Research Article
Published: 21 Jan, 2019

Journal of Dentistry Forecast

Association between Periodontitis and *Helicobacter pylori* (HP) Infection: A Case –Control Study of HP Gastric Infection Population

Mikou S[1]*, Boubdir S[1], Hamza M[2], Zouir S[3] and Kissa J[1]

[1]*Department of Periodontology, University of Hassan II of Casablanca, Morocco*

[2]*Department of Pediatric Prevention. University of Hassan II of Casablanca, Morocco*

[3]*Doctor in Dentistry, Private Practice, Casablanca, Morocco*

Abstract

Objectives: ***Helicobacter pylori*** *(H. pylori),* a pathogen inducing peptic disease, is recently found to be binding to the progress of periodontitis**,**the present study was to evaluate periodontal health status of patients with *HP* gastric infection, and the correlation between *H. pylori* infection and periodontal disease.

Methods: A total of 51 patients (test group) with *HP* gastric infection (28 males and 23 females) were recruited from Gastroenterology service –CHU IBN ROCHD CASABLANCA- 102 patients (control group) with healthy periodontium were selected, including 61 males and 41 females. The clinical parameters assessed were plaque index (PI), probing pocket depth and clinical attachment by the same physician using the method described in literature.

Results: The result indicated that patients with *H. pylori* infection differ in plaque index (PLI) and bleeding index (BI) ($p > 0.05$), while patients with *H. pylori* exhibited deeper probing depth (PD) and more attachment loss (AL) than those without ($p < 0.05$).

Conclusion: *H. pylori* infection may be correlated with the incidence of periodontal disease, producing an impact on the depth of periodontal pockets and pathological degree of periodontitis

Keywords: Periodontitis; *H. pylori* infection; Oral cavity

OPEN ACCESS

Correspondence:
Mikou S, Department of Periodontology, University of Hassan II of Casablanca, Morocco.
E-mail: *mikousalwa12@gmail.com*

Received Date: *06 Dec 2018*
Accepted Date: *17 Jan 2019*
Published Date: *21 Jan 2019*

Citation: *Mikou S, Boubdir S, Hamza M, Zouir S, Kissa J. Association between Periodontitis and Helicobacter pylori (HP) Infection: A Case – Control Study of HP Gastric Infection Population. J Dent Forecast. 2019; 2(1): 1016.*

ISSN 2643-7104

Introduction

Periodontal diseases are inflammatory chronic diseases affecting teeth supportive tissues, and have infectious bacterial etiology [1]. They are initiated by oral bacterial biofilms developing on dental roots. Period onto pathogenic bacteria are mainly anaerobic, Gram negative strains developing subgingivally in periodontal pockets. This subgingival environment is particularly favorable for the survival and proliferation of non-aerobic strains.

Helicobacter pylori is a Gram-negative, spiral- shaped, microaerophilic, flagellated bacterium implicated in the etiology of numerous gastrointestinal diseases such as peptic, duodenal ulcers, gastric carcinoma, and mucosa-associated lymphoid tissue lymphoma [2,3].

Human infection by this pathogen could involve an oral route, and the oral cavity is an anatomical region with a potential to harbor *HP*, in particular dental plaque (oral biofilm).

Most studies are concerned with the pathogenicity of dental plaques, but less concerned with the correlation between periodontal health status and *H. pylori* infection. Zhu et al. [4] believed that *H. pylori* in the stomach may be involved.

The aim of the present study was to evaluate periodontal health status of patients with *HP* gastric infection, and to determine the possible association between H. *pylori* infection and periodontal disease.

Materials and Methods

51 patients with *HP* gastric infection (28 males and 23 females) were recruited from Gastroenterology service –CHU IBN ROCHD CASABLANCA-102 control cases with healthy periodontium were selected, including 61 males and 41 females. All the eligible cases, who

Table 1: Descriptive statistics of plaque index (PI) in control and test groups.

	HP+ (n=51)		Control cases (n=102)		p
PI	Size	Percentage	Size	Percentage	
[0,1-1]	7	13,7%	71	69,6%	
[1,1-1,9]	39	76,5%	26	25,5%	0,0000001*
[2-3]	5	9,8%	5	4,9%	

PI: Plaque Index, *Significance.

Table 2: Descriptive statistics of bleeding index (BI) in control and test groups.

