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THE EFFECT OF A RESIN-BASED DESENSITIZER CONTAINING GLUTARALDEHYDE ON CERVICAL DENTIN HYPERSENSITIVITY: A RANDOMIZED CONTROLLED CLINICAL TRIAL

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ABSTRACT

INTRODUCTION: Non-carious enamel loss is becoming more prevalent due to modern habits which lead to increased levels of dentinal hypersensitivity (DH). DH manifests through dental abrasion, erosion, etc. When dentin is exposed, external stimuli can cause excessive pulpal pain response. Dental adhesives/restorations and desensitizers have been developed for obliteration of dentinal tubules (DTs) and treatment of DH.

OBJECTIVES: To clinically evaluate the effectiveness of a glutaraldehyde-based desensitizer (Gluma™ Desensitizer Heraus-Kulzer, Hanau, Germany) vs conventional universal bonding agent (Scotchbond™ Universal Adhesive) in minimizing cervical dentin hypersensitivity (CDH) throughout a 6-months follow-up period.

MATERIAL AND METHODS: 14 patients having a minimum of two contralateral teeth with CDH were allocated for a split-mouth, double-blind, randomized controlled trial. Each Patient received Gluma™ Desensitizer on one side vs Scotchbond™ Universal Adhesive on the other side. A total of three desensitization sessions were performed at 5 days interval. For assessment of hypersensitivity levels, air-blast and tactile Visual Analog Scale (VAS) sensitivity scores were evaluated at baseline (T0), immediately after each desensitizing session (T1,T2,T3), and at the 1st (T4), 3rd (T5), 6th (T6) months of follow-up.

RESULTS: Both agents reduced CDH significantly over the course of the study. At T6, mean air-blast sensitivity VAS scores demonstrated statistically nonsignificant difference between groups (p=0.493). Probe sensitivity VAS scores recorded significant statistical difference between groups.

CONCLUSIONS: Gluma desensitizer and Scotchbond Universal Adhesive resulted equally in a reduction of pain intensity for patients with DH. No advantage was detected for the use of one material over the other.

KEYWORDS: Hypersensitivity, glutaraldehyde, desensitizer, bonding agent.

RUNNING TITLE: Glutaraldehyde-based desensitizer clinical effect on cervical dentin hypersensitivity.

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INTRODUCTION

DH is a frequent dental condition characterized by pain emerging from exposed dentin. It affects between 1.3–92.1% of worldwide population, with an average prevalence of 33.5%, which underlines the wide heterogeneity of the studies in sample size selection, age group, risk factor exposure, as well as regional, cultural, and social context (1, 2).

In the majority of research age was considered to be a risk component, and since the population has become proportionally older, cervical defects have become more prevalent as a result of aging, gingival recession and tooth wear (3). Yet, the ratio of erosions with exposed dentin appears to increase in younger adults, frequently leading to DH. Also the incidence of DH was observed to be slightly higher in females than in males, which may represent various dietary and oral hygiene habits (4).

With the advances of dental science and promotion of oral hygiene, the prevalence of DH has been on the rise where a higher number of adults retain their teeth, causing an increase in the frequency of denuding root surfaces following dental recession and periodontal surgery (5). Canines, premolars, and incisors are more frequently affected than molars by DH in the buccal cervical region of permanent teeth, also maxillary teeth showed a higher extent of affection than mandibular teeth (4).

Despite the large number of research that has been conducted to explain the impact of various risk

factors on the development of non-carious cervical lesions (NCCLs), this objective has not yet been attained. A causal link between NCCLs and the alleged risk factors is not supported by compelling scientific data (2).

Teeth with exposed dentin or receded gingiva are highly subjected to DH. Physiological and mental changes are both involved in the complicated phenomenon of CDH. It manifests as sudden, sharp, localized pain of varying severity that is brought on by any irritant, including chemical (sweets/acids), thermal (hot/cold), bacterial or tactile stimulation, and can't be attributed to any other dental illness (6).

Studies suggest that microscopic changes occur in the structure of sensitive dentin compared with normal dentin. The diagnosis of DH requires meticulous clinical examination, history taking and eliciting response using different stimuli (4).

