|| ISSN(online): 2589-8698 || ISSN(print): 2589-868X ||
International Journal of Medical and Biomedical Studies
Available Online at www.ijmbs.info
NLM (National Library of Medicine ID: 101738825)
Index Copernicus Value 2019: 79.34
Original Research Article
Volume 5, Issue 1; January: 2021; Page No. 23-28

COMPARATIVE EVALUATION OF THE EFFICACY OF DIODE LASER, GLUMA DESENSITIZER AND DESENSITIZING MOUTH RINSE IN THE TREATMENT OF DENTINE HYPERSENSITIVITY – CLINICAL STUDY

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Article Info: Received 20 November 2020; Accepted 3 January 2021
DOI: https://doi.org/10.32553/ijmbs.v5i1.1614
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Conflict of interest: No conflict of interest.

Abstract

Introduction: Dentine hypersensitivity is characterized by acute, sharp pain arising from the exposed dentine, most commonly in response to thermal, tactile, or chemical stimuli, and which cannot be linked to any other pathological changes in the tooth or the environment. Therapy uses various impregnating agents in the form of solutions or gels and, in more recent times, lasers. Gluma Desensitizer, Desensitizing mouthrinse has been used previously in a dentifrice or gel to alleviate dentinal hypersensitivity.

Aim: To compare the efficacy of Diode laser, Gluma desensitizer and desensitizing mouth rinse in the treatment of dentine hypersensitivity.

Materials and Methods: The study contained 60 individuals (42 males and 18 females) of dentinal hypersensitivity. There were three groups in the study i.e., patients treated with laser, Gluma Desensitizer and Desensitizing mouth rinse. Before starting any treatment, the hypersensitivity was checked with the help of cold air blast and probe test according to VAS (Visual analog scale) the responses were evaluated from the patients.

Results: Group I (Laser) showing the highly significant results compared to group 2 (Gluma desensitizer) and group 3 desensitizing mouth rinse (3% Potassium Nitrate) in decreasing the dentinal hypersensitivity.

Keywords: dentine hypersensitivity, desensitizing agent, diode laser, laser therapy. Gluma Desensitizer; mouth rinses

Introduction

Tooth sensitivity is a very common clinical presentation which can cause considerable concern for patients. This condition is frequently encountered by dentists, endodontists, periodontists, hygienists and dental therapists. This condition generally involves the facial surfaces of teeth near the cervical aspect and is very common in premolars and canines.[1] It is a condition often termed as *"enigma being frequently encountered but poorly understood"* with a prevalence of 4-57 % and mostly occurs in the age group of 30-40 years.[2]

Dentin hypersensitivity has been defined as "*A short, sharp pain arising from exposed dentinal in response to stimuli, typically thermal, evaporative, tactile, osmotic or chemical and which cannot be ascribed to any other dental defect or pathology".*[3]

A modification of this definition was suggested by the Canadian Advisory Board on Dentin Hypersensitivity in 2003, which suggested that 'disease' should be substituted for 'pathology'.[4]

Various theories have been put forth to explain the mechanism of dentin hypersensitivity (DH) which includes Odontoblastic transduction theory, Neural theory, Hydrodynamic theory. The most widely accepted theory for dentin hypersensitivity is *hydrodynamic theory* given by GYSI[5] in 1900 and later explained by BRANNSTROM[6] in 1963 according to that the exposure of dentinal tubules allows movement of intra-dentinal fluid leading to stimulation of A-δ fibres causing hypersensitivity. Hence, occlusion of the tubules is supposed to block the hydrodynamic mechanism and reduce dentin sensitivity.

Conventional therapies for the treatment of dentinal hypersensitivity comprehend the topical use desensitizing agent, either professionally or at home such as nerve desensitizers (potassium nitrate), protein precipitators (glutaraldehyde, silver nitrate, zinc chloride, strontium chloride), dental tubule plungers (sodium fluoride, stannous fluoride, strontium chloride, potassium oxalate, calcium phosphate, calcium carbonate, bioactive glasses), dentin adhesive sealers (fluoride varnishes, oxalic acid and resin, glass ionomer cements, composites, dentin bonding agents) and recently lasers that include neodymium: yttrium aluminum garnet laser (Nd:YAG), gallium-aluminum-arsenide laser, erbium-YAG laser. high-power lasers such as: diode 980nm and 808 nm, KTP 532 nm, Nd: YAG 1064 nm, CO2 10600 nm, Er, Cr: YSGG 2780 nm, and Er: YAG 2940nm act on DH provoking a melting effect with crystallization of dentine inorganic component and the coagulation of fluids contained into the dentinal tubules.

