Effect of Oxalic Acid–Based Desensitizing Agent on Cervical Restorations on Hypersensitive Teeth: A Triple-Blind Randomized Controlled Clinical Trial

Camila Corral, PhD(c), MClinDent, BDS
Professor
Department of Restorative Dentistry
Faculty of Dentistry
University of Chile
Santiago, Chile

Patricio Vildósola Grez, DDS, DS, PhD
Assistant Professor
Department of Restorative Dentistry
Faculty of Dentistry
University of Chile/Universidad Nacional Andrés Bello (UNAB)
Santiago, Chile
and School of Dentistry
University Estadual Paulista – UNESP
São Paolo, Brazil

Matías Letelier, DDS
Assistant Professor
School of Dentistry
Universidad de La Serena
La Serena, Chile

Edson Alves dos Campos, DDS, PhD
Professor
Department of Restorative Dentistry
Faculty of Dentistry
University of Chile
Santiago, Chile

Alessandro Loguercio Dourado, DDS, MsC, PhD
Associate Professor
Department of Restorative Dentistry
Faculty of Dentistry
Universidade Estadual de Ponta Grossa
Ponta Grossa, Brazil

Eduardo Fernández G., DDS, DS, PhD
Associate Professor
Department of Restorative Dentistry
Faculty of Dentistry
University of Chile
Santiago, Chile
and School of Dentistry
University Estadual Paulista – UNESP
São Paolo, Brazil

Correspondence to:
Dr Eduardo Fernández G.
Department of Restorative Dentistry
Universidad de Chile, Dental School
Sergio Livingstone Pohlhammer 943, Independencia
Santiago, Chile
Fax: +56229462929
Email: edofdez@yahoo.com

Aims: To assess the effects of application of an oxalic acid–based desensitizing agent before restoration of noncarious cervical lesions (NCCLs) with either a silorane-based or a methacrylate-based composite resin on decreasing the absolute risk and intensity of dentin hypersensitivity over the course of a 1-year follow-up. **Methods:** NCCLs in 31 patients (age range 24–66 years) were selected and randomly divided into four groups (n = 31 in all groups). In the Z250 and P90 groups, the restorations were performed with a methacrylate-based composite resin (Filtek Z250) and a silorane-based composite resin (Silorane P90), respectively. In the Z250 + OA and P90 + OA groups, the same composite resins were used, but an oxalic acid–based desensitizing agent (Desenssiv, SSWhite) was first applied. All NCCLs were evaluated before restoration (BR) and at 30, 60, 90, 180, and 360 days after treatment. Teeth sensitivity to evaporative and tactile stimuli was measured by a visual analog scale (VAS). The results were analyzed with statistical tools including Wilcoxon and Friedman tests for within-group comparisons and ANOVA and Bonferroni post hoc tests for between-group comparisons ($P < .05$). **Results:** Reduction in dentin hypersensitivity was observed for all treatment groups; however, these reductions were more pronounced when oxalic acid was applied before restoring the NCCL ($P < .001$). Complete elimination of pain was not achieved by any treatment modalities for the first 6 months; afterwards, in the groups that had received application of the oxalate-based desensitizing agent, the absolute risk of dentin hypersensitivity was significantly reduced ($P < .01$). **Conclusion:** The restoration of sensitive NCCLs with composite resins reduces dentin hypersensitivity. This reduction is more pronounced if an oxalic acid–based desensitizing agent is applied prior to the restoration. In addition, its application reduces the absolute risk of dentin hypersensitivity after 6 months of treatment. *J Oral Facial Pain Headache 2016;30:330–337. doi: 10.11607/ofph.1676*

Keywords: *dental pain, dentin hypersensitivity, noncarious cervical lesion, oxalic acid, randomized clinical trial*

Dentin hypersensitivity is a common problem in adults, with studies reporting a prevalence ranging from 3% to 57%.[1] It is defined as "short, sharp pain arising from exposed dentin in response to stimuli that are typically thermal, evaporative, tactile, osmotic or chemical and which cannot be ascribed to any other form of dental defect or pathology."[2] The phenomenon is associated with the exposure of dentin tubules due to traumatic tooth brushing, acid erosion, and/or gingival recession.[3,4] Currently, the most accepted explanation for dentin hypersensitivity is the hydrodynamic theory proposed by Brannstrom, which suggests inward or outward movement of fluid within the dentin tubules as the mechanism of transduction, stimulating A-delta fibers and explaining the pain in pulpal tissue.[5]

Nowadays, there are two main treatment methods for dentin hypersensitivity: dentin tubular occlusion and nerve desensitization. These methods can be carried out with the use of in-office and over-the-counter products.[3,6] For in-office treatments, several agents for dentin tubule blocking are proposed, including fluorides, oxalates, and resin-based systems, among others.[6] These treatments have been evaluated in clinical studies; however, the results are difficult to compare