The etiopathogenesis of CDH remains unclear and different theories have been proposed to explain the condition; however, the hydrodynamic hypothesis, which is based on the hydraulic conductivity of dentin, has been the most accepted and regarded one (Brannstrom and Astrom, 1972) (7). This theory proposes that gingival recession and/or loss of cementum/enamel, presumably from poor/improper dental hygiene techniques, erosion, abrasion, attrition, abfraction or a combination with other factors, may result in dentin exposure to the environment (8). During this process, the movement of fluids inside DTs in reaction to external stimuli innerves the dentino-pulpal unit at the odontoblastic cell layer in response to pressure changes inside the pulp resulting in pain (8).

The vast array of methods and therapeutic options available for the palliation of CDH reflect the difficulty of treating this condition. A number of techniques have been suggested to block dentinal tubule (DT) openings on the basis of the hydrodynamic hypothesis, including the use of various dentin adhesives, fluoride applications, laser therapy, and restorations made of glass-ionomer and composite resins (9).

Recently new products have been recommended for their desensitizing effect. The results of several topical desensitizing medications, however, have reportedly been transitory because they do not attach to the dentin surface (10). Studies have revealed that although the first therapy of choice is to apply desensitizer solutions and dentifrices containing ferric aluminium and potassium oxalates, their effects are transient.

The persistence of these topical therapies is influenced by a number of factors, most typically the desensitizer's dissolution by saliva and oral secretions (10). Therefore, experimental alternatives to treatment are being considered. In the event of recurrences, several treatments, including adhesives, varnishes, and bonding agents, can be used or reapplied. Composite and resin-based restorations are occasionally favored.

Thus, the purpose of this study is to evaluate the influence of desensitizing agents in reducing CDH. The null hypothesis to be tested is that there is no statistically significant difference in the desensitizing effect of different desensitizing materials for managing DH.

MATERIAL AND METHODS

Trial design

A single-center split-mouth randomized controlled clinical trial that was statistician, assessor, and patient blinded. A 1:1 allocation ratio was used during the randomization process. The guidelines of the Consolidated Standards of Reporting Trials (CONSORT) were followed throughout this trial (**Fig. 1**) (11).

Ethical issue

At the Faculty of Dentistry, Alexandria University (IRB NO 00 105 56 - IORG 00 88 39), the Institutional Review Board provided their ethical approval on the clinical trial (**Ethical Approval Number: 0244 - 19/05/2021**). All subjects or their legal guardians provided their informed consent. All methods were carried out in accordance with the relevant guidelines and regulations.

Patient screening

From the outpatient clinics of the faculty of dentistry of Alexandria University, 30 patients were assessed for eligibility from which 14 individuals (with an average age of 33.01 years 1.3) were selected. A minimum allocation of two contralateral teeth per patient was conducted, and an average was determined for each patient during evaluation (**212 teeth in total**). Participants eligible had good oral hygiene and at least 2 contralateral upper and/or lower teeth with sound exposed cervical dentin on the facial surface that is hypersensitive to timed application of compressed-air or cold-water in the anterior/premolar region. Exclusion criteria was: teeth with cavities, restorations, irreversible pulpitis or necrosis, recent use of professional or over-the-counter desensitizing treatments within the last six weeks, enamel defects (such as hypoplasia, amelogenesis imperfecta, etc.), pregnancy or nursing, and prolonged use of analgesic, anti-inflammatory, and psychoactive medications (12).

Interventions

Seven visits were planned altogether. Initial recruiting began in August 2021, and the trial ran through March 2022 (6 month follow-up). Individual "patient diagnostic charts" were used to identify the dietary and oral hygiene practices that were connected with erosion and abrasion as well as other causative and predisposing factors.

Patients were informed about the trial prior to treatment, and their informed consent was acquired following a comprehensive description and assurance of the safety and possible benefits of such intervention (13).

After initial evaluation, the teeth were thoroughly washed and polished with fluoride-free slurry

(pumice), rinsed, and dried for subjective and objective recording of DH.

The VAS scores were then recorded according to degree of hypersensitivity. Using a split-mouth design, subjects were randomly assigned into 2 groups (n=14), with labial cervical aspect of each tooth in both jaws—from the central incisor to second premolars on either side—being taken into consideration.

