

# Association between Gastric Helicobacter Pylori Infection and Periodontal Disease

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## KEY WORDS

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## ABSTRACT

**Statement of Problem:** Colonization of Helicobacter pylori (Hp) in the oral cavity may increase in the presence of the periodontal disease. It has been suggested that one of the possible mechanisms of reinfection is the recolonization from dental plaque.

**Purpose:** The purpose of this study was to determine whether periodontal disease was associated with Hp infection.

**Materials and Method:** Sixty-five consecutive patients (34 males and 31 females) undergoing endoscopy for investigation of dyspepsia were included in this descriptive cross-sectional study. The mean age of the patients was 41.3±13.5 years. Periodontal examination and Rapid Urease Test (RUT) were done for all of the participants. Statistical analysis was done, using Chi-square test.

**Results:** 53.8% of the patients had periodontal disease and 61.5% of them had gastric Hp infection. There was no statistically significant association between periodontal disease and gastric Hp infection ( $p=0.455$ ).

**Conclusion:** This study revealed that the presence of periodontal disease in patients with gastric Hp infection could be an independent status.

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## Introduction

Helicobacter pylori (Hp) is a micro-aerophilic, gram-negative spiral shaped bacterium [1-2]. It is an important gastro-intestinal pathogen associated with gastritis, peptic ulcers and increased risk of gastric carcinoma [1-6]. Gastric infection of Hp can be successfully treated by the use of antibiotics, but unfortunately re-infection of the stomach can be seen after this treatment [1, 6-8].

The oral cavity has been pointed out as a region with great potentiality to harbor Hp and originate re-

infection. It has been the focus of a number of studies [1-4, 7- 8]. As shown by many investigations, it seems that dental plaque has a more important role for harboring Hp [9-12]. Therefore, different gingival clinical states may have an effect on the presence of this bacterium in the dental plaque [7]. Periodontal disease is one of the most common oral diseases and high amounts of plaques are found in affected patients [8]. Gebara et al. provided evidence that patients with periodontal disease show a high prevalence (43%) of Hp in their dental plaque [7]. Also, Umeda et al. sho-

wed that 41.2% of patients who harbored Hp in the stomach or duodenum had periodontal pockets more than 4mm [5]. Bruce et al. in a survey of 4504 participants concluded that periodontal pockets with a depth of 5 mm or more are associated with increased odds of Hp seropositivity [13].

However, investigators such as Annand et al., Chitsazi et al. and Kamat et al. reported that there was no correlation between periodontal disease and Hp infection [6, 14-15].

The occurrence of Hp gastric infection reaches over 90% in developing countries. The purpose of our study was to evaluate the association between periodontal disease and gastric infection with Hp which has not been studied before in our country [3].

### Materials and Method

The present research is a descriptive cross-sectional study. The ethical committee of Medical University of Kerman approved this study. Our research participants were volunteer patients who attended the gastroenterology division of Kerman Medical University (KMU) (Afzalipour hospital). The patients reported dyspeptic symptoms and were candidates for endoscopic examination of the superior digestive tract. Dyspepsia was defined as pain or discomfort in the epigastric region [3].

The patients were selected by convenience sampling and they were over 20 years of age. Exclusion criteria were: 1- The use of antimicrobials, inhibitors of proton pump, H2 blockers and bismuth derivatives within 1 month before the study. 2- Previous upper digestive hemorrhage and gastric cancer. 3- Presence of any underlying systemic disease such as diabetes mellitus, 4- edentulous patients or patients with aggressive periodontitis, and 5- any previous scaling and root planning or periodontal therapy in the last 6 months [1, 5, 13].

First, periodontal examination was done for all the participants. Probing depth was recorded, using a Williams periodontal probe [5]. Patients presenting

more than 3 mm probing depth within at least four sites and exhibiting bleeding on probing were diagnosed as periodontitis, and patients presenting <3 mm probing depth within at least four teeth with or without bleeding were allocated to the normal group [1, 6].

