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EVALUATION AND COMPARISON OF GLUMA DESENSITIZER AND POTASSIUM OXALATE DESENSITIZER IN THE TREATMENT OF DENTINAL HYPERSENSITIVITY INDUCED BY PERIODONTAL THERAPY

Periodontology

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KEYWORDS

INTRODUCTION

Periodontal therapy for treatment of chronic periodontitis needs nonsurgical or surgical therapy to create healthy periodontal status. But active periodontal therapy can result in unwanted sequelae like gingival recession or dentinal tubule exposure, which results in the onset of dentin hypersensitivity (DH). Dentinal hypersensitivity (DH) is a common clinical condition, usually seen after periodontal treatment, especially scaling and root planing. Removal of cementum during the procedures exposes dentinal tubules, resulting in acute sensitivity to thermal, tactile, chemical, and osmotic stimuli. The condition, characterized as sharp, fleeting pain from exposed dentin, cannot be explained by other dental defects or pathologies (Canadian Advisory Board on Dentin Hypersensitivity, 2003) [1]. Dentin hypersensitivity has been termed as "A short, sharp pain occurring from exposed dentinal in reaction to stimuli, commonly thermal, evaporative, tactile, osmotic or chemical and which cannot be attributed to any other dental imperfection or disease.".

At present, "Root Sensitivity" (RS) is a term used to refer to the sensitivity caused by periodontal pathology and its treatment [2,3]. Research indicates an incidence of DH to be 36.8% to 98% in the immediate post-therapy period, Lin and Gillam in a systematic review documented that the incidence of root sensitivity after periodontal therapy was 36.8% at 1st week, 33.4% at 2nd week, and 29.6% at 4th week of the periodontal surgical interventions. Making its efficient management essential for patient comfort and compliance with oral hygiene procedures (Gillam & Orchardson, 2006; Lin & Gillam, 2011) [4,5,6].

The generally accepted hydrodynamic hypothesis of Brännström (1963) blames the pain on movement of fluid in exposed dentinal tubules. Alterations in fluid dynamics in tubules due to stimuli result in depolarization of nerve terminals. More distress is usually generated by outward movement of fluid, e.g., cold exposure, than by fluid movement inward by heat.[7]

Management of DH is directed towards two major strategies: dentinal tubule occlusion or diminution of nerve excitability. Several treatment modalities such as sodium fluoride, potassium nitrate, adhesive resins, and lasers have been utilized with variable success. [8,9]. Gluma® and potassium oxalate are some of the most commonly used desensitizing agents. Gluma Desensitizer is a 5% glutaraldehyde and 35% Hydroxyethyl methacrylate containing preparation used as a one-step, chair-side simple procedure for the prevention and treatment of dentinal hypersensitivity. [10]. Gluma® includes glutaraldehyde and hydroxyethyl methacrylate (HEMA) that occlude the dentinal tubules by protein coagulation, providing a barrier to stimuli [11].Potassium oxalate, meanwhile, precipitates out to form calcium oxalate crystals occluding tubules and limiting fluid flow [12].

Advances in laser-assisted treatments have further increased treatment possibilities for DH. Diode and Nd:YAG lasers, which are high-power lasers, work by melting and recrystallizing dentin, sealing the tubules effectively. Low-power lasers, through photobiomodulation, increase tertiary dentin formation by stimulating odontoblast activity, providing additional therapeutic potential [13] Combination treatments, using lasers with fluoride-based desensitizing agents, are intended to maximize the occlusion of dentinal tubules for better efficacy.

There has been limited comparative clinical research identifying the effectiveness of these treatments despite their availability for DH after periodontal treatment. This current study aims to compare and determine the effectiveness of Gluma® and potassium oxalate on DH after periodontal therapy. Through integration of patient-completed questionnaires and clinical readings, this investigation will provide evidence-based recommendations to ensure the optimal management of DH, enhancing the clinical protocol as well as treatment of patients. [14,15]

MATERIAL AND METHODS

This study was a randomized, double-blind, controlled clinical trial conducted at a dental college & hospital with a sample size of 60 systematically healthy patients. The inclusion criteria consisted of patients aged between 20 and 60 years, diagnosed with chronic periodontitis, and indicated for either non-surgical or surgical periodontal therapy. Among these, 30 patients underwent non-surgical periodontal therapy, while the remaining 30 received surgical periodontal therapy. The selected patients reported dentinal hypersensitivity on the 7th postoperative day, affecting at least two teeth in two quadrants, with hypersensitivity scored at or above 2 cm on the visual analog scale (VAS).The primary objective was to evaluate and compare the efficacy of Gluma desensitizer and Potassium oxalate desensitizer in reducing dentinal hypersensitivity across various time points post-treatment.

