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Original Article

Effect of a modified methyl methacrylate-p-styrene sulfonic acid copolymer-based gel desensitizer on dentin permeability and tubule occlusion in human dentin *in vitro*

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KEYWORDS
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Abstract *Background/purpose:* Tubular occlusion is an effective method to treat dentin hypersensitivity. This study aimed to determine the effect of a modified methyl methacrylate-p-styrene sulfonic acid copolymer-based gel desensitizer on dentin permeability and tubule occlusion in extracted human premolars.
Materials and methods: Hydraulic conductance (HC) measurement (n = 50) and scanning electron microscopy (SEM; n = 64) were performed. Tooth specimens were divided into 6 groups and treated with: G1, distilled water for 30 s; G2, distilled water for 5 min; G3, gel desensitizer for 30 s; G4, gel desensitizer for 5 min; G5, 3% potassium oxalate for 30 s; G6, 3% potassium oxalate for 5 min. HC of dentin were measured before and after 30 s-etching with 35% phosphoric acid, at 0, 30, 60 min after group treatment and after 5-min acid challenge with 6% citric acid. The degree of tubule occlusion and the penetration depth of each agent were also determined. ANOVA and multiple comparison tests were used for data analysis.
Results: G3, G4, G5 and G6 significantly decreased in HC after group treatment every observation period, compared to after acid-etching ($P < 0.001$), and had 100% degree of tubule occlusion with penetration depth about 7.62, 7.94, 8.59 and 8.66 μm, respectively. However, G6 showed the greatest reduction in HC ($P < 0.05$).

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Conclusion: Gel desensitizer treatment, for only 30 s, could reduce dentin permeability and completely occlude the dentinal tubules, even though after acid challenge. However, 5-min treatment with potassium oxalate showed the greatest decrease in dentin permeability.

Introduction

Dentin hypersensitivity is one of the common dental problems in adults, and defined as short, sharp dental pain and discomfort due to dentinal tubules exposed by the loss of enamel or cementum, which probably associated with excessive brushing force, too much acidic food consumption and periodontal therapy.[1–3] From current systematic review and meta-analysis, the estimated prevalence of dentin hypersensitivity was 11.5%, which ranged from 1.3% to 92.1%.[4] The high prevalence of cervical dentin hypersensitivity was found among the patients with some specific conditions such as gingival recession, non-carious cervical lesion and tooth wear.[5] Also, the prevalence increased in young adults with parafunctional habits and favouring acidic diets.[6] In addition, previous studies reported that the prevalence of root dentin sensitivity was 9–23% before and 54–55% after non-surgical periodontal therapy,[7] and was about 76.8–80.4% following surgical periodontal therapy.[3] According to the hydrodynamic theory of dentinal pain, the root dentin exposed by periodontal therapy could be activated by painful stimuli such as cold, heat, tactile, osmotic or abrupt changes in temperature, leading to an increase in dentinal fluid flow rate and a generation of action potential in intradental nerve, finally produced dentinal pain or root dentin sensitivity.[8]

There are two main strategies for the treatment of dentin hypersensitivity: 1) desensitization of the intradental nerve and 2) occlusion of the exposed dentinal tubules.[9] Based on the observation in cats, the high level of potassium ions could decrease the intradental nerve activity; therefore, desensitizing agent containing high concentration of potassium salt could be recommended as a possible treatment of dentin hypersensitivity.[10] However, the topical application of high concentration of potassium chloride to the dentin cavity at atmospheric pressure was an effective method to produce significant but temporary decrease in the sensitivity of dentin in man.[11] Ideally, the restoration of the impermeability of dentinal tubules should be done to treat and prevent dentin hypersensitivity.[9] The treatment by dentinal tubule occlusion, such as fluoride, oxalate, dental adhesives, helps to prevent fluid movement in dentinal tubules, or reduce dentin permeability.[12,13] In vivo studies, the application of 3% potassium oxalate on hypersensitive teeth could reduce dentin hypersensitivity.[14,15] Additionally, the application of potassium oxalate could occlude dentinal tubules at root surface immediately and might help the dentist in treatment of patients with root hypersensitivity.[16]

Recently, a modified methyl methacrylate-p-styrene sulfonic acid copolymer-based gel desensitizer (Sun medical, Shiga, Japan) was newly developed from desensitizing agents of MS coat series and available commercially as 'Gel desensitizer'.[17] It consists of methacrylate-p-styrene sulfonic acid (MS) polymer, oxalic acid, potassium salt and sodium fluoride, and presents as gel-form in a syringe for easier application and higher effectiveness in managing dentin hypersensitivity. Previous studies reported that the conventional MS polymer based desensitizer (MS Coat One) showed the better results in the closure of dentinal tubules when compared to VivaSens and Gluma desensitizer,[18] and the MS polymer based desensitizer with sodium fluoride (MS Coat F) had ability in reducing root dentin demineralization under acid challenge.[19] In addition, the bovine dentin treated with gel desensitizer (the current inherited MS coat series) had the sustainable effect of dentinal tubule occlusion after immersion in artificial saliva for 1 week, but not found the such effect in the dentin treated with MS Coat One and MS Coat F.[20] However, the efficacy of gel desensitizer at different time treatment on dentin permeability before and after acid challenge is still not verified yet.

