

Harsukh Educational Charitable Society

International Journal of Community Health and Medical Research

Journal home page: www.ijchmr.com

doi: 10.21276/ijchmr

Official Publication of "Harsukh Educational Charitable Society" [Regd.]

ISSN E: 2457-0117

ISSN P:2581-5040

RNI No. - PUNENG/2017/75049

Index Copernicus value 2016 = 52.13

Original Research

Analogizing the Hormonal and Metabolic Effect on Periodontal Health After Non- Surgical Therapy Amongst Women

Manan Vyas

Associate Professor, Department of Dentistry, Ananta Institute Of Medical Sciences and Research Centre Rajsamand, Udaipur, Rajasthan

ABSTRACT

Background: The effect of diabetes and hormones is well known on periodontitis. Female sex hormones cannot independently alter periodontal tissue, however influences reaction to plaque and thus circuitously plays role in periodontal disease. The present study evaluated the role of diabetes and menopause on the periodontal condition of the females after non-surgical therapy. **Materials and methods:** The study consisted of 80 females reporting with periodontal problems. The study was divided into four groups. The required periodontal indices were studied in all groups and the results were analyzed using SPSS software. **Results:** The mean baseline gingival index amongst Group A was 2.3 ± 0.98 and at 3 months was 0.9 ± 1.1 . The mean baseline plaque index amongst Group B was 1.03 ± 0.17 and at 3 months was 0.71 ± 0.09 . The periodontal indices showed improvement in all the four groups. The periodontal conditions were worse amongst post-menopausal diabetic females. **Conclusion:** All the groups showed considerable improvement after non- surgical therapy. The study emphasizes that irrespective of influence of female sex hormones and impaired metabolism, the periodontal index could be recuperated with noninvasive therapy and proper plaque control measures. **Clinical Significance:** Menopause and poorer glycemic control are accountable for changes in periodontium as they are responsible for altering inflammatory processes, thus deteriorating periodontal health. The influence of metabolic disorders on periodontium can be reduced with non- surgical periodontal therapy.

Keywords: Diabetes; Gingival; Menopause; Periodontal; Surgical

Corresponding Author: Dr. Manan Vyas, Associate Professor, Department of Dentistry, Ananta Institute Of Medical Sciences and Research Centre Rajsamand, Udaipur, Rajasthan

This article may be cited as: Vyas M Analogizing the Hormonal and Metabolic Effect on Periodontal Health After Non- Surgical Therapy Amongst Women. HECS Int J Comm Health Med Res 2018; 4(3):77-80

INTRODUCTION

Periodontitis is the commonest pervasive ailments that has characteristics features of destruction of connective tissue and supporting bone after an inflammatory host response subordinate to infection caused by periodontal bacteria.^{1,2} Severe periodontitis may also cause tooth loss and is seen in 5–20% of adult populations around the globe.³⁻⁵ Humans have a complex endocrine system that plays a role in causation of periodontitis. During the menopause females undergo significant hormonal alterations that is associated with both oral and systemic manifestations.^{6,7} There is sudden decline in the level of oestrogen that is responsible for osteoporosis and also has affect on bones of jaw.^{8,9} It has been postulated that reduction in the mineral density of bone is a contributing factor for periodontal disease progression.¹⁰ The alterations in the oral tissues that are seen in menopausal women are chiefly due to hormonal changes and less

commonly as a result of physiological changes.¹¹ During menopause, the gingival epithelium begins to thin and therefore increases susceptibility to inflammatory disorders.¹² Another common metabolic disorder is diabetes mellitus that is characterized by increase in blood sugar levels due to malfunctioning of insulin.¹³ Periodontitis and gingivitis are the commonly seen oral presentations of diabetes. Subjects with poorly controlled type I and type II diabetes are at an elevated risk of periodontal disorders. Although the initiation of periodontal disease necessitates the presence of bacteria but no characteristic differences have been observed in the microflora amongst the diabetic and other subjects. Although studies conducted in the initial years have shown higher number of Capnocytophaga amongst subjects with diabetes.¹⁴ Majority of culture studies have shown the microflora at diseased sites of diabetic patients and non-diabetic patients were similar.^{15,16} The present study was

conducted to study the effect of noninvasive periodontal therapy on post and premenopausal diabetic and non-diabetic subjects.