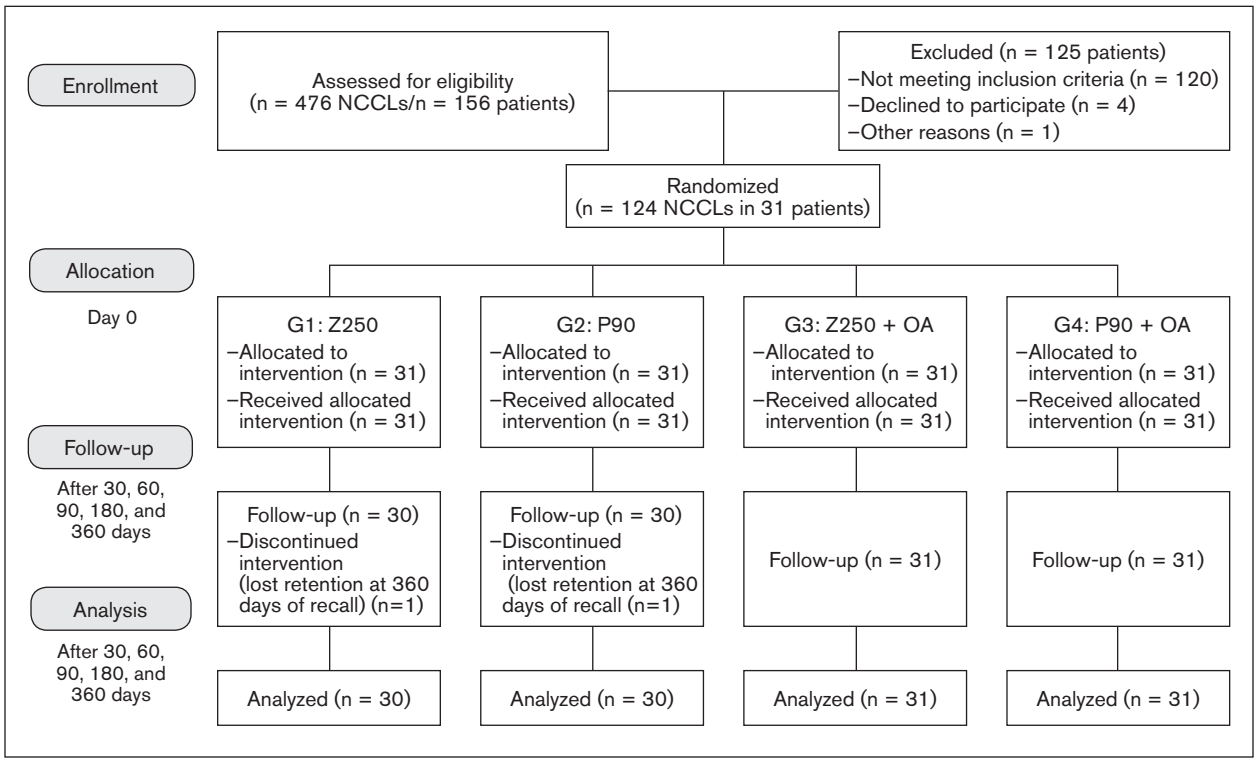

Fig 1 CONSORT trial flow diagram of the clinical trial.

due to differences in approach and technique, and conflicting outcomes are frequently reported.[2,7] Therefore, due to the large number of options available and the uncertain effectiveness of these agents, it is challenging for the clinician to select the most appropriate treatment for a certain clinical case.[6]

One of the options currently used is the application of oxalates. It is believed that their mechanism of action involves calcium from dentin reacting with the oxalates to form insoluble calcium oxalate crystals that precipitate in the dentinal tubules, thereby blocking them.[8] A few clinical trials have evaluated the desensitizing effect when it is applied prior to restoration; however, the results are controversial[9,10] and a closer examination of two of these recent studies[10,11] shows that dentin sensitivity at baseline was not one of the key inclusion criteria. This means that the authors focused on measuring one outcome that previously was nonexistent. Barrientos et al[9] have shown a greater reduction in dentin hypersensitivity when oxalates were applied prior to composite resin restorations; however, this follow-up was only 4 months, and data on whether this effect is preserved after long-term follow-up are not available in the literature.

Another interesting option is to use oxalates in conjunction with low-shrinkage, silorane-based composite resins, as methacrylate-based composite resins suffer significant shrinkage during polymerization and this may cause postoperative sensitivity.[12,13] Despite this advantage, only one clinical study has evaluated silorane-based materials in noncarious cervical lesions (NCCLs) and reported a high rate of success on all the parameters studied.[14] Therefore, this randomized, triple-blind, controlled clinical trial was initiated to assess the effects of an oxalic acid–based desensitizing agent applied before restoring NCCLs with a silorane-based or a methacrylate-based composite resin on decreasing the absolute risk and intensity of dentin hypersensitivity over the course of a 1-year follow-up.

