Assessment of the effect of non-surgical periodontal treatment on the dentin hypersensitivity

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Abstract

Objective: The aim of this study was to evaluate the periodontal treatment effect on dentin hypersensitivity. **Methods:** This study comprised 20 patients diagnosed and treated of chronic periodontitis in the Dental Clinic of Uningá School of Dentistry (Maringá, Brazil). Patients were evaluated for dentin sensitivity by VAS scale before non-surgical periodontal treatment and after 10 to 14 days of its conclusion. **Results:** According to the VAS scale, patients presented an average sensitivity of 3.05 + 3.00 before the beginning of periodontal treatment. At the end of the treatment there was a significant reduction to 1.00 + 1.45 in the average dentin hypersensitivity. **Conclusion:** Based on the outcomes of the present study, it can be concluded that non-surgical periodontal treatment, associated to a modification of oral hygiene habits, was able to reduce dentin hypersensitivity in subjects with chronic periodontitis.

Keywords: Root planing. Dentin sensitivity. Periodontitis.

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Introduction

The dentinal hypersensitivity, also called hyperesthesia, is a momentary and stimulated discomfort reaching the dentinal tubules exposed by the loss of enamel and cementum structures, with unprotected dentin while suffering degradation. This causes pain in response to predisposing factors.[1]

Several factors may favor the onset of this hypersensitivity in which the erosion and abrasion are the most important factors due to a diet rich in acids and improper toothbrushing with excessive use of force and/or hard bristle brushes.[2] This condition reaches subjects of several age groups, but most carriers are in the age range between 20 and 40 years.[3] It occurs more in women than in men on the buccal surfaces of canines and premolars.[4]

The mechanism responsible for the presence or absence of pain through the exposure of dentinal tubules has led students to propose theories to explain the onset of hypersensitivity. Among them, the hydrodynamic theory proposed by Brännström in the 60's is the most accepted.[19] According to the author, when the dentinal tubules are exposed and there is stimulus on the tooth surface, the dentinal fluid inside the tubules moves. This displacement of intratubular fluid can activate recipient mechanics in the intratubular nerves and stimulate and distort nerve fibers present among odontoblasts, producing a painful feeling.[5]

With the development of periodontal diseases, and the consequent destruction of the periodontium, the formation of gingival recessions occurs. These gingival exposures cause the onset of dentin hypersensitivity.[6,7]

The prevalence of dentin hypersensitivity in adults varies considerably.[7-10] Epidemiological studies which considered the sensitivity reported by the patient indicate prevalence of 9 to 52%, with higher incidences occurring in studies using clinical tests as an air jet or mechanical stimuli.[6,10]

In patients evaluated in the section of Periodontics at a post-graduation hospital, Chabanski et al[9] reported the prevalence is much higher, with complaint of sensitivity to mechanical stimuli ranging from 73 to 98%.

In a systematic review, von Troil et al[13] reported scientific evidence is scarce regarding the effect of periodontal treatment on the dentin hypersensitivity. Therefore, the objective of this study was to evaluate the effect of periodontal disease and its treatment on the dentin hypersensitivity.

Material and Methods

A field research was performed in patients from the Dental Clinic of Uningá School of Dentistry, and this clinical trial was approved by the Independent Ethics Committee of the school, after the patients had signed consent forms for participation in the study.

Twenty patients diagnosed with chronic periodontitis, aged between 35 and 57 years, of both sexes, who are systemically healthy were evaluated.

The patients underwent the non-surgical periodontal treatment performed up to 4 sessions, depending on the extension of the periodontal disease. This treatment consisted of oral hygiene instruction and motivation; coronal scaling and root planing of sites with probing depth higher than 4 mm and with presence of bleeding on probing, using Gracey curettes (Hu-Friedy Manufacturing, USA) under local anesthesia as required (2% mepivacaine with epinephrine 1:100,000).

The parameter of dentine hypersensitivity was evaluated with according to the VAS scale before the beginning

of the periodontal treatment and about 10 to 14 days after its completion. VAS scale has a scale from 0 to 10, where 0 is considered as no sensitivity and 10 as an intolerable sensitivity to any type of stimuli, such as cold, heat, wind, speech, mastication, occlusion and others.

Descriptive analysis was expressed as mean values and standard deviations, and data on hypersensitivity considered to be non-parametric, using the Wilcoxon test to evaluate the difference between before and after the periodontal treatment. Significance level for rejecting the null hypothesis was defined at 5%.

Table 1 - Dentin hypersensitivity reported by patients before and after the non-surgical periodontal treatment with according to the VAS scale.

