Original Research

Comparative evaluation of the efficacy of the desensitizing and remineralizing agent in the reduction of dentin hypersensitivity after orthodontic debonding - a randomized clinical trial

Sasipriya Vatturu, Vivek Reddy Ganugapanta, Naga Ravi Teja, Gowri Sankar Singaraju, Prasad Mandava, JS Yamini Priyanka

Orthodontics Department, Narayana Dental College, Nellore, Andhra Pradesh, India

Abstract

Introduction. Enamel loss is a common problem during various orthodontic procedures. The study aims to compare the efficacy of a desensitizer and remineralizer in the reduction of the dentin hypersensitivity (DH) associated with enamel microcracks after orthodontic debonding.

Methods. A unicentric two arm parallel study with 30 subjects randomly assigned to each groups following debonding. *Group-1* subjects were treated with *Gluma® desensitizer* (5% glutaraldehyde and 35% hydroxyethyl methacrylate (HEMA)) and the *Group-2* intervention included a remineralizing agent *GC Tooth Mousse Plus®* (casein phospho peptide and amorphous calcium Fluro phosphate (CPP:ACFP)). The Visual Analogue Scale (VAS) was utilized to evaluate DH as subjective perception of pain following the Air blast test and Cold test. The VAS scale was indexed from 0-10 markings based on the intensity of perception. Five different time points T0 and T1 - immediately after debonding and intervention on day 1, T2 - 48 hours, and T3 after 72 hours were taken for the assessment of VAS scores.

Results. The VAS scores for the airblast test for group 1 were (2.73, 0, 0.06, 0.03) and group 2 (2.46, 0, 0.16, 0.13) at different periods. The sensitivity scores for the cold blast test for group 1 were (2.73, 0, 0.13, 0.03) and for group 2 (2.46, 0, 0.16, 0.13). There was 98 percent reduction in DH between T0 and T3 and was statistically significant ($p<0.05$) for both the groups.

Conclusion. Gluma® desensitizer and GC Tooth Mousse Plus® are equally effective in the reduction of DH in the orthodontic patient following debonding.

Keywords: dentin hypersensitivity, debonding, enamel cracks, orthodontic, CPP-ACFP, HEMA

Introduction

Orthodontic treatment, along with improving aesthetics and function, also carries with it some associated iatrogenic risks. Structural damage to enamel/ enamel loss is one among them, which can be reversible in the majority of cases if proper precautions are taken. Enamel, being a hard protector of the teeth can be chipped and cracked exposing the underlying dentinal tubules during different procedures in carrying out orthodontic treatment, which predisposes to DH. The surface texture of enamel is irreversibly affected [1], with a definite reduction in the thickness of enamel before and after orthodontic treatment [2]. There is qualitative as well as a quantitative loss of enamel reported from invitro studies related to the factors such as the initial prophylaxis [3,4], type of etchant used [5], duration of etching [1], adhesive characteristics such as filled and unfilled [3,5], self etching primer and enamel clean-up methods [6] light cured v/s self cure [7],

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Address for correspondence:
drgowrisankar@gmail.com

type of adhesive retention [8] and type of bracket, metallic or ceramic [8,9]. Further, during debonding, the enamel loss is evident due to some mechanical and chemical factors involved [10,11]; the base design, type of debonding force and the bracket surface area [12], the type of debonding plier and the bonding strength of the adhesive [7].

Debonding of brackets after orthodontic treatment carries the risk of enamel damage in the form of cracks, scratches, or enamel loss which accounts for 25%-40% enamel breakouts after debonding [13,14]. Enamel Micro-Cracks (EMCs), a form of enamel damage, not only compromise the appearance of the teeth but also cause stains and promote the accumulation of plaque on fractured surfaces. There is a significant increase in the number, length and width of enamel cracks after debonding with different pliers [7,14,15]. The enamel loss during various steps and in various forms, especially in the form of enamel cracks that is caused during orthodontic debonding generally attributes to carious lesions and dentinal hypersensitivity (DH) at the end of the orthodontic treatment [7,16,17].

