Restorative Dentistry & Endodontics

RDE

Research Article

Check for updates

Single-session associative protocol for dentin hypersensitivity management: a 1-year randomized, blinded clinical study

Thayna Carolina Zeni (ID), Poliana Maria de Faveri Cardoso (ID),*
Rafael da Silva Vanolli (ID), Márcio José Mendonça (ID), Julio Katuhide Ueda (ID),
Veridiana Camilotti (ID)

Department of Restorative Dentistry, School of Dentistry, Western State University of Paraná, Cascavel, PR, Brazil

🔓 **OPEN ACCESS**

*Correspondence to
Poliana Maria de Faveri Cardoso, DDS
Department of Restorative Dentistry, School of Dentistry, Western State University of Paraná, 2069 Universitaria St. Cascavel, PR 858191110, Brazil.
Email: polif1704@gmail.com

ABSTRACT

Objectives: This study aimed to establish a single-session associative protocol for non-restorative management of dentin hypersensitivity (DH).

Materials and Methods: Twenty-four individuals with DH and a minimum sensitivity level of 4 on the visual analog scale (VAS) were selected. The study was conducted in a split-mouth design, with each participant ($n = 20$) having at least 1 affected tooth in all quadrants. The management protocols consisted of control group: universal adhesive, Neural Desensitizing Protocol group: 5% potassium nitrate, Mixed Desensitizing Protocol (PAM) group: 5% sodium fluoride and 5% potassium nitrate, Remineralizing Desensitizing Protocol (PDR) group: surface-partially reacted glass technology photopolymerizable varnish. Evaluations were performed immediately after application, at 1 week, 1 month, 2 months, and 12 months using the VAS sensitivity test.

Results: The scores were subjected to statistical analysis using the Friedman test ($p < 0.05$), Durbin-Conover test ($p < 0.05$), and Wilcoxon test ($p < 0.05$). At the 12-month evaluation, all groups showed statistically significant differences compared to the initial assessment. For the evaluation after 12 months, there was a statistically significant difference between the PAM group, the control group, and the PDR group.

Conclusions: It can be concluded that all groups were effective in controlling DH, but there were significant results in the control group and PDR group. The clinical relevance of this study is to demonstrate that the application of single-session desensitizing protocols can be effective in controlling DH for up to 12 months.

Trial Registration: Brazilian Clinical Trials Registry Identifier: RBR-4r63d7s

Keywords: Clinical trial; Dental research; Dentin hypersensitivity; Quality of life

INTRODUCTION

Dentin hypersensitivity (DH) is a prevalent clinical manifestation characterized by acute and short-lasting pain without association with any pathology [1-3]. Its etiology can be associated

ORCID iDs

Thayna Carolina Zeni
https://orcid.org/0000-0001-8917-4258
Poliana Maria de Faveri Cardoso
https://orcid.org/0000-0002-7073-278X
Rafael da Silva Vanolli
https://orcid.org/0000-0002-4670-8878
Márcio José Mendonça
https://orcid.org/0000-0002-6953-7135
Julio Katuhide Ueda
https://orcid.org/0000-0002-8664-942X
Veridiana Camilotti
https://orcid.org/0000-0002-3004-3939

with dentin exposure, resulting from the loss of dental enamel or exposure of root surfaces [4,5]. This loss can occur after periodontal therapy, parafunctional habits, acidic diets, or inadequate brushing [6].

DH is commonly associated with non-carious cervical lesions, which are the manifestation of dental structure loss in the cementoenamel junction (CEJ) and not related to dental caries [7]. The loss of dental structure in the CEJ leads to a response to external stimuli (tactile, thermal, chemical, or osmotic), causing discomfort and reducing the quality of life [8]. The most accepted theory for describing DH is the hydrodynamic theory proposed by Braennstroem and Astroem [8], in which intratubular fluid movement occurs when dentin is exposed to stimuli, resulting in acute and short-lasting pain.

The diagnosis can be made through clinical tests such as the evaporative test, tactile test, or by excluding other painful pathologies [9-11]. The main methods of control involve the use of neural agents, obliterating agents, mixed approaches, and associated protocols. Neural agents act on the transmission of nerve impulses by depolarizing the extracellular concentration of ions in neural membranes, preventing repolarization, and consequently reducing the symptoms of DH [12]. Obliterating agents work by sealing dentinal tubules through protein precipitation, remineralizing the structure, sealing the tubules, and reducing fluid flow within the tubules [13].

Recently, a photopolymerizable varnish with bioactive properties has been introduced in the dental market. This varnish releases ions that assist in the control of DH through the process of remineralization, in addition to providing a hermetic seal on the dental surface [14]. This material contains surface-partially reacted glass particles, composed of a bioactive trilaminar structure, and ions such as sodium, aluminum, borate, silicate, fluoride, and strontium, each with specific actions that work synergistically to control DH and prevent dental caries by remineralizing the dental surface [15-17].