MATERIALS AND METHODS

The present observational interventional study was conducted in Department of periodontology RKDF Dental College and Research Centre, Bhopal for a period of 2 years from January 2015 to December 2017. All the subjects that were enrolled in the study were completely informed and a written consent was obtained from all. The ethical committee clearance was obtained from the institutional ethical board. All the concerned evaluations were done by single periodontist to avoid operator error. The menstrual history of all the subjects were noted and the blood sugar levels were obtained. Based on these values subjects were divided into four groups:

Group A: premenopausal diabetic females

Group B: premenopausal non- diabetic females

Group C: postmenopausal diabetic females

Group D: postmenopausal non-diabetic females

The subjects between 30-60 years with moderate chronic periodontitis were included in the study. Only non-smoker subjects were enrolled in the study. Subjects younger than 30 years, with presence of oral tumors or pathology, on steroid or hormone replacement drugs were excluded from the study. Pregnant females were also excluded from the study. Three periodontal parameters namely gingival index, plaque index and periodontal index were recorded at the time of first visit and 3 months after the scaling. SPSS software was used to perform the statistical analysis. Paired t test was used for intra group evaluation and student t test for inter group evaluation. Probability value of less than 0.05 was considered significant.

RESULTS

A total of 80 subjects, with 20 subjects in each group were enrolled in the study. The mean age of the subjects was 37.98± 1.87 years. The mean baseline gingival index amongst Group A was 2.3 ± 0.98 and at 3 months was 0.9± 1.1. The mean difference was 1.4. There was a statistically significant improvement in the gingival index after treatment. The mean baseline plaque index amongst Group A was 1.32±0.14 and at 3 months was 0.95±0.11. The mean difference was 0.37. The mean baseline periodontal index amongst Group A was 5.70±0.54 and at 3 months was 3.41±0.23. The mean difference was 2.29. There was a statistically significant improvement in the periodontal index after treatment (table 1).

The mean baseline gingival index amongst Group B was 1.3±0.53 and at 3 months was 0.7±0.3. The mean difference was 0.7. There was no statistically significant improvement in the gingival index after treatment. The mean baseline plaque index amongst Group B was 1.03±0.17 and at 3 months was 0.71±0.09. The mean difference was 0.59. The mean baseline periodontal index amongst Group B was 3.50±0.24 and at 3 months was 2.31±0.12. The mean difference was 1.19. There was a statistically significant improvement in the periodontal index after treatment (table 2). Table 3 shows a significant improvement in all the indices amongst Group C subjects after treatment. The baseline gingival index improved from 3.9± 1.01 to 1.8±0.93. The plaque index showed a considerable improvement of 1.75 between baseline and postoperative values. There was a significant improvement of 2.07 in the periodontal index also.

Table 4 clearly indicates significant improvement in all the three variables amongst post-menopausal non-diabetic females. The gingival index showed an improvement of 1.4 from the baseline values. The plaque index improved from 2.11±1.03 to 1.02±0.45. The periodontal index showed an improvement of 2.27. Table 5 shows the comparison between all the four groups. The table clearly demonstrates that the periodontal status was best amongst the Group B subjects (pre-menopausal non-diabetic females). Although improvement was seen in all the 4 groups. The periodontal condition was worse amongst the Group C subjects (postmenopausal diabetic females).

