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Clinical evaluation of dentin hypersensitivity before and after application of desensitizing agents- An in vivo study

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Abstract

Objective: This in vivo study determines whether the application of different desensitizing agents on exposed dentin surfaces was effective in reducing dentin hypersensitivity.

Materials and Methods: 14 patients with 75 teeth having history of sensitivity were selected in the study. The teeth were isolated and initial sensitivity was recorded using Verbal Rating Scale (VRS) with two stimuli, probing and air stimulation. Then they were treated with following desensitizing agents Group 1- Oxa-Gel, Group 2- Seal and Protect, Group 3- Placebo Gel. Immediately after application the scores were recorded and patient was recalled after 7 days for 3 weeks.

Results: According to paired t-test all treatments even the placebo gel were capable of reducing sensitive scores for both stimuli. There was no significant difference between all treatments.

Conclusion: Both the desensitizing agents were effective in reducing Dentin hypersensitivity.

Keyword: Dentin Hypersensitivity, Oxa-Gel, Seal and Protect, Verbal Rating Scale

Introduction

Dentine hypersensitivity (DHS) is one of the most commonly encountered dental problems. When the stimuli is applied to exposed dentin like typically thermal, evaporative, tactile, osmotic or chemical it is characterized but short, sharp pain and which cannot be ascribed to any other dental defects or pathology.[1] Hypersensitivity may present in one area of the mouth, or on one specific tooth or on several tooth.[2] Dentin is covered by enamel in normal conditions and doesn't get affected to any of the direct stimulus.[3] The exposure of dentin tubules due to enamel loss by abrasion, erosion, abfraction or root surface exposure caused by gingival recession, periodontal treatment or a combination of both may produce strong dentin sensitivity.[3,4]

To explain the mechanism involved in dentine hypersensitivity several theories have been proposed over more than a century.[5] The most accepted mechanism of dentine hypersensitivity is the hydrodynamic theory which has been proposed by Brännström in 1964.[6] This theory claimed that the displacement of the tubule contents is rapid enough to deform nerve fiber in pulp or predentin or damage odontoblast cell. Both of these effects appear capable of producing pain.[7] This rapid fluid movement in turn activates the mechanoreceptor nerves of A group in the pulp.

Various studies showed that the incidence of DHS in most population's ranges between 10-30% of the general population and the age range varies from 20-50 years.[8] The higher incidence of DHS is reported in females than in males which may reflect hormonal influence and dietary practices.[9] In contrary, one study showed no difference in prevalence of dentine hypersensitivity in either gender, suggesting overall that as many males as females are susceptible.[10] The most frequently affected teeth are premolars (68.8%), followed by molars, canines and incisors.

Potassium oxalate is a desensitizing agent which obliterates the dentinal tubules, with the calcium oxalate crystals precipitation on the surface and inside the dentin tubules, and also nerve endings gets depolarized. The desensitizing agents act in short- and long term bases with this mechanism.[11] It seems impossible to get various resins to adhere to exposed dentin (acid-etched or not) that has been wet from inside of the tooth. Resin will not stay in place for any long time. However, current in vivo experiments have indicated that it may be possible to get the resin to penetrate the dentinal tubules.[12] Both approaches have shown appreciating results in treating dentin hypersensitivity.

The aim of this study is to evaluate the dentin hypersensitivity before and after application of different desensitizing agents.

Materials and Methods

The total number of patients was 14 of both genders (4 male, 10 female) which composed of 75 teeth with mean age of 30years who had atleast 4 cervical lesions with dentin hypersensitivity. Caries-free patients who had at least 4 cervical lesions with clinical diagnosis of moderate or severe dentin hypersensitivity, adequate oral hygiene, and absence of periodontal disease or parafunctional habits were considered as eligible for this study. Radiographic and clinical examination was done for selecting the patients.[25]

Diagnosis was done clinically by using a uniform source of light, having a conventional operating dental

light system, mouth mirror, explorer and periodontal probe. By using a film holder, two bitewing radiographs were taken for diagnostic purpose on each side of the mouth.[25] The teeth included should not have caries, cracks or fractures, extensive or unsatisfactory restorations, recent restorations involving the buccal surface, prosthesis or orthodontic appliances.[26] The power of paired-sample t-test was calculated to be 75% for a sample size of 14, the default significance level (alpha level) was set at 0.05, and the alternative was 2-sided.

According to Pereira JC et.al the degree of hypersensitivity was determined based on Verbal Rating Scale – VRS from 0 to 3, in which: 0=no discomfort, 1=minimum discomfort, 2=mild discomfort, and 3=intense discomfort.[13] The values were collected before and after the application of treatments. Two stimuli were given to each tooth: clinical probing (tactile stimulus) and air blast (thermal evaporative stimulus).[25] Slight manual pressure was given for probe stimuli on the cervical area of the tooth in the mesio distal direction. Air blast was applied with an air syringe for 1 s at the distance of 1 cm of the tooth surface to avoid desiccating the dentin surface.