Fluoride, borate, aluminum, and silicate have antibacterial properties, while strontium, sodium, and aluminum neutralize acids produced by bacteria, reducing the formation of dental biofilm [17-20]. Fluoride transforms hydroxyapatite into fluorapatite, and strontium binds to hydroxyapatite, forming strontium-apatite, providing dentin protection and greater resistance to temperature and pH changes [21,22].

The application of an associative protocol involving the use of neural agents followed by obliterating agents in a single clinical session may represent a promising alternative to maximize their benefits [23]. This sequence cannot be altered due to the mechanism of action of the agents used, where the neural agent is applied first to depolarize the nerve fibers, followed by the obliterating agent to seal the dentinal tubule openings. The hypothesis of this study is that the application of an associative protocol using neural agents followed by obliterating agents in a single clinical session will result in a significant reduction in DH due to the synergistic combination of the depolarizing and sealing effects on dentinal tubules provided by the agents used.

MATERIALS AND METHODS

Ethics approval and protocol registration

The experimental design followed the Consolidated Standards of Reporting Trials (CONSORT) statement (Sarkis) and was registered in the Brazilian Clinical Trials Registry (RBR-4r63d7s). The study protocol was reviewed and accepted by the Local Ethics Committee on Investigations Involving Human Subjects (CAAE 25889819.6.0000.0107). All patients who met the selection criteria were informed of the study's objectives, procedures, risks, and benefits and expressed consent to participate by signing the Terms of Free and Enlightened Consent.

Trial design, settings, and recruitment

This randomized, prospective, double-blind, and parallel clinical trial was conducted between September 2021 to October 2022. This randomized clinical trial had dentin sensitivity as the outcome evaluated, and the variation factor was follow-up times. The participants were recruited through advertisements published in the local community.

Randomization, allocation concealment, and blinding

This controlled clinical trial had an equal allocation rate to the groups. Based on the inclusion and exclusion criteria, the selected teeth were divided into 4 groups using block randomization (4 × 5) of the Research Randomizer software version 4.0. During the application of the interventions experiments, the patient did not know to which experimental group belonged, not allowing this to interfere with the patient's perception of sensitivity. According to the CONSORT flow diagram, the distribution and dynamics of the groups are shown in **Figure 1**.

Eligibility criteria

Individuals aged 18 to 60 years, both sexes, without systemic compromise, non-smokers, with at least 4 teeth with DH (at least 1 tooth per quadrant) on the buccal surface, sensitive to evaporative stimuli, with gingival recession equal to or less than 2 mm, without using methods to control DH in the last 6 months, without using antibiotics in the last 6 months, and with a visual analog scale (VAS) value of sensitivity greater than 4 cm were included.

Patients with the following conditions were excluded from the study: teeth covered by prosthetics or with endodontic treatment; patients in constant use or with a medical history marked by chronic use of analgesics; anti-inflammations and psychotropic drugs; patients with orthodontic appliances; patients who have used desensitizing products in the past 3 months; patients who have undergone restorative treatment on the sensitive element in less than a month ago; removable partial denture abutment teeth; the presence of lesions with great depth (> 3 mm) who needed pulp protection; the presence of carious cervical lesion; pregnant or lactating [20].