Dentin permeability test, by measuring the hydraulic conductance of dentin prior and post treatment, is a well-accepted laboratory method for screening desensitizing products.[21] Based on the hydrodynamic theory of dentin hypersensitivity, if any agent has a good ability in reducing the functional radius of dentinal tubules by occluding the tubules, it should greatly decrease a hydraulic conductance of dentin, therefore reducing the sensitivity of dentin.[21] 3% potassium oxalate solution showed the greatest reduction in hydraulic conductance of dentin (by 72%), when compared to desensitizing dentifrices containing pro-argin or strontium acetate.[12] Oxalate treatment also produced an immediate and stable effect on a decrease in hydraulic conductance of dentin, when the measurements were observed for 60 min.[15] Previously, the conventional MS polymer based desensitizer also had ability to decrease dentin permeability immediately, after 1 day, 1 week and 1 month *in vitro*.[22] Under SEM observations, the product containing potassium oxalate, MS Coat One and MS Coat F are all capable of occluding dentinal tubules to varying degree after dentin treatment.[19,23] However, there were no studies that compare the effect of the current modified MS copolymer-based gel desensitizer and 3% potassium oxalate solution on dentin permeability and tubule occlusion before and after acid challenge. Furthermore, it was well documented that many organic acids in foods could dissolve the smear layer or occluding agents, thus reopening the dentinal tubules and increasing dentin permeability and sensitivity.[2,24,25] Therefore, a desensitizing agent should resist to acid attack for the long-lived desensitizing effect.

Hence, this study aimed to determine the effect of the current modified MS copolymer-based gel desensitizer on dentin permeability and tubule occlusion in extracted intact permanent premolars at time 0, 30 and 60 min before and after acid challenge. The null hypotheses of the study were 1) the dentin treatment with gel desensitizer for 30 s or 5 min would not produce a decrease in the hydraulic conductance of dentin and occlude the dentinal tubules at time 0, 30, 60 min post treatment and after 5-min acid challenge with 6% citric acid. 2) There would be no difference between gel desensitizer and 3% potassium oxalate solution in their ability to decrease the hydraulic conductance of dentin and to occlude dentinal tubules at time 0, 30, 60 min post treatment and after acid challenge.

Materials and methods

The study protocol was approved by the Institutional Review Board, Faculty of Dentistry/Faculty of Pharmacy, Mahidol University, Bangkok, Thailand (COE.No.MU-DT/PY-IRB 2021/011.1405), and complied with the principles of the Declaration of Helsinki. The experiments were performed on 114 human intact permanent premolars which were scheduled for extraction as part of orthodontic treatment, and obtained from Oral and Maxillofacial Surgery Clinic, Dental Hospital, Faculty of Dentistry, Mahidol University. All the teeth were fully erupted, intact, without dental caries, crack or craze line and any restorations. The teeth with periodontal disease, history of endodontic treatment, crown fracture, attrition, abrasion, erosion, root resorption, or developmental anomalies were excluded. The teeth were then stored in 0.9% normal saline solution containing amoxicillin (500 mg/L) and used within two weeks after tooth extraction.[2]

Tooth preparation

All 114 premolars were cut below cemento-enamel junction 2 mm using diamond disc (type 917; Komet, Lemgo, Germany). Then, coronal dental pulp was removed by the help of tissue forceps and each pulp cavity was cleaned with distilled water to remove pulpal remnant. In each tooth, a cylinder dentin cavity with size 3 mm in diameter and 3 mm in depth was prepared at the buccal cusp using round and fissure diamond bur (No. 201 and No. 204; Intensive®, Viganello-Lugano, Switzerland) in an air-rotor handpiece with adequate water spray. Following the protocol of Kijsamanmith et al., each crown specimen was glued with cyanoacrylate cement (Alteco Inc., Osaka, Japan) to an acrylic block and connected to the fluid filtration system.[2,12]

Experimental design

The experiments were divided into 2 parts; part 1: hydraulic conductance (HC) measurement (n = 50) and part 2: scanning electron microscopy (SEM; n = 64). In each part of experiments, the prepared specimens were randomly divided into 6 groups as follow:

Group 1 (G1): Dentin was treated with distilled water for 30 s (HC, n = 5; SEM, n = 8).
Group 2 (G2): Dentin was treated with distilled water for 5 min (HC, n = 5; SEM, n = 8).
Group 3 (G3): Dentin was treated with a modified methyl methacrylate-p-styrene sulfonic acid copolymer-based gel desensitizer (sun medical®, Shiga, Japan) for 30 s (HC, n = 10; SEM, n = 12).
Group 4 (G4): Dentin was treated with a modified methyl methacrylate-p-styrene sulfonic acid copolymer-based

Table 1 Desensitizing agents used in the study.

Material used (Lot No)	Composition	pH	Manufacturer	Mode of application
Gel desensitizer (SW11)	Nano-sized methyl methacrylate-p-styrene sulfonic acid copolymer, oxalic acid, sodium fluoride (900 ppm), potassium salts, water	2.0	Sun Medical, Shiga, Japan	1. Dispense the desensitizer onto the dentin surface (standard quantity per tooth is the size of a grain of rice, diameter of 3 mm). 2. Leave it for 30 s (G3) or 5 min (G4). 3. Rinse with water thoroughly.
3% potassium oxalate solution (0001446770)	Potassium tetraoxalate dihydrate, distilled water	2.5	Sigma—Aldrich, St. Louis, MO, USA	1. Freshly prepare 3% potassium oxalate solution and use it within 24 h. 2. Fill the dentin cavity with 3% potassium oxalate solution and leave it for 30 s (G5) or 5 min (G6). 3. Rinse with water thoroughly.

gel desensitizer (sun medical®) for 5 min (HC, n = 10; SEM, n = 12).
Group 5 (G5): Dentin was treated with 3% potassium oxalate (Sigma–Aldrich, St. Louis, MO, USA) for 30 s (HC, n = 10; SEM, n = 12).
Group 6 (G6): Dentin was treated with 3% potassium oxalate (Sigma–Aldrich) for 5 min (HC, n = 10; SEM, n = 12).