Dentin hypersensitivity is characterized by a short, sharp pain arising from exposed dentine in response to tactile, evaporative, chemical or thermal stimuli and which cannot be ascribed to any other dental defect or pathology 18]. There is a need to provide treatment or relief of symptoms of DH caused after debonding. Specific treatment can be provided if the mechanisms of pain generation in DH are exactly known. Dentinal hypersensitivity results from the loss of enamel and exposure of dentinal tubules to the external environment and the stimuli. The universally accepted hydrodynamic theory as proposed by Brannstrom et al. [19] provides the presumable explanation for the mechanism of DH. The pressure changes within the dentin result when an open dentinal tubule is exposed to external stimuli resulting in the changes in the flow of fluid within the tubule leading to neural stimulation. Two stages must occur in the succession to cause DH: the exposure of the dentin followed by the opening of the dentinal tubules. Most of the therapeutic agents are developed empirically to reduce the movement of the dentinal fluid inside the tubules or block the neural transmission within the tubule and hence the pulpal response. Therefore, the plugging of the open tubules should abolish dentinal pain symptoms effectively [17].

A wide range of treatment options is available for treating this dentin hypersensitivity, arising from non-orthodontic conditions. The goal of treating DH is the immediate and permanent cessation of pain by using chemical agents that reduce pain by occluding the dentinal tubules mechanically. These can be applied either by the dentist (in-office treatment) or used by the patient as a home application. These treatment options include the application of various chemicals (desensitizing agents) such as potassium or ferric oxalates [20-22], Potassium nitrate [23,24], Fluorides [21-27], Calcium sodium phosphosilicate [28-30] and a biomimetic mineralization system (BIMIN) [31].

Beneficial effects can be produced by a combination of one or more agents. One such agent is an aqueous solution composed of 5% glutaraldehyde and 35% hydroxyethyl methacrylate (HEMA) [21,32,33]. Glutaraldehyde is a biological fixative and intrinsically blocks dentinal tubules. HEMA is a hydrophilic monomer, and it blocks the tubules by the coagulation of dentinal fluid proteins within the tubules, thereby counteracting the pain transmission that arises due to fluid movements.

Another agent used in the treatment is a remineralizer composed of CPP-ACFP (Casein phosphopeptide-Amorphous calcium Fluro phosphate). It is a calcium phosphate-based varnish with sodium fluoride. It is based on RECALDENT technology where the amorphous calcium stabilizes the phosphate phase and is capable of restoring the tooth integrity by a continuous supply of calcium, phosphate and fluoride ion deposition from the external source to the tooth [33]. This promotes remineralization of the tooth structure and occludes the dentinal tubules thus reducing DH [25,26,34-38]. A previous study by Lata 2010 [35] showed that CPP-ACP can effectively remineralize enamel subsurface lesions.

The review of literature reveals that there is a scarcity of data comparing the efficacy of desensitizing agents and remineralizing agents in reducing the DH after orthodontic debonding. Thus the current study was aimed to evaluate the sensitivity that is caused after orthodontic debonding in patients with visible enamel micro-cracks and compare the clinical efficiency of 5% glutaraldehyde and 35% HEMA with that of the remineralizer CPP-ACFP in the reduction of the resulting DH.

Methods

Study design

This is a uni-centered, two-arm parallel randomized clinical trial, each arm representing an individual treatment group. The study was carried out in the Department of Orthodontics and Dentofacial Orthopedics from 1st October 2018 to 30th September 2019. The protocol of this in vivo study was reviewed and approved by the Institutional Review Board (Reg. No. D178408011; Ref No. NDC/IECC/ORT/12-17/05 dated 05/12/2017).

Sample size determination

A minimum of 60 subjects is required as a sample size based on the previous studies [36,37]. A sample size of 28 subjects per each of the two treatment groups provides ≥80% power with a 5% confidence interval (CI) and a 90% confidence level to detect a minimal standardized effect size of the difference in the sensitivity intensity of '1' when measured on the VAS scale from 0 – 10 with a 1mm interval between any two readings. The cohort group for this study were the subjects due for debonding after comprehensive orthodontic treatment by fixed appliance therapy. After providing the written informed consent, subjects underwent study-specific screening procedures. The materials, methods, interventions, and protocols that were previously applied and verified as safety standards were followed in this study. The design of the study was shown in the flowchart (Figure 1).