Sample size calculation

The sample calculation was performed using the G*Power program (version 3.1.9.2 - University of Düsseldorf, Düsseldorf, Germany), based on probability distributions of the F family, with a repeated measures design, including within- and between-factor interactions. An effect size of 0.80 was obtained, measured in the between-groups comparison, resulting in 22 participants to achieve a power of 90%, with a significance level α of 5%.
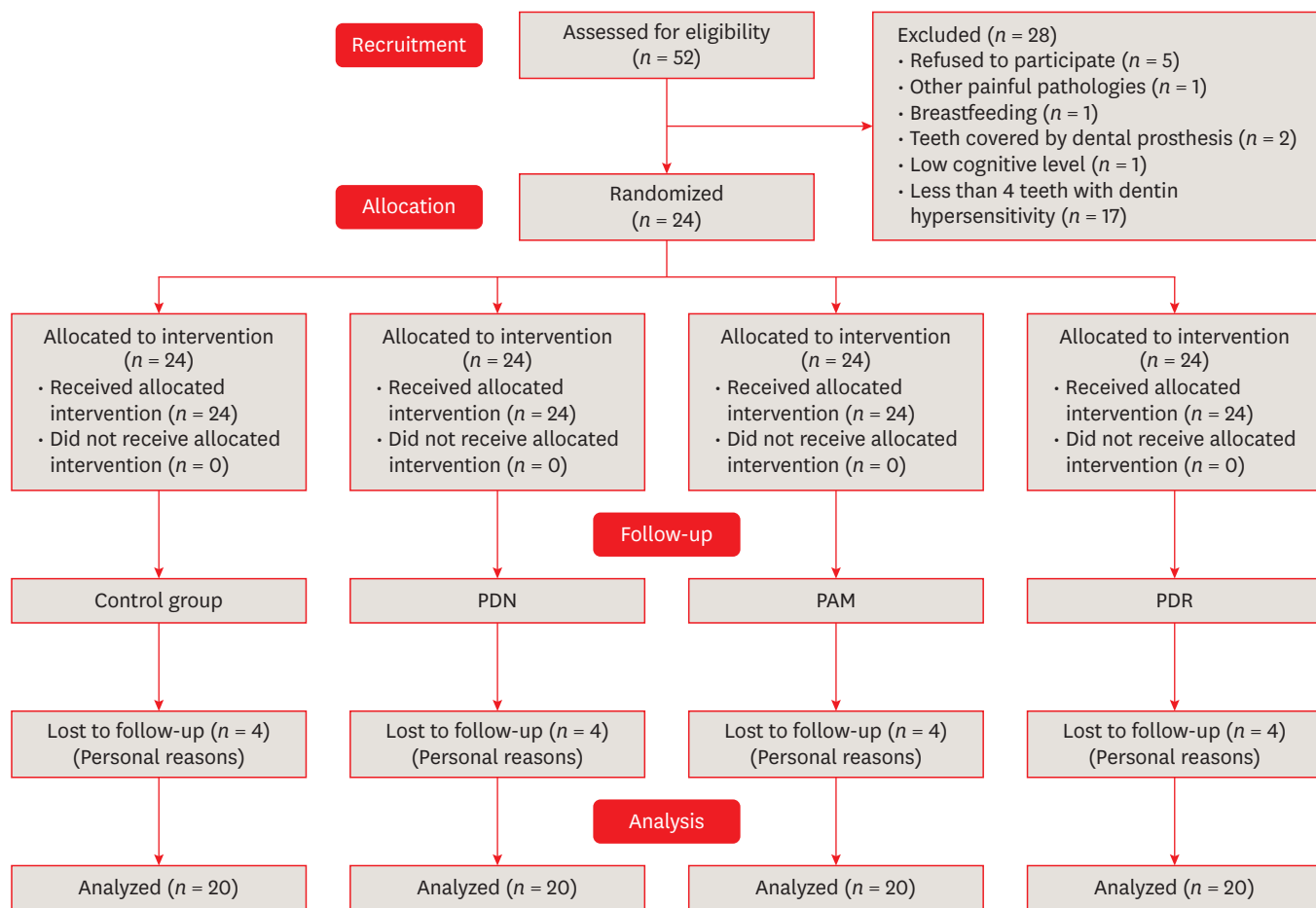
Figure 1. Search method flowchart.
PDN, Neural Desensitizing Protocol; PAM, Mixed Desensitizing Protocol; PDR, Remineralizing Desensitizing Protocol.

DH assessment

The stimulus adopted was the evaporative stimulus (triple syringe).and tactile (probing) stimuli. The VAS with measurements from 0 to 10 was used, in which the volunteer indicated their pain. Zero (0) refers to "no pain" and 10 corresponds to "unbearable pain" [24]. The clinical evaluation involved applying a triple syringe air jet perpendicular to the cervical region of the tooth, at a distance of 1 cm. This stimulus lasted for 2 seconds. Adjacent teeth were isolated using cotton rolls to prevent any interference with the measurement of the specific tooth. Immediately following the evaporative stimulus test, the patient indicated the level of sensitivity experienced on a VAS, and this information was documented in the patient's clinical chart. The examiner responsible for assessing the level of cervical DH had undergone calibration prior to the evaluation.

All treatments were administered by the same researcher (operator 1). The stimuli and pain measurements were carried out by a previously calibrated examiner (operator 2). To minimize errors and prevent bias, operator 2, who was unaware of the treatments applied, assessed the response of each tooth to the air stimuli. Subsequently, the levels of DH were measured and recorded [25].

1. Interventions

After undergoing clinical examination and providing their consent to participate in the study, patients received treatment based on their assigned group. Two weeks prior to the start of the study, participants entered a wash-out period, during which they exclusively used oral hygiene products recommended by the researchers. These products were to be used consistently until the conclusion of the study. The oral hygiene kit included a soft toothbrush (Professional Lab Series, Colgate Palmolive Company, New York, NY, USA), a fluoride toothpaste (Colgate Total 12, 1,450 ppm F, Colgate Palmolive Company), and a dental floss (Colgate, Colgate Palmolive Company).

Prior to treatment, dental prophylaxis was conducted on all teeth using a rubber cup, 2% chlorhexidine, and a pumice stone. The area was subsequently rinsed with an air/water spray and dried using cotton. To ensure relative isolation, cotton rolls were used, and treatments were then administered based on the assigned groups. The group distribution, as well as the composition and usage instructions provided by each manufacturer, are presented in **Table 1**.

