

Gingival status and gingival fluid flow in diabetic and non-diabetic patients

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ABSTRACT

Background: The aim of this study was to estimate the role of smoking as a risk factor on the gingival status of the diabetic and non-diabetic patient as measured by both gingival fluid flow (G.F.F) and gingival fluid pH (G.F.pH) and clinical parameters (plaque index and gingival index).

Material and method: All the patients were smokers with mean age of 45 years. Thirty-two sites were selected from each group. Clinical examination including PL.I and GI were evaluated for each patient, G.F.F and G.F.pH were evaluated to in the selected sites.

Results: The PL.I, G.I, G.F.F and G.F.pH for smoker diabetic patients were slightly higher than smoker non-diabetic patients, but no statistical difference was found.

Conclusion: There was a positive relationship between the clinical parameters and G.F.F and G.F.pH in both diabetic and non diabetic patient. It was also found that smokers are high risk group for periodontitis.

Key words: Gingival status, gingival fluid, diabetes. (J Bagh Coll Dentistry 2006; 18(1) 63-65)

INTRODUCTION

Periodontal disease affects individuals at some points during life and is a major cause of tooth loss in adult.⁽¹⁾ The distribution of periodontitis in the population suggests that a subset of individuals is highly susceptible to this infection, while the remaining majority exhibit varying degree of resistance and moderate susceptibility.⁽²⁾ Current data indicates that dental plaque is the cause of periodontitis and microbiological studies have shown that specific bacterial species are associated with diseased sites and disease activity.⁽³⁾ It has been suggested that the relationship between pathogens, the host and pathogenesis is complex and that host responses to dental plaque are important determinate of periodontal health.⁽⁴⁾ Cigarette smoking is known as risk factor among disease and mounting evidence suggests that smoking adversely influences periodontal health.^(5,6,7) As well as diabetes is a clear risk factor for periodontitis. Epidemiological data demonstrate that the prevalence and severity of periodontitis is significantly higher in diabetic patient than those without diabetes.⁽⁸⁾

The aim is to study the role of smoking as a risk factor on the gingival health of diabetic and non-diabetic patient by measuring G.F.F, G.F.pH as well as through evaluation of clinical indices.

MATERIALS AND METHOD

The sample consisted of 24 patients (12 diabetic and 12 non-diabetics) attending the department of periodontal in the college of dentistry, university of Baghdad with age ranged 35-55. The diabetic patients were controlled according to the level of HbA1C, which was not more than 8.5. The entire diabetic and not diabetic was smoker not less than 10 years and not less than 10 cigarettes per day.

Thirty-two sites were selected from each patient and each site showed pocket depth not more than 5 mm. Clinical examination include PL.I (Silness & Loe 1964)⁽⁹⁾ and G.I (Loe and Silness 1963)⁽¹⁰⁾ followed by the estimation of G.F.F (Brill 1962)⁽¹¹⁾. After good isolation of the area with cotton roll and drying with air syringe, the standardized paper point size 30 was placed gently in each selected site until resistance was felt. Each paper was left for 3 minutes the removed and stained with 0.2-2% alcoholic solution of ninhydrin to facilitate measuring the wet area of paper point, since the staining make the column clearer to be measured by vainer. Then the G.F.pH was measured by using standardization indicator paper, each strip was carefully inserted into the selected site until resistance was felt. The strip was removed after remaining in place for 3 minutes and the color was compared with standardized universal pH.

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RESULTS

The mean PL.I and G.I in smoker diabetic patient were 1.72 and 1.58 respectively, while mean PL.I and G.I in smoker non-diabetic patient was 1.69 and 1.37 respectively as shown in table (1). No significant difference was found between them.

The mean G.F.F and G.F.pH of smoker diabetic were 1.6 and 6.98 respectively, while the corresponding value of smoker non-diabetic were 1.3 and 6.64 respectively as shown in table (2). Also no significant deference was found between these two values.

Table 1: Mean PL.I and G.I in smoker diabetic and non-diabetic

	No	PL.I	G.I
Diabetic	12	1.72 ± 0.65	1.58 ± 0.4
Non-diabetic	12	1.69 ± 0.59	1.37 ± 3.7

P> 0.05 non significant

Table 2: Mean gingival fluid flow and gingival fluid pH in smoker diabetic and non-diabetic

	No	G.F.F	G.F. pH
Diabetic	12	1.6 ± 0.15	6.98 ± 0.21
Non-diabetic	12	1.3 ± 0.21	6.64 ± 0.33

DISCUSSION

Although the mean PL.I and G.I of smoker diabetic patient was slightly more than smoker non-diabetic patient but statistically not significant this result is in agreement with pervious studies ^(12,13), who found that the effects of smoking among diabetic patient were similar to that observed in the non-diabetic patient.

The mean values of G.F.F and G.F.pH in the elected sites were slightly higher in diabetic smokers in comparison to non-diabetic smokers but no statistical difference was found.

This study support Borden et al ⁽¹⁾ and Becket et al ⁽¹⁵⁾, who found that the mean value of G.F.F was increased with increasing gingival inflammation. This could be explained on the bases that in gingival inflammation the GCF is considered as a transudate of the interstitial tissue accompanied by the inflammatory exudate of the serum ⁽¹⁶⁾. So, in increasing gingival inflammation there is an increase in the amount of exudate ending with increasing G.F.F.