The desensitizing agents used in this study are listed in Table 1.

Hydraulic conductance measurement

Fifty prepared specimens were used for HC measurement. One end of the fluid filtration device was connected to a manometer for setting the pressure in the system. The pressure of the system was set at 11 mmHg above atmospheric pressure to simulate the normal intrapulpal pressure during any dentin treatment,[26] and it was reset at 50 mmHg above atmospheric to represent hydrostatic pressure stimuli during HC measurement.[15] Time and distance of air bubble movement in the glass capillary (diameter of 300 μm, DADE®, Miami, FL, USA) of the fluid filtration device were observed and calculated for the HC of dentin.

For each specimen, the HC of dentin with smear layer was firstly measured. After that, the dentin cavity was etched with 35% phosphoric acid (3M ESPE, St. Paul, MN, USA) for 30 s to remove smear layer,[2] and the HC of etched dentin was measured again. Thereafter, each specimen was treated with the agent as assigned above. After each dentin treatment, the treated dentin was rinsed with distilled water thoroughly, and the HC of treated dentin was measured at time 0, 30, 60 min. Further HC measurement was made after 5-min acid challenge with 6% citric acid (Loba Chemie, Mumbai, India).[24,25]

Thereafter, the percentage (%) changes of HC of treated dentin at time 0, 30, 60 min and after acid challenge were calculated by the following formula: % change of HC (0, 30, 60 min or after acid challenge) = {[HC of etched dentin − HC of treated dentin (0, 30, 60 min or after acid challenge)]/HC of etched dentin} × 100%.[24,25]

At the end of the experiment, all specimens were longitudinally sectioned through the dentin cavity, and the remaining dentin thickness of each specimen was measured between the dentinal cavity floor and the closest pulpal horn using digimatic caliper (Mitutoyo, Kanagawa, Japan) to ensure that there was no variation in dentin depth which might affect the results of dentin permeability among the treatment groups.

Scanning electron microscopy

The additional 64 prepared specimens were used in the SEM study. The dentin cavity of each specimen was etched with 35% phosphoric acid (3M ESPE) for 30 s to remove smear layer.[2] Then, the specimens were randomly divided into 6 groups, and received dentin treatment as described above. After dentin treatment, each group was divided into 2 equal-sized subgroups: subgroup 1, no acid challenge and subgroup 2, acid challenge with 6% citric acid (Loba Chemie) for 5 min. To dehydrate the specimens, all specimens were dried in a desiccator. The specimens in each subgroup were bucco-lingual vertically fractured into 2 halves using a diamond disc (Komet) and a sharp chisel. After that, all specimens were mounted onto metal stubs, and sputter-coated with gold/palladium in a sputter coater (SC7620; Quorum Technologies Ltd, East Sussex, England). Thereafter, the specimens were examined under a scanning electron microscope (JSM-6610LV; JEOL, Tokyo, Japan). For cross sectional views, SEM images at magnification of x3000 were assessed independently by three reviewers to score the level of dentinal tubule occlusion as grade 1, 2, 3, 4 or 5, when each grade was defined as 100, 75, 50, 25 or 0 % of dentinal tubules being occluded, respectively.[27]

For longitudinal views, the penetration depth within dentinal tubules of each treatment group was the average of the distances (μm) of desensitizing agent occluded in dentinal tubules which were measured from the top of dentin surface to the deepest points of occluding agent found in dentinal tubules. The penetration depth of each group was presented as mean and standard deviation (SD).

Statistical analysis

The HC values of dentin, the percentage changes of HC of treated dentin in each group and the penetration depths of precipitation within dentinal tubules are represented as means and standard deviations (SDs).

In each group, the mean HC values of dentin before and after etching, at 0, 30, 60 min after agent treatment, and after acid challenge were compared using one-way RM repeated measure analysis of variance (1-way RM ANOVA). Among the groups (upon to the different agents, treatment times and observation periods), the mean percentage changes of HC were compared using three-way repeated measure analysis of variance (3-way RM ANOVA). Where this showed a significant effect, the Tukey test was used for pairwise multiple comparisons. *P* values less than 0.05 were considered significant.

To analyze the results of SEM study, two-way analysis of variance (2-way ANOVA) were used to compare the mean penetration depths of precipitation within dentinal tubules among the groups, with regarding the effect of different agents and treatment times. Where this showed a significant effect, the Tukey test was used for pairwise multiple comparisons. *P* values less than 0.05 were considered significant.

Results

Effect of different treatments on the hydraulic conductance of dentin

For each treatment group, the means and SDs of HC values of smeared dentin, acid-etched dentin, and treated dentin at 0, 30, 60 min post treatment and after acid challenge are shown in Fig. 1A–F.

In all treatment groups, the mean HC of dentin increased significantly after etching with 35% phosphoric acid, when compared to the mean HC of smeared dentin ($P < 0.001$).