Initially, a topical anesthetic (Benzotop 200g/mg, DFL, Rio de Janeiro, RJ, Brazil) was applied to assist in controlling any discomfort in the participants' lips for the placement of a lip retractor (Expandex White Lip Retractor, Maquira, Maringá, PR, Brazil). Participants received prophylaxis with a paste made of pumice stone (Pumice Stone, Biodinâmica, Ibiporã, PR, Brazil) and 2% chlorhexidine gluconate (Chlorhexidine Solution, Maquira), in a 1:1 ratio, using a rubber cup (Microdont, São Paulo, SP, Brazil) attached to a low-speed handpiece (Kavo, Joinville, SC, Brazil) on the teeth included in this study, followed by relative isolation using cotton rolls on the adjacent teeth. The application of all protocols was performed in a single session, and the steps of the clinical procedures are summarized in **Table 2**.

The treatments were carried out in 1 session. The effectiveness of the products was evaluated immediately after each treatment session using the VAS. Participants were scheduled for follow-up visits at 7, 30-, 60-, and 360 days post-treatment, during which the VAS level was assessed using the same evaporative and tactile stimuli.

Table 1. Description of application and composition of products used according to each manufacturer

Product	Application	Composition
Enamelast (Ultradent, South Jordan, UT, USA)	Prophylaxis, relative isolation, thoroughly dry the area and apply the product to the sensitive region, wait for 1 min. Remove excess with cotton.	Five percent sodium fluoride in varnish suspension.
Desensibilize KF 2% (FGM, Joinville, SC, Brazil)	Prophylaxis, relative isolation, thoroughly dry the area and apply the product to the sensitive region and wait for 15 min. Remove excess with cotton and rinse abundantly with water.	Active ingredients: 5% potassium nitrate, 2% sodium fluoride.
Single Bond Universal (3M ESPE, St. Paul, MO, USA)	Prophylaxis, relative isolation, dry the region, apply Single Bond by rubbing for 20 sec, light air blasts for 5 sec, light-cure for 10 sec with Bluephase LED light-curing unit (Ivoclar Vivadent, Barueri, SP, Brazil) at an intensity of 1,200 mW/cm².	HEMA, ethanol, water, initiators, silane, filler, dimethacrylate, MDP, Vitrebond Copolymer.
PRG Barrier Coat (Shofu, Kyoto, Japan)	Prophylaxis, relative isolation, dry the region. Add a drop of activator into the capsule with the base. Remove excess material from the brush on the edges of the capsule. The working time of the material is about 2 min. Apply a uniform layer on the surface, from the cervical to the incisal margin. Leave for 3 sec. In case of saliva contamination, remove with gauze and repeat the procedure. Light-cure for 10 sec with LED or other light-curing units. The formed film is approximately 15 µm thick without aesthetic compromise.	Base: fluoroaluminosilicate glass-based S-PRG filler, distilled water, methacrylic acid monomer, and other components. Active: phosphoric acid monomer, methacrylic acid monomer, Bis-MPEPP, carboxylic acid monomer, TEGDMA, polymerization initiator, and other components.

LED, light-emitting diode; HEMA, 2-hydroxyethyl methacrylate; MDP, methacryloyloxydecyl dihydrogen phosphate; S-PRG, surface-partially reacted glass; Bis-MPEPP, bisphenol A polyethoxymethacrylate; TEGDMA, triethylene glycol dimethacrylate.

Table 2. Description of application of management protocols

Protocol	Step 1	Step 2
Control group	Application of adhesive system (Single Bond Universal, 3M ESPE, St. Paul, MO, USA)	N/A
PDN group	Application of neural desensitizing agent (Desensibilize KF 2, FGM, Joinville, SC, Brazil)	Application of adhesive system (Single Bond Universal)
PAM group	Application of neural desensitizing agent (Desensibilize KF 2%)	Application of obliterating agent (Enamelast, Ultradent, South Jordan, UT, USA)
PDR group	Application of neural desensitizing agent (Desensibilize KF 2%)	Application of remineralizing agent (PRG Barrier Coat, Shofu, Kyoto, Japan)

PDN, Neural Desensitizing Protocol; PAM, Mixed Desensitizing Protocol; PDR, Remineralizing Desensitizing Protocol; PDN group, application of the neural desensitizing agent followed by the application of the adhesive; PAM group, application of the neural desensitizing agent followed by the application of the obliterating agent; PDR group, application of the neural desensitizing agent followed by the application of the remineralizing agent.

Statistical analysis

The analysis followed the intention-to-treat protocol and involved all participants who received the management. The results were subjected to statistical analysis using JAMOVI software, version 1.2.24 (The Jamovi Project, Sydney, Australia) [26].