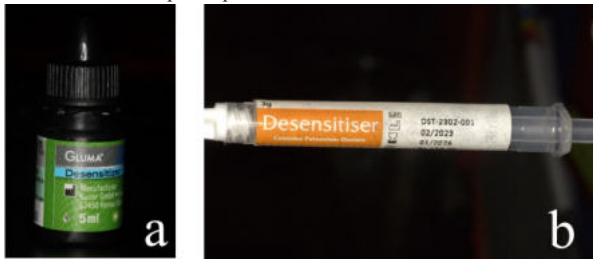

Fig 1: a: Gluma desensitizer, b: Potassium oxalate desensitizer

The ethical approval for the study was obtained from the institutional

review board, and informed consent was secured from all participants. To ensure a controlled and unbiased distribution of the desensitizing agents, patients were randomized using the envelope method into two groups. In each patient, Gluma desensitizer was applied to one randomly selected quadrant, while Potassium oxalate desensitizer was applied to the other. The double-blind design ensured that both the clinician administering the treatment and the patient were unaware of which desensitizer was used in each quadrant, thus minimizing bias.

Hypersensitivity was assessed at five distinct time points: immediately postoperative, one week after treatment, two weeks post-treatment, one-month post-treatment, and three months post-treatment. The evaluation of dentinal hypersensitivity was performed using three distinct stimuli: tactile, cold, and air. For the tactile stimulus, a UNC-15 probe was used, moving in a mesiodistal direction along the buccal surface of the hypersensitive teeth. The patients' responses were recorded using the Visual Analog scale (VAS) ranging from 1 to 10. Cold stimulation was administered by isolating the hypersensitive teeth, followed by the application of 0.5 ml of freshly melted ice-cold water using a 2-ml disposable syringe with a 24-gauge needle. The VAS was used to record the pain response. For the air stimulus, a three-way syringe attached to the dental chair was employed, delivering an air blast at a distance of 1 cm from the tooth surface for three seconds, after which the VAS score was recorded.

Following baseline hypersensitivity assessment, Gluma desensitizer and Potassium oxalate desensitizer were applied according to the manufacturer's instructions. A disposable brush applicator was used for uniform application of the desensitizing agents, ensuring proper coverage. Patients were then recalled at the designated intervals—immediately postoperative, after one week, after two weeks, after one month, and after three months—to reassess the degree of hypersensitivity using the same stimuli and VAS scoring system. A five-minute interval was maintained between the application of each stimulus to ensure consistent and accurate pain assessment.

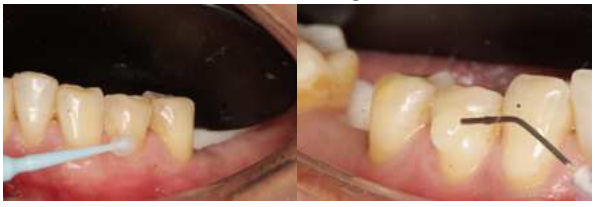

Fig 2: a: Application of Gluma desensitizer, b: Application of Potassium oxalate desensitizer

The collected VAS scores were analyzed statistically to determine the effectiveness of each desensitizer. Descriptive statistics, including mean and standard deviation, were calculated for the VAS scores within each group at each time point. Intragroup comparisons were performed using paired t-tests to assess changes in sensitivity over time within each group. Intergroup comparisons between the Gluma and Potassium oxalate groups were conducted using Analysis of Variance (ANOVA), with a significance level set at 0.05.

