Helicobacter pylori in the dental plaque: Is it of diagnostic value for gastric infection?

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ABSTRACT

Aim: The aim of the present study was the assessment of association of helicobacter pylori of dental plaque and stomach in a more homogenous population and also to determine the diagnostic value of dental plaque for gastric infection. *Materials and Methods:* Based on the results of Rapid urease test (RUT) on specimens from gastric antrum, 88 patients with symptoms of dyspepsia were assigned into two groups of infected and non-infected with helicobacter pylori. Supragingival plaque samples were collected from mandibular first and second molar area using and sterile curette and were investigated using RUT. Statistical analysis of data was performed using chi-square test and independent t-test. *Results:* H.pylori was detected in 34.1 % of dental plaque specimens. The prevalence of helicobacter pylori infection in dental was 31.8 % and 36.4 % in patients with and without gastric infection (P=0.6). Also the interaction of age and infection was not significant but the prevalence of H. pylori both in the dental plaque and the stomach of male patients was higher than female patients.

Conclusion: there is not any significant association between the helicobacter pylori of the dental plaque and the stomach. Also the dental plaque can not be used as a primary diagnostic aid for gastric infection.

Key words: Helicobacter pylori, dental plaque, rapid urease test.

INTRODUCTION

Helicobacter pylori infection is considered as one of the most prevalent infectious diseases throughout the world; the carriage rate of *Helicobacter pylori* is reported to be 20-80% for adults in the developed world, and more than 90% in the developing world (1). This infection has a central role in the development and progress of peptic ulcers (2,3). It has also been correlated with gastric cancer (4,5). After the discovery of this association, the recurrence rate of peptic ulcers, which was 80% formerly, has declined to 20% (6). Taken together, early detection and eradication of this bacterium would be helpful either in the prevention or the treatment of peptic ulcers and also in reducing the economical burden

imposed to the health systems by the disease. By the same token, early diagnostic approaches which are accessible and less expensive would be highly beneficial.

Dental plaque is a soft gelatinous mass adhering to the tooth surface and other hard surfaces and comprising of bacteria embedded in an organic matrix. After isolation of helicobacter pylori form dental plaque, controversies regarding the existence of this bacterium in the dental plaque -as a probable secondary reservoir for it – have been never ceased (7-9). While the presence of helicobacter pylori in dental plaque was a common finding in some studies (10-13), it was not detected in others as frequently (14,15); the latter correlate the residence of the bacterium in the oral

cavity with gastroesophagal reflux (16,17). Co-existence of helicobacter pylori in the dental plaque and the stomach is also a matter of debate (18,19). Although this coincidence has been reported by some authors (20,21), other researchers did not find any association between these two potential sources of the bacterium (22,23). The issue still remains a question today. Is there any relationship between helicobacter pylori of dental plaque and gastric infection? If any, is it_of diagnostic value for the clinician?

One of the factors aggravating the present relative lack of agreement among the researchers is the lack of strict criteria in selecting study population, design of the study and collection of the specimens. For instance, despite the interaction of cariogenic bacteria with helicobacter pylori (24), caries status of study population has not been taken into account in the design of previous studies. The aim of the present study was the assessment of association of two potential sources of helicobacter pylori, namely stomach and dental plaque in a more homogenous study design by eliminating some of the most important confounding factors which could affect the results of the study enormously. Also we investigated whether helicobacter pylori of dental plaque can be of value in the diagnosis of gastric infection.

METHODS AND MATERIALS

Study population

Present study was conducted at the departments of endocrinology and periodontology at Tabriz University of Medical Sciences. The study population was comprised of the patients referring to the department of endocrinology with a complaint of dyspepsia. All of the selected patients were non-smokers. A history of the following confounding factors was ruled out in patients as an important step toward achievement of a more homogenous study population: previous treatment of peptic ulcer, cancer, antibiotic therapy or the use of oral antibacterial mouth rinse for any reason during the past year, surgical and non-surgical periodontal therapy since a year ago, signs of active periodontal disease, severe dental caries or recent use of proton pump inhibitors and non-steroidal anti-inflammatory drugs. The last two factors are important since potential interactions of dental and periodontal pathogens with H. pylori could act as a source of bias.

Grouping and laboratory procedures

Endoscopic survey of stomach was performed. Biopsy specimens were taken from the gastric antrum at a distance of 2cm from pyloric canal in order to retain site specifity. Samples were examined using rapid urease test (RUT) for the detection of H. pylori. Those samples showing a color change from yellow to red within an hour were considered to be positive for H. pylori infection.

Based on the results of RUT, subjects were divided into two groups (a group with gastric infection and another group without infection, each consisting of 44 patients). For the detection of H. pylori in the dental plaque, supragingival plaque samples were collected from mandibular first and second molar area using and sterile curette and were investigated using RUT as mentioned previously. The examiner was unaware of the grouping of the samples.

Statistical analysis

All figures were expressed as mean \pm SD (Standard deviation). Chi-square test was employed for the analysis of detection frequency of Helicobacter pylori in dental plaque of patients with and without gastric infection; and the association of sex with the results of RUT in the dental plaque and the stomach. Independent t-test was used for the analysis of the association of the age with the results of RUT test of the dental plaque and stomach. In the present study P<0.05 was considered to indicate statistical significance.