This randomization was performed by listing all the selected teeth and recording the scores as per the patient response before applying the desensitizing agents and placebo. Then the three treatments were done to the same patient to correlate the date regarding his/her sensitivity threshold. In each patient the teeth were randomly divided into 3 groups according to the desensitizing agent used in the study: Group 1-Oxa-Gel (Kota Import's Ltda, São Paulo, SP, Brazil) (23 teeth), Group 2-Seal&Protect (Dentsply DeTrey GmbH, Konstanz, Germany) (25 teeth), Group 3- Placebo Gel (22 teeth). Immediately after the application of the agents the scores were recorded by the examiner.

There are 2 persons for conducting this study, Person-1(operator) – applies desensitizing agents to the selected teeth. Person-2 (examiner)- examines the teeth and do not know which teeth was treated with particular agent. The composition of the agent and procedure of application was discussed in Table 1 (T I). Before the application of desensitizing agents, all the selected tooth surfaces were cleaned with rubber cup and pumice. Air and water spray is used to remove the pumice remnants. Cotton rolls and suction device were used to isolate the operating field.

Table 1

Group	Composition	Manufacturers Instruction
Group 1 Oxa-Gel (n=23)	3% potassium oxalate monohydratate (pH 4) solution, carboxymethylcellulose gel	The solution was passively applied on the surface for 2 min followed by water rising
Group 2 Seal & Protect (n=25)	PENTA, nanofillers, triclosan, acetone (resin-based material)	Apply for 20 seconds. • Volatilize the acetone with a gentle stream of air. • Light-cure for 10 seconds. • Apply the second coat and light-cure.
Group 3 Placebo Gel (n=22)	carboxymethylcellulose gel, distilled water	The gel was applied on the surface for 2 min followed by water rising

The subjects were recalled after 7 days interval for 3 weeks from the time of application of agents. The subject responses were recorded according to VRS in the same manner and with the same order as done before.

Results

According to the paired t-test, all treatments for both stimuli showed reducing sensitivity scores. Table 2 (T2) According to ANOVA, the scores before treatment procedure for both the stimuli and those obtained after treatment showed statistical significant difference ($p<0.05$). The values are reduced after treatment procedure.

Table 2: Sensitive scores (mean ± SD) before and after treatments according to the groups and stimulus

Groups	Air blast stimulus scores			Probing stimulus scores		
	Before	After	p-value	Before	After	p-value
Oxagel	2.4 ± 0.41	2.12 ± 0.988	0.574	1.96 ± 0.97	1.88 ± 0.92	0.161
Seal and protect	2.36 ± 1.22	2.10 ± 1.19	0.664	2.04 ± 1.06	1.94 ± 0.91	0.327
Placebo	2.28 ± 1.20	2.24 ± 1.12	0.714	1.80 ± 0.91	1.78 ± 0.89	0.327

p-values obtained with paired t-test ($p< 0.05$) was considered to be significant

Discussion

This study was aimed at comparing the response of tooth before and after the application of two desensitizing agents and placebo gel as a control group.

Monopotassium-monohydrogen oxalate is the main component of Oxa-Gel desensitizing agent. Calcium oxalate crystals are precipitated in dentin tubules by the action of these agents. Pashley et. Al[14] finalised that the formulation of calcium oxalate crystals occurs 30 sec after the application oxalate-based solutions, thus decreasing the dentin permeability. It was also observed that these solutions were proven even more effective when applied for 60sec. In the present study we applied desensitizing agent for 2 min as per the Oxa-Gel manufacture instructions. So, it is sufficient time for crystal precipitation.

Various studies[15,16] found that dentin permeability was decreased by 75% by potassium oxalate formulation indicating the effectiveness of these products. Laboratory tests were conducted to evaluate the effectiveness of desensitizing agents or to know their clinical performance. But, laboratory tests do not show the clinical performance of the material, so clinical evaluations are mandatory to confirm the efficacy of the product.[3,13,17]

Seal and protect also reduced the permeability in a similar manner with Oxa-Gel. in hypersensitive teeth it seals the open dentinal tubules. Seal & Protect is a resin-based material that does not contain HEMA. It is a self-adhesive, light curing, translucent sealing material and contains nanofillers. The manufacturer has claimed that these nanoparticles can easily penetrate into open dentin tubules.

Some clinical studies have reported that the placebo effect influence how dentinal hypersensitivity is treated.[19,20,21,22] This effect is described as a complex physiological and psychological interaction that depends to a large extent on the relationship between the patient and professional.[19,23] A positive attitude towards treatment may attribute the central system pain inhibitors, which controls the painful stimuli of the periphery by releasing endorphins.[20] Although the placebo formulation consists of inert substances, the hypothesis of a mechanical tubules occlusion by the particles of the carboxymethyl cellulose cannot be discarded.

The positive results achieved in the placebo group suggest the need for not only further clinical studies in order to better understand such intriguing event, but also for standard clinical protocols for the study of dentin hypersensitivity.[24]

Conclusion

All the experimental desensitizing agents used in this study acted by blocking the dentinal tubules and were equally efficient in relieving dentinal hypersensitivity. There was no statistical significant difference among the three desentitizing agents. Placebo treatment reduced the degree of hypersensitivity, but not as equal as other two groups, suggesting that its effect plays an important role in this scenario and should be further investigated.

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