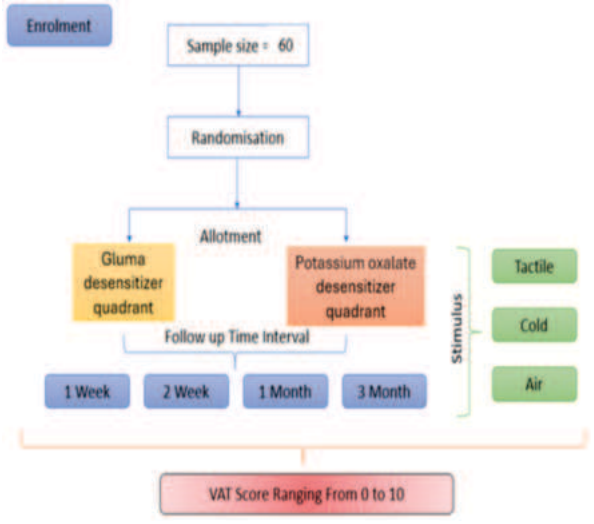

Fig 3: Flowchart of methodology of the study

RESULTS

Table 1. Intragroup comparison of mean VAS score among Gluma group at different time period

Days	N	Stimulus			F	p value
		Tactile Mean±SD	Cold Mean±SD	Air Mean±SD		
1 week	60	7.21±1.92	7.75±1.66	7.75±1.66	2.384	0.095
2 weeks	60	3.23±1.81	3.35±1.91	3.25±1.80	0.070	0.932
1month	60	1.41±1.33	1.45±1.38	1.41±1.33	0.012	0.988
3 months	60	0.18±0.39	0.18±0.39	0.18±0.39	0.000	1.000

Table 1 depicts the response (tactile, cold, and air) in 60 participants by using Gluma Desensitising agent over 1 week, 2 weeks, 1 month, and 3 months. At 1 week, mean sensitivity scores were high (7.21–7.75), with an F-value of 2.384 and a p-value of 0.095, indicating no significant differences between stimuli. By 2 weeks, scores decreased significantly (3.23–3.35), with an F-value of 0.070 and a p-value of 0.932. At 1 month, sensitivity further dropped to 1.41–1.45, with negligible variance (F = 0.012, p = 0.988). By 3 months, sensitivity was minimal (0.18), with F = 0.000 and p = 1.000, confirming uniform desensitization.

Table 2: Intragroup comparison of mean VAS score among potassium oxalate group at different intervals of time

Days	N	Stimulus			F	p value
		Tactile Mean±SD	Cold Mean±SD	Air Mean±SD		
1 week	60	7.03±1.98	7.76±1.65	7.03±1.99	3.020	0.051
2 weeks	60	3.91±1.90	4.03±1.93	3.90±1.90	0.086	0.917
1month	60	1.96±1.43	1.98±1.46	1.96±1.43	0.003	0.997
3 months	60	0.48±0.53	0.48±0.53	0.48±0.53	0.000	1.000

Table 2 depicts the response (tactile, cold, and air) in 60 participants by using potassium oxalate desensitising agent over 1 week, 2 weeks, 1 month, and 3 months in 60 participants. At 1 week, mean sensitivity scores were 7.03–7.76, with an F-value of 3.020 and a p-value of 0.051, indicating a trend towards significance but no substantial differences between stimuli. By 2 weeks, sensitivity reduced to 3.90–4.03, with an F-value of 0.086 and a p-value of 0.917, showing no significant differences. At 1 month, sensitivity dropped further to 1.96–1.98, with negligible variance (F = 0.003, p = 0.997). By 3 months, scores were minimal (0.48), with F = 0.000 and p = 1.000, indicating consistent desensitization across all stimuli types.