Table 1: periodontal parameters comparison in Group A (premenopausal diabetic females)

Group A	Mean ±SD	Mean difference	P value
Gingival Index		1.4	<0.05
Baseline	2.3 ±0.98		
3 months	0.9±1.1		
Plaque Index		0.37	>0.05
Baseline	1.32±0.14		
3 months	0.95±0.11		
Periodontal Index		2.29	<0.05
Baseline	5.70±0.54		
3 months	3.41±0.23		

Table 2: Periodontal parameters comparison in Group B (premenopausal non- diabetic females)

Group B	Mean±SD	Mean difference	P value
Gingival Index		0.6	>0.05
Baseline	1.3 ± 0.53		
3 months	0.7±0.3		
Plaque Index		0.59	>0.05
Baseline	1.03±0.17		
3 months	0.71±0.09		
Periodontal Index		1.19	<0.05
Baseline	3.50±0.24		
3 months	2.31±0.12		

Table 3: Periodontal parameters comparison in Group C (postmenopausal diabetic females)

Group C	Mean ±SD	Mean difference	P value
Gingival Index		2.1	<0.05
Baseline	3.9 ±1.01		
3 months	1.8±0.93		
Plaque Index		1.75	<0.05
Baseline	2.80±1.15		
3 months	1.05±0.88		
Periodontal Index		2.07	<0.05
Baseline	6.50±0.44		
3 months	4.43±0.33		

Table 4: Periodontal parameters comparison in Group D (postmenopausal non- diabetic females)

Group D	Mean ±SD	Mean difference	P value
Gingival Index		1.4	<0.05
Baseline	2.8 ± 1.00		
3 months	1.4±0.83		
Plaque Index		1.09	<0.05
Baseline	2.11±1.03		
3 months	1.02±0.45		
Periodontal Index		2.27	<0.05
Baseline	6.20±0.43		
3 months	3.93±0.23		

Table 5: Periodontal parameters comparison between Groups

Periodontal parameters	Mean	P value
Gingival Index		<0.05
Group A	1.6	
Group B	1.0	
Group C	4.8	
Group D	3.5	
Plaque Index		<0.05
Group A	1.79	
Group B	1.38	
Group C	3.32	
Group D	2.62	
Periodontal index		<0.05
Group A	7.27	
Group B	4.65	
Group C	8.71	
Group D	5.06	

DISCUSSION

Periodontitis is an inflammatory progression that leads to episodic loss of periodontal attachment system, ultimately leading to tooth loss in susceptible subjects. The inflammatory retort in periodontitis include activation of leucocytes, T-lymphocytes and plasma bodies with production of antibodies, lipopolysaccharides and chemical mediators of inflammation like cytokines, chemokines and CRP. The cell wall of gram negative bacteria has lipopolysaccharides that act as potent stimulants of host response. It has been now universally accepted that periodontal disorders are due to mixed microbial invasion and in them there exists specific groups of pathogenic bacteria.¹⁷⁻¹⁹ The commencement and development of periodontal disease is dependent on the number of pathogenic bacteria, response of host and risk factors. These risk factors include systemic factors like poorly controlled diabetes mellitus, external stimuli like smoking, intrinsic issues and local influences. These include oral hygiene practices, sex, socioeconomic position, age group, use of drugs etc Hormonal oscillations in the female subject may lead to alteration the grade of periodontal fitness.²⁰ Such fluctuations may happen during puberty, menstrual cycle, pregnancy, or menopause. Changes may also arise with the use of oral contraceptives. Deficiency of estrogen alters the bone formation and resorption and this has also paved the way towards its effect on the alveolar bone amongst

postmenopausal females.²¹ Normally it exerts an anti-inflammatory effect on the periodontal structures but with its deficiency its effect is lost and the tissue becomes compromised. Studies have proven that the tooth retention^{22,23} and reduction in attachment loss and gingival inflammation amongst the females on the hormone replacement therapy.²⁴ These hormones exert a direct or indirect effect on the proliferation, differentiation and growth of cells like keratinocytes and fibroblasts of gingiva. Studies have also shown connection between ovarian abnormality and the incidence of periodontitis.²⁵ The result of our study demonstrated that the periodontal status was worse amongst the postmenopausal diabetic females demonstrating that blood sugar and hormone levels have a well elucidated role in the progression of periodontitis.