The data were assessed for normal distribution using the Shapiro-Wilk test, which yielded a non-normal distribution. Based on this prerequisite analysis, statistical tests were performed to evaluate the presence of statistically significant intra-group differences using the Friedman repeated measures analysis of variance (Friedman's analysis of variance [ANOVA]) followed by the Durbin-Conover post-test, with a significance level set at $p < 0.05$. For the analysis of data related to the degree of sensitivity among participants in the intra-group assessment, the Friedman repeated measures ANOVA was conducted, followed by the Durbin-Conover follow-up test ($p < 0.05$). In turn, for the inter-group analysis comparing the same time interval, the Wilcoxon test was performed ($p < 0.05$).

RESULTS

A total of 52 individuals were assessed for sample selection, and 28 were excluded for not meeting the inclusion criteria. In the end, 20 participants were randomized, treated, and evaluated (**Figure 1**).

The demographic characteristics are presented in **Table 3**. The mean age ranged from 19 to 60 years, with the majority being of Caucasian ethnicity and female sex.

Table 3. Patient data regarding sex, race, and age

Variables	Values
Sex	
Female	19 (79.17)
Male	5 (20.83)
Race	
Caucasian	20 (83.33)
Non-Caucasian	4 (16.67)
Age (yr)	
11–20	1 (4.17)
21–30	9 (37.50)
31–40	6 (25.00)
41–50	5 (20.83)
51–60	3 (12.50)

Values are presented as number (%).

Table 4. Median values and interquartile ranges of sensitivity level during the execution of the tooth whitening protocol using desensitizing agents control group, PDN group, PAM group, and PDR group at different assessment times

Time	Control group	PDN group	PAM group	PDR group
T1	7.00 ± 2.12^{Aab}	7.17 ± 2.12^{Aab}	8.00 ± 1.57^{Aa}	6.67 ± 3.00^{Ab}
T2	3.54 ± 2.6^{Ba}	3.93 ± 4.00^{Ba}	3.80 ± 4.00^{Ba}	3.73 ± 2.57^{Ba}
T3	3.83 ± 2.5^{Ba}	4.42 ± 3.7^{Ca}	4.00 ± 2.5^{Ba}	3.83 ± 2.5^{BCa}
T4	3.00 ± 4.63^{Ba}	3.25 ± 3.25^{Da}	3.00 ± 3.75^{Ba}	3.00 ± 3.45^{Ca}
T5	1.67 ± 2.2^{Ca}	2.50 ± 2.77^{BDab}	2.71 ± 3.77^{Cb}	1.67 ± 3.18^{Da}

PDN, Neural Desensitizing Protocol; PAM, Mixed Desensitizing Protocol; PDR, Remineralizing Desensitizing Protocol. Different uppercase letters in the same column indicate significant differences ($p < 0.05$) in the intra-group analysis using the Friedman repeated measures analysis of variance test. Different lowercase letters in the same row indicate significant differences ($p < 0.05$) in the inter-group analysis using the Wilcoxon test.

Analysis of dentin sensitivity level

The results were subjected to statistical analysis using the non-parametric Friedman repeated measures ANOVA test ($p < 0.05$), followed by the Durbin-Conover *post hoc* test ($p < 0.05$) for intra-group evaluation. For inter-group analysis, the Wilcoxon test ($p < 0.05$) was used. Overall, in most evaluations, there was no statistically significant difference between the tested desensitizing agents at different reassessment times.

The evaluation of sensitivity level generally showed a statistically significant difference between the initial time points and the other evaluated times in the intra-group analysis. Another important finding is that for the 12-month evaluation (T5), all groups showed a statistically significant difference compared to the initial evaluation. In terms of inter-group analysis, there was a statistically significant difference between the Mixed Desensitizing Protocol (PAM) group and Remineralizing Desensitizing Protocol (PDR) group at the initial evaluation (T1). For the assessment at 12 months (T5), there was a statistically significant difference between the PAM group and the control group, and PDR group. The data are presented in **Table 4**.

DISCUSSION

This study demonstrated that all protocols were effective in reducing DH. A higher incidence of DH was observed in women, which is consistent with findings from other studies [26,27]. However, another study reported a higher prevalence of DH in males, with 68 out of 90 participants being affected [28]. Thus, it can be inferred that in the Brazilian context, DH predominantly affects females, unlike in other countries. The study population consistently exhibited similar baseline characteristics across all treatment groups, which aligns with a clinical study conducted by Lopes *et al.* [29], who also evaluated different management protocols for DH.

The results of this study provide evidence in support of the formulated hypothesis, suggesting that the application of an associative protocol using neural agents followed by obliterating agents in the same clinical session would lead to a significant reduction in DH. This hypothesis is based on the synergistic combination of the depolarizing and tubule-sealing effects provided by the agents used. The study findings confirmed this hypothesis, as a substantial reduction in hypersensitivity was observed throughout the evaluation period. Thus, the associative protocol proved to be an effective approach in managing DH.

The results of this study regarding SBU are similar to those found by other researchers. The positive effect may be related to its composition, as the chemical interaction of the dental

substrate with 10-methacryloyloxydecyl dihydrogen phosphate (10-MDP) forms an MDP-Ca salt, which is responsible for improved adhesion and greater stability in an aqueous environment [29]. The reduction and control of DH occur because the protein precipitates present on the dental surface are not permeable to the monomers present in SBU's composition, and the presence of phosphorylated monomers allows for bonding with dentin when in an aqueous environment, enabling the obliteration of open tubules [30].