Inclusion and exclusion criteria

A two-step selection procedure was made for the selection of the subjects for the final study. In the first step of the study, the eighty-two (82) participants who met the inclusion and exclusion criteria as mentioned below, were selected out of the ninety-two (92) enrolled participants in the predebonding phase. The inclusion and exclusion criteria for the first stage selection were adapted from the previous study by Dumbryte [16]. Inclusion criteria included age between 18-30 years, with sound mental and physical health. Patients bonded with 0.22 slot metal brackets with uniform base dimensions were selected. These brackets were bonded after conditioning the enamel with 34.5% phosphoric acid gel and utilizing light cure adhesive; duration of the active treatment did not exceed 24 months. Patients with pre-treatment history of enamel hypoplasia, periodontal surgery, on drug therapy for more than two months for medical and health problems were excluded from the study. Patients who underwent teeth whitening treatment and/or a previous history of using over the counter (OTC) or prescribed professional desensitizing treatments were not considered for inclusion. After debonding, in the second step, patients with visible enamel microcracks with individual sensitivity of 1 or above on the VAS scale 1-10 only were considered for the study.

Randomization and allocation

Following the second step examination for microcracks, a final sample of 60 subjects were found to be eligible for the study. The selected subjects (n=60) were randomly assigned to one of the two groups using SNOSE (Sequentially Numbered Opaque Sealed Envelope) method

- Group-1 (n=30): treated with combination product Gluma® desensitizer (Heraeus Kulzer GmbH, Wehreim, Germany); an aqueous solution of 5% glutaraldehyde and 35% hydroxyethyl methacrylate
- Group-2 (n=30): treated with remineralizing agent GC Tooth Mousse Plus® (Recaldent; GC India corp, India); composition - Calcium, potassium, fluoride 950 ppm, casein phosphopeptide and amorphous calcium phosphate (CPP:ACFP).

These assignments were carried out by nursing staff (GK) after a brief formal training regarding the procedure. After the intervention, the outcome is assessed by a secondary investigator (YPR) or an assessor who was blind to the allocation. Though basically, it was a single-blinded study, in essence, it was made as a double-blind procedure as the participant, and the score assessor was blinded for the study. For obvious reasons, the primary investigator (VSP) cannot be blinded to the interventions applied as he is the person directly involved in treating the subjects of the individual group.

Methodology

Predebonding procedures included the removal of the orthodontic archwire two weeks before the debonding procedure, and the dietary instructions, such as avoidance of hard solid, extremely hot or cold foods, were given. On the day of debonding, the teeth were kept out of occlusion (biting on a cotton roll) during the debonding. Debonding was done with the conventional utility Weingart pliers (Dentaurum, Ispringen, Germany) by hand (the mesial edge and distal edges of the bracket wings were squeezed gently until the bracket became free. Following debonding procedures, all visible residual adhesive was carefully removed using a slow-speed handpiece, and a 12 bladed carbide finishing bur and all the subjects irrespective of their participation in the study were admitted to the clinical evaluation for dentinal hypersensitivity. Debonding of all the brackets was done exclusively by the primary investigator (VSP).

Assessment of dentin hypersensitivity (DH)

At immediate post debonding stage 'T0', measurement of dental hypersensitivity was done using evaporative air stimulus and thermal cold stimulus as mentioned by Sowinski in his study [39]. Subjective assessment of Dentinal sensitivity was measured on a quantitative scale, perceived by the patient as pain sensation. Visual Analog Scale (VAS) was utilized, which is a 10 point numeric rating from 1-10 with intervals between each unit. This is followed by the application of treatment procedures.

The DH may vary depending upon the stimuli, so it is recommended that at least two hydrodynamic stimuli should be used. Usually, the least severe stimulus should be applied first [18,39]. Accordingly, for each patient, initially, the air blast test followed by the cold test was utilized. A cooling period of 10 minutes was given between the tests to minimize the interaction between stimuli. The site on the tooth surface from which the air blast test reading was measured was noted down. The same site was used to measure the subsequent cold test reading. In the follow-up visit also, the same tooth and same site were measured for both the stimuli for the assessment of DH.

Measurement of DH score

Subjects were asked to mark their level of sensitivity or discomfort by placing a mark at a point on a scale from 0 to 10 where '0' was 'no pain', and '10' was the 'worst possible pain' as a measure of response for both air and cold stimuli. The score was measured by the assessor by using VAS (Visual Analog Scale - a 10-point Numeric Rating Scale from 0-10 with an interval of '1' between two units of measurement).

Number of interventions and time periods of evaluation

Only a one time intervention at the start of study immediately after debonding was given and the total duration of the study was one week. The evaluation of the patient was completed at the following time intervals: T0 - Day 1 - Immediately after debonding before allocation at the second step elimination-pre-intervention; T1 - Day 1- after debonding - 10-30 seconds after intervention on the same day; T2 - on the 3rd day or 48 hours after T1; T3 - on the 7th day after first, 3 days after T2 measurement. At all time periods, the VAS scale was measured for both air and cold stimuli.