Materials and Methods

This study was a single-center, split-mouth, placebo-controlled, triple-blind (blinded operators, patients, and examiners) randomized clinical trial with parallel groups designed according to CONSORT recommendations[15,16] (Fig 1). It was conducted in the Dental Clinic of the University of Chile in accordance with the Declaration of Helsinki of 1975 (revised in 2000) and Guidelines for Good Clinical Practice. The study was conducted from March 2012 to July 2013.

Following ethical approval of the Ethics Committee (Dental School, University of Chile), 31 patients were selected from the Dental Clinic of the University

of Chile. They were recruited for 2 months with the help of a poster campaign around the Clinic. An informed consent was obtained from the patients after explaining the rationale and purpose of the study (N° 2012/09/ClinicalTrials.gov Identifier/NCT02306486).

Inclusion criteria were: Patients 18 years or older with at least four NCCLs (surface loss on buccal/facial aspect of canines, premolars, or molars, with each cervical lesion located in a different quadrant of the mouth, 2 to 3 mm of occlusogingival height and < 3 mm in depth) showing moderate to severe dentin hypersensitivity according to scores on a 100-mm visual analog scale (VAS; 0–25 mm = mild, 26–50 mm = moderate, 51–75 mm = severe, and 76–100 mm = very severe) when tactile and evaporative stimuli were applied.

Exclusion criteria: Recent periodontal surgery, orthodontic treatment, or desensitizing treatment within the last 3 months, undergoing treatment with a nonsteroidal, anti-inflammatory drug (NSAID), patients with systemic diseases related to chronic pain, pregnancy or breastfeeding, and/or having teeth with caries or restorations.

Interventions

A single dose of NSAID was prescribed prior to restoring the NCCL, as recommended by the Ethics Committee, Dental School, University of Chile.

Two calibrated examiners carried out the clinical examinations and sensitivity tests to assess the sensitivity of patients with NCCLs and dentin hypersensitivity at baseline (interexaminer kappa > .7 measurements for tactile and evaporative stimuli were taken 1 week apart and the results were compared to the intra- and interexaminer kappa values). The assessment of dentin sensitivity at baseline (ie, before restoration [BR]) was based on tactile and evaporative stimulation-evoked responses. The tactile stimulus consisted of probing the buccal cervical surface of the teeth with a North-Carolina periodontal probe (CP-15 Probe, Hu-Friedy). The evaporative stimulus was direct application of air pressure with an air-dry syringe perpendicular to the tooth for 1 second at 60 psi at room temperature, 1 cm away from the surface, protecting the adjacent teeth with cotton rolls. Patients quantified their dentin hypersensitivity caused by both stimuli through the use of a 100-mm VAS[17] with 0 cm indicating no sensitivity and 100 indicating extreme sensitivity or discomfort.

The randomization process of the NCCLs within the groups was performed using computer software (NCSS PASS11) by a staff member not involved in the research protocol. Details of the allocated groups were recorded on cards contained in sequentially numbered, opaque, sealed envelopes. These were also prepared by a staff member not involved in the clinical trial. The allocation assignment was revealed by opening the envelope on the day of the restorative procedure.

Operators (M.L., E.F.) and evaluators (three) were lecturers with more than 10 years experience in the Restorative Dentistry course at the University of Chile. The operators were trained and calibrated to standardize the steps in the procedure. Evaluators were calibrated with in vitro pressure and distance exercises, and later evaluations were conducted on patients to confirm the reliability of the evaluators' assessments, as reflected in the evaluations producing a kappa value of > 0.75 in previous exercises in the tactile and evaporative measurements.

Before placing a rubber dam, the operators anesthetized the teeth with a 3% mepivacaine solution (Scadiacaina, Septodont, France) and all lesions were cleaned with pumice and water in a rubber cup followed by rinsing and drying. Following the guidelines of the American Dental Association, the operators did not prepare any additional retention or bevel.

There were 31 NCCLs in each of the four groups. The NCCLs received either a methacrylate-based composite resin Filtek Z250 (Z250 group, 3M ESPE) associated with a two-step etch-and-rinse adhesive using Adper Single Bond 2 (3M ESPE) or a silorane-based composite resin Filtek Silorane P90 (P90 group, 3M ESPE) associated with a two-step self-etch adhesive using Adper Silorane system adhesive (3M ESPE).