RESULTS

A total of 88 patients with a mean age of 34.9(SD: 11.23) participated in this study. Of these patients, 52 were male and 36 were female. The mean age of patients was 34.9 (SD: 11.23). H.pylori was detected in 34.1% of dental plaque specimens (30 patients).

Table 1 shows the prevalence of H.pylori in the dental plaque of patients with and without gastric infection. The association of H.pylori infection of stomach and simultaneous existence of this bacterium in the dental plaque was not significant (P=0.6).

The prevalence of with H.pylori both in the dental plaque and the stomach of male patients was higher than female patients (dental plaque: $\chi^2=3.8$, P=0.04 stomach: $\chi^2=4.8$, P=0.03). While 31 male patient had stomach infection, only 13 female patients were infected. Also 22 male and 8 female patients had helicobacter pylori in the dental plaque. Eight patients demonstrated simultaneous existence of helicobacter pylori in stomach and dental plaque.

There was not any significant interaction of age and the infection in the stomach and the dental plaque (P=0.7 for both).

Table 1. The association of helicobacter pylori infection of the dental	
plaque and the stomach.	

Sex	Infection of dental plaque	Infected stomach	Non-infected stomach	Total
Male	Negative	13 69.9%	17 54.8%	30 57.7%
Male	Positive	8 38.1%	14 45.2%	22 42.3%
Total		21 100%	31 100%	52 100%
Female	Negative	15 65.2%	13 100%	28 77.8%
Female	Positive	8 34.8%	0 0%	8 22.2%
Total		23 100%	13 100%	36 100%

The first figure in each cell shows the number of patients with the associated characteristic and the second one demonstrates the percentage.

DISCUSSION

The findings of the present study show that there is not any association between H.pylori of dental plaque and stomach as two potential and important sources of this microorganism. While 31.8% of patients with H.pylori infection of the stomach demonstrated the microorganism in the dental plaque, 36.4% of patients not affected by the stomach infection were found to have the microorganism in the dental plaque. There was not any statistical difference between these two groups (p=0.6).

In several studies there was not any association between H.pylori infection of dental plague and stomach (19,22,23). Some authors believe that H.pylori should be categorized as the normal microflora of the oral cavity (25,26), while others suggest that the existence of this microorganism in oral cavity is temporary and could be related to the occupational exposure to the bacterium (27,28). The results of aforementioned surveys are in agreement with the finding of the present study that demonstrated low prevalence of helicobacter pylori in the dental plaque. Some researches insist on the positive association of H.pylori in the dental plaque and the stomach (20,21). We did not find such a strong association in our study population. Some researchers believe in the lack of such an association (22,23). This discrepancy may reflect the difference in study method and study population.

Another finding of this study was the higher prevalence of H.pylori infection both in the dental plaque and the stomach in male subjects. Similar findings, showing higher and more consistent prevalence of helicobacter pylori infection in male sex, have been reported previously (29). This issue has several potential implications. It has been suggested that the existence of H.pylori in dental plaque is temporary and that it could be related to the gastroesophageal reflux (16,17). They believe that oral cavity is not a primary source of this bacterium. Since gastroesophageal reflux is more prevalent in female sex (30), it can not explain the male dominant pattern of helicobacter pylori infection observed in the present study. However, smoking (31), social contacts and occupational exposure to the bacterium, all of which are associated with high risk of helicobacter pylori infection, are found more in men in the developing countries. A fact that is concordant with the results of the present study.

Several factors are involved in partial lack of agreement between the results of different researchers. Strict criteria in study design and population selection, collection of specimen, utilized tests, different blood groups (32), serum lipid profile (33) and maturation stage of dental plaque –which affects its lipid content and also potential bacterial interactions- and active periodontal diseases (34), are among these factors. In this study we minimized the effects of these confounding factors. However, there are other issues that should be meticulously considered while conducting any study of this kind. The number of bacteria for maintenance of active infection is not known yet. Also further studies are needed to determine whether the presence of the bacteria in oral cavity is permanent or not. Moreover, local factors contributing to the growth of bacteria in the oral cavity and possible interactions of this bacterium with indigenous bacteria of oral cavity should be investigated. The investigation of serotypic similarity of H.pylori in dental plaque and stomach is also of vital importance.

CONCLUSION

In conclusion, the results of present study with a relatively homogenous study population and study design shows that there is not any association between H.pylori in dental plaque and stomach. Also it seems that the presence of H.pylori is not of any diagnostic value for gastric infection.

REFERENCES

1. Taylor DN, Blaser MJ. The epidemiology of Helicobacter pylori infection. Epidemiol Rev 1991;13:42-59.

2. Lee Å, Fox J, Hazell S. Pathogenecity of Helicobacter pylori: a perspective. Infect Immune 1993;61:1601-10.

3. Dixon MF. Helicobacter Pylori and peptic ulceration: Histopathological aspects. J Gastroenetrol hepatol 1991;6:125-30.