Periodontitis is regarded as the sixth impediment of diabetes.²⁶ Several studies found an increased prevalence of periodontitis amongst diabetic subjects than amongst healthy controls.²⁷ The progress of periodontitis is more rapid in poorly controlled diabetics,²⁸ and early onset age of the disorder is a potent risk factor for more severe conditions.²⁹ Contrarywise, well-controlled diabetic subjects can maintain good periodontal health and respond constructively to periodontal treatment.³⁰ A longitudinal gingivitis study³¹ has shown more rapid and distinct development of gingival inflammatory condition in well-controlled type 1 diabetic subjects compared to non-diabetic controls, even in the presence of similar plaque accumulation level and similar bacterial load of plaque, indicating a hyperinflammatory response in diabetes.

CONCLUSION

The study emphasized on the effects of noninvasive periodontal therapy amongst diabetic and menopausal females. All the groups showed improvement in the periodontal health after non-surgical therapy. Although the periodontal status of postmenopausal females was not good and the study demonstrates that the periodontal index could be improved with proper plaque control measures and noninvasive therapy.

REFERENCES

1. Bascones-Martínez A, Muñoz-Corcuera M, Noronha S, Mota P, Bascones-Ilundain C, Campo-Trapero J. Host defence mechanisms against bacterial aggression in periodontal disease: Basic mechanisms. *Med Oral Patol Oral Cir Bucal.* 2009 Dec 1;14(12):e680-5.
2. Zhang L, Henson BS, Camargo PM, Wong DT. The clinical value of salivary biomarkers for periodontal disease. *Periodontology* 2000. 2009 Oct;51(1):25-37.
3. Albandar JM. Epidemiology and risk factors of periodontal diseases. *Dental Clinics.* 2005 Jul 1;49(3):517-32.
4. Haynes DR. Emerging and future therapies for the treatment of bone loss associated with chronic inflammation. *Inflammopharmacology.* 2006 Dec 1;14(5-6):193-7.
5. Khalili J. Periodontal disease: an overview for medical practitioners. *Likars' ka sprava.* 2008(3-4):10-21.
6. Vitiello D, Naftolin F, Taylor HS. Menopause: Developing a rational treatment plan. *Gyneco Endocrinol* 2007;23:682-691.