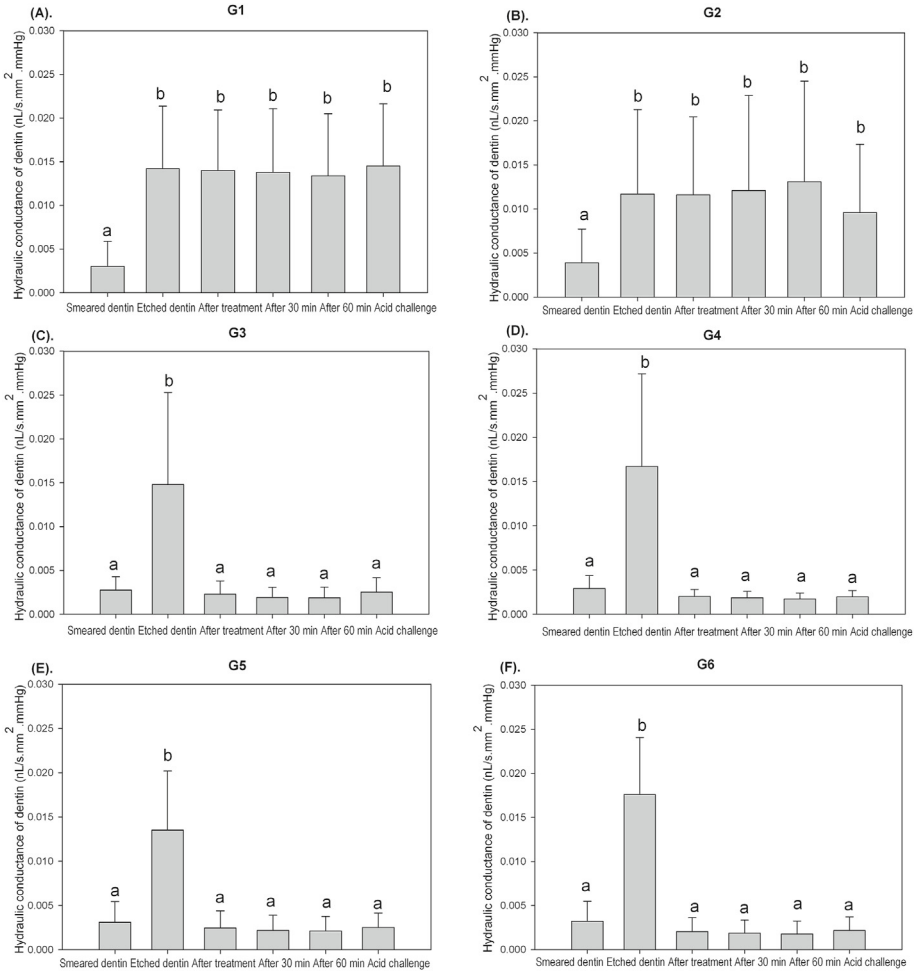

Figure 1 Mean (±1 SD) hydraulic conductance values of dentin before and after etching, after treatment with distilled water for 30 s (G1), distilled water for 5 min (G2), gel desensitizer for 30 s (G3), gel desensitizer for 5 min (G4), 3% potassium oxalate for 30 s (G5) and 3% potassium oxalate for 5 min (G6) at time 0, 30 and 60 min, and after acid challenge. The same lowercase letter represents no significant difference between observation periods in each group ($P > 0.05$, repeated measure, 1-way ANOVA).

After G1 and G2 (control) treated with distilled water for 30 s and 5 min, respectively, no significant differences were found between the mean HC values of treated dentin and the mean HC values of etched dentin every observation period post treatment ($P > 0.05$). Meanwhile, in experimental groups, dentin either treated with gel desensitizer for 30 s (G3) or 5 min (G4), or treated with 3% potassium oxalate for 30 s (G5) or 5 min (G6), the mean HC values of treated dentin at 0, 30, 60 min post treatment and after acid challenge were significantly lower than the mean HC value of etched dentin ($P < 0.001$). However, there were no significant differences between the mean HC of smeared dentin (before acid-etching) and the mean HC of treated dentin at 0, 30, 60 min post treatment and after acid challenge ($P > 0.05$).

The percentage reductions of HC of dentin treated with different desensitizing agents at different treatment times and observation periods at 0, 30, 60 min after treatment and after acid challenge are presented as mean (±SD) as shown in Table 2. The results of three-way ANOVA and Tukey test indicated that there were not significant effects of the types of desensitizing agents ($F = 1.414$, $P = 0.236$) and the observation periods before and after acid challenge ($F = 0.867$, $P = 0.460$), but there was a significant effect of the treatment time ($F = 7.735$, $P = 0.006$) on the percentage reduction of the HC of dentin. The interactions between the tested factors were not significant ($P > 0.05$).

Considering the percentage reductions of HC at 0, 30, 60 min post treatment and after acid challenge in each experimental group (Table 2), G3 had significant differences between 30 min post treatment and after acid challenge ($P < 0.001$), and between 60 min post treatment and after acid challenge ($P < 0.001$). In contrast, G4, G5 and G6 had no significant difference in the percentage decrease of HC among the observation periods at 0, 30, 60 min post treatment and after acid challenge ($P > 0.05$).

When comparison in gel desensitizer groups, there was no significant difference between G3 and G4 ($P = 0.535$). Meanwhile, in oxalated treatment groups, G6 showed a greater reduction in HC than G5 ($P < 0.001$).

In comparison between gel desensitizer and oxalate treatment groups within treatment time 30 s, there was no significant difference between G3 and G5 ($P = 0.613$). However, within treatment time 5 min, G6 had a greater

Table 2 The means and standard deviations (SD) of percentage reductions of hydraulic conductance of treated dentin with different desensitizing agents at different treatment times and observation periods at 0, 30, 60 min after treatment and after acid challenge, when compared to the hydraulic conductance of acid-etched dentin and the results of three-way ANOVA test showing F values.