Gluma Desensitizer is a preparation containing 5% glutaraldehyde and 35% Hydroxyethyl methacrylate and is

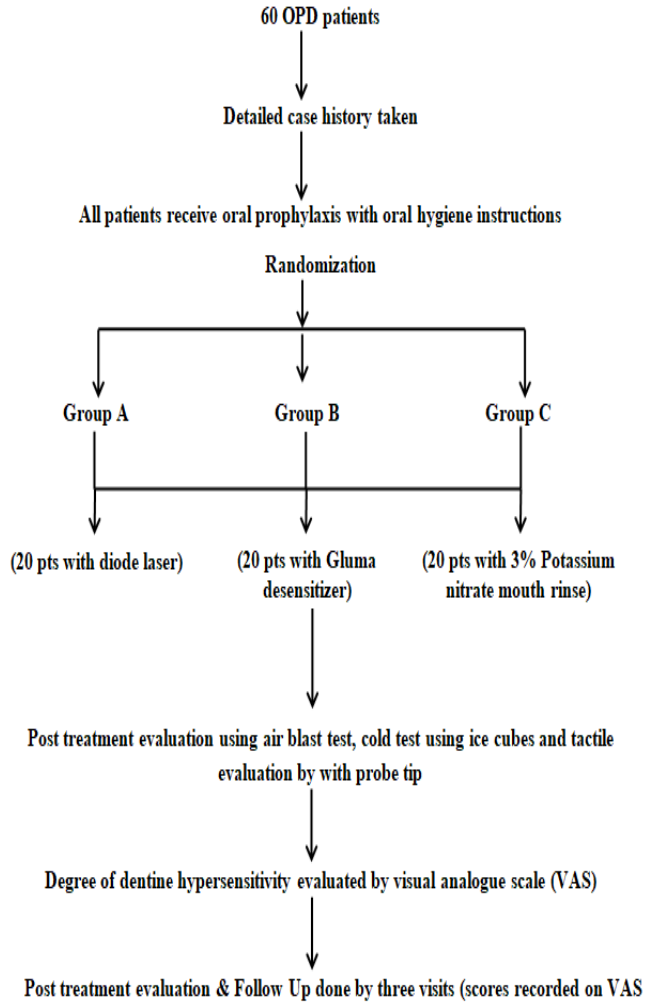
used as a simple, one-step chair-side procedure for treating and preventing dentinal hypersensitivity. Its desensitizing effect by precipitation of plasma proteins in the dentinal tubules which reduces dentinal permeability and occludes the peripheral tubules.[7] The presence of glutaraldehyde causes irreversible stiffening of collagen, inhibits dentin demineralization thus preventing caries development and imparts an antibacterial effect to Gluma Desensitizer. It has proved to be superior desensitizing agent. [8]

Potassium salts, including potassium nitrate, are the most popular substance used for the treatment of DH and act by disrupting the nerve transmission leading to sensations of pain. Potassium nitrate as an effective desensitizing agent and claims of its safety and efficacy date back to the testimonial report of Hodosh.[9] Few published studies on the use of 3% potassium fluoride and sodium fluoride mouthwash to alleviate dentinal hypersensitivity.

Taking into consideration that the application of laser and desensitizing agents is a non-invasive treatment, its potential in reducing the fluid movement through the narrowing or occlusion of tubule openings, the present study was undertaken to compare the efficacy of diode laser, Gluma desensitizer and desensitizing mouth rinse in the treatment of dentine hypersensitivity.