It has been shown the flow of GCF is a sensitive parameter of gingival inflammation ⁽¹⁷⁾. Cimasoni noted a positive correlation was

always found between the clinical appreciation of gingival inflammation and the amount of gingival fluid ⁽¹⁷⁾. The amount of gingival fluid is greater when inflammation is present ⁽¹⁸⁾. The results of present studies agree with the above findings as there was a steady increase in the GCF level with an increase in severity of periodontal disease, and significant reduction was observed in follow-up ⁽¹⁹⁾. Also it is in agreement with Egbert et al ⁽²⁰⁾ who found that the range of G.FpH per person was 6-9.3 at gingival sites with mean G.I: 1.4. These finding are also in agreement with Ringelberg et al ⁽²¹⁾ who found that children with diabetic had more gingival inflammation and mean value of G.F.F than the children with non-diabetic and this is due to the higher susceptibility of diabetic patients to infection rather than the non-diabetic individuals.

Regarding smoking, the results of this study is in consistent with the results of Ketobi ²², who stated that, although gingival fluid has been observed to increase upon smoking, it decreases over extended time period as a result of the vasoactive effects of nicotine.

In relation to diabetes and gingivitis, Diabetic patients are more susceptible to gingival diseases and the likelihood of periodontal disease increases when diabetes is poorly controlled ⁽²³⁾. People with well-controlled diabetes, with good oral hygiene and on a regular maintenance schedule have the same chance of developing severe periodontitis as people without diabetes ⁽²⁴⁾.

REFERENCES

1. Burt BA, Ismail A. Periodontal disease, tooth loss and oral hygiene among older american. *Comm Dent Oral Epidem* 1985; 13: 93-6.
2. Johnson NW, Wilson. Detection of high-risk group and individual for periodontal disease: Evidence for the existence of high-risk groups and approaches to their detection. *J Clin Periodontol* 1988; 15: 276-90.
3. Tanner A, Bauldin H. The microbiota of early periodontitis lesion in adult. *J Clin Periodontol* 1989; 16: 467-71.
4. Gunsolley J, Ranney R. A.A in families afflicted with periodontitis. *J Periodont* 1990; 61: 643-8.
5. Arno A, Lovdal A. Alveolar bone loss as a function of tobacco consumption. *Acta Odontol Scand* 1959; 17:3-10.
6. Vander Weijden GA, Siegte C, Vander Velden U. Periodontitis in smoker and non smoker: Intra oral distributions of pockets. *J Clin Periodont* 2001; 28:955-60.
7. American Academy of periodontology: Position paper. Epidemiology of periodontal disease. *J Periodont* 1996; 67: 935-45.

8. Tervonen T, Oliver RC. Long term control of diabetes mellitus and periodontitis. *J Clin Periodont* 1993; 20:431-5.
9. Silness J, Loe H. Periodontal disease in pregnancy. *Acta Odont Scand* 1964; 22: 121-35.
10. Loe H, Silness J. Periodontal disease in pregnancy 1-prevalence and severity *Acta Odont Scand* 1963; 21: 531-3.
11. Brill N. The gingival pocket fluid studies of its occurrence composition and effect. *Acta Odont Scand*. 1962; 20:1-115.
12. Birnick H, Cohen W. Dental disease in children with diabetic mellitus. *J Periodont* 1975; 46: 241-5.
13. Haber J, Crowley M. Evidence for cigarette smoking as major risk factor for periodontitis. *J Periodont* 1993, 64: 16-23.
14. Borden H, Goluble L. The effect of age and sex on the relationship between cervicular fluid and gingival inflammation in human. *J Periodont* 1977; 2:160-5.
15. Bicket L, Cimasoni G. The pH of gingival crevice fluid measured by aneul micro analytical technique. *J Periodont. Res* 1985; 20:35-40.
16. Garehe S, Griffiths H. Formation, collection and significance of gingival crevice fluid. *Periodontology* 2000; 13: 23-42.
17. Cimasoni G. Monograph in Oral Science the Crevicular Fluid Update, 1983; vol 12: Switzerland, S. Karger.
18. Brex MC, Schlegel K, Gehr P, Lang NP. Comparison between histological and clinical parameters during human experimental gingivitis. *J Periodontal Res* 1987; 22::50-7.
19. Amas K, Suleman A, Quadairi A. The effect of oral hygiene instructions on diabetic type 2 male patients with periodontal diseases. *J Contemp Dent Pract*. 2003 ; 4:1-7
20. Eggert H. The pH of gingival crevice in children teenagers and adult. *J Dent Res* 1989; (Abst) 183.
21. Ringelberg D. Comparison of gingival health and gingival crevicular fluid flow in children with and without diabetes. *J Dent Res* 1977; 56: 108-11.
22. Genevieve B. Periodontal disease: Part I- Biological mechanisms and risk factors. *P P A D*. 2003; 11(1): 53-62.
23. Seppala B, Ainamo J. A site-by-site follow-up study on the effect of controlled versus poorly controlled insulin-dependent diabetes mellitus. *J Clin Periodontol* 1994; 21:161-5.
24. Debors CM. Periodontal medicine: A new paradigm. *Journal de l'Association dentaire canadienne* 2000; 66: 488-91.