Patient	Initial hypersensitivity	Final hypersensitivity
1	3	1
2	1	0
3	0	0
4	3	1
5	5	1
6	8	6
7	2	0
8	0	0
8	7	1
10	9	1
11	0	0
12	2	1
13	0	2
14	2	2
15	0	0
16	0	0
17	5	2
18	3	1
19	3	3
20	8	0
mean ± s.d.	**3.05 ± 3.00**	**1.00 ± 1.45**

Results

Table 1 shows data on hypersensitivity using VAS scale for the 20 patients evaluated.

Initially, before the periodontal treatment, patients had mean hypersensitivity of 3.05±3.00, six patients experienced no hypersensitivity; eight experienced low hypersensitivity (1 to 3 points); two, moderate hypersensitivity (4 to 6 points); and four, severe hypersensitivity (7 to 10 points).

About 10 to 14 days after the completion of the periodontal treatment, the mean hypersensitivity decreases significantly ($p = 0.0026$) to 1.00±1.45: Eight patients experienced no hypersensitivity; eleven experienced low hypersensitivity (1 to 3 points), and, one experienced moderate hypersensitivity (4 to 6 points). At reassessment, no patient reported severe hypersensitivity (7 to 10 points).

Only one patient experienced an increased hypersensitivity, twelve experienced a decreased sensitivity and seven continued with the same grade of sensitivity, including those who had no baseline sensitivity.

Discussion

This study evaluates the condition of dentine hypersensitivity in patients with periodontal disease, and also the influence of periodontal treatment on the grade of hypersensitivity. The results show periodontal disease alone can cause dentine hypersensitivity — since 70% of patients had some initial grade of initial sensitivity - and the periodontal treatment can reduce significantly this hypersensitivity

Karlsson and Penney[14] demonstrated the sensitivity process after scaling and root planing in dogs. The sensitivity was measured by electrical stimuli in the treated root surfaces. The onset of the reduction of

dentine hypersensitivity occurred 2 to 4 days after completion of scaling and root planing; from the tenth day, the sensitivity decreased gradually compared to the baseline measurement. Some clinical studies have shown an increase in the dentin sensitivity within 2 to 4 days after supra and subgingival scaling; however, this sensitivity decreases after about two weeks.[15,16] This study showed the non-surgical periodontal treatment decreased dentin the hypersensitivity, considering the final assessment was performed 10 to 14 days after the completion of non-surgical treatment, such results corroborated by other studies.

A decrease of dentin hypersensitivity can occur by reducing the plaque index — which is considered by many periodontists as causal factor of increased sensitivity. In contrast, the new calculus formed on the root surface obstructs the dentinal tubules and facilitates the formation of reparative dentin, which possibly explains a desensitization.[17] Other mechanisms have been suggested to contribute for natural process of desensitization, as reviewed by Pashley.[17] However, bacteria accumulated at that site release their metabolic products, mainly organic acids, causing wear of the dentinal tubules, which make them more open and cause potential dentine sensitivity at any time.[12] Thus, the periodontal treatment performed using supra or subgingival scaling and proper oral hygiene instruction, as performed in this study, removes the bacterial biofilm with no release of metabolic products or organic acids, avoiding exposure of dentinal tubules and promoting the regression of the dentine sensitivity condition, providing comfort and relief to the patient in response to offending agents.

Another explanation for the changes in dentine sensitivity after periodontal therapy is proposed by Addy et al,[18] which demonstrated an in vitro study in which the surfaces treated by scaling and root planing are covered by smear layer, described as microcrystals from cementum and dental debridement, changing the sensitivity. Clinical observations have shown this smear layer becomes the teeth less sensitive to stimuli, and the covered areas have less sensitivity compared to those more exposed to the oral cavity. 19 The longevity of smear layer is unknown, but the loss over the time is assigned to the effects of acid diet and brushing.[18]

In addition, many patients have exposed root surfaces but they have no dentine hypersensitivity.[7,8,9] Some explanations for this fact may be given through obstruction of dentinal tubules, and then decreased external stimuli. Studies with scanning electron microscopy showed most dental tubules in a site with higher hypersensitivity are less obstructed; and sites with lower sensitivity, the tubules are more obstructed. These findings explain the fact some patients do not have dentine hypersensitivity, even with the dentin surface exposed. The presence of cementum on the root surface and its incomplete extraction after the root scaling procedure may be another reason for the absence of hypersensitivity in these subjects.[20]

Another explanation for the changes in dentin hypersensitivity in different patients is on the size and shape of the exposed tooth area which depends on the offending agent and it may influence the sensitivity. Studies of dentin anatomy revealed differences between the dentin with sensitivity and that without sensitivity.[20,21] In hypersensitive teeth, wider dentinal tubules in larger amount compared to those without sensitivity were found.[21]

Symptoms of dentin hypersensitivity may regress without treatment, as well as the dentin permeability can decrease spontaneously. But, in case this and desensitization do not occur by natural processes — such as reparative dentin, sclerotic dentin and calculus

formation on dentin surface — the therapy itself is defined according to the severity of the problem.[11,12] The treatment is quite diversified; however, the diet control and toothbrushing habits of the patient are of paramount importance. It should control the amount of acids ingested, as well as the interval between the alimentation and brushing. Any treatment may fail if these factors are not controlled, since they are notable primary etiological factors.[13]