The infection of the stomach was estimated, using Rapid Urease Test (RUT). After the oral examination, endoscopic examination was carried out by Olympus GIF- XQ240 gastro-duodenoscope. By endoscopy, two biopsy specimens were obtained from the anterior and posterior surfaces of the antrum, and then RUT test was carried out on these samples [6, 10]. The samples were placed into a RUT kit (Fooman chemie-Iran). Urease was produced by Hp in biopsy specimens. Urease is produced by H.pylori in biopsy specimens. Urea was hydrolyzed with the production of ammonium ions. This resulted in a rise in PH which was detected by color change of the indicator (phenol red) from yellow to red within one hour and this was considered positive [10]. Statistical analysis was done, using the Chi-square test and  $p < 0.05$  was considered as statistically significant.

### Results

Among the 65 subjects under the study, there were 34 males (52.3%) and 31 females (47.7%). The age among the case group ranged from 21-67 years with the mean and standard deviation of  $41.3 \pm 13.5$  years. Thirty five patients (53.8%) had periodontal disease and Hp was detected in 40 patients of all (61.5%) (Table 1).

Twenty three out of 40 patients (57.5) harboring Hp in their stomach showed concomitant periodontal disease, whereas 17 patients (42.5%) were periodontally normal (Table 2).

**Table 1** Frequency of periodontal disease and gastric Hp infection among the studied patients.

Variable	Sex	Positive (%)	Negative (%)	Sum
Periodontal disease	Male	17 (50)	17 (50)	34
	Female	18 (58.1)	13 (41.9)	31
Gastric Hp infection	Male	22 (67.7)	12 (35.3)	34
	Female	18 (58.1)	13 (41.9)	31

**Table 2** Frequency of periodontal disease in the patients infected with Hp

Gastric HP infection	Periodontal disease	
	Negative (%)	Positive (%)
Negative	13 (52)	12 (48)
Positive	17 (42.5)	23 (57.5)

There was no statistically significant association between periodontal disease and H. pylori gastric infection ( $p = 0.455$ ). The odds ratio (OR) was also calculated for this association and it was 1.4666 with a confidence interval of 0.537-4.001.

### Discussion

We studied the potential association between the presence of Hp in the stomach and periodontal disease. No significant association between these two was found in the studied population. Gastric Hp infection and periodontal disease are common diseases among the general population [2, 4]. However, there are conflicting data about the correlation between these two. These discrepancies indicate that the potential relationship between them is more complex.

Some investigators suggested that Hp in the dental plaque might present a risk factor for gastrointestinal reinfection and ulcer relapse after antibiotic therapy [16-22]. The results of the Umeda et al.'s study indicate that careful attention should be paid to patients with periodontal diseases who harbor Hp in their oral cavities [5]. Very high prevalence of Hp in the dental plaque of the patients with dyspepsia in Butt et al.'s study makes an attractive hypothesis [12], as it indicates that colonizing in the oral cavity can be an important source of gastric reinfection after effective Hp eradication therapy [23-26].

Nevertheless, other investigators have indicated that there is no correlation between disease attributes and Hp infection. Some researchers proposed that the cases of gastritis showing negative dental plaque for Hp are not significant and the bacteria have entered the stomach with the food in these cases [10]. These findings may suggest that oral presence of Hp may be

only transient and intermittent [4]. Our findings in the present study are similar to the investigations done by the second group.

Variations in the methods of detecting Hp in the stomach among the sampled population and also the technical difficulties may account for the different data in the studies [3, 7]. Some investigators used PCR technique for detection of the Hp [1, 3, 5, 7, 26]. Although PCR is non-invasive, it gives the highest detection rate and plays a significant role in the early detection of Hp infection [27-28]. PCR's high sensitivity can lead to false positive reactions as a result of sample contamination by PCR products [26]. Microbiological culture is the gold standard method for identifying the presence of Hp but some forms of microorganisms are difficult or impossible to culture [26, 29].