Consort flow diagram:

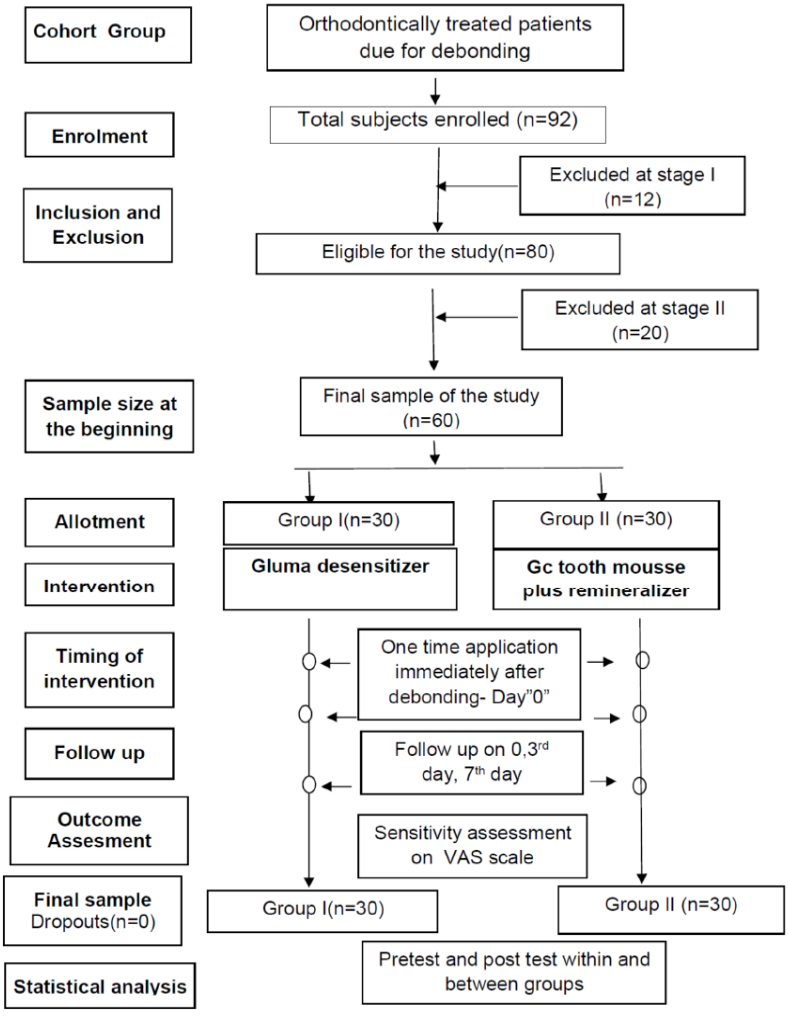

Figure 1. The design of the study was shown in the flowchart.

Intervention procedure

The Group-1 Gluma group (n=30) received the combination product GLUMA Desensitizer as a treatment procedure. Pumicing was done with pumice powder using a polishing brush. A small amount of Gluma desensitizer, which comes as a liquid in a small bottle, was applied onto the tooth surface using small cotton pellets as per the manufacturer's instructions and left for 30-60 seconds.

The surface was then gently dried by careful application of a stream of compressed air until the fluid film had disappeared, and the surface was no longer shiny, followed by rinsing with water thoroughly.

The Group-2 GC Tooth Mousseplus (n=30) received a remineralizing agent as follows: The material was applied by the examiner to the tooth surface using a clean, dry finger or cotton tip, and the material was left undisturbed for a minimum of 3 minutes. Then the patient was instructed to use the tongue to spread the remaining material throughout the mouth and asked to hold the material in the mouth as long as possible (an additional 1-2 minutes) by avoiding expectoration and delaying swallowing. Then the patient was asked to expectorate and if possible avoid rinsing. Any remaining material on the surface could be left to dissipate gradually. In both the groups the subjects were advised not to eat or drink for 30 minutes following the application of the desensitizing agent.

