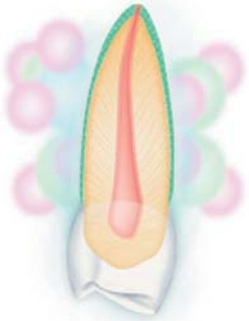
the Journal of CONTEMPORARY DENTAL PRACTICE at www.thejcdp.com
Volume 5 Number 2 May 15, 2004 Spring Issue

The Comparison of the Thickness of the Cementum Layer in Type 2 Diabetic and Non-diabetic Patients

Kamer Gokhan, PhD; Nurullah Keklikoglu, PhD; Mete Buyukertan, PhD



Abstract

It has been suggested diabetes plays an important role in tooth loss. Studies examining the periodontal structures and the alveolar bone architecture in diabetics have revealed some alterations. However, mechanisms responsible for these alterations have not been elucidated completely. Established relationships between the cementum layer, which is resistant to resorption, and diabetes are few in number. The aim of this study was to compare the thickness of the cementum layer in Type 2 diabetic and non-diabetic subjects in order to improve the understanding of dental mobility in Type 2 diabetes and its effect on tooth loss. A total of 46 male patients with a mean age of 61.72 ± 5.45 yr. were included in this study (Type 2 diabetics, n=23; non-diabetics, n=23); undecayed. Single rooted premolar teeth extracted from 46 male patients were used to assess the alterations in the cementum layer in Type 2 diabetics. Histological preparations from extracted teeth were examined under light microscopy. In each tooth, the thickness of the cementum layer was measured by an oculometer in 4 different sites on the specimen with the largest pulp space including the pulp chamber and root canals. Statistical analyses were performed with student's *t* test.

The average thickness of the cementum layer in the decalcified teeth extracted from non-diabetic patients was compared to the average thickness of the cementum layer in Type 2 diabetic patients. A significant difference was observed in every site of measurement between Type 2 diabetic and non-diabetic patients with regard to the thickness of the cementum layer ($p < 0.05$). The cementum layer was thicker at the apical part of the root and at the midpoint of the apical half, and thinner at the central part of the root and at the midpoint of the coronal half in Type 2 diabetic patients.

Keywords: Cementum, diabetes, resorption

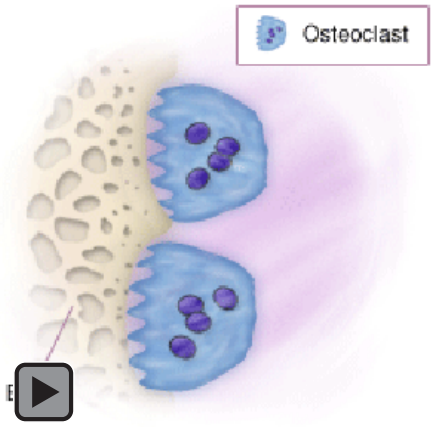
Citation: Gokhan K, Keklikoglu N, Buyukertan M. Comparison of the Thickness of the Cementum Layer in Type 2 Diabetic and Non-diabetic Patients. J Contemp Dent Pract 2004 May;(5)2:124-133.

Introduction

Several trials have demonstrated a relationship between diabetes and tooth loss.[1-10] The status of the periodontal disease was also examined in diabetics, and significant alveolar bone loss was observed.[11] However, structural changes in the cementum of diabetics has not been evaluated adequately with regard to its relationship with mobility of the teeth and eventual tooth loss.