Sensitivity assessment

At baseline (T0), subjects meeting the eligibility criteria had a specific clinical evaluation linked to DH (**Fig. 1**). Cotton rolls were used to separate the test teeth from neighboring teeth. A stream of air was delivered from a standard dental unit air-syringe (14) at 45 to 60 psi placed 2 mm distant from and perpendicular to the affected buccal aspect of the tooth for three seconds (15). A dental explorer was also used to provide tactile stimulation by scratching the exposed dentin in a mesio-distal orientation (14).

The respondents' level of pain was measured using a VAS, labelled from 0 (absence of pain) to 10 (severe, intolerable pain) (**Fig. 2**) (13). Each group was assessed before application (T0), directly after each desensitizing session (T1, T2, and T3), and one, three, and six months later (T4, T5, and T6).

After confirming the diagnosis of CDH, it was necessary to remove risk factors, such as limiting acid intake, altering dietary patterns, improving dental hygiene practices, etc (16). Therefore all patients were given oral hygiene instructions and informed not to use any desensitizing toothpastes (12, 13). Next, rubber dam isolation was applied for the affected teeth and dentin surfaces were washed with water spray for 5 seconds before being dried with cotton pellets. (Fig. 3: A, Fig. 4: A)

Application of the test materials to Dentin

Each Patient randomly had each contralateral teeth desensitized in a split-mouth design as follows (17): For Group I The affected cervical dentinal surface was treated with a little amount of **Gluma desensitizer** using a micro-brush, and the treatment was kept on for **30 seconds** (**Fig. 3: B).** A stream of compressed-air was then used to dry the area until the fluid layer had vanished and the surface was no longer glossy (**Fig. 3: C**). The application of the agent was repeated for three times in each session. The substance was reapplied at five days interval with a total number of 3 applications (T0, T1, T2). For group II (control) the affected cervical aspect received **3M™ Scotchbond™ Universal Adhesive** which was **rubbed** in for **20 seconds** (**Fig. 4: B**). The adhesive was gently air-**dried** for approximately 5 seconds to evaporate the solvent (**Fig. 4: C**) then light-cured for 10 seconds (**Fig. 4: D**). The process was repeated for three times in each session. The agent was reapplied at five days interval as for the other group. The patients did not know which type of treatment corresponded to each tooth.

Study Parameters

Outcome measures

The patients were evaluated and the outcomes of the pain and sensitivity changes after being exposed to airblast and probe stimuli were recorded. In order to estimate the magnitude of the pain, individuals had to rate their level of discomfort along a VAS-continuum. Another skilled dentist with competence in conservative and preventive dentistry evaluated and gathered the outcome measurements at baseline (T0), immediately after each desensitizing session (T1, T2, T3), and at the 1st (T4), 3rd (T5), 6th (T6) months of follow-up.

Sample size computation

5% alpha error and 80% research power were used to estimate the sample size. The mean pain score after 6 months for the dentine bonding agent group was 2.54 (5) compared to 1.545 (17) in the Gluma agent group. Based on comparison of two dependent means and pooled SD= 1.155 (17), sample size was calculated to be 13 sides per group, increased to 14 sides to make up for loss to follow-up. Total Sample = number per group x number of groups = 14 x 2 = 28 sides. Due to the split-mouth design, 14 patients were required.

Randomization, blinding and allocation concealment

A split-mouth approach with a 1:1 allocation ratio was used to randomly place each contralateral tooth of each patient who met the eligibility requirements in one of the two groups, which are the test group I (**Gluma™** desensitizer) and control group II (**Scotchbond™ Universal Adhesive**). Randomization and blinding were carried out using the sealed envelope method. The random numbers were taken from a computer-generated table. Cards providing information about each group were made and placed in sealed, opaque envelopes by a neutral party who was not participating in the study. After the patient was checked for meeting the inclusion criteria and the main investigator had recorded the baseline assessments, this impartial person opened the envelope and revealed the allocation assignment. One qualified examiner performed all clinical procedures. The statistician, patients, and data assessor were not informed of the treatment allocation.