Procedure

Sensitivity was assessed at the point of Time (T1) measured on the VAS scale immediately after the initial

intervention to both the type of stimulus - air and cold, as mentioned earlier for 'T0'. Instructions were given to avoid foods with extreme temperatures above the oral temperature (37°C). A dietary chart was given to note down the diet history of the patient for the next week. The subjects were recalled for a follow-up visit on the 3rd day or 48hrs after the intervention by giving reminders via a phone call one day before the appointment. On each of the recall visits, the dietary chart of the patient was assessed; the possibility of the patient taking food that might affect the outcome of the measurement of the sensitivity was ruled out. At 'T2' and 'T3', DH stimuli tests were repeated using the same procedure as was done at 'T0' and 'T1'. No intervention was given except the assessment of the sensitivity at this timepoint. Necessary instructions were given and patients were asked to continue the filling of the dietary chart at T2. A final assessment of the sensitivity was repeated on the 7th day (T3), and it marked the endpoint of the study. After the study, the patients without sensitivity were eliminated from the study with the necessary retention protocols. In those patients with sensitivity greater than '1' after 'T3' a re-application of the intervention treatment was administered. No adverse effects were observed in any of the subjects enrolled in the study.

Statistical analysis

All the values on the pain scale were entered on the numerical scale as continuous data on a *Microsoft* excel sheet 2007. Data analysis was performed using the Statistical Package for Social Sciences (SPSS version 21). Basic descriptions were presented in the form of Mean and Standard deviation. The Independent Sample 't' test was used to analyze the difference between Group 1 and Group 2. The paired 't' test was used to assess the difference at different time intervals within the two groups. The level of significance was set at $p<0.05$ for all tests.

Results

A total of 92 subjects were enrolled in the study, of which 60 subjects were evaluated (male-28 and female-32) in the final run. The mean age of the Desensitizer group and the Remineralizer group were 20.2±2.2 and 19.8±2.13, respectively. There was no statistically significant difference between the two groups regarding ages (Table I).

The mean VAS scores in both the groups for both the tests were highest at 'T0', recording a zero response at 'T1'. Further, there was an increase in the scores at 'T2', which finally declined at 'T3' or at the end of the 7th day. There was no statistically significant ($p<0.05$) difference in VAS score values at the different points of time when evaluated against each of the groups or for a different type of stimuli (Table II, Table III, Figure 2, Figure 3).

Individual interpair differences within each group between different time periods were evaluated. The treatment outcome was statistically evaluated by a paired 't' test from T0 to T1, T0 to T2 and T0 to T3. There was a statistically significant ($p<0.05$) change in the VAS score values for both the air blast test and cold test (Table IV) in both the two groups.

Table I. Age and gender distribution among both groups.

	Group -1 Desensitizer		Group-2 Remineralizer		p value
N	30		30		
Mean age	20.2 ± 2.2		19.8 ± 2.13		0.477
Gender	Male	Female	Male	Female	
	11	18	17	14	
VAS score	1.12 ± 0.24				Not assesed

Table II. Intragroup comparison of mean VAS scores at different time points in the two groups for the air blast and cold test.

Group	Timepoint	Air blast test	Cold test	p value
		Mean ± SD	Mean ± SD	
Group-1 (Desensitizer)	T0	2.73 ± 0.63	2.73 ± 0.63	1.000**
	T1	0	0	-
	T2	0.06 ± 0.25	0.13 ± 0.34	0.398**
	T3	0.03 ± 0.18	0.03 ± 0.18	1.000**
Group-2 (Remineralizer)	T0	2.46 ± 0.50	2.46 ± 0.57	1.000**
	T1	0	0	-
	T2	0.16 ± 0.46	0.16 ± 0.37	1.000**
	T3	0.13 ± 0.34	0.13 ± 0.34	1.000**

*Independent Sample 't' test: *p <0.05 (significant), **p >0.05 (Not significant) ; Group-1 - Desensitizer; Group-2 - Remineralizer;T0 - Sensitivity immediately after debonding; T1 - 10 to30 seconds after intervention on the same day; T2 - On the 3rd day or 48 hours after T1; T3 - On the 7th day after first, 3 days after T2 measurement.*

Table III. Comparison of the mean VAS scores of the two groups at different time points in the two groups for the air blast and cold test.