However, when comparing Gluma Desensitizer, Gluma Comfort Bond + Desensitizer, and SBU, the adhesive showed less effective results in controlling DH [31]. The application of a double layer of SBU in this study may have contributed to the effectiveness of sensitivity control.

A 4-session protocol for reducing DH, using Desensibilize KF 2% (FGM, Joinville, SC, Brazil), Clinpro XT Varnish, and Photo Lase III, demonstrated that Desensibilize KF 2% was effective in controlling sensitivity, as observed in this study [26]. The immediate efficacy of neural action products is attributed to the presence of potassium nitrate in their composition, which depolarizes the cells on the dental surface and in the dentinal tubules, thus preventing sensitivity [12,32]. This immediate reduction efficiency was demonstrated in an *in vitro* study, where potassium nitrate was able to penetrate the dental pulp within 5 minutes [33].

Obliterative agents are also used in the control of DH, and in this study, Enamelast (Ultradent, South Jordan, UT, USA) was chosen, which contains sodium fluoride that deposits fluoride ions and obliterates the dentinal tubules, preventing external stimuli from causing hypersensitivity [33]. This deposition of ions on the dental surface was confirmed in an *in vitro* study, where Enamelast showed higher microhardness values and a higher concentration of fluoride in its composition compared to Duraphat (Colgate Palmolive Company) and Clinpro White Varnish (3M ESPE, St. Paul, MO, USA) [34,35].

In this study, a mixed protocol was proposed, where the neural agent Desensibilize KF 2% was applied first, followed by the obliterative agent Enamelast. The neural agent's function is to depolarize the nerve fibers, preventing the transmission of nerve impulses, thereby controlling sensitivity. It is important to apply the correct sequence due to the mechanism of action of the agents used. If the dentinal tubule orifice is sealed before the depolarization of the nerve fibers, the desired efficacy of this protocol is compromised [12,33,36].

By combining agents with different mechanisms of action, it is possible to achieve a more effective and long-lasting result, as these actions can interact positively. The present associative protocol demonstrated efficacy in treating DH in all evaluations, showing a reduction from the first assessment. In a previous study that used potassium oxalate, an agent with neural and obliterative properties for DH control, a significant reduction in hypersensitivity complaints was observed in all assessments [27].

The clinical study used Profluorid Varnish, Admira Protect, and PRG-Barrier Coat (PBC) for DH control, evaluated through evaporative and tactile tests immediately after, 1 week, and 1 month after the application of the agents. The study showed that all products had positive results in reducing DH at all time intervals, which differs from the results of this research where the PBC desensitizer was more effective in reducing DH after 7 days of application [15]. This discrepancy may have been caused by the difference in prophylaxis procedures, as the authors used a Robinson brush instead of a rubber cup, which was different from this study.

Similar results were found in a clinical study comparing PBC and BiFluorid 10 for reducing DH, where PBC showed a significant difference only in the evaluation conducted 1 week after application [37]. The fact that the reduction in DH does not occur immediately after the application of PBC may be related to its mechanism of action, which occurs gradually when in contact with oral fluids, by binding ions to collagen fibers, while fluoride and strontium bind to apatite present in the dental structure, improving its resistance to acid attack [22].

The results obtained in this study were significant for the management of DH at all proposed time points, and they particularly stood out in the PDR and control groups. It should be mentioned that PBC is a promising agent that has demonstrated its long-lasting effect in managing DH due to its mechanism of ion release and precipitation, aiding in remineralization and obliteration of dentinal tubules, as observed after 12 months of application in this study, as well as in the Control group [37]. This is attributed to the characteristics of SBU, with the presence of 10-MDP, which stabilizes in an aqueous medium and chemically bonds with dentin, obliterating the dentinal tubules [32].

The scarcity of clinical trials and lack of scientific evidence pose a challenge in providing precise information on the long-term clinical efficacy of materials used in the management of DH. However, the results of this study demonstrate that these materials hold promise for the treatment of DH. Further clinical trials are recommended to compare the desensitizing effect among different materials, in order to maximize their benefits and improve their utilization in clinical practice. Additionally, future studies can explore the influence of variables such as age, sex, and specific oral conditions on the effectiveness of these associative protocols, further enhancing the understanding and management of DH.

CONCLUSIONS

Based on this study, it can be concluded that all the protocols employed were effective in reducing DH from the initial evaluation, as observed within each group, up to 12 months. In the intergroup evaluation, the control and PDR groups demonstrated greater efficacy in controlling DH after 12 months.

The clinical relevance of this study is to demonstrate that the application of single-session desensitizing protocols can be effective in controlling DH for up to 12 months, as demonstrated by statistical analysis.