Analytical statistics

Shapiro-Wilk test, box plots, and descriptives were used to evaluate normality. The means, standard deviations, medians, and minimum and maximum values were used to illustrate the variables. According to the study's split-mouth design, a Wilcoxon Sign Rank test was used to compare the two groups. The Friedman test was employed to determine changes in sensitivity scores across the time intervals. P value of 0.05 was chosen as the significance level. Every test had a two-tailed distribution. SPSS for Windows, version 23, was used to analyze the data. The intention to treat analysis was used to examine all groups.

The intra-class correlation (ICC) test results for Examiner 1 ranged from 0.995 to 0.996, indicating excellent reliability. Based on a single measurement, absolute-agreement, and 2-way mixed-effects model, ICC estimates and their 95% confidence intervals were computed using IBM SPSS statistics version 28.0.0 (SPSS Inc, Chicago, IL).

RESULTS

Patients flow

The study included 14 patients of both genders (with a mean age of 33.01 years ± 1.3). A split-mouth design was used for the study and a total of 212 teeth (106 in each treatment agent group) were treated with Gluma desensitizer or Universal Scotchbond. About 6 to 20 teeth per patient were treated. In total 13 participants (with an average age of 32.8 years ± 1.3) having 192 teeth (96 in each group) were available at the end of the trial. Of the included subjects, only one person dropped out and 2 missed 2 appointments at (T5, T6). Patients were enrolled between **August 1st** and **September 20th, 2021**. The trail commenced on **October 3rd, 2021**, and ended on **March 27th, 2022**, with the final follow-up appointment. No significant adverse events were reported throughout the trial.

Sample description

The mean age of the study sample was 33.01 and standard deviation (SD) was 1.3. Gender, age, and a number of other standard characteristics of the sample were comparable.

Primary outcome

VAS – airblast sensitivity test

At baseline, pain was comparable in both groups with no significant difference ($p > 0.05$). The finding of Friedman test demonstrated that both treatments reduced pain intensity at each stage of the trial, shown in **Table 1, Fig. 5.** The mean VAS scores from baseline through the review periods showed a significant decrease in pain intensity for both agents over a 6 months follow-up period.

The mean VAS-measured pain reduction for airblast sensitivity was not statistically significant at different stages between both groups except at two time points: the tenth day and the sixth month, where **Scotchbond** group showed significant greater pain reduction compared to Gluma group. **Gluma** showed greater mean VAS nonsignificant pain reduction only on the first day **(Table 1, 2)** compared to Scotchbond. For **Gluma** group, mean VAS rating decreased from 5.0 ± 2.3 at baseline to 2.7 ± 2.0 at first day **(Table 1)** with percent reduction of **-42.83%** (34.42) as shown in **Table 2,** while for **Scotchbond** the rating **decreased** from 5.1 ± 2.3 at baseline to 3.2 ± 2.2 at first day with percent reduction of **-34.57%** (35.35). However at the remaining time points, **Scotchbond** group showed greater pain reduction than did **Gluma** group (**Table 1**).

The mean VAS scores from baseline through the review periods showed a significant decrease in pain intensity for both agents over a 6 months follow-up period. For **Gluma** group, mean VAS rating fell from 5.0 ± 2.3 at baseline to 1.1 ± 1.3 at T6 with a total reduction of **-78.80%** (24.34). Also **Scotchbond** group successfully reduced mean pain scores from 5.1 ± 2.3 at baseline to 0.9 ± 1.1 at T6 with a total reduction of **84.75%** (17.87). The difference between the two groups had a p value of 0.074 which was not statistically significant.