Test	Time point	Group-1 Desensitizer	Group-2 Remineraliser	t value	p value
Air blast test	T0	2.73 ± 0.63	2.46 ± 0.50	1.789	0.079**
	T1	0	0	-	-
	T2	0.06 ± 0.25	0.16 ± 0.46	1.041	0.302**
	T3	0.03 ± 0.18	0.13 ± 0.34	1.401	0.167**
Cold test	T0	2.73 ± 0.63	2.46 ± 0.57	1.703	0.094**
	T1	0	0	-	-
	T2	0.13 ± 0.34	0.16 ± 0.37	0.356	0.723**
	T3	0.03 ± 0.18	0.13 ± 0.34	1.401	0.167**

*Independent Sample 't' test: *p <0.05 (significant), **p >0.05 (Not significant); Group-1 - Desensitizer; Group-2 - Remineralizer; T0 - Sensitivity immediately after debonding; T1 - 10 to 30 seconds after intervention on the same day; T2 - on the 3rd day or 48 hours after T1; T3 - on the 7th day after first, 3 days after T2 measurement.*

Table IV. Comparison of the difference between the VAS scores for air blast test within the two groups – at different periods – using the paired 't' test.

	Group-1 Desensitizer				Group-2 Remineraliser		
	Time period	Mean difference	t value	p value	Mean difference	t value	p value
Air blast test	T0-T1	2.73	23.404	< 0.001*	2.46	26.626	< 0.001*
	T0-T2	2.66	26.718	< 0.001*	2.30	17.940	< 0.001*
	T0-T3	2.70	24.814	< 0.001*	2.33	21.073	< 0.001*
	T1-T2	0.06	1.439	0.161**	0.16	1.980	0.057**
	T1-T3	0.03	1.000	0.326**	0.13	2.112	0.043*
	T2-T3	0.03	1.000	0.326**	0.03	0.571	0.573**
Cold Test	T0-T1	2.73	23.404	< 0.001*	2.46	23.647	< 0.001*
	T0-T2	2.60	25.284	< 0.001*	2.30	27.028	< 0.001*
	T0-T3	2.70	24.814	< 0.001*	2.33	26.655	< 0.001*
	T1-T2	0.13	2.112	0.043*	0.16	2.408	0.023*
	T1-T3	0.03	1.000	0.326**	0.13	2.112	0.043*
	T2-T3	0.10	1.795	0.083**	0.03	1.000	0.326**

*Paired 't' test: *p <0.05 (significant), **p >0.05 (Not significant); sign convention is not considered T0 is taken as zero reference. Group-1 - Desensitizer; Group-2 - Remineralizer; T0 to T1 - time period between debonding and after intervention; T0 to T2 - 3 days; T0 to T3 - 7 days; T1 to T2 - 3 days; T1 to T3 - 7 days; T2 to T3 - 3 days.*

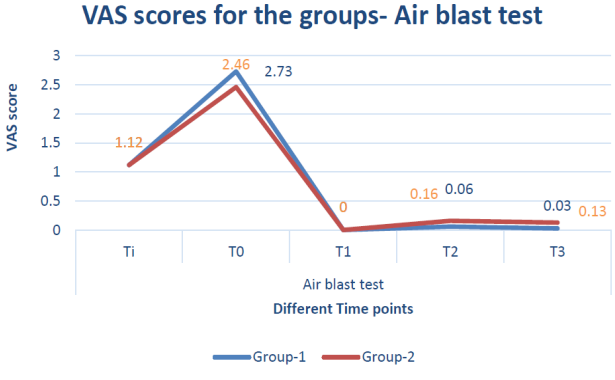

Figure 2. Comparison of VAS scores for the Air Blast test at different time intervals.

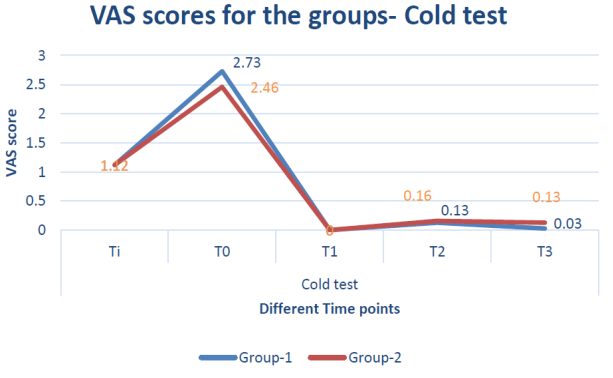

Figure 3. Comparison of VAS scores for the Cold Test at different time intervals.