Our method was similar to that of Ozdemir et al. for detecting Hp and it was RUT. This test has the advantage of speed, simplicity and low cost. It has near 100% specificity and between 70-80% sensitivity [8, 30]. Similar to the study of Guzik et al., although our data were obtained in relatively small groups, the large epidemiological investigation using urease test and performed on over 10,000 subjects revealed that periodontal disease significantly increases the risk of gastric Hp infection [2, 4]. Another limitation of the present study, like that of Bruce et al. is that it was cross-sectional. Because both Hp infection and periodontal disease are considered as chronic disorders, the design of these studies does address temporal relationships [13].

### Conclusion

This study revealed that the presence of periodontal disease in patients with gastric Hp infection could be an independent status.

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## References

- [1] Gebara EC, Faria CM, Pannuti C, Chehter L, Mayer MP, Lima LA. Persistence of *Helicobacter pylori* in the oral cavity after systemic eradication therapy. *J Clin Periodontol* 2006; 33: 329-333.
- [2] Czeńnikiewicz-Guzik M, Karczewska E, Bielański W, Guzik TJ, Kapera P, Targosz A, et al. Association of the presence of *Helicobacter pylori* in the oral cavity and in the stomach. *J Physiol Pharmacol* 2004; 55: 105-115.
- [3] Kignel S, de Almeida Pina F, André EA, Alves Mayer MP, Birman EG. Occurrence of *Helicobacter pylori* in dental plaque and saliva of dyspeptic patients. *Oral Dis* 2005; 11: 17-21.
- [4] Czesnikiewicz-Guzik M, Bielanski W, Guzik TJ, Loster B, Konturek SJ. *Helicobacter pylori* in the oral cavity and its implications for gastric infection, periodontal health, immunology and dyspepsia. *J Physiol Pharmacol* 2005; 56: 77-89.
- [5] Umeda M, Kobayashi H, Takeuchi Y, Hayashi J, Morotome-Hayashi Y, Yano K, et al. High prevalence of *Helicobacter pylori* detected by PCR in the oral cavities of periodontitis patients. *J Periodontol* 2003; 74: 129-134.
- [6] Anand PS, Nandakumar K, Shenoy KT. Are dental plaque, poor oral hygiene, and periodontal disease associated with *Helicobacter pylori* infection? *J Periodontol* 2006; 77: 692-698.
- [7] Gebara EC, Pannuti C, Faria CM, Chehter L, Mayer MP, Lima LA. Prevalence of *Helicobacter pylori* detected by polymerase chain reaction in the oral cavity of periodontitis patients. *Oral Microbiol Immunol* 2004; 19: 277-280.
- [8] Ozdemir A, Mas MR, Sahin S, Sağlamkaya U, Ateşkan U. Detection of *Helicobacter pylori* colonization in dental plaques and tongue scrapings of patients with chronic gastritis. *Quintessence Int* 2001; 32: 131-134.
- [9] Berroteran A, Perrone M, Correnti M, Cavazza ME, Tombazzi C, Goncalvez R, et al. Detection of *Helicobacter pylori* DNA in the oral cavity and gastroduodenal system of a Venezuelan population. *J Med Microbiol* 2002; 51: 764-770.
- [10] Siddiq M, Haseebur Re, Mahmood A. Evidence of *Helicobacter pylori* infection in dental plaque and gastric mucosa. *J Coll Physicians Surg Pak* 2004; 14: 205-207.
- [11] Chaudhry S, Iqbal HA, Khan AA, Izhar M, Butt AK, Akhter MW, et al. *Helicobacter pylori* in dental plaque and gastric mucosa: correlation revisited. *J Pak Med Assoc* 2008; 58: 331-334.
- [12] Butt AK, Khan AA, Khan AA, Izhar M, Alam A, Shah SW, et al. Correlation of *Helicobacter pylori* in dental plaque and gastric mucosa of dyspeptic patients. *J Pak Med Assoc* 2002; 52: 196-200.
- [13] 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: 1809-1815.
- [14] Chitsazi MT, Fattahi E, Farahani RM, Fattahi S. *Helicobacter pylori* in the dental plaque: is it of diagnostic value for gastric infection? *Med Oral Patol Oral Cir Bucal* 2006; 11: 325-328.
- [15] Kamat AH, Mehta PR, Natu AA, Phadke AY, Vora IM, Desai PD, et al. Dental plaque: an unlikely reservoir of *Helicobacter pylori*. *Indian J Gastroenterol* 1998; 17: 138-140.
- [16] Hu W, Cao C, Meng H. *Helicobacter pylori* in dental plaque of periodontitis and gastric disease patients. *Zhonghua Kou Qiang Yi Xue Za Zhi* 1999; 34: 49-51.
- [17] Riggio MP, Lennon A. Identification by PCR of *Helicobacter pylori* in subgingival plaque of adult periodontitis patients. *J Med Microbiol* 1999; 48: 317-322.
- [18] Sheu BS, Cheng HC, Yang YJ, Yang HB, Wu JJ. The presence of dental disease can be a risk factor for recurrent *Helicobacter pylori* infection after eradication therapy: a 3-year follow-up. *Endoscopy* 2007; 39: 942-947.
- [19] Hu W, Cao C, Meng H, Zhang J, Ma D, Zhang L.