Group	Agent	Treatment time	After treatment			After acid challenge
			0 min	30 min	60 min	
G3	Gel desensitizer	30 s	80.885^{aA} (15.014)	84.082^{bA} (11.076)	84.748^{bA} (10.366)	78.975^{aA} (14.102)
G4		5 min	82.382^{aA} (11.904)	83.581^{aA} (11.643)	85.026^{aA} (10.784)	83.400^{aA} (10.977)
G5	Potassium oxalate	30 s	79.920^{aA} (10.682)	81.979^{aA} (9.325)	82.638^{aA} (8.704)	79.454^{aA} (9.072)
G6		5 min	88.179^{aB} (6.845)	89.176^{aB} (6.533)	89.754^{aB} (6.639)	87.486^{aB} (6.749)
Three-way ANOVA			*F* values			
Agent			1.414^{NS}			
Treatment time			7.735*			
Observation period			0.867^{NS}			
Agent × treatment time			3.626^{NS}			
Agent × observation period			0.025^{NS}			
Treatment time × observation period			0.160^{NS}			
Agent × treatment time × observation period			0.075^{NS}			

Abbreviations: NS: not significant. Level of significance $^{*}P < 0.05$. Different lowercase letters indicate significant differences within each row, and different uppercase letters indicate significant differences within each column at $P < 0.05$ by Tukey test.

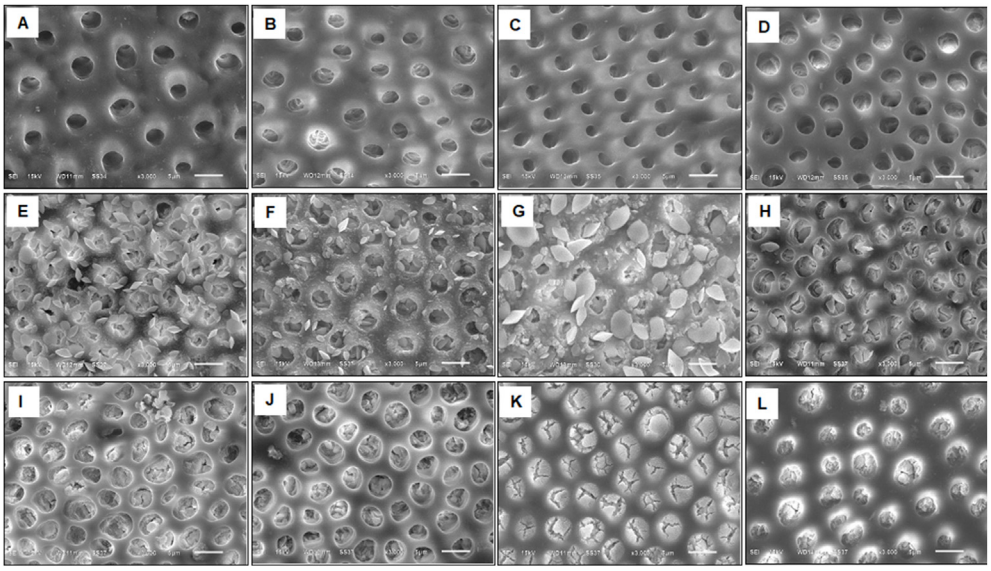

Figure 2 (A–L) Scanning electron micrographs of cross sectional views of human dentin (original magnification X3000, scale bar = 5 μm). (A, B) treated with distilled water for 30 s; (C, D) treated with distilled water for 5 min; (E, F) treated with gel desensitizer for 30 s; (G, H) treated with gel desensitizer for 5 min; (I, J) treated with 3% potassium oxalate for 30 s; (K, L) treated with 3% potassium oxalate for 5 min; (A, C, E, G, I, K) before acid challenge; (B, D, F, H, J, L) after acid challenge.

reduction in HC than G4 ($P = 0.029$). G6 showed the greatest reduction in HC, when compared to the other groups ($P < 0.05$).

Effect of different treatments on dentinal tubule occlusion

Scanning electron micrograph findings

Scanning electron micrographs of G1 and G2 showed clean dentinal surfaces with open dentinal tubules (score 5; Fig. 2A–D and Fig. 3A–D). Meanwhile, SEM images of G3, G4, G5 and G6 showed totally occluded dentinal tubules (score 1; Fig. 2E–G, I, K and Fig. 3E–G, I, K), even after acid challenge (score 1; Fig. 2F–H, J, L and Fig. 3F–H, J, L). G3 and G4 found various sizes of rhombus crystals precipitated on dentinal surfaces (Fig. 2E–H) and smaller size of rhombus crystals occluded within dentinal tubules (Fig. 3E–H). G5 and G6 found homogenous unspecified crystals deposited within dentinal tubules (Fig. 2I–L and Fig. 3I–L). However, G6 exhibited denser precipitation of oxalate crystals in tubules (Fig. 2K and L).