The Z250 + OA and P90 + OA groups received a methacrylate- or silorane-based composite resin in addition to an oxalic acid–based desensitizing agent (OA; Desenssiv, SSWhite). The desensitizing agent was applied after the acid-etching process in the Z250 + OA group[18] and before the restorative procedure in the P90 + OA group. The materials, compositions, application procedures, and other details are described in Table 1.

In the Z250 and P90 groups, a vehicle control of distilled water was used in place of oxalic acid. To ensure the blind nature of both operators and patients, bottles of oxalic acid and distilled water, as well as the syringes of composite resins, were covered with an opaque adhesive tape so the operator could not recognize them. Likewise, the patients were also not aware of which tooth corresponded with which treatment.

The oxalic acid or distilled water was vigorously agitated on the entire dentin surface in all groups for approximately 1 minute and then rinsed with water. Subsequently, the restorations were made following the manufacturer's instructions (Table 1).

All light-curing procedures were performed using a Radii Cal (SDI) at 1,000 mW/cm^2. The restorations were finished immediately with fine diamond burs (KG Sorensen). Polishing was performed with rubber

Table 1 Materials, Compositions, and Procedures

Materials (batch no.)	Composition	Procedure	Manufacturer
Desenssiv (Lot No. 0030907)	.5% potassium oxalate, potassium nitrate 4%, potassium fluoride 4%	Applied after acid-etching for the Z250 group and applied before for the P90 group; 1 minute, rinse and dry.	SS White
Silorane System Adhesive (Lot No. 4773P, 8AP)	Phosphorylated methacrylates, Bis-GMA, HEMA, water, ethanol, silane-treated silica filler (Primer). Hydrophobic dimethacrylate, phosphorylated methacrylates, TEGDMA, silane-treated silica filler, initiators, stabilizers (Bond), Self-Etch Primer and Bond	*First step:* Apply primer for 15 s, gentle air and light curing for 10 s. *Second step:* Apply bond, gentle air and light curing for 10 s.	3M ESPE
Adper Single Bond 2 (Lot No. 51202, 9YA)	Bis-GMA, HEMA, dimethacrylates, polyalkenoic acid, copolymer, initiators, water, ethanol -Total Etch Adhesive	After etchant apply 2–3 coats of adhesive for 15 s with gentle agitation, gently air thin for 5 s and light cure for 20 s.	3M ESPE
Gel Scothbond etchant	Phosphoric acid 37%	Apply etchant for 15 s, rinse for 30 s and blot excess water.	3M ESPE
Filtek Silorane P90 (Lot No. 4772A3, 8BF)	Silorane-based composite/resin	2-mm layers and light cure for 20–30 s.	3M ESPE
Filtek Z250 (Lot No. 1370A2, 7AU)	Methacrylate-based composite resin, TEGDMA, UDMA, and Bis-EMA	2-mm layers and light cure for 20–30 s.	3M ESPE

Bis-EMA = ethoxylated bisphenol-A glycidyl methacrylate; Bis-GMA = bisphenol A diglycidyl methacrylate; HEMA = 2-hydroxyethyl methacrylate; TEGDMA = triethylene glycol dimethacrylate; UDMA = urethane dimethacrylate.

points (Astropol, Ivoclar Vivadent) 1 week after placement of the restorations.

Clinical Evaluations

The same two calibrated examiners at baseline who were not involved with the placement of the restorations, and therefore blinded to the group assignment, performed the evaluation of hypersensitivity after 30, 60, 90, 180, and 360 days of placing the restorations during 2012 to 2013. The absolute risk of dentin hypersensivity, as well as dentin hypersensitivity scored on a 100-mm VAS, were recorded after application of the tactile and evaporative stimuli. This process was performed in the same manner as at baseline (ie, BR), but the tactile stimulus was performed by probing around the tooth/restoration margin with a North-Carolina periodontal probe.

Statistical Analyses

Considering the sensitivity measured with VAS as the principal outcome according to previous studies by the authors,[9] the sample size was calculated with a statistical power of 95% and a confidence level of 95%, resulting in a necessary sample size of 28, considering the reported dropout was an added 5%. SPSS statistical software (SPSS) was used for the statistical analysis of data. Differences within groups were analyzed with the Wilcoxon and Friedman tests and those between groups by ANOVA and the Bonferroni post hoc test. The significance level was set at .05.[19] The absolute risk of pain was calculated considering the occurrence or not of dentin hypersensitivity in the groups and was compared by chi-square tests. The statistical power for all comparisons between groups was corroborated post hoc by using G-Power v. 3.1.7 software.[20]

Results

Characterization of the Initial Sample

A total of 31 patients (16 women and 15 men) with a mean age of 46.8 years (ranging from 24 to 66 years old) were recruited. A total of 124 NCCLs associated with dentin hypersensitivity were assessed at baseline.