Table 3: Inter group comparison of mean VAS scores at different time intervals

Stimulus	Days	N	Gluma Mean±SD	Pot Mean±SD	95% CI	t	p value
Tactile	1 week	60	7.21±1.92	7.03±1.98	-.52 to .88	.514	.608
	2 weeks	60	3.23±1.81	3.91±1.90	-1.35 to .00	-2.010	.047
	1 month	60	1.41±1.33	1.96±1.43	-1.05 to -40	-2.174	.032
	3 months	60	.18±.39	.48±.53	-.46 to -.13	-3.503	.001
Cold	1 week	60	7.75±1.66	7.76±1.65	-.61 to .58	-.055	.956
	2 weeks	60	3.35±1.91	4.03±1.93	-1.37 to -.01	-1.944	.054
	1 month	60	1.45±1.38	1.98±1.46	-1.04 to -.01	-2.049	.043
	3 months	60	. 18±.39	. 48±.53	-.46 to -.13	-3.503	.001
Air	1 week	60	7.03±1.99	7.03±1.99	-.72 to .72	.000	1.000
	2 weeks	60	3.25±1.80	3.90±1.90	-1.31 to .01	-1.923	.057
	1 month	60	1.41±1.33	1.96±1.43	-1.05 to -.04	-2.174	.032
	3 months	60	. 18±.39	.48±.53	-.46 to -.13	-3.503	.001

Table 3 compares the mean VAS scores for tactile, cold, and air stimuli between the Gluma and potassium oxalate groups at different time intervals. At baseline, both groups exhibited similar responses for all

three types of stimuli. However, during follow-up, the Gluma group consistently demonstrated significantly greater reductions in VAS scores compared to the potassium oxalate group. The most notable differences were observed at 3 months for all stimuli, with the Gluma group showing superior reductions in VAS scores (p = 0.001). This highlights the greater efficacy of Gluma in reducing sensitivity over time across all stimulus types.

Table 4: Interaction between treatment groups, stimulus and time on VAS scores

Independent variables	Dependent variable - VAS scores Mean square	F	P value
Treatment groups	48.034	20.811	.000
Days	3298.660	1429.161	.000
Stimulus	6.801	2.946	.053
Groups*days	9.230	3.999	.008
Groups*Stimulus	.072	.031	.969
Days*stimulus	4.157	1.801	.095
Groups*days*stimulus	.104	.045	1.000

Table 4 presents the interaction effects of treatment groups, stimuli, and duration on mean VAS scores. Both the treatment groups and the duration had a statistically significant impact on the mean VAS scores (p = 0.000). Additionally, the interaction between treatment groups and duration was also statistically significant (p = 0.008). Overall, the study demonstrated that the mean VAS scores decreased over the 3-month period with the application of both Gluma and potassium oxalate, highlighting the effectiveness of both treatments in reducing sensitivity over time.

DISCUSSION

Dentinal hypersensitivity (DH) is a common occurrence as a result of non-surgical periodontal treatment, including scaling and root planing, in which exposed root surfaces elicit pain upon contact with thermal, acidic, or sweet stimuli. [3] Patients often avoid cleaning oral hygiene in the involved sites due to this condition, resulting in a build-up of plaque and a cycle of sensitivity and inflammation. The evidence from systematic review shows that the peak of root sensitivity occurs within the first week after surgery, with patients complaining of severe discomfort. Such hypersensitivity not only undermines the efficacy of periodontal treatment but also affects the patient's overall quality of life.[2]

Treatment of postoperative DH is necessary to guarantee the success of periodontal therapy and patient compliance with oral hygiene regimens. A good intervention should be one that offers relief early, ideally within 24–48 hours, to resolve symptoms and allow for the return to effective oral care.[9] This emphasizes the clinical significance of recognizing and applying effective desensitizing agents to reduce DH and maximize therapeutic results.

Table 5: Theories of Dentin Hypersensitivity

Theory	Key Concept	Evidence/Limitations
Direct Innervation Theory	Nerves extend through dentin to the dentin-enamel junction, and direct stimulation activates them.	• Lack of evidence for nerve presence in outer dentin. • Newly erupted teeth sensitive despite absence of nerve plexus. • Local anesthetics ineffective on dentin.
Odontoblast Receptor Theory	Odontoblasts act as sensory receptors, relaying signals to nerve endings.	• Odontoblasts are matrix-forming cells, not excitable. • No synaptic connections found between odontoblasts and nerves.
Hydrodynamic Theory	Fluid movement within dentinal tubules triggers mechanosensitive Aδ nerve fibers in the pulp-dentin complex.	• Widely accepted mechanism. • Open tubules and a thin smear layer facilitate fluid movement. • Cold and drying stimuli induce centrifugal fluid flow and intense pain.

