Libyan International Medical University

Faculty of Basic Medical Science

2017-2018

Tooth Sensitivity in Fluorotic Teeth

Submitted by: Ahmed Fathi Shalloof

Submitted to: Dr. Fatima Altarhony

Submitter's Number: 1385

Submission Date: 08/26/2018.

This report is set to fulfill the requirements of 2nd year Dentistry

Abstract:

The aim of this study was to compare the demographic and clinical features of tooth sensitivity (TS) in subjects with and without fluorosis. A total of 2249 subjects (378 subjects with fluorosis and 1871 subjects without fluorosis) were examined for TS during a study period of one year and TS was determined in 122 subjects. The level of TS was evaluated on a visual analogue scale (VAS). The sensitivity evaluation was made by applying tactile and cold air stimuli. In teeth sensitive to any stimuli, the plaque index (PI), gingival index (GI), gingival recession (GR) and periodontal pocket depth (PPD) were recorded. Fluorosis was assessed using the Dean Index. One hundred and twenty-two participants were found to have TS (5.42%). The frequency of TS in subjects with fluorosis was 9.26%, while the frequency of TS in subjects without fluorosis was 4.65%. There were no significant differences between the groups for periodontal parameters except PI. The results of the study showed that the subjects with fluorosis may have been suffering from TS more than the subjects with normal dentition.

Introduction:

Tooth sensitivity also known as dentin hypersensitivity affects the tooth or exposed root surfaces. This occurs when the enamel that protects our teeth gets thinner, or when gum recession occurs, exposing the underlying surface, the dentin, thus, reducing the protection the enamel and gums provide to the tooth and root. Tooth sensitivity affects up to 57% of the population. The chief symptom of tooth sensitivity is rapid, sharp pain against tactile (i.e. tooth brushing), thermal (hot or cold) and chemical (acids and sweet) stimuli, as well as exposure to air.

Tooth quality relates to the tooth's ability to fulfil its function and is evaluated by measuring mechanical and structural properties of tooth material.[2] It was demonstrated in studies conducted on teeth with molar incisor hypomineralization that the mechanical and structural properties of tooth material are also related to other tooth characteristics such as TS.[3,4] Different properties of teeth and the effects of intrinsic and extrinsic factors on tooth quality have been investigated in several studies.[5–10] Despite the caries preventive effectiveness of fluoride, it was found to have some negative effects on tooth quality.[2]

Dental fluorosis is a common disorder of teeth associated with high fluoride intake, especially from drinking water containing high concentrations of fluoride. The adverse effect of excessive exposure to fluoride is dental fluorosis, which is a permanent hypomineralization in the subsurface of enamel, characterized in its mildest form by small, clearly visible, white flecks found on the cusp tips and on facial surfaces of permanent dentition. Fluorosis is mostly found on permanent teeth surfaces ranging from obvious white opaque areas (moderate form) to darkly stained and pitted enamel (severe form).[11]

Discussion:

The principal findings of this study were the higher TS frequency and number of sensitive teeth per subject in the fluorosis group compared to the non-fluorosis group. These results may be related to the effects of fluorosis on the structure of teeth. Mild to moderate enamel fluorosis makes the enamel more resistant to dental caries. However, recent research revealed that a systemic fluoride intake could have the opposite effect on dentin, making dentin more susceptible to dental caries and other defects such as tooth fractures. Dentin fluorosis has been found to distort the intertubular collagen network in dentin, thereby causing detrimental hypermineralization of dentin, resulting in a higher susceptibility to acid degradation.[14] In addition,the dentin in fluorotic teeth was characterized by

a highly mineralized sclerotic pattern when compared to healthy teeth or fluorotic enamel lesions. In response to the effects of severe fluorosis in the enamel, the dentin showed hypermineralization, as seen in other enamel disorders. Furthermore, it was also shown that there was a positive correlation between the dentin fluoride concentration and the dentin tubule size, demonstrating wider dentin tubules in teeth with higher levels of fluoride in the dentin.[2]The cause of the higher prevalence of TS in the fluorosis group may be associated with the changes in dentin tubule size and the both the enamel and dentin. But histological alterations in the teeth related to fluorosis were not investigated in this study. Studies using a questionnaire approach with the patients self-reporting their sensitivity levels without any subsequent clinical examination are likely to grossly overestimate the prevalence, as the sensitivity reported could be the result of a number of different pathologies.[3] Actually, in this study, the number of subjects who positively answered the question 'Have you any sensitivity to hot and/or cold foods, cold air, brushing, or sweet and/or sour foods in your teeth in your daily life?' was 645 (28.7%). However, the number of subjects with clinically determined TS was only 122 (5.4%).Dentin hypersensitivity (DH), which cannot be ascribed to any other form of dental defect or pathology, has been typically described as a 'short, sharp pain' arising from exposed dentin in response to thermal, evaporative, tactile, osmotic or chemical stimuli.[14]Studies regarding patients of periodontology clinics indicated that the prevalence of DH was higher compared to the general dental population.[10,11,12,13] The results of these studies showed that the prevalence of DH ranged from 60.3% to 98%. Periodontitis and periodontal treatment results in gingival recession and increases DH.[7,9] In order to discriminate between TS and DH, the participants in our study were not periodontally treated before recording their clinical measurements in order to eliminate a possible increase in sensitivity due to periodontal therapy. In addition, in order to eliminate the negative effects of a periodontal breakdown, the teeth with a clinical attachment loss of more than 3 mm were also excluded from our study.The frequency of TS in the fluorosis group was 9.26%, which was significantly higher than the prevalence noted in the non-fluorosis group in this study (P=0.0003) and also higher than the DH prevalence of the general dental populations noted in other studies.[5,6,7]In the present study, the participants were asked what the initiating factor for their TS was and they were allowed to make a choice of one or more from; hot or cold foods, sour or sweet foods, cold air, and brushing. The most reported initiating factor was hot or cold food in both groups. Similarly, cold was the provoking factor that was most frequently cited in the literature.[1-4]The second most prevalent provoking factor reported in the literature was heat.[4]The PI of the fluorosis group was found to be lower than those of the non-fluorosis group. This result is consistent with the results of our previous study.[6] It was found that plaque accumulation, gingival bleeding and inflammation were lower in subjects with fluorosis who were resident in Isparta compared to subjects with normal dentition who were resident in Konya, which is a non- fluorosis area in Turkey.[11] Similarly, it was reported that as the concentration of fluoride in drinking water increased, plaque accumulation on tooth surfaces decreased.[10] Moreover, it was shown that high level of fluoride in dentifrices reduces de novo plaque formation on tooth surfaces[13] because high fluoride concentrations inhibit the metabolic and physiological pathways of biofilms.[12] For this reason, although the enamel surfaces of fluorotic teeth have a high porosity, the amount of plaque deposited on these surfaces is lower than on nonfluorotic enamel surfaces.In our study, the percentage of subjects in the fluorosis group who smoked was higher than in the non-fluorosis group (P=0.009). However, there was no difference between the groups regarding GR. Smoking is