The increased frequency of tooth loss in diabetics has been associated with periodontitis, its accompanying tooth mobility, and deep pocket formation.[12] Diabetes has been implicated in the development of significant periodontal disease and with other significant local effects on the oral cavity that contribute to periodontal disease.[13] Systemic diseases and hormonal changes have also been associated with complications of periodontal diseases. It has been demonstrated diabetes was a risk factor for periodontal disease.[12]

The effects of metabolic diabetes are multifaceted including plaque microbiology, vascular changes, and alterations in the metabolism of collagen tissues and in immunological responses.[14] Compared to non-diabetic controls, glucose concentration in blood and gingival fluid in diabetics is higher; this results in qualitative changes in bacteria.[12] The concentrations of the nucleotide, cAMP (adenosine 3',5'-cyclic monophosphate), in diabetics is reduced and cAMP suppresses inflammation.[12] Despite all these studies, the exact role of microorganisms in tooth loss is unknown.[12]



Studies in Type 2 diabetic patients with periodontal disease that have examined the leukocyte function have shown an impairment in chemotaxis and phagocytic capabilities.[15] These contribute to the development of periodontitis. Another finding in Type 2 diabetics with periodontitis is a significant increase in the concentration of immunoglobulins.[16,17] Human leucocyte antigen complexes and cytokines also increase in periodontal diseases.[18] The most important potential consequence of these alterations is the stimulation of osteoclastic resorption.[18-20] Diabetes itself is not a direct cause of periodontal disease, but rather it facilitates the development of gingivitis and periodontitis through local pathological changes in the oral cavity.[21]

While periodontitis in diabetics is the subject of ongoing research, the histological changes in human teeth associated with diabetes have not been adequately studied. The thickness of the cementum layer in healthy subjects is thinnest in the cervical region at the junction of cementum and enamel (20-50 microns) and thickest in the apical region (150-200 micron).[22] Age, local trauma, and hyperactivity influence the thickness of the cementum layer. The cementum layer is resistant to resorption, and it has the capability to restore itself after resorption in those instances when resorption occurs.[12]

This study was conducted to compare the thickness of the cementum layer in Type 2 diabetic and non-diabetic subjects in order to improve the understanding of dental mobility in Type 2 diabetes and its effect on tooth loss.

Materials and Methods

Teeth extracted for various reasons (periodontal and prosthetic) from Type 2 diabetic (n = 23) and non-diabetic (n = 23) male patients being treated at the Bahcelievler Department of Oral and Dental Medicine (Istanbul) were studied. The mean age was 61.7 ± 5.4 yr. (minimum age 55; maximum age 74) Only one single rooted first or second premolar tooth from each participant was used for the assessments. Upon extraction, teeth were placed in a 10% buffered neutral formaldehyde solution and were kept there for 24 hours. After fixation, teeth were decalcified in 8% formic acid solutions, which were replaced every day for 7 days.

Cross-sections of 5 microns were prepared in the longitudinal axis of paraffin embedded teeth. The preparations were stained with H&E (Hematoxylin and Eosin) while measurements and a histological examination were performed with light microscopy (X100, with immersion oil) (Olympus Bx50, Japan) and metric oculars (oculometer). In each tooth, the thickness of the cementum layer was measured at four different sites on the specimen with the largest pulp space including the pulp chamber and root canals (Figure 1).

These sites are as follows:

1- The thickest layer of cementum at the apex (Figure 2).
2- The midpoint of the apical half of the root (Figure 3).
3- The central part of the root (Figure 4).
4- The midpoint of the cervical half of the root.

Statistical analyses were performed with SPPS 10.0, Excel (Prentice Hall) software. The statistical level of significance was set at $p < 0.05$. Between-group comparisons were performed with a student's *t* test. The thickness of the cementum layer was compared between Type 2 diabetic and non-diabetic subjects.

Findings

The average thickness of the cementum layer in the decalcified teeth extracted from non-diabetic patients was 113.1 ± 23.4 µm in the apical region, 98.9 ± 11.7 µm in the midpoint of the apical half of the root, 87.5 ± 13.1 µm in the central part of the root, and 40.2 ± 6.8 µm in the midpoint of the cervical half of the root.