The current clinical trial design is a double-blind, split-mouth and randomised design. The study design is quantified as a benchmark in assessing the no difference hypotheses among management techniques. It proved that Gluma and potassium oxalate desensitizing products significantly alleviated dentinal hypersensitivity (DH) within a 3-month duration, where Gluma proved to be more effective in early follow-up phases. These results complement previous studies, which explained mechanisms of action and clinical efficacy for these agents but also shed some light on disparities found in the literature.

The mechanism of action for Gluma includes glutaraldehyde as a primary molecule that reacts with serum albumin within dentinal fluid, initiating coagulation followed by precipitation in the dentinal tubules. This reaction creates hydroxyethyl methacrylate polymerization (HEMA), which creates deep tags that close off the tubules, thus impeding fluid flow and nerve stimulation. [10,11] This biochemical process, evidenced by research like that of Schüpbach et al. (2006) and Duran and Sengun (2004), accentuates Gluma's capacity to offer fast and efficient relief from hypersensitivity. [16,17] Furthermore, Yilmaz et al. (2011) proved in an SEM study that Gluma's active components successfully occlude dentinal tubules, thus proving its long-term efficacy in the treatment of DH.[18]

Potassium oxalate works by a different mechanism, diffusing along dentinal tubules to deposit calcium oxalate crystals. These physically occlude the tubules, hindering fluid movement and decreasing the excitability of intradental nerve fibers. Research by Hodosh (1974) and Tarbet et al. (1980) supports this mechanism of action, comparing the soothing effect of potassium ions on nerve fibers to an anesthetic-like effect. The effectiveness noted in this research is consistent with that of Kim (1986) and Al-Tayeb (2008), who proved that potassium ions can penetrate dentinal tubules and travel to nerve endings, thus reducing hypersensitivity. [19,20] In addition, Gillam et al. (2002) pointed out the use of potassium salts to reduce nerve excitability, highlighting their role in DH management.[9]

The comparative study among these agents revealed stable desensitization effects over time. Gluma revealed greater VAS score reduction during the early phases, consistent with its mechanism of rapid tubule occlusion. Potassium oxalate, although effective, showed a marginally slow response, possibly due to its dependency on crystallization in the tubules. This is of clinical importance in those instances necessitating rapid relief. The findings of this study are substantiated by Dondi dall'Orologio et al. (1999), where Gluma proved to decrease DH, and Sengun and Duran's report of sustained effectiveness.[21,17]

Literature contradictions, including results by De Assis et al. (2006), who found no effect of Gluma in periodontally treated patients, and Jalalian et al. (2010), who found Gluma to be less effective than potassium nitrate for post-crown preparation sensitivity, reflect the inconsistency in results. [22,23] Such inconsistencies could be due to differences in study protocols, patient populations, or procedural techniques. Moreover, the placebo effect and the physiological remineralization by secondary dentin deposition can also account for some portion of reported sensitivity decreases, postulated by Pearce et al. (1994) and West et al. (1997). Such physical and psychological effects highlight the need for well-controlled experiments to define the actual effectivity of desensitizing products. [24,25]

Other desensitization modalities, including laser treatment, fluoride varnishes, bioactive glass, and sodium fluoride products, have been found to be effective in decreasing DH. Laser treatment, as commented by Moraschini et al. (2017), coagulates proteins in dentinal tubules and inhibits nerve conduction.[26] Fluoride varnishes occlude tubules and add strength to enamel, as underscored by Ritter et al. (2006).[27]

The findings of this research highlight the clinical relevance of both desensitizing agents, with Gluma being more rapid in relief and potassium oxalate showing long-term effects. Both agents were well tolerated with no adverse effects reported, indicating their suitability for chronic use. Future studies need to address the mechanisms behind these differences and the use of novel imaging methods to measure tubule occlusion and fluid dynamics. Adding placebo-controlled designs might further determine the roles of physiological and psychological influences in desensitization.

In total, the research reinforces the efficacy of Gluma and potassium oxalate in DH management, especially post-periodontal therapy. Though both agents offer notable desensitization, the quick action of Gluma provides a clear edge in its use in clinical situations of urgent relief. This complete view of their mechanisms and relative efficacy offers important insights to direct treatment planning towards

individual patient needs.

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