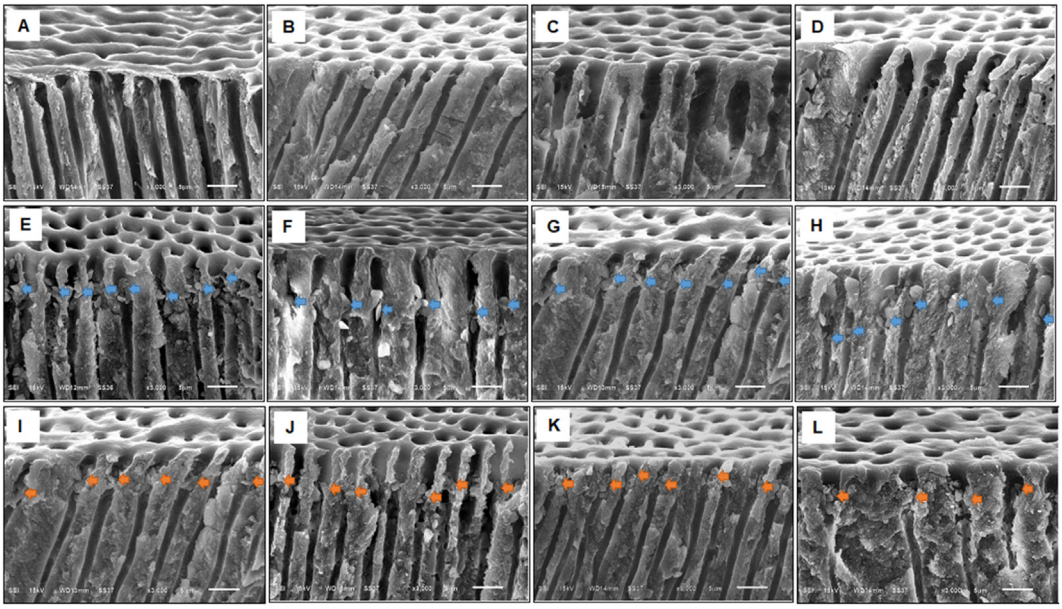

Figure 3 (A–L) Scanning electron micrographs of longitudinal views of human dentin (original magnification X3000, scale bar = 5 μm). (A, B) treated with distilled water for 30 s; (C, D) treated with distilled water for 5 min; (E, F) treated with gel desensitizer for 30 s; (G, H) treated with gel desensitizer for 5 min; (I, J) treated with 3% potassium oxalate for 30 s; (K, L) treated with 3% potassium oxalate for 5 min; (A, C, E, G, I, K) before acid challenge; (B, D, F, H, J, L) after acid challenge. Arrows showing precipitates formed within dentinal tubules.

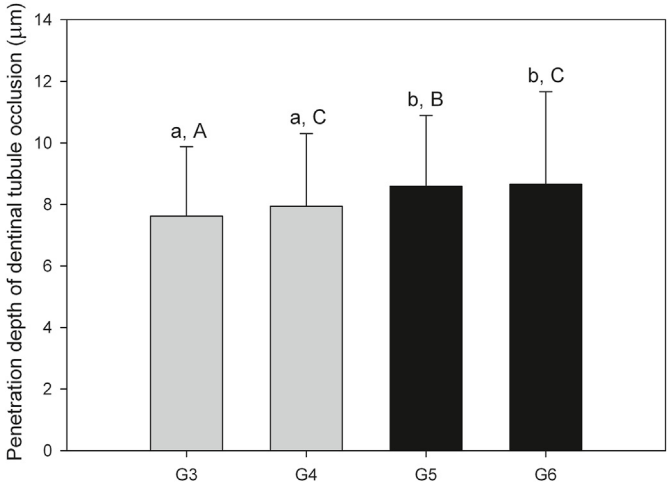

Figure 4 Mean (±1 SD) penetration depths of dentinal tubule occlusion after treatment with gel desensitizer (gray column) for 30 s (G3) and for 5 min (G4), and after treatment with 3% potassium oxalate (black column) for 30 s (G5) and for 5 min (G6). The same lowercase letter represents no significant difference between treatment times, and the same uppercase letter represents no significant difference between desensitizing agents ($P > 0.05$, 2-way ANOVA and Tukey test).

Penetration depth of dentinal tubule occlusion

After desensitizing treatment, the means and SDs of penetration depths of dentinal tubule occlusions of G3, G4, G5 and G6 were 7.62 ± 2.26, 7.94 ± 2.36, 8.59 ± 2.30 and 8.66 ± 3.01 μm, respectively (Fig. 4). Comparison between gel desensitizer and 3% potassium oxalate, within treatment time for 30 s, G3 gave lesser penetration within dentinal tubules than G5 ($P = 0.006$). Meanwhile, within treatment time for 5 min, there was no significant difference between G4 and G6 ($P = 0.061$). Regarding to the different level of treatment times (30 s vs 5 min), there were no significant differences both within gel desensitizer groups (between G3 and G4; $P = 0.399$) and within 3% potassium oxalate groups (between G5 and G6; $P = 0.856$).

Discussion

The present study demonstrated that the application of gel desensitizer on acid-etched dentin, even for as little as

30 s, could effectively reduce dentin permeability by occluding the dentinal tubules with crystalline precipitation immediately after treatment, and its effect remained stable throughout 60-min observation period. The tubule occlusion after gel desensitizer treatment was also resistant to 5-min acid challenge with 6% citric acid. Thus, the first null hypothesis was rejected.

