A randomized split mouth clinical trial of the application of the desensitizer agents for tooth sensitivity

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ABSTRACT

Background: Dentin hypersensitivity (DH) affects 3%-75% of the people and is one of the morbid tooth conditions. Hence in the present study we aim to examine the clinical effectiveness of 3 different desensitizing agents in decreasing pain of DH in time of 1 month.

Methods: Fifty subjects with cervical DH in at least one tooth in any three of the 4 quadrants were selected. VAS was used to note the pain. Each quadrant in an individual was randomly assigned. Profluorid varnish, Admira protect, and PRG-Barrier coat was used. VAS scores for the tactile and air stimuli were noted immediately after application, 1 week, and after 1 month. The data was analyzed keeping p<0.05 as significant.

Results: VAS significantly reduced for all three groups from the base line (p<0.001). Admira protect showed significant reduction of hypersensitivity scores at 1 month compared to other groups (p<0.001).

Conclusions: Admira protect was better at lowering the pain due to DH than PRG-barrier coat and Profluorid varnish after 1 month of application.

Keywords: Random clinical trial, Desensitizing agents, Dental sensitivity

INTRODUCTION

Dentin hypersensitivity (DH) may be caused by both physiological and pathological factors. 1 Incidence ranges from 3%-75%. 2-4 Also there is a noticed increasing trend in DH due to increased tooth retention protocols. There are various theories that tried to explain the pathogenesis of the DH. 5-10 The chief objective is the pain reduction in these cases, that might give relief to the patient. 11-13

Hence in the present study we aim to examine the clinical effectiveness of 3 different desensitizing agents in decreasing pain of DH in time of 1 month. The null hypotheses tested were that (1) the desensitizing agents are not able to reduce the pain resulting from DH and (2) the desensitizing effects do not differ between the tested agents when tactile and evaporative stimuli are applied.

METHODS

We conducted prospective study, at the department of endodontics, government dental college, Vijayawada. The study was conducted from June 2019 to July 2019.

Fifty subjects in the age groups of 25-70 years with DH in at least one tooth in any three of the four quadrants were selected. Patients exhibiting pain scores of ≥3 on the visual analog scale (VAS) were considered for the study.
We excluded those patients who have teeth with active carious lesions or who required restorative treatment, patients who were receiving periodontal treatment, patients who received desensitizing treatment within the last 6 months, patients who were using anti-inflammatory drugs, pregnant patients, or smokers.

The subjects were randomly assigned to one of the three treatment groups based on computer-generated random number. All the three quadrants in each individual were randomly allocated to one of the three treatment groups based on computer-generated random number. The study deployed two different operators: one operator recorded the baseline sensitivity scores for the teeth after evaporative and tactile stimuli by visual analog score system and the second operator who was not aware of the baseline values applied the desensitizing agents and recorded the sensitivity scores for both the stimuli. Thus, the study was double blinded (patient and the examiner).

The teeth were cleaned with pumice and rotary brush using slow-speed handpiece and isolated with cotton pellets and suction. Tactile stimulus was applied with an explorer in mesiodistal direction across the cervical area, and VAS score was recorded. For evaporative stimuli, the tooth was isolated from the adjacent teeth with cotton rolls. A 1 s blast of air from the three-way syringe at 40-65 Psi at 1-3 mm away and perpendicular to the cervical area was applied. The sensitivity was recorded using VAS scale.

Later the teeth were air-dried and isolated by cotton pellets and suction. In each quadrant, a single application of different desensitizing agents was randomly applied according to the manufacturer’s instructions (Table 1).

This study used a split-mouth model using at least 3 quadrants, and placebo was not included due to ethical reasons. The patients were instructed to avoid eating/drinking for 2 h and avoid brushing for 12 h. Hypersensitivity assessment was done immediately after application of desensitizing agents, after 1 week, and after 1 month using the tactile and evaporative stimuli.