4. Forman D, Newell DG, Fullerton F. Association between infection with Helicobacter pylori and risk of gastric cancer: evidence from a prospective investigation. BMJ 1991;302:1302-5.

5. Parsonnet J, Vandersteen D, Goates J, Sibley RK, Pritikin J, Chang Y. Helicobacter pylori infection in intestinal- and diffuse-type gastric adenocarcinomas. J Natl Cancer Inst 1991;302:1302-5.

6. Catherine M. Dental implications of helicobacter pylori. J Can Dent Assoc 2002;68:489-93.

7. Checchi L, Felice P, Acciardi C, Ricci C, Gatta L, Polacci R, et al. Absence of Helicobacter pylori in dental plaque assessed by stool test. Am J Gastroenterol 2000;95:3005-6.

8. Mazumder DN, Ghoshal UC. Dental plaque as reservoir, and determinants of pathogenic potential of Helicobacter pylori: the controversy remains. Indian J Gastroenterol 1998;17:123-5.

9. Siddiq M, Haseeb UR, Mahmood A. Evidence of Helicobacter pylori infection in dental plaque and gastric mucosa. J Coll Physicians Surg Pak 2004;14:205-7.

10. 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.

11. Nguyen AM, Engstrand L, Genta RM, Graham DY, el-Zaatari FA. Detection of Helicobacter pylori in dental plaque by reverse transcription-polymerase chain reaction. J Clin Microbiol 1993;31:783-7.

12. Song Q, Lange T, Spahr A, Adler G, Bode G. Characteristic distribution pattern of Helicobacter pylori in dental plaque and saliva detected with nested PCR. J Med Microbiol 2000;49:349-53.

13. Yang HT. Nested-polymerase chain reaction in detection of Helicobacter pylori in human dental plaque. Zhonghua Yi Xue Za Zhi 1993;73:750-2, 774.

14. Bickley J, Owen RJ, Fraser AG, Pounder RE. Evaluation of the polymerase chain reaction for detecting the urease C gene of Helicobacter pylori in gastric biopsy samples and dental plaque. J Med Microbiol 1993;39:338-44.

15. Hardo PG, Tugnait A, Hassan F. Helicobacter pylori infection and dental care. Gut 1995;37:44-6.

16. Savoldi E, Marinone MG, Negrini R, Facchinetti D, Lanzini A, Sapelli PL. Absence of Helicobacter pylori in dental plaque determined by immunoperoxidase. Helicobacter 1998;3:283-7.

17. Mattana CM, Vega AE, Flores G, de Domeniconi AG, de Centorbi ON. Isolation of Helicobacter pylori from dental plaque. Rev Argent Microbiol 1998;30:93-5.

18. 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;923:127-33.

19. Sahin FI, Tinaz AC, Simsek IS, Menevse S, Gorgul A. Detection of Helicobacter pylori in dental plaque and gastric biopsy samples of Turkish patients by PCR-RFLP. Acta Gastroenterol Belg 2001;64:150-2.

21. Cellini L, Allocati N, Piattelli A, Petrelli I, Fanci P, Dainelli B. Microbiological evidence of Helicobacter pylori from dental plaque in dyspeptic patients. New Microbiol 1995;18:187-92.

22. 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;22:174-7.

23. Cheng LH, Webberley M, Evans M, Hanson N, Brown R. Helicobacter pylori in dental plaque and gastric mucosa. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;81:421-3.

24. Okuda K, Ishihara K, Miura T, Katakura A, Noma H, Ebihara Y. Helicobacter pylori may have only a transient presence in the oral cavity and on the surface of oral cancer. Microbiol Immunol 2000;44:385-8.

25. Shankaran K, Desai HG. Helicobacter pylori in dental plaque. J Clin Gastroenterol 1995;21:82-4.

26. Song Q, Lange T, Spahr A, Adler G, Bode G. Characteristic distribution pattern of Helicobacter pylori in dental plaque and saliva detected with nested PCR. J Med Microbiol 2000;49:349-53.

27. 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;17:138-40.

28. Santamaria MJ, Varea C, V, Munoz Almagro MC. Dental plaque in Helicobacter pylori infection. An Esp Pediatr 1999;50:244-6.

29. Replogle ML, Glaser SL, Hiatt RA, Parsonnet J. Biologic sex as a risk factor for Helicobacter pylori infection in healthy young adults. Am J Epidemiol 1995;15:56-63.

30. de Oliveira SS, dos Santos Ida S, da Silva JF, Machado EC. Gastroesophageal reflux disease: prevalence and associated factors. Arq Gastroenterol 2005;42:116-21.

31. Cardenas VM, Graham DY. Smoking and Helicobacter pylori infection in a sample of U.S. adults. Epidemiology 2005;16:586-90.

32. Kanbay M, Gur G, Arslan H, Yilmaz U, Boyacioglu S. The relationship of ABO blood group, age, gender, smoking, and Helicobacter pylori infection. Dig Dis Sci 2005;50:1214-7.

33. Trampenau C, Muller KD. Affinity of Helicobacter pylori to cholesterol and other steroids. Microbes Infect 2003;5:13-7.

34. 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;92:1809-15.