No desensitizing agent or technique completely fills the definition of an optimal product.[7] This can be explained by the fact that the exact mechanism of dentine hypersensitivity is unclear. However, predisposing factors related to the causes of dentine hypersensitivity should be controlled, modified or eliminated through the patient's education regarding the diet, in other words, recommendations to reduce the excessive consumption of acidic foods, and also instruction of toothbrushing and occlusal analysis.[1]

Conclusion

Within the limits of this study, it can be concluded that periodontal disease produces dentine hypersensitivity and non-surgical periodontal treatment can decrease sensitivity levels.

REFERENCES

1. Yui KCK, Benetti AR, Valera MC, Gomes APM. Hipersensibilidade dentinária: etiologia, mecanismo de ação, diagnóstico e tratamento. Rev Odontol UNICID. 2004;16(1):63-71.
2. Estrela C, Pesce HF, Silva MT, Fernandes JMA, Silveira HDP. Análise da redução da dor pós-tratamento da hipersensibilidade dentinária. ROBRAC. 1996;6(17):4-9.
3. Furlan AM, Sallum AW, Sallum EA, Nociti-Junior FH, Casati MZ, Ambrosano GMB. Incidência da recessão gengival e hipersensibilidade dentinária na clínica de graduação da FOP-UNICAMP. Periodontia. 2007;17(1):53-61.
4. Sallum AW, Bueno R, Silva Filho W. A dor que tira o prazer e o sorriso. J Assoc Bras Odontol. 2010;124:6-7.
5. Aranha AC. Hipersensibilidade dentinária e lesões cervicais não-cariosas: causas, etiologia e prevalência. Rev Assoc Bras Odontol Nac. 2009;17(4):5-9.
6. Fischer C, Fischer RG, Wennberg A. Prevalence and distribution of cervical dentine hypersensitivity in a population in Rio de Janeiro, Brazil. J Dent. 1992;20(5):272-6.
7. Sallum AW, Rösing CK, Lotufo, RFM. Guia prático etiologia, diagnóstico e manejo clínico da hipersensibilidade dentinária cervical. Odontol Baseada Evid Colgate. 2008;1(1):1-26.
8. Murray LE, Roberts AJ. The prevalence of self reported hypersensitive teeth. Arch Oral Biol. 1994;39(suppl.):129S.
9. Chabanski MB, Gillam DG, Newman HN. Prevalence of cervical dentine sensitivity in a population of patients referred to a specialist Periodontology Department. J Clin Periodontol. 1996;23(11):989-92.
10. Graf H, Galasse R. Morbidity, prevalence and intraoral distribution of hypersensitive teeth. J Dent Res. 1977;56(Spec Issue A):479-62.
11. Vale ISD, Bramante AS. Hipersensibilidade dentinária: diagnóstico e tratamento. Rev Odontol Univ São Paulo. 1997;11(3):207-13.
12. Aranha ACC, Marchi GM. Hipersensibilidade dentinária: aspectos gerais e tratamento. J Bras Clin Odontol Int. 2004;8(44):179-84.
13. von Troil B, Needleman I, Sanz M. A systematic review of the prevalence of root sensitivity following periodontal therapy. J Clin Periodontol. 2002;29(Suppl 3):173-7.
14. Karlsson UE, Penney DA. Natural desensitization of exposed root in dogs. J Dent Res. 1975;54(5):982-6.
15. Fischer C, Wennberg A, Fischer RG, Attström R. Clinical evaluation of pulp and dentine sensitivity after supragingival and subgingival scaling. Endod Dent Traumatol. 1991;7(6):259-65.
16. Tammaro S, Wennström JL, Bergenholtz G. Root-dentin sensitivity following non-surgical periodontal treatment. J Clin Periodontol. 2000;27(9):690-7.
17. Pashley DH. Dentin permeability, dentin sensitivity, and treatment through tubule occlusion. J Endod. 1986;12(10):465-74.
18. Addy M, Absi ED, Adams D. Dentine hypersensitivity: the effects of acids and dietary compounds on burred and root planed dentine. J Clin Periodontol. 1987;14:274-9.
19. Brannstrom M. The surface of sensitive dentine. Odont Rev. 1965;16:293-9.
20. Yoshiyama M, Masada J, Uchida A, Ishida H. Scanning electron microscopic characterization of sensitive vs. insensitive human radicular dentin. J Dent Res. 1989;68(11):1498-502.
21. Absi EG, Addy M, Adams D. Dentine hypersensitivity. A study of the patency of dentinal tubules in sensitive and non-sensitive cervical dentine. J Clin Periodontol. 1987;14(5):280-4.