The ethical clearance from the institutional committee was taken. Also, the patient consent was taken. The intergroup comparison was done using repeated measure ANOVA and post hoc Tukey’s multiple comparison tests (p<0.05 was considered statistically significant). The within-group comparison was done using repeated measure ANOVA and post hoc multiple comparison was done using Tukey’s honest significant difference (p<0.05 was considered statistically significant).

**RESULTS**

All the desensitizing agents showed significant (p<0.001) reduction in DH immediately after application, at 1 week, and 1 month compared to baseline mean VAS scores for both tactile and evaporative stimuli. There were 8 dropouts in this study.

Between the 3 agents soon after application, there was no significant difference between all 3 groups.

At 1-week follow-up, Admira protect (mean VAS, 0.00) and PRG-barrier coat (mean VAS, 0.30) groups were significantly effective in reducing (p<0.001) DH compared to Profluorid varnish group (mean VAS, 2.00).

At 1-month follow-up, Admira protect (mean VAS, 1.100) was significantly better (p<0.001) than Profluorid varnish (mean VAS, 2.500) and PRG-barrier coat (mean VAS, 1.700) for both tactile and evaporative stimuli (Table 1).

| Evaluation  | Test groups       | N  | Tactile/touch stimuli, mean (SD) | Significance | Air stimulus, mean (SD) | Significance |
|-------------|-------------------|----|---------------------------------|-------------|------------------------|-------------|
| Baseline    | PRG-barrier coat  | 50 | 2.20 (0.61)                     | 0.741       | 2.50 (0.7)             | 0.032       |
|             | Admira protect    | 50 | 2.30 (0.91)                     |             | 3.00 (1.45)            |             |
|             | Profluorid        | 50 | 2.41 (0.81)                     |             | 3.60 (1.2)             |             |
| Instantly   | PRG-barrier coat  | 50 | -                               | 0.341       | -                      | 0.000       |
|             | Admira protect    | 50 | -                               |             | -                      |             |
|             | Profluorid        | 50 | 0.11 (0.40)                     |             | 0.34 (0.80)            |             |
| 1 week      | PRG-Barrier coat  | 50 | -                               | <0.001      | -                      | <0.001      |
|             | Admira protect    | 50 | 0.21 (0.60)                     |             | 0.13 (0.69)            |             |
|             | Profluorid        | 50 | 1.40 (0.90)                     |             | 2.10 (1.20)            |             |
| 1 month     | PRG-barrier coat  | 50 | 0.50 (0.80)                     | <0.001      | 1.10 (1.01)            | <0.001      |
|             | Admira protect    | 50 | 1.50 (0.81)                     |             | 1.70 (1.32)            |             |
|             | Profluorid        | 50 | 1.81 (0.60)                     |             | 2.41 (1.20)            |             |
DISCUSSION

DH is described by sharp pain caused by the thermal or evaporative stimuli on exposed dentinal surfaces. Conferring to the hydrodynamic theory by Brannstrom, stimulation of dentin results in a flow movement in the dentinal tubules, either toward or away from the pulp which can cause a mechanical deformation of nerve endings in dentin or in dentin/pulp interphase resulting in pain transmission. Management for DH is chiefly focused on the occlusion of dentinal tubules. Other therapeutic modalities may work by a neural blocking mechanism.

Various mechanisms have been projected for occlusion of dentinal tubules. Occlusion can be done by the precipitation of proteins present in dentinal tubular fluid, precipitation of amorphous particles over exposed dentin surfaces and/or inside tubules, or by the formation of a superficial pellicle which may penetrate into the dentin tubules. The neural blocking method is done by the direct diffusion of potassium ions through dentin increasing its concentration in the pulp tissue which can block nerve impulse conduction by alteration of action potentials.

It may be hard to correctly quantify DH as it is a subjective condition. Earlier reported methods to provoke and quantify pain of DH are the evaporative method and the tactile method. The tactile method using a probe tip can cause movement of dentinal fluid as a result of dentin compression. An air blast can decrease the temperature at the exposed dentin surface and can cause evaporation of fluid inside the tubules. Dentinal fluid movement can also occur due to both these effects.