The average thickness of the cementum layer in Type 2 diabetic patients was 175.0 ± 33.7 µm at the apical region, 159.1 ± 31.3 µm at the midpoint

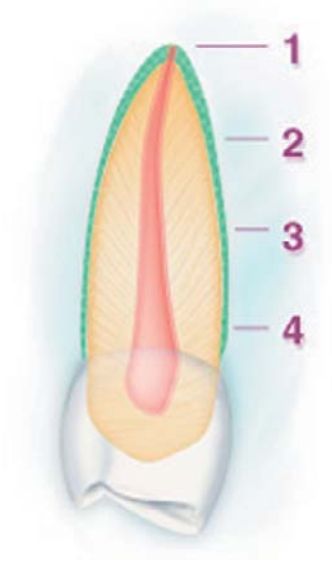


Figure 1. Measurement sites.

1-The thickest layer of cement at the apex.
2-The midpoint of the apical half of the root.
3-The central part of the root.
4-The midpoint of the cervical half of the root.

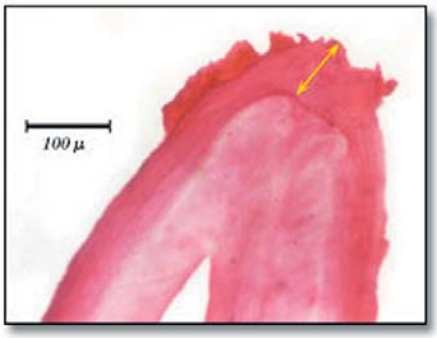


Figure 2. A histological section of a tooth of a diabetic person at the apical region with an arrow which is showing the site of the measurement.

of the apical half of the root, 54.0 ± 24.1 µm at the central part of the root, and 24.5 ± 7.5 µm at the midpoint of the cervical half of the root (Figure 5).

The comparison of the thickness of the cementum layer between the groups revealed the following: (Table 1)

1. A statistically significant difference with regard to the thickness of the apical cementum layer between Type 2 diabetic and non-diabetic subjects ($p < 0.05$) ($t = -4.994$).
2. A statistically significant difference with regard to the thickness of the cementum layer in the apical half of the root between Type 2 diabetic and non-diabetic subjects ($p < 0.05$) ($t = -5.969$).
3. A statistically significant difference with regard to the thickness of the cementum layer in the central point of the root between Type 2 diabetic and non-diabetic subjects ($p < 0.05$) ($t=4.044$).
4. A statistically significant difference with regard to the thickness of the cementum layer in the cervical half of the root between Type 2 diabetic and non-diabetic subjects ($p < 0.05$) ($t=5.132$).

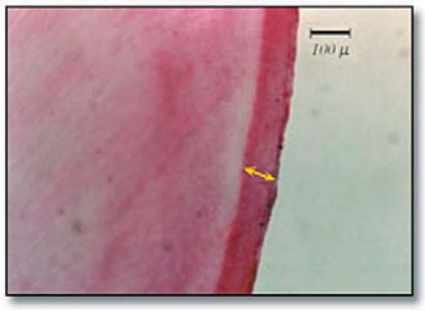


Figure 3. A histological section of a tooth of a diabetic person at the midpoint of the apical half of the root with an arrow which is showing the site of the measurement.

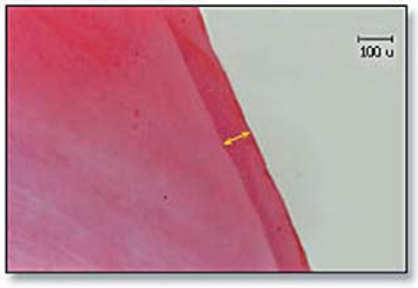


Figure 4. A histological section of a tooth of a diabetic person at the central part of the root with an arrow which is showing the site of the measurement.