As mentioned earlier, all included patients showed moderate to severe dentin hypersensitivity (VAS score of > 40 mm) prior to the restoration of the NCCLs. The means of the VAS scores (± standard deviations [SDs]) of dentin hypersensitivity to the tactile and thermal/evaporative stimuli were 7.16 ± 1.08 and 7.67 ± 1.06, respectively.

The recall rate after 30, 60, 90, 180, and 360 days after treatment was 100% for all time points.

Treatment Group Comparisons

Analysis of Dentin Hypersensitivity After Treatment

After the intervention, all treatment groups showed a reduction in their VAS scores. The mean VAS scores after 30 days of intervention were 2.25 ± 0.93 (tactile stimulus) and 2.46 ± 0.94 (evaporative stimulus),

Table 2 VAS Scores for Tactile Stimuli

	BR	30 d	60 d	90 d	180 d	360 d
Z250	7.31 (± .92)	2.58 (± .99)*	2.48 (± .72)*	2.91 (± .48)*	2.60 (± .77)*	2.97 (± 1.18)*
P90	7.76 (± 1.11)	2.69 (± .90)*	2.80 (± .66)*	3.06 (± .55)*	2.54 (± 1.13)*	2.90 (± 1.03)*
Z250 + OA	7.94 (± 1.09)	2.73 (± 1.02)*	1.99 (± .53)*	2.05 (± .38)*	1.70 (± .77)*	1.16 (± .96)*
P90 + OA	7.67 (± 1.07)	1.84 (± .53)*	1.74 (± .65)*	1.81 (± .46)*	1.56 (± .54)*	.75 (± .61)*

Mean and standard deviation (SD) of 10-cm VAS scores for tactile stimulation test during all evaluation periods for all treatment groups.
*$P \leq .0001$ for comparison between scores at the different times and before restoration (BR) (Wilcoxon test).

Table 3 VAS Scores for Evaporative Stimuli

	BR	30 d	60 d	90 d	180 d	360 d
Z250	6.85 (± .96)	2.23 (± .91)*	2.31 (± .74)*	2.62 (± 53)*	1.92 (± .64)*	2.61 (± 1.11)*
P90	7.46 (± 1.17)	2.50 (± .80)*	2.52 (± .64)*	2.75 (± .56)*	2.43 (± .90)*	2.88 (± 1.03)*
Z250 + OA	7.17 (± .99)	2.56 (± 1.09)*	1.84 (± .54)*	1.80 (± .39)*	1.34 (± .80)*	.94 (± .81)*
P90 + OA	7.17 (± 1.13)	1.70 (± .66)*	1.52 (± .67)*	1.50 (± .44)*	1.15 (± .52)*	.55 (± .51)*

Mean and standard deviation (SD) of 10-cm VAS scores for tactile stimulation test during all evaluation periods for all treatment groups.
*$P \leq .0001$ for comparison between scores at the different times and before restoration (BR) (Wilcoxon test).

Table 4 Statistical Comparisons of Dentin Hypersensitivity Between Groups With and Without Oxalic Acid Application

	Z250 / Z250 + OA			P90 / P90 + OA		
	P	Effect size	Power (1 − β)	*P*	Effect size	Power (1 − β)
Tactile BR	1	.32	.23	1	.25	.24
Evaporative BR	.121	.62	.65	1	.08	.09
Tactile 30 d	.875	.32	.23	**.003**	1.09	**.99**
Evaporative 30 d	1	.14	.08	**.002**	1.15	**.99**
Tactile 60 d	**.036**	.72	**.78**	**.000**	1.52	**.99**
Evaporative 60 d	**.0021**	.96	**.95**	**.000**	1.61	**.99**
Tactile 90 d	**.000**	1.76	**.99**	**.000**	2.48	**1**
Evaporative 90 d	**.000**	1.98	**1**	**.000**	2.46	**.99**
Tactile 180 d	**.0015**	.8	**.85**	**.000**	1.74	**.99**
Evaporative 180 d	**.000**	1.16	**.99**	**.000**	1.10	**.99**
Tactile 360 d	**.000**	1.71	**.99**	**.000**	2.86	**1**
Evaporative 360 d	**.000**	1.68	**.99**	**.000**	2.53	**1**

Comparison by the post hoc Bonferroni test. *P* value, effect size, and statistical power post hoc are expressed. Significant values are presented in bold.

reflecting mild to moderate pain. The difference in VAS scores between the BR values and the values at all later time points were significant for all four treatment groups ($P \leq .0001$). The results are shown in Tables 2 and 3.