Pain due to DH using tactile and evaporative stimuli was determined by VAS. VAS has been reported to be the most appropriate method to diagnose pain levels as it allows for translation of subjective feedback into objective data.

In our trial study, patients with DH in at least three quadrants were selected. We evaluated Profluorid varnish, protect, and PRG-barrier coat as treatment modalities for DH by their application in different quadrants in the same patient.

Fluoride-containing compounds such as sodium monofluorophosphate, sodium fluoride, stannous fluoride, and fluorosilicate have been evaluated as therapeutic agents to treat dentin hypersensitivity. Fluoride varnish that adheres to dentin preserves the fluoride as long as possible. Immediate desensitization can be seen with the use of fluoride varnish, but since they exhibit low adhesion, they can be removed by saliva or by toothbrush abrasion. In the present study, Profluorid varnish which contains sodium fluoride was used.

Admira protect contains bisphenol A diglycidyl ether dimethacrylate and 2-hydroxyethyl methacrylate monomers, organic acids, and ormocer. Ormocer materials contain inorganic-organic copolymers and inorganic silanated filler particles. According to the manufacturer, it can bond to dentin similar to a self-etching adhesive. However it has been reported that it may not contain chemicals needed for polymerization. It may induce precipitation of proteins inside dentinal tubules thus reducing fluid movement. The fillers may enhance the wear resistance thereby resisting removal.

PRG-barrier coat is a light-curable varnish/desensitizer which is supplied as a base and active solutions. According to the manufacturer, the surface-partially reacted glass (S-PRG) filler is a bioactive trilaminar structure with a multifunctional glass core embedded in resin matrix. It can release and recharge fluoride ions.

All the desensitizing agents showed significant (p<0.001) reduction in DH immediately after application, at 1 week, and 1 month compared to baseline mean VAS scores for both tactile and evaporative stimuli. In addition, immediately after application, there was no significant difference between all three groups.

Similarly, Torres et al reported a significant reduction immediately after the application of Admira protect, Bifluorid 12, and Colgate pro-relief. Yu et al have also reported that one-bottle self-etching adhesives, gluma desensitizer, and Bifluorid 12 can cause an immediate reduction in DH. Samuels et al. have also had three agents and have reported a significant immediate reduction in DH in their study. Therefore, it can be interpreted that most desensitizing agents will cause an immediate and significant reduction in DH.

At 1 week and 1 month, all the desensitizing agents showed significant (p<0.001) reduction in DH compared to baseline mean VAS scores for both tactile and evaporative stimuli. However, Admira protect and PRG-barrier coat groups were significantly effective in reducing DH compared to Profluorid varnish group at 1 week. At 1-month follow-up, Admira protect was significantly better than Profluorid varnish and PRG-barrier coat for both tactile and evaporative stimuli.

Similarly, Torres et al reported a significant reduction in DH at 1 week using Admira protect, Bifluorid 12, or Colgate pro-relief. Another study reported the use of calcium, sodium phosphor-silicate desensitizer resulted in significant hypersensitivity reduction after 1 week and 4 weeks compared to baseline values. Erdemir et al reported that the three desensitizing agents (Pain-free, BisBlock, and seal and protect) used in their study provided effective desensitization for 4 weeks.

In the present study, there were few limitations. The gender bias was not considered. Only few desensitizing agents were compared. Admira protect showed better
reduction in pain of dentin hypersensitivity compared to other products evaluated in this study. Further studies using Admira protect for a longer period of time will have to be done to evaluate long-term performance in the treatment of DH.

CONCLUSION

From our study it was noted that Profluorid varnish, Admira protect, and PRG-barrier coat can lower dental hypersensitivity immediately after application, at 1 week, and 1 month compared to baseline mean VAS scores for both tactile and evaporative stimuli.

Admira protect and PRG-barrier coat groups were significantly more efficient in reducing DH as compared to Profluorid varnish group at one week. At 1-month follow-up, Admira protect was significantly better than Profluorid varnish and PRG-barrier coat for both tactile and evaporative stimuli.

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