Discussion

The structural changes that occur in enamel during and after orthodontic treatment is a cause of concern to both the patient and the orthodontist. Enamel loss can be seen in various stages of the orthodontic treatment, starting from the preparation for orthodontic bonding to the post debonding phase. This enamel loss during various steps and in various forms, especially in the form of enamel cracks that is caused during orthodontic debonding, generally attributes to dentinal hypersensitivity (DH). There is a significant increase in the number and length of enamel cracks after debonding with different pliers [7]. The chances of enamel surface damage after orthodontic debonding are increased if the visible enamel micro-cracks (EMC) are inclined less than 30-45 degrees and extending more than one- third of the buccal/labial surface of the tooth [14,16]. In such cases, there is increased susceptibility of the patient to carious lesions and Dentin Hypersensitivity (DH) at the end of orthodontic treatment. In a recent study it was shown that visible EMCs are associated with an increased DH compared to the patients without EMCs. The sensitivity was measured on the VAS scale at each time interval till the end of the 7th day, where it has reached normal levels in both the groups [16].

Dentinal hypersensitivity develops in two phases: Lesion localization (LL) and Lesion initiation (LI). LL occurs by loss of protective covering over dentin, thereby exposing it to the external environment. It includes loss of enamel by various reasons and gingival recession. When this protective natural veneer is lost, there is an exposure of dentinal tubules. This is followed by the removal of the smear layer, dentinal tubules open up, and the lesion is initiated, resulting in DH [17].

Various agents have been used for occluding the dentinal tubule in the process of treatment for DH problems. The obstructed tubules are generally feeble in contesting regular tooth erosion and abrasion. The search for a natural desensitizing agent with long-lasting effects has led to the observation that calcium phosphate minerals obstruct dentinal tubule orifices mimicking the natural process of sclerosis. Recently, milk protein casein has been used to develop a remineralizing agent CPP-ACFP which provides a continuous supply of calcium and phosphate ions in the immediate milieu of the dentinal tubules. Another combination product is Gluma Desensitizer Powergel contains 35% HEMA and 5% glutaraldehyde which coagulates the serum albumin in dentinal fluid. The reaction of glutaraldehyde with albumin induces polymerization of HEMA which plugs the dentinal tubule in succession [40,41]. A review of the literature shows that there is lacunae comparing the efficacy of sensitivity reducing agents after orthodontic debonding in patients with visible enamel micro-cracks. Thus, this study is aimed at the assessment of the efficacy of the desensitizer and remineraliser in reducing the DH during the post debonding phase of orthodontic treatment.

It is generally recommended that more than one type of stimulus should be used in clinical studies for evaluating dentine hypersensitivity [39]. This would enhance the sensitivity of the measurement of DH. Thus, in the present study, two types of stimuli, the air blast test, and the cold test, were utilized. Results of this study showed that teeth with visible enamel micro-cracks were equally sensitive to both air stimuli and thermal stimuli at baseline (after debonding) as well as at different time points (T1, T2, T3) in both the groups (Table II). The sensitivity values that were recorded after debonding decreased effectively immediately after treatment shows that both the agents are equally efficient in providing immediate relief after application irrespective of the stimuli. Both *Gluma* and *GC tooth mousse* plus were effective in reducing DH immediately and up to one week, which was evident by a decrease in VAS scores compared to baseline (Figure 2 and Figure 3).

Within group 1 there is no statistically significant difference between mean VAS scores (T0, T1, T2, T3) for the air blast test (2.73, 0, 0.06, 0.03) and the cold test (2.73, 0, 0.13, 0.03) at different time points (Table II). Within group 2, there is no statistically significant difference between mean VAS scores for air blast test (2.46, 0, 0.16, 0.13) and cold test (2.46, 0, 0.16, 0.13) at different time points (Table III). The results of the current study are not in concordance with the study of Pamir [42] and Dumbryte [16], wherein, VAS scores of the thermal stimuli caused higher patient discomfort than evaporative stimuli. There is an absolute reduction in VAS scores to zero in both the groups from baseline (T0) to immediately after intervention (T1) on day 1. Though there is a minute increase in scores as noticed at 'T2', it was negligible, and by the end of the seventh day (T3), the values remained close to zero. This was observed with both the stimuli tested (Table-IV, Figure 2 and Figure 3).

Both the *Gluma* and *GC tooth mousse plus* displayed a faster relieving action of sensitivity. An *in vitro* study of Joshi [16], revealed that after the initial application, Gluma desensitizer produced a greater number of partially occluded tubules and fewer completely occluded tubules. This occlusion might be due to the mechanism of Glutaraldedyde, which reportedly is based on total or partial closure of the tubules by protein coagulation and precipitation upon reaction with glutaraldehyde and hydroxyethyl methacrylate [43]. Regarding group-2, the results of this study are in accordance with a previous study; *GC Tooth Mousse* showed a rapid and sustained desensitizing action and was effective in reducing the cervical dental hypersensitivity [25]. The paste could have stayed on the surface of the tubule, possibly delivering Ca-P particles into the tubules. The tubular ratio and hydraulic pipeline could have been reduced, thus explaining the reduced pain and the drop of VAS values immediately on the application of the agent [44].

