# Original Article Relation between periodontitis and helicobacter pylori infection

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Abstract: Objective: The correlation between periodontitis and *Helicobacter pylori* (*H. pylori*) infection in the mouth was analyzed. Method: 70 elderly patients with periodontitis treated at our hospital from January 2013 to December 2014 were recruited. Dental plaques and gargle were collected for *H. pylori* detection using PCR technique. Periodontal health status of the patients was recorded. 70 control cases with healthy periodontium were also included. The symptoms of *H. pylori* infection in the mouth were compared between the two groups, and the results were analyzed statistically. Results: The positive rate of urease C gene of *H. pylori* in the periodontitis group was 71.4%; the positive rate of cagA gene was 35.7%. The positive rate of urease C gene of *H. pylori* in the control group was 34.3% and that of cagA gene was 12.9%. The two groups did not show significant differences in these two indicators (P<0.05). The positive detection rate of urease C gene of *H. pylori* in subgingival plaques was higher than that in supragingival plaques, and the difference was of statistical significance (P<0.05). The positive detection rate of *H. pylori* in patients with moderate and severe periodontitis was obviously higher than that of patients with mild periodontitis (P<0.05). Conclusion: Periodontal health status of elderly people with periodontitis correlated with *H. pylori* infection in the stomach.

Keywords: Periodontitis, Helicobacter, pylorus, stomach, elderly people

# Introduction

Periodontal disease is a common oral disease and also one of the major causes of tooth decay in adults, especially in elderly people [1, 2]. Periodontal disease usually occurs to tooth supporting tissues, including the gum disease that only affects the gum and periodontitis that affects periodontal tissues (periodontal membrane, alveolar bone and cementum). Early symptoms of periodontal disease can be easily ignored, thereby leading to chronic, recurrent infection of periodontal tissues, which impairs the chewing function and poses threat to people's health. Helicobacter pylori (H. pylori) is a helical-shaped, microaerophilic Gram-negative bacillus, which has a rigorous requirement for growth condition [3, 4]. H. pylori was first isolated from gastric mucosal biopsy in patients with active chronic gastritis in 1983. It is believed that H. pylori infection is the main cause of chronic gastritis, with an infection rate of 80%-95% in active chronic gastritis [5, 6]. H. pylori infection is also associated with chronic gastritis, peptic ulcer, gastric cancer, mucosa associated lymphadenoma, gastroesophageal reflux disease and functional dyspepsia. The correlation between H. pylori infection and periodontal disease has attracted growing attention. Most studies are concerned with the pathogenicity of dental plaques [7-9], but less concerned with the correlation between periodontal health status and H. pylori infection. Zhu et al. [10] believed that H. pylori in the stomach may be involved pathogenically in chronic periodontal disease. We detected H. pylori infection in the mouth and stomach of elderly people with periodontitis and discussed the correlation between periodontal health status and *H. pylori* infection in the stomach.

#### Data and methods

## General data

70 elderly patients with chronic periodontitis treated at our hospital from January 2013 to December 2014 were recruited, including 43

**Table 1.** Comparison in PCR detection results of Hp between two groups

| Group             | N  | Urease C gene   |                 | cagA gene       |                 |
|-------------------|----|-----------------|-----------------|-----------------|-----------------|
|                   |    | Positive (n, %) | Negative (n, %) | Positive (n, %) | Negative (n, %) |
| 0                 | 70 |                 |                 | ( , ,           |                 |
| Control group     | 70 | 24 (34.3)       | 46 (65.7)       | 9 (12.9)        | 61 (87.1)       |
| Observation group | 70 | 50 (71.4)       | 20 (28.6)       | 25 (35.7)       | 45 (64.3)       |
| Р                 |    | <0.001          |                 | <0.001          |                 |

**Table 2.** Detection of Hp in subgingival plaques and supragingival plaques

| Group             | Ν  | Distribution          | Urease C<br>gene | cagA<br>gene    |  |
|-------------------|----|-----------------------|------------------|-----------------|--|
|                   |    |                       | Positive (n, %)  | Positive (n, %) |  |
| Control group     | 70 | Supragingival plaques | 6 (8.6)          | 4 (5.7)         |  |
|                   |    | Subgingival plaques   | 18 (18.6)*       | 5 (7.1)         |  |
| Observation group | 70 | Supragingival plaques | 21 (30.0)        | 11 (15.7)       |  |
|                   |    | Subgingival plaques   | 29 (41.4)*,#     | 14 (20.0)       |  |
|                   |    |                       |                  |                 |  |

<sup>\*</sup>Compared to supragingival group, P<0.05; \*Compared to control group, P<0.05.

males and 27 females aged  $(63.4\pm1.2)$  years old. The course of disease was 4-33  $(14.2\pm4.2)$  months, and 33, 30 and 7 cases were classified as mild, moderate and severe periodontitis, respectively. 70 control cases with healthy periodontium were selected, including 44 males and 26 females aged  $(64.1\pm2.3)$  years old. Two groups did not show significant differences in gender and age (P>0.05).