The gel desensitizer used in this study consists of four main ingredients: methacrylate-p-styrene sulfonic acid (MS) polymer, oxalic acid, potassium salt, and sodium fluoride. As the gel desensitizer has the strong demineralization effect in relation to its strong acidity (pH 2), the effectiveness of gel desensitizer on dentinal tubule occlusion is thought to be due to the reaction of the MS polymer and oxalic acid with the calcium ions released from demineralized dentin. The MS polymer, or a water-soluble copolymer, could react chemically with the calcium ions supplied by demineralized dentin to form an insolubilized MS polymer–calcium complex, covering the exposed dentin surface and occluding the dentinal tubules.[19,20] Additionally, the oxalic acid could react with the calcium ions to form insoluble calcium oxalate crystals which obliterated the dentinal tubules.[23] In agreement with the previous studies, dentin permeability was immediately reduced after a single application of MS polymer based desensitizer.[22,28] Furthermore, the present study showed that gel desensitizer had sustainable effect on a reduction of dentin permeability over 60-min observation period and could resist to acid challenge with 6% citric acid. For the acid resistance of treated dentin, fluoride in gel desensitizer could remain in contact with the dentin surface and diffuse into the deep dentin, then facilitating the inhibition of dentin demineralization.[30] In addition, oxalic acid in gel desensitizer could also play a role in an acid-resistant layer of treated dentin, as its ability to generate insoluble calcium oxalate crystals covering the dentin surface.[31]

However, in group of dentin treated with gel desensitizer for 30 s (G3), the percentage change in dentin permeability increased after acid challenge when compared to the percentage values at 30 and 60 min before acid challenge. Meanwhile, in group of dentin treated with gel desensitizer for 5 min (G4), the acid challenge could not increase the percentage change of dentin permeability, suggesting that dentin treated with gel desensitizer for 5 min might resist to acid challenge better than that treated for 30 s. Due to the time dependent effect, fluoride in gel desensitizer could be more uptake into dentin and showed a greater acid resistance.[30]

When compared between gel desensitizer and 3% potassium oxalate solution, dentin treated with 3% potassium oxalate for 5 min (G6) exhibited the highest reduction in dentin permeability. Consistent with the SEM study, G6 had deeper penetration and denser precipitation of oxalate crystals within dentinal tubules of treated dentin. Thus, the second null hypothesis was rejected.

The significantly smaller calcium oxalate crystals, produced by the reaction between 3% potassium oxalate solution and available ionized calcium within dentinal tubules, were more effective in occluding dentinal tubules, resulted in a greater decrease in dentin permeability after the 5-min application of 3% potassium oxalate.[12] In agreement with the previous studies, dentin treated with 3% potassium oxalate had the sustainable effect of dentinal tubule occlusion and a reduction in dentin permeability over 60-min observation period,[15] and was acid resistant.[29] Meanwhile, dentin treated with gel desensitizer showed dentinal tubules occluded with various sizes of rhombus-shaped crystals, bigger sizes mostly found on the dentinal surface and smaller sizes mostly deposited within dentinal tubules. The prior studies by Kameyama et al., using the scanning electron microscopy and energy dispersive X-ray spectroscopy analysis of bovine dentin treated with gel desensitizer for 30 s, showed that the granular crystals on the dentin surface were predominantly composed of calcium, oxygen and carbon; therefore, it was reasonable to assume that the crystals on the dentin surface are calcium oxalate (CaC_2O_4) crystals.[20] Also, it was detected that the crystals occluding the dentinal tubules had the presence of calcium, phosphorus and fluoride, hence, it was presumed that the crystals in dentinal tubules could be the crystals of calcium phosphate [$Ca_3(PO_4)_2$] and calcium fluorophosphates ($CaFO_3P$).[20] Thus, the efficacy of desensitizing agent depends upon the pattern of crystalline precipitation in dentin.

However, potassium oxalate has a relatively short-term effect, since calcium oxalate is dissolved by saliva.[32] Moreover, the application of potassium oxalate to occlude root dentinal tubules to reduce root sensitivity was relatively short-lived because by 7 days after treatment the root tubules had reopened.[16] Meanwhile, the application of gel desensitizer produced complete tubule occlusion and remained occluded after a 1-week immersion in artificial saliva, as a result of the active ingredient 900 ppm sodium fluoride and the thickener in gel desensitizer.[20] Hence, further studies are needed to evaluate their effects on dentin permeability after long term immersion in artificial saliva. In addition, as gel desensitizer also contains potassium salts to enhance its desensitizing effect, the clinical experiments should be performed to evaluate its effectiveness in the treatment of hypersensitive dentin.

Within the limitation of the study, it was concluded that gel desensitizer treatment, for only 30 s, could reduce dentin permeability and completely occlude the dentinal tubules, even though after acid challenge. However, due to the time-dependent effect, 5-min treatment with potassium oxalate showed the greatest decrease in dentin permeability.

Declaration of competing interest

The authors have no conflicts of interest relevant to this article.