REFERENCES

1. Favaro Zeola L, Soares PV, Cunha-Cruz J. Prevalence of dentin hypersensitivity: systematic review and meta-analysis. J Dent 2019;81:1-6. **PUBMED | CROSSREF**

2. Addy M. Dentine hypersensitivity new perspectives on an old problem. Int Dent J 2002;52:367-375. **CROSSREF**

3. 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. **PUBMED**

4. Davari A, Ataei E, Assarzadeh H. Dentin hypersensitivity: etiology, diagnosis and treatment; a literature review. J Dent (Shiraz) 2013;14:136-145. **PUBMED**

5. Ishihata H, Finger WJ, Kanehira M, Shimauchi H, Komatsu M. In vitro dentin permeability after application of Gluma® desensitizer as aqueous solution or aqueous fumed silica dispersion. J Appl Oral Sci 2011;19:147-153. **PUBMED | CROSSREF**

6. García-Delaney C, Abad-Sánchez D, Arnabat-Domínguez J, Valmaseda-Castellón E, Gay-Escoda C. Evaluation of the effectiveness of the photobiomodulation in the treatment of dentin hypersensitivity after basic therapy. A randomized clinical trial. J Clin Exp Dent 2017;9:e694-e702.   **PUBMED** | **CROSSREF**

7. Aw TC, Lepe X, Johnson GH, Mancl L. Characteristics of noncarious cervical lesions: a clinical investigation. J Am Dent Assoc 2002;133:725-733.   **PUBMED** | **CROSSREF**

8. Braennstroem M, Astroem A. A study on the mechanism of pain elicited from the dentin. J Dent Res 1964;43:619-625.   **PUBMED** | **CROSSREF**

9. Douglas-de-Oliveira DW, Vitor GP, Silveira JO, Martins CC, Costa FO, Cota LO. Effect of dentin hypersensitivity treatment on oral health related quality of life - a systematic review and meta-analysis. J Dent 2018;71:1-8.   **PUBMED** | **CROSSREF**

10. Teixeira DNR, Zeola LF, Machado AC, Gomes RR, Souza PG, Mendes DC, et al. Relationship between noncarious cervical lesions, cervical dentin hypersensitivity, gingival recession, and associated risk factors: a cross-sectional study. J Dent 2018;76:93-97.   **PUBMED** | **CROSSREF**

11. Liu XX, Tenenbaum HC, Wilder RS, Quock R, Hewlett ER, Ren YF. Pathogenesis, diagnosis and management of dentin hypersensitivity: an evidence-based overview for dental practitioners. BMC Oral Health 2020;20:220.   **PUBMED** | **CROSSREF**

12. Shah D, Mital K. The role of trypsin:chymotrypsin in tissue repair. Adv Ther 2018;35:31-42.   **PUBMED** | **CROSSREF**

13. Joshi S, Gowda AS, Joshi C. Comparative evaluation of NovaMin desensitizer and Gluma desensitizer on dentinal tubule occlusion: a scanning electron microscopic study. J Periodontal Implant Sci 2013;43:269-275.   **PUBMED** | **CROSSREF**

14. Ma S, Imazato S, Chen JH, Mayanagi G, Takahashi N, Ishimoto T, et al. Effects of a coating resin containing S-PRG filler to prevent demineralization of root surfaces. Dent Mater J 2012;31:909-915.   **PUBMED** | **CROSSREF**

15. Ravishankar P, Viswanath V, Archana D, Keerthi V, Dhanapal S, Lavanya Priya KP. The effect of three desensitizing agents on dentin hypersensitivity: a randomized, split-mouth clinical trial. Indian J Dent Res 2018;29:51-55.   **PUBMED** | **CROSSREF**

16. Rabiabasree R, Krishnakumar R, Prabhu AS, Naik NS, Shashibhushan KK, Janarthanan K. Inhibitory effect of a resin coat-containing prereacted glass fillers on the enamel demineralization of the primary teeth: an *in vitro* pilot study. J Indian Soc Pedod Prev Dent 2019;37:146-150.   **PUBMED** | **CROSSREF**

17. Spinola MD, Moecke SE, Rossi NR, Nakatsuka T, Borges AB, Torres CRG. Efficacy of S-PRG filler containing varnishes on enamel demineralization prevention. Sci Rep 2020;10:18992.   **PUBMED** | **CROSSREF**

18. Suzuki M, Yamada A, Saito K, Hino R, Sugawara Y, Ono M, et al. Application of a tooth-surface coating material containing pre-reacted glass-ionomer fillers for caries prevention. Pediatr Dent J 2015;25:72-78.   **CROSSREF**

19. Miki S, Kitagawa H, Kitagawa R, Kiba W, Hayashi M, Imazato S. Antibacterial activity of resin composites containing surface pre-reacted glass-ionomer (S-PRG) filler. Dent Mater 2016;32:1095-1102.   **PUBMED** | **CROSSREF**