- Detection and analysis of *Helicobacter pylori* in oral cavity and stomach from chronic gastritis patients. *Zhonghua Yi Xue Za Zhi* 2002; 82: 1037-1041.
- [20] Suk FM, Chen SH, Ho YS, Pan S, Lou HY, Chang CC, et al. It is difficult to eradicate *Helicobacter pylori* from dental plaque by triple therapy. *Zhonghua Yi Xue Za Zhi (Taipei)* 2002; 65: 468-473.
- [21] Chumpitaz Conde J, Gutiérrez Manay J, Córdova Acosta R, Sánchez Medina M, Vásquez Valverde N, Rivadeira Malaga C, et al. Isolation of *Helicobacter pylori* in dental plaque in patients with gastritis at "Angamos" clinic. *Rev Gastroenterol Peru* 2006; 26: 373-376.
- [22] Ogunbodede EO, Lawal OO, Lamikanra A, Okeke IN, Rotimi O, Rasheed AA. *Helicobacter pylori* in the dental plaque and gastric mucosa of dyspeptic Nigerian patients. *Trop Gastroenterol* 2002; 23: 127-133.
- [23] Cammarota G, Tursi A, Montalto M, Papa A, Veneto G, Bernardi S, et al. Role of dental plaque in the transmission of *Helicobacter pylori* infection. *J Clin Gastroenterol* 1996; 22: 174-177.
- [24] Dore-Davin C, Heitz M, Yang H, Herranz M, Blum AI, Corthensy TI. *Helicobacter pylori* in the oral cavity reflects handling of contaminate but not gastric infection. *Digestion* 1999; 60: 196-202.
- [25] Nasrolahei M, Maleki I, Emadian O. *Helicobacter pylori* colonization in dental plaque and gastric infection. *Rom J Gastroenterol* 2003; 12: 293-296.
- [26] Cheng LH, Webberley MW, Evans M, Hanson N, Brown R. *Helicobacter pylori* in dental plaque and gastric mucosa. *Oral Surg Oral Med Oral Pathol Radiol Endod* 1996; 81: 421-423.
- [27] Patthy A, Rakoczi G, Vegh A. Diagnosis of *Helicobacter pylori* infection from dental plaque. *Fogorv Sz* 2002; 95: 33-34.
- [28] Teoman I, Ozmeric N, Ozcan G, Alaaddinoglu E, Dumlu S, Akyon Y, Balos K. Comparison of different methods to detect *Helicobacter pylori* in the dental plaque of dyspeptic patients. *Clin Oral Investig* 2007; 11: 201-205.
- [29] Nguyen AH, El-zaateri FA, Graham DY. *Helicobacter pylori* in the oral cavity. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1995; 76: 705-709.
- [30] Yakoob J, Jafri W, Abid SH, Jafri N, Abbas Z, Hameed S, et al. Role of Rapid Urease Test and Histopathology in the diagnosis of *Helicobacter pylori* infection in developing country. *BMC Gastroenterol* 2005; 5: 38.