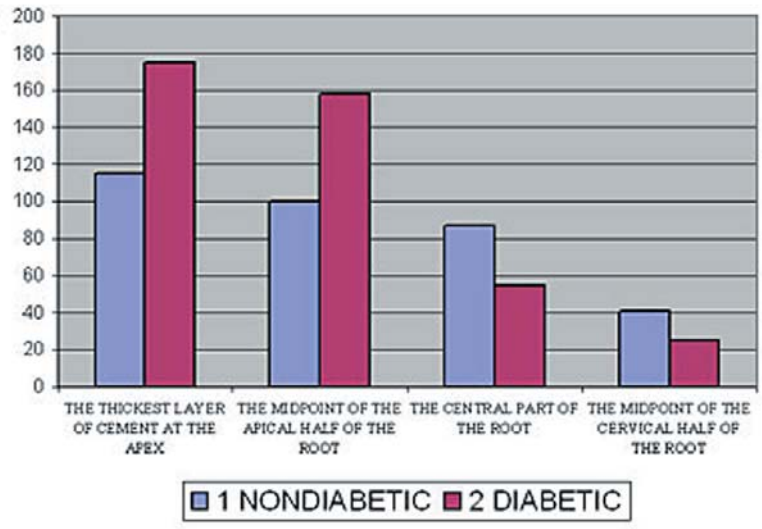


Figure 5. Graph comparing the cement thickness of non-diabetic and diabetic persons.

Table 1. Comparision of cementum thickness between diabetic and non-diabetic groups towards different places of the root.

Variable	Diabetic Group (n=23) Mean ± SD	Control Group (n=23) Mean ± SD
Apical cementum thickness	175.09* ± 5.74 µm	113.18* ± 4.78 µm
Apical middle cementum thickness	159.18* ± 5.53 µm	98.91* ± 3.38 µm
Middle cementum thickness	54.09* ± 4.85 µm	87.55* ± 3.58 µm
Cervical middle cementum thickness	24.55* ± 2.71 µm	40.27* ± 2.58 µm

*P<0.05 (student t test)

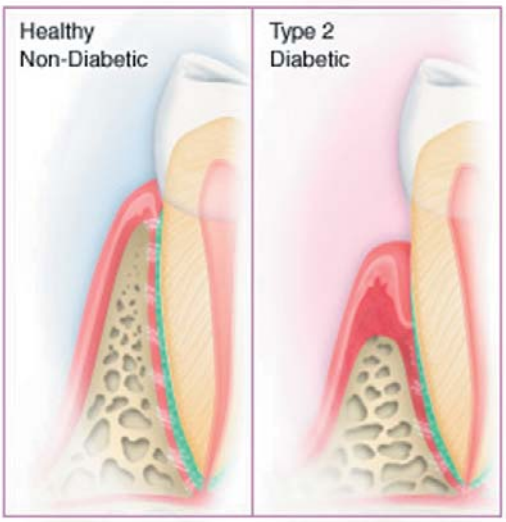
Discussion

Despite studies demonstrating an association between periodontal diseases and Type 2 diabetes[1-9], the mechanisms responsible for this association are not clear[23] and there have been controversial reports. Data from previous studies suggest impairment of humoral and cellular immune responses probably contributes to the development of periodontal disease in subjects with Type 2 diabetes. However conclusive evidence is lacking.[16,17,24,25,26] Premature tooth loss due to systemic diseases is usually the result of the changes in the immunologic system, or connective tissues.[27] The relationship between tooth loss and systemic factors have also been evaluated, and no correlation between Type 2 diabetes and the absence of teeth in mandible and maxilla has been established.[28]

In addition to periodontitis, Type 2 diabetes is related to other complications in the oral cavity including tooth decay, dry mouth, fungal infections, and oral and peripheral neuropathies.[29,30] The incidence of tooth loss is 15% higher in Type 2 diabetic subjects compared to healthy controls, and it has been suggested this difference can be accounted for by alveolar bone resorption, loss of attachment of the periodontal ligament to the cementum, and alveolar bone and alterations in the structure and thickness of the cementum layer.[12] Thus, our aim was to investigate the abovementioned potential mechanisms of increased tooth loss in Type 2 diabetics. In this study we measured the thickness of the cementum layer in the apical region, at the midpoint of the apical half of the root, at the central point of the root, and at the midpoint of the cervical half of the root.