7. Friedlander AH. The physiology, medical management and oral implications of menopause. *The Journal of the American Dental Association*. 2002 Jan 1;133(1):73-81.
8. Becker C. Pathophysiology and clinical manifestations of osteoporosis. *Clin Cornerstone* 2006;8:19-27.
9. Lerner UH. Bone Remodeling in Post-meno-pausal Osteoporosis. *J Dent Res* 2007; 85: 584-595.
10. Wactawski-Wende J. Periodontal diseases and osteoporosis: association and mechanisms. *Ann Periodontol* 2001;6:197-208.
11. Lopez BC, Perez MG, Soriano YJ. Dental considerations in pregnancy and menopause. *J Clin Exp Dent* 2011;3:e135-44.
12. Forabosco A, Criscuolo M, Coukos G, Uccelli E, Weinstein R, Spinato S, *et al*. Efficacy of hormone replacement therapy in postmenopausal women with oral discomfort. *Oral Surg Oral Med Oral Pathol* 1992;73:570-4.
13. Tan M, Daneman D, Lau D, and others. Diabetes in Canada: strategies towards 2000. In: *Canadian Diabetes Advisory Board*; 1997; Toronto; 1997. p. 3.
14. Research, Science and Therapy Committee of The American Academy of Periodontology. Position Paper; Diabetes and Periodontal Diseases. *Journal of periodontology*. 1999 Aug 1;70(8):935-49.
15. Sastrowijoto S, Hillemans P, van Steenberg T, Abraham-Inpijn L, de Graaff J. Periodontal condition and microbiology of healthy and diseased periodontal pockets in type 1 diabetes mellitus patients. *J Clin Periodontol* 1989;16:316-322.
16. Zambon JJ, Reynolds H, Fisher JG, Shlossman M, Dunford R, Genco RJ. Microbiological and immunological studies of adult periodontitis in patients with non-insulin dependent diabetes mellitus. *J Periodontol* 1988;59:23-31.
17. Blandino G, Milazzo I, Fazio D, Puglisi S, Pisano M, Speciale A, Pappalardo S. Antimicrobial susceptibility and β -lactamase production of anaerobic and aerobic bacteria isolated from pus specimens from orofacial infections. *Journal of Chemotherapy*. 2007 Oct 1;19(5):495-9.
18. Pussinen PJ, Paju S, Mantyla P, Sorsa T. Serum microbial-and host-derived markers of periodontal diseases: a review. *Current medicinal chemistry*. 2007 Sep 1;14(22):2402-12.
19. Ruby J, Barbeau J. The buccale puzzle: the symbiotic nature of endogenous infections of the oral cavity. *Canadian Journal of Infectious Diseases and Medical Microbiology*. 2002;13(1):34-41.
20. Lopez-Marcos JF, Garcia-Valle S, Garcia-Iglesias AA. Periodontal aspects in menopausal women undergoing hormone replacement therapy. *Medicina oral, patologia oral y cirugia bucal*. 2005;10(2):132-41.
21. Almeida M, Laurent MR, Dubois V, Claessens F, O'Brien CA, Bouillon R, Vanderschueren D, Manolagas SC. Estrogens and androgens in skeletal physiology and pathophysiology. *Physiological reviews*. 2016 Nov 2;97(1):135-87.
22. Grodstein F, Colditz GA, Stampfer MJ. Post-menopausal hormone use and tooth loss: a prospective study. *J Am Dent Assoc* 1996;127:370-377.
23. Taguchi A, Sanada M, Sueti Y, Ohtsuka M, Nakamoto T, Lee K, Tsuda M, Ohama K, Tanimoto K, Bollen AM. Effect of estrogen use on tooth retention, oral bone height, and oral bone porosity in Japanese postmenopausal women. *Menopause* 2004; 11: 556-562.
24. Ronderos M, Jacobs DR, Himes JH, Pihlstrom BL. Associations of periodontal disease with femoral bone mineral density and estrogen replacement therapy: cross-sectional evaluation of US adults from NHANES III. *J Clin Periodontol* 2000; 27: 778-786.
25. Pizzo G, Guiglia R, Licata ME, Pizzo I, Davis JM, Giuliana G. Effect of hormone replacement therapy (HRT) on periodontal status of postmenopausal women. *Medical science monitor: international medical journal of experimental and clinical research*. 2011;17(4):PH23.
26. Loe H. Periodontal disease. The sixth complication of diabetes mellitus. *Diabetes Care* 1993; 16(1):329-34.
27. Firatli E. The relationship between clinical periodontal status and insulin-dependent diabetes mellitus. Results after 5 years. *J Periodontol* 1997; 68(2):136-40.
28. Seppälä B, Seppälä M, Ainamo J. A longitudinal study on insulin-dependent diabetes mellitus and periodontal disease. *Journal of clinical periodontology*. 1993 Mar;20(3):161-5.
29. Thorstensson H, Hugoson A. Periodontal disease experience in adult long-duration insulin-dependent diabetics. *Journal of Clinical Periodontology*. 1993 May;20(5):352-8.
30. Pucher J, Stewart J. Periodontal disease and diabetes mellitus. *Current Diabetes Reports*. 2004 Feb 1;4(1):46-50.
31. Salvi GE, Kandyaki M, Troendle A, Persson GR, Lang NP. Experimental gingivitis in type 1 diabetics: A controlled clinical and microbiological study. *J Clin Periodontol* 2005;32:310-316.

Source of support: Nil

Conflict of interest: None declared

This work is licensed under CC BY: *Creative Commons Attribution 3.0 License*.