	HP+ (n=51)		Control cases (n=102)		p
BI	Size	Percentage	Size	Percentage	
[0-1]	7	13,7%	56	54,9%	0,00001*
[1,1-2]	44	86,3%	46	45,1%	
[2,1-3]	0	0%	0	0%	

BI: Bleeding index, *Significance.

volunteered, were informed about the nature, potential risks, and benefits of their participation in the study and written informed consent was obtained from all the cases.

Selection criteria

The inclusion criteria of cases were as follows:

1. Patients with HP gastric infection (diagnostic confirmed).
2. Systemically healthy patients (Except HP gastric infection).
3. Patients with more than twenty teeth.

The Exclusion criteria of control cases were as follows:

1. Chronic gastritis patients were excluded in the present study based on rapid urease test (RUT).
2. Patients who were under medications such as antibiotics, prolonged use of anti-inflammatory drugs, or the use of antacids in the past 6 months.
3. Patients who had undergone professional tooth cleaning or any other periodontal treatment within the past 6 months.
4. Patients with less than twenty teeth.
5. Smokers.
6. Aggressive periodontitis patients.

Clinical examination

Full-mouth periodontal examination was carried out in all cases. The clinical parameters assessed were plaque index (PI), probing pocket depth, recessions, clinical attachment and mobility by the same physician using the method described in literature.

Statistical analysis

Analysis of variance test was applied to determine the intergroup differences in clinical parameters between the test and control groups. Statistical analyses were done using Chi-2 test and Student test. The $P< 0.05$ was considered to be statistically significant.

Results

Table 1 shows the descriptive statistics of clinical parameters including plaque index (PI) of both groups (control and test groups).

In the test group more than 70% of patients with HP+ infection had plaque index [1,1-1,9] Statistically significant differences were observed in plaque index ($p < 0.05$) between both groups.

Table 3: Descriptive statistics of probing depth (PD) in control and test groups.

	HP+ (n=51)		Control cases (n=102)		P
Probing depth (PD)	Size	Percentage	Size	Percentage	
[0 mm-3mm]	16	31,4%	76	74,5%	0,000001*
[4mm-6mm]	31	60,8%	21	20,6%	
> 6mm	4	7,8%	5	4,9%	

PD: Probing Depth, *Significance.

Table 4: Descriptive statistics of Attachment loss (AL) in control and test groups.

	HP+ (n=51)		Control cases (n=102)		p
Attachment loss (AL)	Size	Percentage	Size	Percentage	
< 3mm	17	33,3%	81	79,4%	0,0000006
[3mm-5mm]	28	54,9%	20	19,6%	
> 5mm	6	11,8%	1	1%	

AL: Attachment Loss, *Significance.

Table 2 shows the descriptive statistics of bleeding index (PI) of both groups (control and test groups). In the test group more than 80% of patients with HP+ infection had bleeding index [1,1-2] Statistically significant differences were observed in the bleeding index ($p < 0, 05$) between both groups.

Table 3 shows the descriptive statistics of probing depth (PD) of both groups (control and test groups). In the test group more than 60% of patients with HP+ infection had periodontal pockets [4mm-6mm] .Statistically significant differences were observed in the bleeding index ($p < 0, 05$) between both groups.

Table 4 shows the descriptive statistics of Attachment loss (AL) of both groups (control and test groups). In the test group more than 10% of patients with HP+ infection had Attachment loss (> 5mm). Statistically significant differences were observed in the bleeding index ($p < 0, 05$) between both groups.

Discussion

The result of our study indicated that patients with *H. pylori* infection differ in plaque index (PLI) and bleeding index (BI) ($p > 0.05$) (Table 1,2), while patients with *H. pylori* exhibited deeper probing depth (PD) and more attachment loss (AL) than those without ($p < 0.05$) (Table 3,4).

Dental plaque consists of a variety of microorganisms which are responsible for the initiation and progression of periodontal diseases. It has been shown that there is a significant increase in the number of these microorganisms once the gingival sulcus is converted into the periodontal pocket [5]. Dental plaque and periodontal pockets can act as reservoirs of *H. pylori* since it is a microaerophilic organism [6].

Umeda *et al.* [7] showed that more than 40% of patients with history of gastritis or peptic ulcers had periodontal pockets (>4mm). Bruce *et al.* [8] found that periodontal pockets ≥5mm are associated with increased odds of *HP* seropositivity. Also, a large epidemiological investigation performed over 10,000 subjects [9], found a positive link between *HP associated* gastric infection and periodontal diseases. The latter may facilitate the *HP* oro-gastric transmission and colonization of the bacteria in the digestive tract. Also, periodontitis microenvironment conditions could be favorable for *HP* multiplication, thus increasing sufficiently to cause gastric infection [10,11].