Material & Methods:

The patients were informed of benefits and risks of study and consent was taken before the commencement of the procedure. Patients were selected as per the inclusion criteria and complete pre-operative records were made. This study includes 60 patients. All patients were asked to assess their level of dentine hypersensitivity using the VAS scale of 0 to 10, where 0 represents “no pain” and 10 represents “greatest pain.” After initial sensitivity was assessed and recorded, treatment initiated. Dentine hypersensitivity stimulated by touching the sensitive teeth with the tip of the probe, with mesial-distal and cervical-apical directionality. In group A diode laser(980nm) used in this study. The laser operated in a continual regime, and 2 W of power applied to the tooth surface for 60 seconds of exposure, 2mm away from the laser (figure 3 and 4). Exposure time (60s) repeated after seven and fourteen days after initial exposure, only on those teeth that still sensitive. While working with the laser, both the therapist and the patient wore protective goggles, and work space appropriately designated and marked. In group B, a single application of Gluma Desensitizer was carried out and sensitivity scores were recorded by VAS at baseline,2,4 and 6 months. The little amount of Gluma Desensitizer applied to the dentin using pellets or brushes and then it was left for 30-60 seconds making sure that it contacts only the area to be treated (Figure 5). The surface was dried carefully with a stream of air until the fluid disappeared and no longer shiny then rinsed thoroughly with water. In group C subjects were instructed to brush their teeth using the toothpaste, twice daily, followed by rinsing with 1 ml of water for 1 minute and then using 15 ml (measuring cap) of the desensitizing mouth rinse (3% potassium nitrate and 0.2% sodium fluoride) for 1 minute before spitting out. Revisited all subjects returned back with bottles of mouthwash at 2 and 6 weeks, at which time replacement products provided. All clinical measurements were recorded at 2 and 6 weeks and final clinical evaluation was done after 6 months (flow chart 1). Post treatment evaluation was done using air blast test (figure 1), cold test using ice cubes and tactile evaluation was with probe tip (Figure 2) and degree of dentine hypersensitivity was evaluated by visual analogue scale (VAS) and the follow-up done by over the span of three visits.



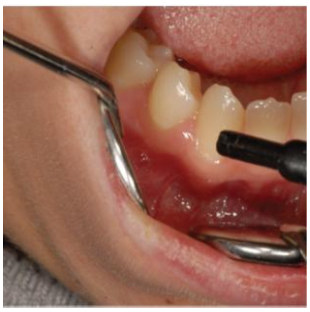


Figure 1: Cold Air Test

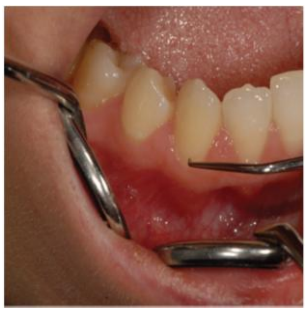


Figure 2: Probintensity Test

Group a patients

(DIODE LASER)

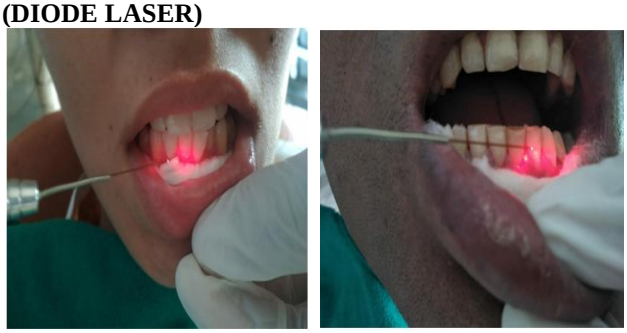


Figure 3 & 4: Application of Diode Laser

Group b patients

(GLUMA DESENSITIZER)

Figure 5: Application of Gluma Desensitizer

Results:

The study included 60 patients, with average age of 27 years. It can be seen that initially fewer sensitive teeth required fewer treatments. ANOVA test was carried out in order to assess whether this difference is significant.

There is a significant difference in tooth sensitivity values measured at baseline, in teeth that had a different number of laser treatments. Based on the obtained results, we can say (with 95%confidence) that teeth which had lower dentine sensitivity at the very beginning will require fewer laser treatments.

In order to determine between which teeth this difference is observed, given the number of treatments, a post-hoc analysis was carried out using Turkey's Honest Significant Difference (HSD) test.

Results revealed that lesions in group A(Laser) showed mean values at baseline 1.97±1.92, 1.13±1.43 at 7 days and 0.74±0.86 at 14days.P(0.00) value showed highly significant. In group B (Gluma desensitizer) showed mean values of 6.2±1.8 at baselie ,0.9±1.1 at 2 months,0.6±0.6 at 4 months and 0.8±0.7 at 6 months. P (0.01) value showed significant results. In group C (3% Potassium nitrate) showed mean values of 6.40±1.47 at baseline,4.00±1.50 at 2 weeks and 2.12±1.54 at 6 weeks. P (0.012) value showed significant (Table 1).