20. Kaga M, Kakuda S, Ida Y, Toshima H, Hashimoto M, Endo K, et al. Inhibition of enamel demineralization by buffering effect of S-PRG filler-containing dental sealant. Eur J Oral Sci 2014;122:78-83.   **PUBMED** | **CROSSREF**

21. Iijima M, Ito S, Nakagaki S, Kohda N, Muguruma T, Saito T, et al. Effects of immersion in solution of an experimental toothpaste containing S-PRG filler on like-remineralizing ability of etched enamel. Dent Mater J 2014;33:430-436.   **PUBMED** | **CROSSREF**

22. Rusnac ME, Gasparik C, Irimie AI, Grecu AG, Mesaroş AŞ, Dudea D. Giomers in dentistry - at the boundary between dental composites and glass-ionomers. Med Pharm Reports 2019;92:1-6.   **CROSSREF**

23. Sarkis-Onofre R, Cenci MS, Moher D, Pereira-Cenci T. Research reporting guidelines in dentistry: a survey of editors. Braz Dent J 2017;28:3-8.   **PUBMED** | **CROSSREF**

24. Camilotti V, Zilly J, Busato PM, Nassar CA, Nassar PO. Desensitizing treatments for dentin hypersensitivity: a randomized, split-mouth clinical trial. Braz Oral Res 2012;26:263-268.   **PUBMED** | **CROSSREF**

25. Lopes AO, Aranha ACC. Comparative evaluation of the effects of Nd:YAG laser and a desensitizer agent on the treatment of dentin hypersensitivity: a clinical study. Photomed Laser Surg 2013;31:132-138.   **PUBMED** | **CROSSREF**

26. Moura GF, Zeola LF, Silva MB, Sousa SC, Guedes FR, Soares PV. Four-session protocol effectiveness in reducing cervical dentin hypersensitivity: a 24-week randomized clinical trial. Photobiomodul Photomed Laser Surg 2019;37:117-123.   **PUBMED** | **CROSSREF**

27. Galvão AD, Zeola LF, Moura GF, Teixeira DNR, Gonzaga RCQ, da Silva GR, et al. A long-term evaluation of experimental potassium oxalate concentrations on dentin hypersensitivity reduction: a triple-blind randomized clinical trial. J Dent 2019;89:103180. PUBMED | CROSSREF

28. Narayanan R, Prabhuji MLV, Paramashivaiah R, Bhavikatti SK. Low-level laser therapy in combination with desensitising agent reduces dentin hypersensitivity in fluorotic and non-fluorotic teeth - a randomised, controlled, double-blind clinical trial. Oral Health Prev Dent 2019;17:547-556. PUBMED | CROSSREF

29. Lopes AO, de Paula Eduardo C, Aranha ACC. Evaluation of different treatment protocols for dentin hypersensitivity: an 18-month randomized clinical trial. Lasers Med Sci 2017;32:1023-1030. PUBMED | CROSSREF

30. Younus MZ, Ahmed MA, Syed AUY, Baloch JM, Ali M, Sheikh A. Comparison between effectiveness of dentine desensitizer and one bottle self-etch adhesive on dentine hypersensitivity. Technol Health Care 2021;29:1153-1159. PUBMED | CROSSREF

31. Askari M, Yazdani R. Comparison of two desensitizing agents for decreasing dentin hypersensitivity following periodontal surgeries: a randomized clinical trial. Quintessence Int 2019;50:320-329. PUBMED | CROSSREF

32. Yoshida Y, Inoue S. Chemical analyses in dental adhesive technology. Jpn Dent Sci Rev 2012;48:141-152. CROSSREF

33. Kwon SR, Dawson DV, Schenck DM, Fiegel J, Wertz PW. Spectrophotometric evaluation of potassium nitrate penetration into the pulp cavity. Oper Dent 2015;40:614-621. PUBMED | CROSSREF

34. Rösing CK, Fiorini T, Liberman DN, Cavagni J. Dentine hypersensitivity: analysis of self-care products. Braz Oral Res 2009;23 Supplement 1:56-63. PUBMED | CROSSREF

35. Godoy CE, Consani S, Guimarães AT, Laurindo BM, Mendonça MJ, Camilotti V. Effect of two desensitizing agents applied previous to in-office bleaching on the degree of whitening and dentin sensitivity: a randomized, controlled, double-blind clinical trial. Am J Dent 2021;34:70-74. PUBMED

36. Grippo JO, Simring M, Coleman TA. Abfraction, abrasion, biocorrosion, and the enigma of noncarious cervical lesions: a 20-year perspective. J Esthet Restor Dent 2012;24:10-23. PUBMED | CROSSREF

37. Ali NY, Hassanein OE, Hamza HS, Baz MAE. Clinical efficacy of giomer versus sodium fluoride varnish for management of hypersensitivity: randomized control trail. Egypt Dent J 2021;67:905-915. CROSSREF