Conclusion

There was a statistically significant difference with regard to the thickness of the cementum layer in every site a measurement was detected ($p < 0.05$). In Type 2 diabetic patients the thickness of the cementum layer at the apex and at the midpoint of the apical half of the root increased, while it was decreased in the central part of the root and at the midpoint of the cervical half of the root. The increase or the decrease in the thickness of the cementum layer can be idiopathic, may result from local factors, or can arise from systemic conditions like Type 2 diabetes. As a conclusion, we suggest histometric changes in cementum by people with Type 2 diabetes, due to loss of attachment of the periodontal ligament to the cementum and alveolar bone, can be considered as a reason for tooth loss.



References

1. Nelson RG, Schlossman M, Budding LM, et. al. Periodontal disease and NIDDM in Pima Indians. Diabetes Care. 1990 Aug;13(8):836-40.
2. Schlossman M, Knowler WC, Pettitt DJ, et. al. Type 2 diabetes mellitus and periodontal disease. J Am Dent Assoc. 1990 Oct;121(4):532-6.
3. Emrich LJ, Shlossman M, Genco RJ. Periodontal disease in non-insulin-dependent diabetes mellitus. J Periodontol. 1991 Feb;62(2):123-31
4. Loe H. Periodontal disease. The sixth complication of diabetes mellitus. Diabetes Care. 1993 Jan;16(1):329-34. No abstract available.
5. Sforza NM, Giardini E, Cicognani Simoncini F, et. al. Diabete mellito e terapia parodontale. Dental Cadmos. 1998;4:31-47.
6. Novaes Jr AB, Silva MA, Batista Jr EL, et. al. Manifestations of insulin-dependent diabetes mellitus in the periodontium of young Brazilian patients. A 10-year follow-up study. J Periodontol. 1997 Apr;68(4):328-34.
7. Grossi SG, Skrepcinski FB, DeCaro T, et. al. Treatment of periodontal disease in diabetes reduces glycated hemoglobin. J Periodontal.1997 Aug;68(8):713-9.
8. Baelum V, Luan WM, Chen X, et. al. A A 10-year study of the progression of destructive periodontal disease in adult and elderly Chinese. J Periodontol. 1997 Nov;68(11):1033-42.
9. Firatli E, Yilmaz O, Onan U. The relationship between clinical attachment loss and the duration of insulin-dependent diabetes mellitus (IDDM) in children and adolescents. J Clin Periodontol. 1996 Apr;23(4):362-6.
10. Kawamura M, Fukuda S, Kawabata K, et. al. Comparison of health behaviour and oral/medical conditions in non-insulin-dependent (type II) diabetics and non-diabetics. Aust Dent J. 1998 Oct;43(5):315-20.
11. Fontana G, Lapolla A, Sanzari M, et. al. An immunological evaluation of type II diabetic patients with periodontal disease. J Diabetes Complications. 1999 Jan-Feb;13(1):23-30.
12. Carranza FA. Clinical Perıodontology. 7. Edition. W.B. Saunders Company, Philadelphia,1 990.pp.446-50.
13. Wilson TG Jr, Higginbottom FL. Periodontal diseases and dental implants in older adults. J Esthet Dent. 1998;10(5):265-71.
14. Slakeski N, Cleal SM, Reynolds EC. Characterization of a Porphyromonas gingivalis gene prtR that encodes an arginine-specific thiol proteinase and multiple adhesins. Biochem Biophys Res Commun. 1996 Jul 25;224(3):605-10.
15. Cutler CW, Eke P, Arnold RR, et. al. Defective neutrophil function in an insulin-dependent diabetes mellitus patients. A case report. J Periodontol. 1991 Jun;62(6):394-401.
16. Anil S, Remani P, Vijayakumar T. Cell-mediated and humoral immune response in diabetic patients with periodontitis. Oral Surg Oral Med Oral Pathol. 1990 Jul;70(1):44-8.
17. Anil S, Remani P, Vijayakumar T, et. al. Total hemolytic complement (CH50) and its fractions (C3 and C4) in the sera of diabetic patients with periodontitis. J Periodontol. 1990 Jan;61(1):27-9.
18. Yang XD, Sytwu HK, McDevitt HO, et. al. Involvement of beta 7 integrin and mucosal addressin cell adhesion molecule-1 (MAdCAM-1) in the development of diabetes in obese diabetic mice. Diabetes. 1997 Oct;46(10):1542-7.
19. Mosmann TR, Coffman RL. Heterogeneity of cytokine secretion patterns and functions of helper T cells. Adv Immunol. 1989;46:111-47. Review. No abstract available.
20. Galbraith GM, Hagan C, Steed RB, et. al. Cytokine production by oral and peripheral blood neutrophils in adult periodontitis. J Periodontol. 1997 Sep;68(9):832-8.
21. Sanguedolce MV, Capo C, Bouhamdan M, et. al. Zymosan-induced tyrosine phosphorylations in human monocytes. Role of protein kinase C. J Immunol. 1993 Jul 1;151(1):405-14.
22. Avery JK. Oral development and histology , 4.th. Ed., Thieme, New York,2002.pp.231-5.
23. Reeves WG, Wilson RM. Infection, immunity and diabetes in Alberti KGMM, Defronzo RA, Keen H, Zimmet P (eds), International Textbook of Diabetes Mellitus. John Wiley and Son, 1992, pp.1165-71.