Analysis of the Effect of Desensitizing Agent on Dentin Hypersensitivity

Statistically significant differences were observed between the groups that involved application of oxalic acid prior to restoration and the groups without its application for both restorative materials ($P < .000$; ANOVA). The differences were evident for the assessment after 1 month for the groups receiving P90 and after 2 months of treatment for the groups receiving Z250. Results of the post hoc analysis are shown in Table 4.

Analysis of Postintervention Dentin Hypersensitivity Over Time

The average VAS values were reduced over time. Considering all time points up to 360 days, this reduction over time was statistically significant for all groups (Tables 2 and 3).

Analysis of the Absolute Risk of Dentin Hypersensitivity

The absolute risk of dentin hypersensitivity (defined as the percentage of teeth with pain relative to the total for each group) is shown in Table 5. There was an absolute risk of 100% for up to 3 months for all treatment modalities; thereafter, the absolute risk decreased. After 1-year follow-up, the absolute risk was significantly lower for restorations with prior oxalic acid application than for restorations without the

Table 5 Absolute Risk of Dentin Hypersensitivity (%) Over Time

	E (BR-30-60-90 d)	E 180	E 360	T (BR-30-60-90 d)	T 180	T 360
Z250	100	96.7	90	100	96.7	90
P90	100	90	93.3	100	96.7	93.3
Z250 + OA	100	87.1	63.5*	100	77.3	61.3*
P90 + OA	100	94.5	71**	100	92.3	71**

Percentage of teeth with sensitivity to stimuli (absolute risk of dentin hypersensitivity) by group and time. E = evaporative; T = tactile; BR = before restoration.
*$P < .01$; comparison between Z250 and Z250 + OA groups; **$P < .01$; comparison between P90 and P90 + OA groups (χ^2 test).

application (between Z250 and Z250 + OA, and between P90 and P90 + OA; $P < .01$).

Post Hoc Corroboration of Statistical Power

To corroborate the statistical power of this study, power was calculated post hoc with the sensitivity values for all measurements of comparisons between groups with or without oxalic acid, or comparison by materials. These are shown in Table 4.

Discussion

This randomized clinical trial demonstrated that restoration with composite resins in NCCLs was effective in decreasing dentin hypersensitivity. Although all treatment groups showed moderate to severe sensitivity reduced to mild, which is in concordance with the results of Barrientos et al,[9] the best results after 60 days of clinical evaluation were observed when an oxalic acid–based desensitization agent was applied prior to restoration. Those groups receiving the desensitization agent showed a more consistent reduction in sensitivity with complete elimination of dentin hypersensitivity after 1 year of treatment in more than 25% of cases (Table 5). Sensitivity was assessed at the first, second, and third months to determine whether there was a direct effect caused by the performance of the restoration on postoperative sensitivity. Measurements at the 6th, 9th, and 12th months were conducted to assess the regression of dentin hypersensitivity and the specific medium-term response to the treatments applied to each study group.[21]

The enhanced reduction in dentin hypersensitivity produced by applying the desensitizing agent in terms of intensity and absolute risk could be explained by the mechanisms of action of the different components of this desensitizing agent. The agent contained potassium oxalate 0.5%, potassium nitrate 4%, and potassium fluoride 4%, which in an acidic pH medium would raise the local calcium ion concentration, resulting in the precipitation of calcium oxalate and calcium fluoride crystals. Both types of mineral deposits could occlude exposed dentinal tubules. The potassium fluoride content could be considered as much an active ingredient for reducing dentin hypersensitivity as the oxalates, especially bearing in mind that the fluoride concentration (1.29%) exceeds the oxalate concentration (0.5%) in this desensitizing agent. This fact would explain the opposing results reported by Sartori et al,[10] who pretreated sensitive NCCLs with BisBlock, a 2.7% potassium oxalate product at pH 2.3, using 5 times more oxalate than in the present study. However, in that study, there was no reduction in postoperative sensitivity to air blasts. The difference in this study may therefore be due to the use of a mixture of oxalate plus fluoride. Moreover, the patients' selection by Sartori et al was different, without a moderate to severe sensitivity at baseline that very likely had an influence on the perception effect.[10] It is expected then that the oxalate and potassium flouride solution allows the sealing of exposed dentinal tubules and in the absence of dental stimulation it is possible for this inflamed tissue to recover in a more effective manner, which explains the results in the present study of decreased sensitivity 1 year after treatment.[22]

The effect of oxalic acid in achieving tubular occlusion after removal of the smear layer, as well as its influence on bond strength when used prior to restoration with composite resin restorations, is well documented.[10] However, its long-term and medium-term effect on pulpal inflammation when oxalic acid is used prior to restoration is not clear. Significant differences were observed when the two composite resins were compared with their respective adhesive systems used; however, this difference was marginal when compared to the effects of the application of oxalic acid.