Secondary outcome

VAS – tactile stimuli

At baseline, there was no discernible difference between the groups in terms of pain. The findings in Friedman tests demonstrated that both treatments reduced pain intensity at each stage during the trial but with a period of stagnation between T3- T6 as shown in **Table 3, Fig. 6.**

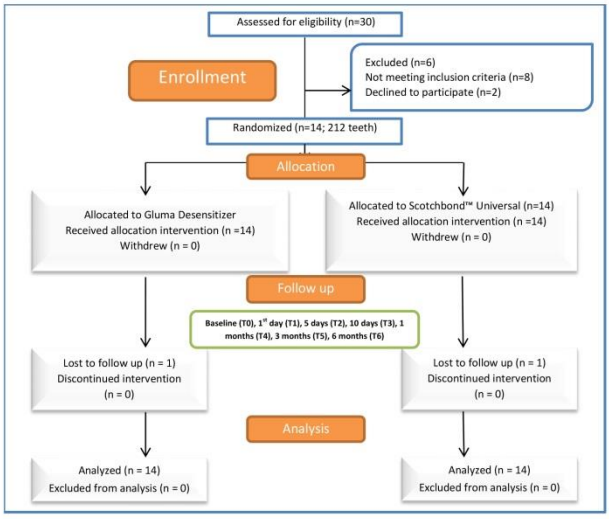

Figure (1): CONSORT flow diagram showing patient flow during the trial.

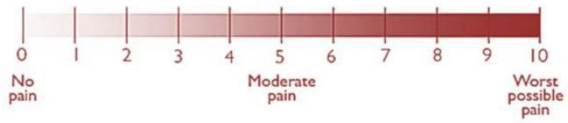

Figure (2): VAS for determining the level of DH.

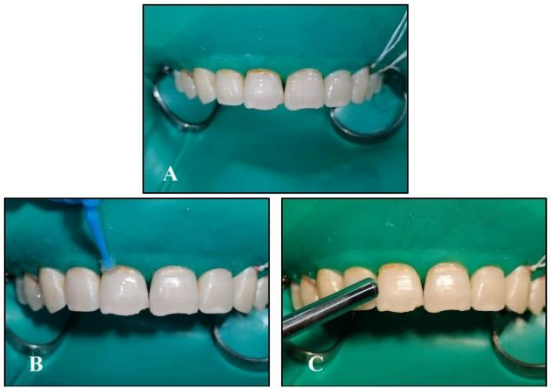

Figure (3): Teeth with CDH isolated with rubber dam (A), and Gluma desensitizer was applied and air-dried till set (B, C)

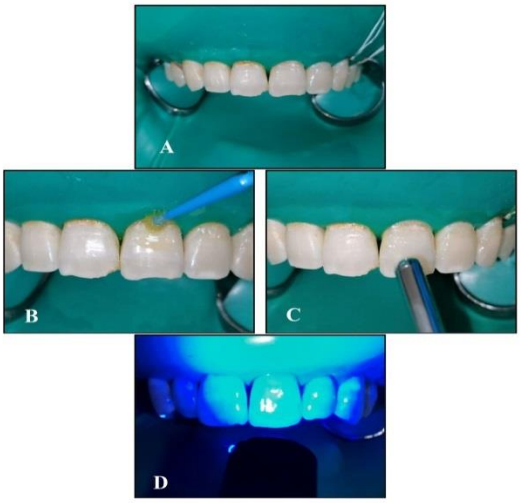

Figure (4): Teeth with CDH isolated with rubber dam (A), and Scotchbond was applied, air-dried and light-cured (B, C, D)

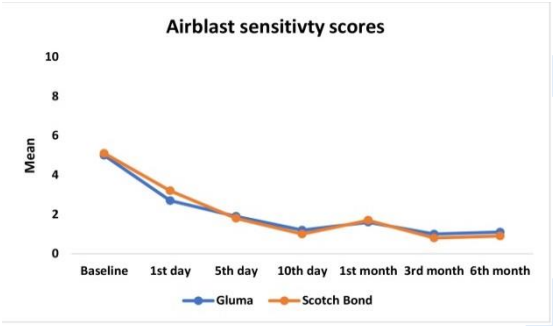

Figure (5): Mean VAS sensitivity scores at different time points in both groups

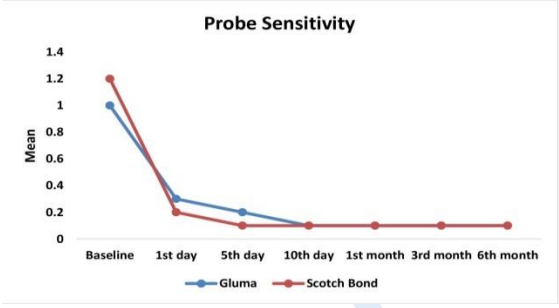

Figure (6): Mean VAS probe sensitivity scores at different time points in both groups