24. Hugoson A, Thorstennson H, Falk H, et. al. Periodontal conditions in insulin-dependent diabetics. J Clin Periodontol. 1989 Apr;16(4):215-23.
25. Zambon JJ, Reynolds H, Fisher JG, et. al. Microbiological and immunological studies of adult periodontitis in patients with noninsulin-dependent diabetes mellitus. J Periodontol. 1988 Jan;59(1): 23-31.
26. Cutler CW, Eke P, Arnold RR, et. al. Defective neutrophil function in an insulin-dependent diabetes mellitus patients. A case report. J Periodontol. 1991 Jun;62(6):394-401.
27. Hartsfield JK Jr. Premature exfoliation of teeth in childhood and adolescence. Adv Pediatr. 1994;41: 453-70. Review.
28. Xie Q, Ainamo A. Association of edentulousness with systemic factors in elderly people living at home. Community Dent Oral Epidemiol. 1999 Jun;27(3):202-9.
29. Finney LS, Finney MO, Gonzalez–Campoy JM. What the mouth has to say about diabetes. Careful examinations can avert serious complications. Postgrad Med. 1997 Dec;102(6):117-26. Review.
30. Oliver RC, Tervonen T. Diabetes--a risk factor for periodontitis in adults? J Periodontol. 1994 May;65(5 Suppl):530-8. Review.

About the Authors

Kamer Gokhan, PhD

Dr. Gokhan graduated from the Istanbul University Faculty of Dentistry in 1995 and obtained her doctoral degree in 2001 from the Department of Experimental Medicine and Research Center Molecular Medicine at Istanbul University. She now serves as a clinical dentist at Bogazici University Bogazici University Mediko Sosyal Merkezi in I˙stanbul, Turkey.

e-mail: kamer.gokhan@boun.edu.tr

Nurullah Keklikoglu, PhD

Dr. Keklikoglu is a specialist in Histology and Embryology in the Department of Histology and Embryology of Faculty of Dentistry at I˙stanbul University in I˙stanbul, Turkey.

e-mail: nkeklik@istanbul.edu.tr

Mete Buyukertan, PhD

Dr. Buyukertan serves in the Department of Anatomy in Faculty of Dentistry at I˙stanbul University in I˙stanbul, Turkey.

e mail: metebuyukertan@hotmail.com