Percentage of reduction in mean sensitivity in cold air test 5.06 at baseline ,3.13 at 2 weeks and 1.35 at 6 weeks. Probe intensity test score were 3.03,1.98,0.99 at baseline,2 weeks and 6 weeks respectively, and overall reduction in mean sensitivity scores were 6.4,3.73,1.88 at baseline ,2 weeks and 6 weeks respectively (figure 6).

Inter group comparison after 6 months showed the results as, Group I (Laser)showed the men value of 0.74, P (0.00), highly significant results compared to group 2 (Gluma desensitizer) mean value of 0.8, P (0.01) and group 3 (3% Potassium Nitrate) mean value of 2.12, P (0.012) in decreasing the dentinal hypersensitivity (figure 7 and 8).

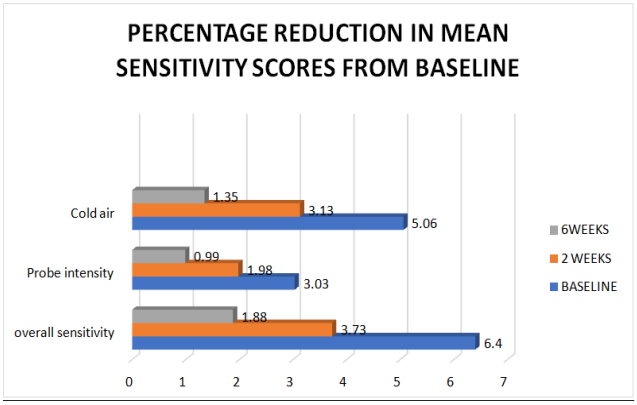


Figure 6: Percentage reduction in mean sensitivity scores from baseline

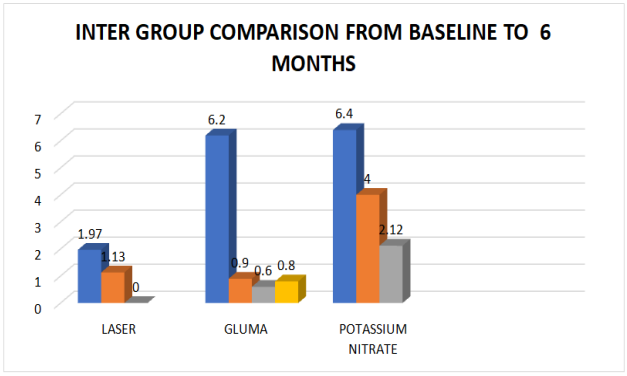


Figure 7: Inter Group Comparison after 6 Months

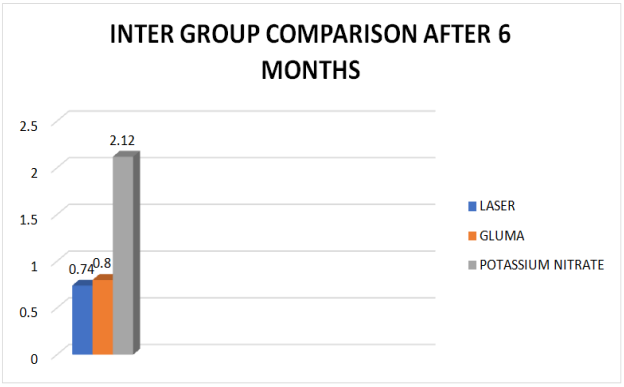


Figure 8:Inter group comparison after 6 months

Table 1: inter group comparison

	Mean +SD	P VALUE	SIGNIFICANCE
GROUP A (LASER**)**			
BASELINE	1.97±1.92		
7 DAYS	1.13±1.43	0.00	Highly significant
14 DAYS	0.74±0.86		
GROUP B(GLUMA)			
BASELINE	6.2±1.8		
2 MONTHS	0.9±1.1		Significant
4 MONTHS	0.6±0.6	0.01	
6 MONTHS	0.8±0.7		
GROUP C (POTASSIUM NITARTE)			
BASELINE	6.40±1.47		Significant
2 WEEKS	4.00±1.50	0.012	
6 WEEKS	2.12±1.54		

Significant ($p < 0.05$)

Discussion:

Chronic Dentinal hypersensitivity subjects feel an intense pain/burning when teeth come in contact with hot, cold, chilled, acidic or sweet liquid and food. Choice of the correct treatment is based on the promise of proven clinical efficacy both in terms of effectiveness and duration of desensitizing effect.