Mean VAS-measured decrease in pain for probe sensitivity was not statistically significant at different stages between both groups except at the first day (T1-T0) and after 6 months (T6-T0), as shown in **Table 3,** where **Gluma** group mean VAS rating fell from 1.0 ± 2.3 at baseline to 0.3 ± 0.05 at first day with percent reduction of **21.07%** (32.58)**,** and **Scotchbond** score was reduced from 1.2 ± 2.5 at baseline to 0.2 ± 0.4 at first day with percent reduction of **30.34% (43.84),** while at **T6-T0 Gluma** group reduced pain with a total of **19.71%** (50.37)% and **Scotchbond** reduced pain with a total of **39.17%** (48.38)%. However at the remaining time points, Scotchbond group showed greater pain reduction than did Gluma group with non-significant difference. Total pain reduction percentage difference between the two groups at the end of the trail was significant (P = 0.007) as shown in **Table 3.**

Table (1): Comparison between Gluma and Scotchbond regarding sensitivity (Air blast)

	Gluma		Scotch Bond		P value
	Mean (SD)	Median (Min -Max)	Mean (SD)	Median (Min -Max)	
Baseline (T0)	**5.0 (2.3)**	**5.0 (1.5 – 8.8)**	**5.1 (2.3)**	**5.1 (1.0 – 10.0)**	0.932
1st day (T1)	**2.7 (2.0)**	**2.0 (0.0 – 7.2)**	**3.2 (2.2)**	**2.7 (0.0 – 8.0)**	0.338
5th day (T2)	**1.9 (2.0)**	**1.6 (0.0 – 8.0)**	**1.8 (2.1)**	**1.3 (0.0 – 8.3)**	0.754
10th day (T3)	**1.2 (1.8)**	**0.7 (0.0 – 8.0)**	**1.0 (1.8)**	**0.4 (0.0 – 8.0)**	**0.045***
1st month (T4)	**1.6 (2.2)**	**0.9 (0.0 – 7.5)**	**1.7 (2.9)**	**0.5 (0.0 – 9.4)**	0.972
3rd month (T5)	**1.0 (1.2)**	**0.6 (0.0 – 4.8)**	**0.8 (1.0)**	**0.5 (0.0 – 3.6)**	0.122
6th month (T6)	**1.1 (1.3)**	**0.6 (0.0 – 4.8)**	**0.9 (1.1)**	**0.3 (0.0 – 3.6)**	**0.037***
P value	**<0.0001***		**<0.0001***		

*Statistically significant at p value ≤ 0.05

Table (2): Comparison beween the Gluma and Scotch bond regarding percent change in VAS score indicating sensitivity (Air blast)

	Gluma		Scotch Bond		P value
	Mean (SD)	Median (Min - Max)	Mean (SD)	Median (Min - Max)	
T1 – T0	-42.83 (34.42)	-40.49 (-100.0 – 33.33)	-34.57 (35.35)	-40.00 (-93.33 – 40.00)	0.545
T2 – T1	-25.86 (64.43)	-19.82 (-100.0 – 100.0)	-38.72 (63.68)	-44.64 (-100.0 – 200.0)	0.162
T3 – T2	-26.85 (48.07)	-31.32 (-100.0 – 100.0)	-37.29 (44.61)	-50.00 (-100.0 – 100.0)	0.089
T4 – T3	30.60 (62.50)	0.00 (-75.00 - 250.0)	49.59 (87.62)	0.00 (-83.33 – 262.50)	0.138
T5 – T4	-16.33 (35.94)	0.00 (-100.0 - 50.0)	22.99 (176.46)	0.00 (-94.83 – 900.00)	0.508
T6 – T5	0.89 (41.10)	0.00 (-100.0 – -14.29)	-4.15 (32.10)	0.00 (-100.0 – 66.67)	0.528
T6 – T0	-78.80 (24.34)	-85.83 (-100.0– -14.29)	-84.75 (17.87)	-92.26 (-100.0 – -40.00)	0.074