known to be a major risk factor for periodontal disease and attachment loss. There were conflicting results in the literature regarding the effect of smoking on DH. Some studies reported a higher frequency of gingival recession and dentin sensitivity in smokers.[9] However, other studies did not support a relationship between smoking and DH.[4] In the present study, the higher number of smokers in the fluorosis group compared to the non-fluorosis group may be coincidental. There were a number of limitations to our study. Since the study population was formed from subjects referred to the periodontology clinic, the sample in our study did not represent the general population. For this reason, the prevalence of TS in subjects with fluorosis was not determined; only the frequency of TS was determined in this population. As tooth sensitivity is a subjective symptom that may vary between individuals, each subject was treated as an experimental unit in this study. However, in this kind of study, it would be better if each tooth was the experimental unit instead.

Conclusion:

The aim of this study was to compare the demographic and clinical features of tooth sensitivity (TS) in subjects with and without fluorosis. And to increase the knowledge of dental trainees, dentists and specialists in the field of dentistry. The results of the study suggest that the subjects with fluorosis may have been suffering more from TS than the subjects with normal dentition. Further studies that use each tooth as an experimental unit, preferably considering the degree of pulpal inflammation, are needed in order to evaluate the effects of fluorosis severity on TS.

References:

1. Orchardson R, Gillam DG. Managing dentin hypersensitivity. J Am Dent Assoc. 2006;137:990–998.
2. Porto IC, Andrade AK, Montes MA. Diagnosis and treatment of dentinal hypersensitivity. J Oral Sci. 2009;51:323–332.
3. Miglani S, Aggarwal V, Ahuja B. Dentin hypersensitivity: Recent trends in management. J Conserv Dent. 2010;13:218–224.
4. Canadian Advisory Board on Dentin Hypersensitivity. Consensus-based recommendations for the diagnosis and management of dentin hypersensitivity. J Can Dent Assoc. 2003;69:221–226.
5. Aranha AC, Pimenta LA, Marchi GM. Clinical evaluation of desensitizing treatments for cervical dentin hypersensitivity. Braz Oral Res. 2009;23:333–339. Wang R, Weiner S. Human root dentin: structural anisotropy and Vickers microhardness isotropy. Connect Tissue Res. 1998;39:269–279.
6. Watts DC, El Mowafy OM, Grant AA. Temperature dependence of compressive properties of human dentin. J Dent Res. 1987;66:29–32.
7. Mahoney E, Holta A, Swain M, Kilpatrick N. The hardness and modulus of elasticity of primary molar teeth: an ultra-micro-indentation study. J Dent. 2000;28:589–594.
8. Kinney JH, Gladden JR, Marshall GW, Marshall SJ, So JH, Maynard JD. Resonant ultrasound spectroscopy measurements of the elastic constants of human dentin. J Biomech. 2004;37:437–441.
9. Kruzic JJ, Nalla RK, Kinney JH, Ritchie RO. Crack blunting, crack bridging and resistance-curve fracture mechanics in dentin: effect of hydration. Biomaterials. 2003;24:5209–5221.

10. Arola D, Reprogel RK. Effects of aging on the mechanical behavior of human dentin. Biomaterials. 2005;26:4051–4061.
11. Den Besten PK. Mechanism and timing of fluoride effects on developing enamel. J Public Health Dent. 1999;59:247–251.
12. 3. Jälevik B, Klingberg GA. Dental treatment, dental fear, and behavior management problems in children with severe enamel hypomineralization of their first permanent molars. Int J Pediatr Dent. 2002;12:24–32.
13. 4. Rodd HD, Boissonade FM, Day PF. Pulpal status of hypomineralized permanent molars. Pediatr Dent. 2007;29:514–520.
14. 2. Vieira APGF, Hancock R, Dumitriu M, Limeback H, Grynpas MD. Fluoride's effect on human dentin ultrasound velocity (elastic modulus) and tubule size. Eur J Oral Sci. 2006;114:83–88.