational level and less regular visits at the dentist than pooled control groups (*Tab. 3*). The number of natural teeth, number and type of prosthetic restorations, periodontal index, as well as total time of daily toothbrushing did not differ between compared groups. Moreover, most peptic ulcer patients, unlike controls, were smokers.

Discussion

The infection of the stomach with *H. pylori* appears to be the most important factor in pathogenesis of peptic ulcer disease. However, many factors in addition to *H. pylori* infection predispose patients to peptic ulcer development, among them are family history, male gender, strenuous work, tobacco smoking, low educational level and low socio-economic status [11-15]. Since stomach infection with *H. pylori* is the main, but not the only, cause of peptic ulcer development, we have attempted to evaluate the role of other potentially important pathogenetic factors of this disease. The aim of the study was to answer the question whether there is association between the prevalence of peptic ulcers and oral health. Assuming that peptic ulcers located both in the stomach and duodenum may sometimes be a side effect of NSAIDs, the present study has been designed to include only patients with peptic ulcers unrelated to NSAIDs consumption. Moreover, since dental status depends upon gender and age, these factors were taken into account in recruitment.

***Table 3*. Data of non-NSAIDs-related peptic ulcer patients and controls**

	Peptic ulcer	Control	
N	93	93	
Age	49.6±14.8	49.9±14.5	
Gender (M/F)	66/27	66/27	
Education (E,J/ S,U)	54/39	37/56	p=0.018
Smokers	59	36	p=0.013
Dentist visits (regular/ non-regular)	12/81	36/57	p=0.0001
Number of teeth	15.9±9.4	17.9±9.0	
Number of decayed teeth	4.7±4.2	2.9±2.8	p=0.0066
Number of filled teeth	2.9±3.8	5.6±4.2	p=0.0000
Plaque index	1.5±0.8	1.1±0.8	p=0.0003
Periodontal index	1.8±2.1	1.2±1.4	
Denture wearers	42	53	
Fixed	8	18	
Removable	26	25	
Fixed and removable	8	10	
Total time of daily toothbrushing			
0 min	29	18	
less than 2 min	15	12	
2 min or more	42	57	

The results of this study have shown that oral health status in *H. pylori* infected subjects with peptic ulcer unrelated to NSAIDs is worse than in controls, and a number of oral health factors could be the cause of this effect. Poor dental status in patients with peptic ulcer unrelated to NSAIDs may be the consequence of less care of oral health. Rare visits to the dentist and short time of daily brushing natural teeth may lead to dental plaque accumulation, and finally to dental decay responsible for early tooth loss. It is of note that patients with gastric and duodenal ulcers were of lower education level than controls and thus quite likely of lower economic status; these both may limit markedly the means and possibility of keeping up standards of oral health care. Moreover, most patients with peptic ulcers were smokers, and this factor may have additional contribution to poor oral heath [20].

Poor dental status may be a consequence of a restricted diet. It is possible that the dietary habits of the ulcer population were different from the general population, and shifts in food selection patterns with insufficient intakes of some nutrients and vitamins might be the cause of dental invalidity [16]. An argument for this is an increase of patient body weight within one year after ulcer healing [17].

On the other hand, poor dental status may be considered as a possible cause of peptic ulcers. Less effective mastication may lead to swallow of food particles of relatively large size which reside in the stomach much longer than well masticated [18,19]; a delayed stomach emptying predisposes to gastritis, and finally to disturbed gastric acid secretion, a crucial pathogenetic factor in peptic ulcer development. The unanswered question remains whether impaired mastication could promote gastric and duodenal ulcer in the same way, as it is known that pathogenesis of these two entities differs at many points.

The limitation of the study is that the control group consisted of dyspeptic patients but not of healthy subjects; however, only this model was possible to be accepted from ethical point of view. Despite limitations, the obtained results confirm the suggestion that poor oral health is associated with the prevalence of peptic ulcers unrelated to NSAIDs, but this association in *H. pylori* infected population is rather incidental.

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