*Statistically significant at p value≤0.05

Table (3): Comparison between the Gluma and Scotch bond regarding percent change in VAS score indicating sensitivity (probe)

	Gluma		Scotch Bond		P value
	Mean (SD)	Median (Min -Max)	Mean (SD)	Median (Min -Max)	
T1 – T0	-21.07 (32.58)	0.00 (-100.0 – 0.00)	-30.34 (43.84)	0.00 (-100.0 – 100.0)	**0.008***
T2 – T1	-2.38 (62.15)	0.00 (-100.0 – 200.0)	-7.65 (37.79)	0.00 (-100.0 – 100.0)	0.417
T3 – T2	0.00 (45.13)	0.00 (-100.0 – 100.0)	-0.60 (36.99)	0.00 (-100.0 – 100.0)	0.862
T4 – T3	-5.36 (39.30)	0.00 (-100.0 – 100.0)	-7.14 (26.23)	0.00 (-100.0 – 0.00)	0.888
T5 – T4	2.38 (33.86)	0.00 (-100.0 – 100.0)	0.00 (0.00)	0.00 (0.00)	0.705
T6 – T5	-1.79 (34.65)	0.00 (-100.0 – 100.0)	0.00 (0.00)	0.00 (0.00)	0.705
T6 – T0	-19.71 (50.37)	0.00 (-100.0 – 100.0)	-39.17 (48.38)	0.00 (-100.0 – 0.00)	**0.007***

*Statistically significant at p value≤0.05

DISCUSSION

Cervical dentin hypersensitivity, a widespread condition that affects people all over the world, is increasing in prevalence and progression over various age groups. This might be a result of an increase in the community's preservation of their teeth with the recently available preventive measures and a parallel increase in life-expectancy. Since CDH is a subjective condition, its treatment can be challenging (18). In different studies, as in this one, a pain rating measure, such as the VAS, has commonly been used to assess CDH (18, 19). Topical desensitizing products, which can be used by oneself or a professional, are currently a readily available noninvasive therapeutic option (18). The mode of action of these treatments depends on physical agents for sealing DTs and blocking fluid flow or altering neural pain response, recently by using lasers.

The most acknowledged hydrodynamic theory states that the pulp tissue is irritated by the fluid's fast flow in the DT. Pulpal fibers are activated by any stimulation that creates fluid turbulence in the DT, which illustrates the reason for an only painful response towards a mechanical, chemical or thermal input (20). Scanning electron microscopic (SEM) analysis demonstrated that in case of hypersensitive dentin DTs are wide open with a count of eight times higher in contrast to normal dentin (21). Moreover, DT diameter in sensitive dentin was found to be twice as large as that of non-sensitive dentin (20).

The efficacy of desensitizing medications, specifically Gluma, in alleviating DH has been demonstrated in numerous investigations. **Gluma®** (Heraeus Kulzer GmbH, Hanau, Germany), which is a commercially available desensitizing solution, not only successfully lowers hypersensitivity of dentin by limiting its permeability along with blocking peripheral DTs, but also has an **antimicrobial influence** (18).

Gluma®, as demonstrated in the **components table** below, comprises **Glutaraldehyde**, which acts as a biologic fixative that coagulates proteins and amino acids available in DTs forming transverse septa in its lumen, and **Hydroxyethyl methacrylate (HEMA)**, which, due to its hydrophilic nature, acts as a carrier for glutaraldehyde allowing in-depth action. Also, by widening the demineralized collagen in hypersensitive dentin, HEMA forms the hybrid layer which promotes dentin attachment and prevents phase separation (22). On polymerization, HEMA stops the intra-tubular fluid flow, which provides the immediate relieving effect. While the clogging effect of HEMA alone is transient and the DTs become unconcealed eventually (20), the complex of components together forms an impermeable dentin surface by eliminating the hydrodynamic effect thus desensitizing teeth (23). However, The HEMA hydrophilic nature may disrupt the Gluma-tooth interface by imbibing water oozing from the opened dentin orifices prior to the resin polymerization or by allowing water absorption after polymerization.

Table: Composition of materials used in the study.