It was revealed by the studies of Lata [35] and Bou Chebel [38] that ACP remineralizes early lesions of enamel beneath the surface. The CPP, a bioactive product released, enhances the bivalent mineral solubility with the formation of a supersaturated solution of calcium and phosphate ions in the saliva and also optimizes the mineral binding ability of the calcium. At the interface of the tooth surface, both calcium and phosphate are able to precipitate hydroxyapatite in a stable manner. The fluoride ions help in remineralization by forming fluorapatite crystals in the presence of calcium and phosphate ions over the enamel surface, which is resistant to dissolution [17]. The effectiveness CPP-ACP on the regression of white spot lesions has been already established [45,46]. Thus *GC tooth mousse plus* can be thus advised in patients with DH after orthodontic debonding enamel cracks on a short term basis and for the regression of white spot lesions after orthodontic treatment, if at all observed.

Effective treatment of DH with long-term results has been related to the formation of intratubular dentin, which reduces the fluid flow rate or seals the tubule lumen [5]. Gluma that was used in the present study can penetrate exposed dentinal tubules up to 200 micrometer [47], resulting in the formation of multiple layers of protein septa that prevent intertubular fluid movements due to osmotic changes. At the same time, it provides a hermetic seal that acts as a microbial barrier, inhibiting bacterial growth and resurrects collapsed collagenous fibers, improving the bond strength of many adhesives. But reversible action of HEMA in Gluma may allow the re-opening of dentinal tubules leading to DH within a short duration [5].

Unfortunately, there is no published data and this hinders the comparison of the results of our study with variables tested. This is the first randomized clinical trial that evaluated the effectiveness of the desensitising agent after orthodontic debonding. The mean sensitivity scores in the present study immediately after debonding with air stimulation (Table III) were 2.73 and 2.46, respectively, in both the groups when measured on a 0-10 mm scale. Dumbryte [16] in his study showed that sensitivity scores after debonding in patients with visible enamel microcracks were 7.60, assessing the sensitivity scores on a 0-100 mm scale. Comparatively, higher values are obtained in our study. However, the sensitivity scores of the cold test (2.73, 2.46) of the present study matched closely with his results, a score 23, when measured immediately after post bonding.

To be considered as mild pain the VAS ratings should fall in between 5 to 44 mm whereas in the present study the recorded values are 2.46 to 2.73 on a 10 mm scale. The findings suggested that DH associated pain can be categorized as 'mild pain' when equated on a 100 mm VAS scale- Dumbryte [48]. The difference in the pain perception can be best assessed as percentage changes in VAS scores rather than the absolute change scores. A 33% decrease in pain is an acceptable standard for determining that a change in pain is meaningful from the patient's perspective as suggested by Jensen [48]. In the current study, there is an approximately 100% reduction in the immediate period and 98% reduction in sensitivity at the end of the 7th day. These findings suggest that both the agents can be effectively prescribed in reducing the immediate increase in DH associated with debonding.

Limitations

The efficacy of drugs was based on the responsive outcome variable measured. Thus, patient-reported outcome measurement (PROM) was measured on a VAS scale rather than a clinical outcome assessment. The performance outcome cannot be measured directly. Duration of the study is short; so long term effects of the desensitizer and remineralizer were not assessed. The present study is aimed at measuring the reduction in the sensitivity only. However, studies with increased sample size with prolonged duration are needed to test the remineralizing capacity of the Gc tooth mousse plus in relation to the reduction in the number of enamel cracks and hence the reduction in the sensitivity. The surface characteristics of the enamel cannot be assessed by this *in vivo* study.

Conclusions

1. Debonding may lead to a short term increase in tooth sensitivity.
2. From this study, it is clear that both the Gluma *desensitizer* and the *GC tooth mousse plus* are equally effective in providing immediate relief, and their effect was maintained up to 7days irrespective of the stimulus used in the study.
3. *GC tooth mousse plus* seems to have the upper hand in providing immediate relief from sensitivity; however, the difference in sensitivity reduction between the two medicaments is not statistically discernible.
4. For evaluating the remineralizing capacity of Gc tooth mousse plus a further study has to be carried out over a long term duration.

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