#### Method

Diagnosis was made according to the diagnostic criteria in *Periodontology*.

## Inclusion criteria

No intake of antibiotics, H2-receptor antagonist, antacids, anti-inflammatory agents or use of gargle within 1 months before sampling; receiving no periodontal treatment within 6 months; no history of stomach surgery.

#### Detection method and indicators

Oral cleaning was performed within 12 h before sampling. Gingival crevice bacteria were collected in near middle of mandibular first molar along with gargle. Probing depth, plaque index (PI) and bleeding index (BI) were measured for the corresponding teeth. All samples were col-

lected by the same physician using the method described in literature. PCR amplification was performed following DNA extraction of the samples.

# Statistical analysis

Statistical analyses were done using SPSS18.0 software. The ratios were compared using  $\times 2$  test and  $\times 2$  statistics was adjusted. Measurement data conforming to normal distribution were expressed as M  $\pm$  SD. P<0.05 for t test was considered as statistically significant.

#### Results

Detection of H. pylori by PCR

As shown in **Table 1**, the positive rates of urease C gene and cagA gene of the periodontitis

group were significantly higher than those of the control group (P<0.05).

Detection of H. pylori in supragingival plaques and subgingival plaques in cases with periodontitis

The positive detection rate of *H. pylori* in subgingival plaques in cases with periodontitis was obviously higher than that in supragingival plaques (P<0.05); the positive detection rate of *H. pylori* in cases with periodontitis was significantly higher than that in the controls (P<0.05). See **Table 2**.

Comparison of positive detection rate of H. pylori in cases with different severity of periodontitis

As shown in **Table 3**, the positive detection rate of *H. pylori* in cases with moderate and severe periodontitis was considerably higher than in those with mild periodontitis (P<0.05).

# Discussion

Periodontal disease poses great threat to the health of teeth and overall health. The typical manifestations of periodontal disease include gingival inflammation, bleeding, periodontal pocket formation, alveolar bone resorption,

**Table 3.** Positive rate of Hp between mild, moderate, and sever pateints

|          | -  |                    |
|----------|----|--------------------|
| Extent   | N  | Hp positive (n, %) |
| Mild     | 33 | 13 (39.4)          |
| Moderate | 30 | 21 (70.0)          |
| Severe   | 7  | 6 (85.7)           |

reduced alveolar bone height, tooth displacement, agomphosis and masticatory atonia, or even shedding or removal of teeth for severe cases. According to literature report [12], H. pylori is the main pathogen causing gastric ulcer, chronic gastritis or even gastric cancer. Periodontal disease may adversely impact the overall health, especially chronic periodontal disease. We detected H. pylori in the mouth and stomach in elderly people with periodontitis and investigated the correlation between periodontal health status and H. pylori infection. Results indicated that the positive rates of urease C gene and cagA gene of H. pylori in the periodontitis group were significantly higher than those of the control (P<0.05). Thus H. pylori infection correlated with periodontitis.

H. pylori is the pathogenic factor of chronic gastritis, gastric ulcer, duodenal ulcer or even gastric cancer. H. pylori can be detected in the periodontal pockets of patients with these gastric diseases and therefore is considered as the risk factor of periodontitis. The positive detection rate of *H. pylori* in patients with gastric diseases is usually higher than in those without gastric diseases. The average probing depth of patients positive for H. pylori infection is correlated with H. pylori. We showed that the positive detection rate of *H. pylori* in supragingival plagues was higher than that in the subgingival plaques. This indicated that subgingival plaques provide a more favorable environment for the growth of *H. pylori*. Periodontal health status is affected by H. pylori, and further damage of periodontal tissues will occur because of H. pylori infection. H. pylori in the stomach may be involved in the pathogenicity of chronic periodontitis, showing a certain correlation with the latter. In the present study, the positive detection rates of *H. pylori* in patients with moderate and severe periodontitis were significantly higher than in patients with mild periodontitis (P<0.05). Thus H. pylori infection was correlated with severity of periodontitis, probing depth and pathological degree of periodontitis.

To conclude, *H. pylori* infection rate is correlated with the incidence of periodontal disease, producing an impact on depth of periodontal pockets and pathological degree of periodontitis. *H. pylori* infection is related to oral environment and oral hygiene. Localized to oral cavity, *H. pylori* is a risk factor of gastric ulcer and duodenal ulcer and enters gastrointestinal infection via swallowing. Maintaining good oral hygiene and removing oral plaques are important for controlling *H. pylori* infection, gastric diseases and periodontal disease.

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#### Disclosure of conflict of interest

None.

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