Three major mechanisms of dentinal sensitivity have been proposed in the literature:[10]

According to **direct innervation theory**, nerve endings penetrate dentine and extend to the dentin enamel junction. Direct mechanical stimulation of these nerves will initiate an action potential.[11] There are many shortcomings of this theory. There is lack of evidence that outer dentin, which is usually the most sensitive part, is innervated. Developmental studies have shown that the plexus of Rashkow and intratubular nerves do not establish themselves until the tooth has erupted; yet, newly erupted tooth is sensitive. Moreover, pain inducers such as bradykinin fail to induce pain when applied to dentine, and bathing dentine with local anesthetic solutions does not prevent pain, which does so when applied to skin.

Odontoblast receptor theory states that odontoblasts act as receptors by themselves and relay the signal to a nerve terminal.[12] But majority of studies have shown that odontoblasts are matrix forming cells and hence they are not considered to be excitable cells, and no synapses have been demonstrated between odontoblasts and nerve terminals.

Brannstrom[13] has proposed that dentinal pain is due to hydrodynamic mechanism, i.e., fluid force. Scanning electron microscopic analysis of "hypersensitive" dentin shows the presence of widely open dentinal tubules. The presence of wide tubules in hypersensitive dentin is consistent with the hydrodynamic theory. This theory is based on the presence and movement of fluid inside the dentinal tubules. This centrifugal fluid movement, in turn, activates the nerve endings at the end of dentinal tubules or at the pulp dentin complex. This is similar to the activation of nerve fibers surrounding the hair by touching or applying pressure to the hair. The response of pulpal nerves, mainly A δ intra-dentinal afferent fibers, depends upon the pressure applied, i.e., intensity of stimuli. It has been noted that stimuli which tend to move the fluid away from the pulp dentin complex produce more pain. These stimuli include cooling, drying, evaporation and application of hypertonic chemical substances. Approximately, 75% of patients with DH complain of pain with application of cold stimuli.[14] In spite of the fact that fluid movement inside the dentinal tubules produces pain, it should be noted that not all exposed dentine is sensitive. As stated before, the "hypersensitive" dentin has more widely open tubules and thin/under calcified smear layer as compared with "non-sensitive" dentine. The wider tubules increase the fluid movement and thus the pain response.

The etiology of the condition (DH/RDS) is multifactorial and not completely understood, although it has been demonstrated by several investigators that the structure of dentine in the affected (sensitive) areas of a tooth is altered, containing a larger number of patent dentine tubules with a wider tubular diameter than unaffected areas (non-sensitive) Etiological and predisposing factors associated with dentine hypersensitivity/root dentine sensitivity.[15]

Etiological and Predisposing Factors[16;] Loss of enamel, Denudation of cementum, Gingival recession, Attrition, Abrasion, Abfraction, Erosion (intrinsic and extrinsic), Tooth malposition, fenestration, absent buccal alveolar bone plate, Periodontal disease and its treatment, Periodontal surgery, Patient habits

Differential diagnosis hypersensitivity for Cracked tooth syndrome, Fractured restorations, Fractured teeth, Dental caries, Post-operative sensitivity, Acute hyperfunction of teeth, Atypical facial odontalgia, Hypoplastic enamel, Congenitally open cementum–enamel junction, Improperly insulted metallic restoration. (British Dental Journal (1985).

Guidelines on management of dentine hypersensitivity/root dentine sensitivity (table 2).