Two different adhesive systems were applied in this study. For the silorane-based composite resin, a self-etching adhesive was used, and for the methacrylate-based composite resin, an etch-and-rinse adhesive system was used. Since the lesions restored had moderate to severe sensitivity at baseline, the etch-and-rinse system could theoretically have had a detrimental effect on postoperative sensitivity. However, in this trial there was no significant difference in sensitivity between the groups ($P > .05$) with different adhesive systems, which is similar to the results obtained by other clinical studies.[23,24]

In addition, in the present study, a silorane-based composite resin was included to restore NCCLs. This decision was based on the possibility that silorane's lower polymerization contraction might decrease postoperative sensitivity and recent publications supporting its use in class V noncarious lesions.[14]

A possible limitation of this study was that it did not specifically address the psychological component of pain that could lead to errors in the data. Despite this, the VAS scale is accepted and widely used in medicine. This is considered the most suitable method to assess pain levels, allowing the translation of subjective feedback on objective data.[25]

Patient selection was complex because the inclusion criteria required NCCLs with moderate to severe sensitivity with various types of teeth included (canines, premolars, molars, etc), which could represent a potential factor of bias. This criterion was chosen to allow that the effect of the treatments used could be clearly observed.

Another limitation of this study was the lack of baseline values. Due to concerns expressed by the Ethics Committee, it was recommended that a single dose of NSAID be administered prior to restoring NCCLs to minimize immediate pain. Therefore, it was decided not to evaluate baseline values (1 week after restoration) because of the possible added confounding effect of the NSAID on dentin hypersensitivity reduction. Nevertheless, the authors believe that this does not alter the results and the reduction in dentin hypersensitivity that was observed.

Dental pain may be caused by different stimuli, such as chemical, mechanical, or thermal, applied to the exposed dentin under oral conditions.[26,27] Holland et al[2] recommended that at least two hydrodynamic stimuli should be used for hypersensitivity evaluation and that a reasonable period of time must be allowed between stimuli. The stimuli must be measurable, reproducible, and clinically relevant.[28] Several studies have used a probe tip as a tactile stimulus because it causes the movement of dentinal fluid as a result of dentin compression. The triple syringe air blast is the most common stimulus used to evaluate dentin hypersensitivity[29] and is generally considered the most similar to naturally evoked pain, having no tendency to cause pain to nonsensitive teeth. The stimulus effects begin when the evaporation of the dentin fluid occurs, increasing fluid flow and activating hydrodynamic processes.[30] The stimulation usually accepted is a 1-second blast[31] (as applied in this study). Longer stimuli may cause odontoblasts to be drawn into the tubules. By using tactile and evaporative stimuli, the sensitivity level can be determined by a VAS.

The four treatment modalities assessed in the present study were confirmed to be effective in reducing the symptoms of dentin hypersensitivity, but a greater reduction was demonstrated when a commercial desensitizing agent was applied prior to restoration. The results are relevant to NCCLs with moderate to severe dentin hypersensitivity and showed a lasting and marked effect of the treatment, eliminating dentin hypersensitivity after 1 year of treatment in more than 25% of cases when a desensitizing agent was used. The prior application of this desensitizing agent was effective, simple, and not time consuming, and desensitizing agents have been shown not to alter the adhesion of the restoration.

Conclusions

The results suggest that restoration of hypersensitive NCCLs with composite resins reduces dentin hypersensitivity. This reduction is more pronounced if an oxalic acid–based desensitizing agent is applied prior to the restoration. This application also significantly reduces the absolute risk of pain after 6 months of treatment.