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References

1. Sehmi H, Olley RC. The effect of toothbrush abrasion force on dentin hypersensitivity in vitro. *J Dent* 2015;43:1442—7.
2. Kijsamanmith K, Surarit R, Vongsavan N. Effect of tropical fruit juices on dentine permeability and erosive ability in removing the smear layer: an in vitro study. *J Dent Sci* 2016;11:130—5.
3. Lin YH, Gillam DG. The prevalence of root sensitivity following periodontal therapy: a systematic review. *Int J Dent* 2012; 2012:407023.
4. Zeola LF, Soares PV, Cunha-Cruz J. Prevalence of dentin hypersensitivity: systematic review and meta-analysis. *J Dent* 2019;81:1—6.
5. Chabanski MB, Gillam DG. Aetiology, prevalence and clinical features of cervical dentine sensitivity. *J Oral Rehabil* 1997;24: 15—9.
6. Grippo JO, Simring M, Coleman TA. Abfraction, abrasion, biocorrosion, and the enigma of noncarious cervical lesions: a 20-year perspective. *J Esthetic Restor Dent* 2012;24:10—23.
7. von Troil B, Needleman I, Sanz M. A systematic review of the prevalence of root sensitivity following periodontal therapy. *J Clin Periodontol* 2002;29(Suppl 3):173—7.
8. Orchardson R, Gillam DG. Managing dentin hypersensitivity. *J Am Dent Assoc* 2006;137:990—8.
9. Kim JW, Park JC. Dentin hypersensitivity and emerging concepts for treatments. *J Oral Biosci* 2017;59:211—7.
10. Markowitz K, Bilotto G, Kim S. Decreasing intradental nerve activity in the cat with potassium and divalent cations. *Arch Oral Biol* 1991;36:1—7.
11. Noparatkailas S, Wanachantararak S, Vongsavan N, Matthews B. The effect of applying potassium chloride solutions at atmospheric pressure on the sensitivity of dentine in man. *Arch Oral Biol* 2009;54:50—4.
12. Kijsamanmith K, Surarit R, Vongsavan N. Comparison of milk and desensitizing dentifrices in reducing hydraulic conductance of human dentin in vitro. Southeast Asian. *J Trop Med Public Health* 2018;49:345—54.
13. Kijsamanmith K, Wallanon P, Pitchayasatit C, Kittiratanaviwat P. The effect of fluoride iontophoresis on seal ability of self-etch adhesive in human dentin in vitro. *BMC Oral Health* 2022;22:109.
14. Muzzin KB, Johnson R. Effects of potassium oxalate on dentin hypersensitivity in vivo. *J Periodontol* 1989;60:151—8.
15. Chinajitphan N, Ajcharanukul O, Kijsamanmith K, Vongsavan N, Matthews B. Time-course of the effect of potassium oxalate in the treatment of hypersensitive dentine in man. *Arch Oral Biol* 2021;126:105109.
16. Kerns DG, Scheidt MJ, Pashley DH, Horner JA, Strong SL, Van Dyke TE. Dentinal tubule occlusion and root hypersensitivity. *J Periodontol* 1991;62:421—8.
17. Sun Medical Company. *Gel desensitizer, desensitizer for hypersensitive teeth. Shiga, Japan: sun Medical*. Available at:. 2016. https://sunmedical.co.jp/english/support/pdf/ifu/gel-desensitizere.pdf. [Accessed 30 May 2021]. Data accessed.
18. Mushtaq S, Gupta R, Dahiya P, Kumar M, Bansal V, Melwani SR. Evaluation of different desensitizing agents on dentinal tubule occlusion: a scanning electron microscope study. Indian. *J Dent Sci* 2019;1:121—4.
19. Oshima M, Hamba H, Sadr A, Nikaido T, Tagami J. Effect of polymer-based desensitizer with sodium fluoride on prevention of root dentin demineralization. *Am J Dent* 2015;28:123—7.
20. Kameyama A, Haruyama A, Nakazawa Y, Furusawa M, Muramatsu T. Micrographic and spectroscopic analyses of the application of methyl methacrylate-p-styrene sulfonic acid copolymer-based desensitizing agents to bovine dentin. *Asian Pac J Dent* 2019;19:51—7.
21. Greenhill JD, Pashley DH. The effects of desensitizing agents on the hydraulic conductance of human dentin in vitro. *J Dent Res* 1981;60:686—98.
22. Zhang Y, Agee K, Pashley DH, Pashley EL. The effects of pain-free desensitizer on dentine permeability and tubule occlusion over time, in vitro. *J Clin Periodontol* 1998;25:884—91.
23. Gillam DG, Mordan NJ, Sinodinou AD, Tang JY, Knowles JC, Gibson IR. The effects of oxalate-containing products on the exposed dentine surface: an SEM investigation. *J Oral Rehabil* 2001;28:1037—44.
24. Kijsamanmith K, Banomyong D, Burrow MF, et al. Effect of conventional and acid-modified casein phosphopeptide-amorphous calcium phosphate crèmes on dentin permeability before and after acid challenge. *Operat Dent* 2019;44: 530—5.
25. Kijsamanmith K, Monthonjulaket N, Kuanpradit N, Thongwong K, Kijprasert N. The effect of iontophoresis delivery of fluoride in stannous fluoride desensitizing toothpaste on dentin permeability in human extracted teeth. *Sci Rep* 2022;12:13615.
26. Ciucchi B, Bouillaguet S, Holz J, Pashley D. Dentinal fluid dynamics in human teeth, in vivo. *J Endod* 1995;21:191—4.
27. Davies M, Paice EM, Jones SB, Leary S, Curtis AR, West NX. Efficacy of desensitizing dentifrices to occlude dentinal tubules. *Eur J Oral Sci* 2011;119:497—503.
28. Hong SW, Park NJ, Park YB, Lee KW. The effects of desensitizing agents, bonding resin and tooth brushing on dentin permeability, in vitro. *J Korean Acad Prosthodont* 2014;52: 165—76.
29. Pashley DH, Galloway SE. The effects of oxalate treatment on the smear layer of ground surfaces of human dentine. *Arch Oral Biol* 1985;30:731—7.
30. Matsuda Y, Altankhishig B, Okuyama K, et al. Inhibition of demineralization of dentin by fluoride-containing hydrogel desensitizers: an in vitro study. *J Funct Biomater* 2022;13:246.
31. Oguma H, Matsuda Y, Yoshihara K, et al. Prevention of root caries using oxalic acid. *Materials* 2023;16:1454.
32. Suge T, Ishikawa K, Kawasaki A, Yoshiyama M, Asaoka K, Ebisu S. Duration of dentinal tubule occlusion formed by calcium phosphate precipitation method: in vitro evaluation using synthetic saliva. *J Dent Res* 1995;74:1709—14.