On the other hand, many studies [12] failed to establish a positive correlation between periodontal disease and gastric infection.

An association between the presence of *H. pylori* in dental plaque and gastritis has been demonstrated. A higher prevalence of *H. pylori* has been observed in dental plaque than in the stomach in patients with chronic gastritis [13].

Although *H. pylori has* been detected in different sites, the periodontal pocket can be regarded as a natural reservoir for *H. pylori* as it provides microaerophilic conditions suitable for its growth. Poor oral health and periodontitis have shown to be associated with high prevalence of gastritis caused by *H.pylori* [14].

Oral hygiene parameters such as the presence of plaque and gingival bleeding have positively correlated with *H. pylori* in the oral cavity.

Thus, it seems conceivable that oral health status and different gingival clinical conditions may have an effect on the presence of *HP* in oral biofilms, hence influencing the process of *HP* gastric infection or reinfection.

Conclusion

H. pylori infection may be correlated with the incidence of periodontal disease, producing an impact on the depth of periodontal pockets and pathological degree of periodontitis. *H. pylori* infection is related to oral environment and oral hygiene. Localized in the 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.

References

1. Pei Z, Weiying Z. Relation between periodontitis and *helicobacter pylori* infection. Int J Clin Exp Med. 2015; 8: 16741-16744.
2. Safavi M, Shams-Ardakani M, Foroumadi A. Medicinal plants in the treatment of *Helicobacter pylori* infections. Pharm Biol. 2015; 53: 939-960.
3. Amizadeh M, Shamsadini A, Arabzadeh A, Jazayeri S. Association of cagA Positive *Helicobacter pylori* Infection and Laryngeal Squamous Cell Carcinoma: A PCR Approach. Indian J Otolaryngol Head Neck Surg. 2015; 67: 51-55.
4. Zhu LF, Long BG, Luo J, Jiang R, Fang HY. Construction of a recombinant Lactobacillus acidophilus expressing high levels of *Helicobacter pylori* adhesin Hp0410. Nan Fang Yi Ke Da Xue Xue Bao. 2010; 30: 334-337.
5. Listgarten MA, Helldén L. Relative distribution of bacteria at clinically healthy and periodontally diseased sites in humans. J ClinPeriodontol. 1978; 5: 115-132.
6. Venkata and coll. Effectiveness of nonsurgical periodontal therapy on the levels of *Helicobacter pylori* in dental plaque and saliva of patients with chronic periodontitis. European Journal of General Dentistry. 2017; 6: 29-35.
7. Umeda M and coll. High prevalence of *Helicobacter pylori* detected by PCR in the oral cavities of periodonti-tis patients. Journal of Periodontology. 74: 129-134.
8. Bruce A, Dye B, 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.
9. Bielanski W. Epidemiological study on Helico-bacter pylori infection and extra gastroduodenal disorders in Polish population. Journal of Physiology and Phar-macology. 1999; 50: 723-733.
10. Czesnikiewicz-Guzik M, Bielanski W, Guzik TJ, Los- ter B and Konturek SJ. *Helicobacter pylori* in the oral cavity and its implications for gastric infection, periodontal health, immunology and dyspepsia. Journal of Physiology and Pharmacology. 2005; 56: 77-89.
11. Czesnikiewicz-Guzik M, Karczewska E, Bielanski W, Guzik TJ, Kapera P, Targosz A, et al. Association of the presence of Helico- bacter pylori in the oral cavity and in the stomach. Journal of Physiology and Pharmacology. 2004; 55: 105-115.
12. Anand PS, Nandakumar K and Shenoy KT. Are dental plaque, poor oral hygiene, and periodontal di- sease associated with *Helicobacter pylori* infection? Journal of Periodontology. 2006; 77: 692-698.
13. Song Q, Lange T, Spahr A, Adler G and Bode G. Characteristic distribution pattern of *Helicobacter pylori* in dental plaque and saliva detected with nested PCR. Journal of Medical Microbiology. 2000; 49: 349-353.
14. Zou QH and Li RQ. *Helicobacter pylori* in the oral cavity and gastric mucosa: a meta-analysis. Journal of Oral Pathology & Medicine. 2011; 40: 317-324.