References

1. Splieth CH, Tachou A. Epidemiology of dentin hypersensitivity. Clin Oral Investig 2013;17 (suppl):s3–s8.
2. Holland GR, Narhi MN, Addy M, Gangarosa L, Orchardson R. Guidelines for the design and conduct of clinical trials on dentine hypersensitivity. J Clin Periodontol 1997;24:808–813.
3. Porto IC, Andrade AK, Montes MA. Diagnosis and treatment of dentinal hypersensitivity. J Oral Sci 2009;51:323–332.
4. Addy M. Tooth brushing, tooth wear and dentine hypersensitivity--are they associated? Int Dent J 2005;55(suppl):261–267.
5. Brannstrom M. The hydrodynamic theory of dentinal pain: Sensation in preparations, caries, and the dentinal crack syndrome. J Endod 1986;12:453–457.
6. Lin PY, Cheng YW, Chu CY, Chien KL, Lin CP, Tu YK. In-office treatment for dentin hypersensitivity: A systematic review and network meta-analysis. J Clin Periodontol 2013;40:53–64.
7. Erdemir U, Yildiz E, Kilic I, Yucel T, Ozel S. The efficacy of three desensitizing agents used to treat dentin hypersensitivity. J Am Dent Assoc 2010;141:285–296.
8. Chidchuangchai W, Vongsavan N, Matthews B. Sensory transduction mechanisms responsible for pain caused by cold stimulation of dentine in man. Arch Oral Biol 2007;52:154–160.
9. Barrientos C, Xaus G, Leighton C, Martin J, Gordan VV, Moncada G. Oxalic acid under adhesive restorations as a means to reduce dentin sensitivity: A four-month clinical trial. Oper Dent 2011;36:126–132.
10. Sartori N, Lopes GC, Vieira LC. Clinical performance of cervical restorations with desensitizing agents: 18-month clinical trial. J Adhes Dent 2012;14:183–189.
11. Albuquerque NL, de Souza AM, de Moraes MD, Medonça JS, Rodrigues LK, Santiago SL. Four-year randomized clinical trial of oxalic acid pretreatment in restorations of non-carious cervical lesions. Clin Oral Investig 2016;20:199–205.
12. Carvalho RM, Pereira JC, Yoshiyama M, Pashley DH. A review of polymerization contraction: The influence of stress development versus stress relief. Oper Dent 1996;21:17–24.

13. Eick JD, Welch FH. Polymerization shrinkage of posterior composite resins and its possible influence on postoperative sensitivity. Quintessence Int 1986;17:103–111.
14. Yaman BC, Doğruer I, Gümüştaş B, Efes BG. Three-year randomized clinical evaluation of a low-shrinkage silorane-based resin composite in non-carious cervical lesions. Clin Oral Investig 2014;18:1071–1079.
15. Hickel R, Roulet JF, Bayne S, et al. Recommendations for conducting controlled clinical studies of dental restorative materials. Science Committee Project 2/98--FDI World Dental Federation study design (Part I) and criteria for evaluation (Part II) of direct and indirect restorations including onlays and partial crowns. J Adhes Dent 2007;9(suppl 1):121–147.
16. Sarkis-Onofre R, Cenci MS, Demarco FF, et al. Use of guidelines to improve the quality and transparency of reporting oral health research. J Dent 2015;43:397–404.
17. Breivik H, Borchgrevink PC, Allen SM, et al. Assessment of pain. Br J Anaesth 2008;101:17–24.
18. Tay FR, Pashley DH, Mak YF, Carvalho RM, Lai SC, Suh BI. Integrating oxalate desensitizers with total-etch two-step adhesive. J Dent Res 2003;82:703–707.
19. Hannigan A, Lynch CD. Statistical methodology in oral and dental research: Pitfalls and recommendations. J Dent 2013;41: 385–392.
20. Faul F, Erdfelder E, Lang AG, Buchner A. G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. Behav Res Methods 2007;39:175–191.
21. Ricucci D, Loghin S, Lin LM, Spångberg LS, Tay FR. Is hard tissue formation in the dental pulp after the death of the primary odontoblasts a regenerative or a reparative process? J Dent 2014;42:1156–1170.
22. Kim SY, Kim EJ, Kim DS, Lee IB. The evaluation of dentinal tubule occlusion by desensitizing agents: A real-time measurement of dentinal fluid flow rate and scanning electron microscopy. Oper Dent 2013;38:419–428.
23. van Dijken JW. A randomized controlled 5-year prospective study of two HEMA-free adhesives, a 1-step self etching and a 3-step etch-and-rinse, in non-carious cervical lesions. Dent Mater 2013;29:e271–e280.
24. Perdigão J, Dutra-Corrêa M, Saraceni CH, Ciaramicoli MT, Kiyan VH, Queiroz CS. Randomized clinical trial of four adhesion strategies: 18-month results. Oper Dent 2012;37:3–11.
25. Pamir T, Dalgar H, Onal B. Clinical evaluation of three desensitizing agents in relieving dentin hypersensitivity. Oper Dent 2007;32:544–548.
26. Camps J, Salomon JP, Meerbeek BV, Tay F, Pashley D. Dentin deformation after scratching with clinically-relevant forces. Arch Oral Biol 2003;48:527–534.
27. Orchardson R, Gillam DG. Managing dentin hypersensitivity. J Am Dent Assoc 2006;137:990–998.
28. McGrath PA. The measurement of human pain. Endod Dent Traumatol 1986;2:124–129.
29. Kleinberg I, Kaufman HW, Confessore F. Methods of measuring tooth hypersensitivity. Dent Clin North Am 1990;34:515–529.
30. Närhi MV. Dentin sensitivity: A review. J Biol Buccale 1985;13: 75–96.
31. Pashley DH. How can sensitive dentine become hypersensitive and can it be reversed? J Dent 2013;41(suppl):S49–S55.