Table 2: Guidelines on management of dentine hypersensitivity/root dentine sensitivity[17]

History and examination to establish diagnosis
Identification of cause
Treatment based on severity of problem incorporation of preventive measures- remove etiological and predisposing factors (Dietary and Oral hygiene advice)
Review the patient regularly for signs of attrition, abrasion, erosion, and abfraction; give dietary advice in line with current thinking particularly in view of the potential effect of erosive materials (food and fizzy drinks) and brushing immediately after meals.
Give oral hygiene instruction and recommend an atraumatic tooth brushing technique to avoid potential damage to both hard and soft tissues.
Mild generalized sensitivity-use of OTC desensitizing products (toothpastes gels, etc.)
Localized moderate to severe sensitivity-use of In-office products (primers, varnishes, sealants, etc.)
Avoid placing subgingival restorations that may retain plaque
Avoid violating the biological width when placing crown margins
Use of periodontal flap surgery (including GTR) in the treatment of exposed root dentin
In severe cases, pulpal extirpation and extraction may be the treatment of choice
Review on an appropriate basic and reassess if pain persists

In the present in vivo study divided into three groups *Group 1* (Laser); *Group 2* (Gluma Desensitizer); *Group 3* (Desensitizing mouth rinse) and compared the efficacy of each agent used in the study. Some of the patients reported pain so severe that it has become a physical and emotional problem that affects their quality of life. Many of them were not able to consume hot or cold foods or liquids, acidic foods or liquids, and even had difficulty with brushing teeth. *Matsumoto et al* showed an 85% improvement in teeth treated with laser; *Aun et al* reported success in laser-irradiated teeth in 98% of their cases; *Yamaguchi et al* noticed improvement index of 60% in the group treated with laser compared to the 22.2% of the control non laser group. *Kumazaki et al* showed an improvement of 69.2% in the group treated with laser compared to 20% in the placebo group. *Gerschman et al*, in a double-blind study, found significant values in the laser-treated group. In fact, sensitivity to thermal stimuli was reduced by 67%, whereas the placebo group had a reduction of 17%, sensitivity to tactile stimuli was reduced by 65%, while the placebo group showed a reduction of 21%. *Brugnera et al* showed the immediate analgesic effect using a diode laser. These results support the results of our study.

A number of studies have reported the efficacy of potassium nitrate for managing dentinal hyper-sensitivity. While the Hodosh[18] study was the first to report that potassium nitrate was a "superior desensitizer" this study was not well controlled and it was not until the studies of Tarbet.[19]Potassium salts act by diffusion along the dentinal tubules and decreasing the excitability of the intradental nerve fibers by blocking the axonic action. According to the FDA, for a potassium nitrate toothpaste to claim to be desensitizing it must contain 5% of the ingredient. Potassium nitrate penetrates the enamel and dentin to travel to the pulp and creates a calming effect on the nerve.[19] This effect can be thought of as "anesthetic-like". Previously it was believed that NO_3 is the active agent is potassium nitrate, however showed that NO_3- anion is not effective as desensitizing agent and K+ is an effective desensitizing agent regardless of the anion with which it is combined and further K+ had a reversible effect, that is, they did not appear to damage the dentinal sensory apparatus.This demonstration of 3% potassium nitrate and 0.2% sodium fluoride as effective desensitizing agents in a mouthwash formulation correlates with the findings of *Gillam et al.* Our findings of reduced symptoms were similar to studies of *Pearce et al, Chesters et al.,* and *West et al.*

Gluma Desensitizer shown promising results in clinical research. This desensitizing agent contains hydroxyethyl methacrylate (HEMA), benzalkonium chloride, glutaraldehyde, and fluoride in its formula. Glutaraldehyde causes coagulation of proteins inside the dentinal tubules, reacting with the albumin in dentinal fluid, thus causing the precipitation of albumin. As a result of this reaction, HEMA polymerization occurs, which is able to form deep tags, so that there is partial or complete obliteration of the dentinal tubules and consequent reduction in dentin hypersensitivity. Our findings of reduced symptoms similar to *Duran, Sengun (2004)* compared the effectiveness of five desensitizer products, including the Gluma Desensitizer, in a split mouth design. The VAS scores at post-treatment evaluation points were significantly decreased compared with baseline data ($p < 0.05$), but they did not use a placebo control. *Dondi dall'Orologio et al (1999)* found Gluma Desensitizer to be successful as well. Within the scope of the conducted study, laser therapy has provided effective results in the treatment of dentine hypersensitivity.

Conclusion:

All the experimental agents– diode laser, Gluma desensitizer and desensitizing mouth rinse (potassium nitrate) were effective in decreasing dentinal hypersensitivity. The percentage (%) of decrease was found to be highest diode laser as compared to the other groups over a period of 6 months. The decrease in the dentinal

hypersensitivity by the Gluma desensitizer and desensitizing mouth rinse (potassium nitrate) showed comparable results over a period of 6 months.

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