Material	Composition	Manufacturer
Gluma Desensitizer	5% glutaraldehyde and 35% hydroxyethyl methacrylate (HEMA)	Heraeus Kulzer, Hanau, Germany
Scotchbond™ Universal Adhesive	10-MDP, phosphate monomer, dimethacrylate resins, HEMA, methacrylate-modified polyalkenoic acid copolymer, filler, ethanol, water, initiators, silane	3M ESPE, Minnesota, united states

For assessing the hypotheses of absence of difference between management techniques, the current randomized clinical trial double-blind, and split-mouth design serves as the gold standard. In this study, Gluma® desensitizer was compared with Scotchbond™, a universal adhesive, for alleviating CDH instantly after each desensitizing session (T1,T2,T3), and at the first (T4), third (T5), and sixth (T6) months of follow-up. Mean VAS scores measured demonstrated that both groups, Gluma and Scotchbond, showed a significant cervical dentin hypersensitivity reduction in the two tests, air-blast sensitivity test and tactile test.

The findings of the present investigation demonstrated that **Gluma®** was successful in lowering CDH by occluding DTs. Dayton et al. (24), and Pereira et al. (25) substantiated the mode of action of **Gluma®** by SEM examination, which revealed that the active components in Gluma® successfully obstructed the DT.

Adhesives, varnishes and resins have long been used for alleviation of DH and were reported by many studies to be effective (26-28). The **single-bottle** self-etching adhesives have been recommended for relieving pain of DH since they create an acid-resistant hybrid layer. As demonstrated in the **components table** above, **one-bottle self-etching adhesive** implements **Ethanol** constituent, a water chaser and solvent for monomers, which provides a sound sealing with better infiltration of the adhesive (29), and **Phosphorylated monomers** in an aqueous solution, which dispenses acidity and promotes the adhesive's attachment to dentin. Such specific chemistry rehydrates the collagen network thus forming a distinct hybrid layer. This yields a durable bond that seals DTs if applied in the self-etch and prevents open tubules and potential sensitivity, therefore reducing pain for patients that are already symptomatic (30).

In our current study, mean VAS scores of air-blast sensitivity tests demonstrated that **Gluma** had a non-significantly higher effect on CDH minimization than did Scotchbond on the **first** day post application, while the accumulated effect of **Scotchbond** universal adhesive over T6-T0 period in **air-blast sensitivity** test was higher for Scotchbond group with **84.75%** pain reduction than for Gluma group with **78.80%** pain reduction with

non-significant difference between the two groups (p value of 0.074). However, for **probe sensitivity test** total pain reduction percentage at the end of the trail was significantly greater (P = 0.007) in Scotchbond group with a total of **39.17%** sensitivity reduction in contrast with Gluma group with a total of **19.71%** sensitivity reduction.

Similarly, An in-vivo double-blind, randomized, parallel, prospective study was designed by Patil et al in **2015** to assess and compare between the clinical efficacy of Single-Bond Universal, which is a one-bottle self-etching adhesive, Gluma® Desensitizer and a combination of Gluma desensitizer + Gluma Comfort Bond adhesive for an in-office management of DH (30). With a single application, all three agents were found to **effectively reduce DH** for up to six weeks. However opposite to our study, Gluma Desensitizer and the combination of Gluma desensitizer + Gluma Comfort Bond adhesive showed greater pain reduction than did Single-Bond universal (30).

Authors interpreted that treating DH with **resins**, **dentin bonding agents** or both created resin tags, by which exposed DTs are physically sealed. On the other hand, Gluma desensitizer, containing both HEMA, a hydrophilic monomer having the potential of penetrating acid etched dentin physically blocking DTs, and Glutaraldehyde, which forms a physiological seal, together successfully occlude DTs at depths of **50-200 µm** (9, 22, 31). Therefore, the one-bottle self-etching adhesive giving less pain reduction compared to the other agents can be due to the presence of hydrophilic and less hydrophobic monomers which might permit water after application causing a limited penetration into DTs (30).

In 2017, Idon et al. conducted a randomized clinical trial to determine the efficacy of **Gluma** in comparison with Copal F, and Pro-Relief in treating DH. Gluma exhibited a more dramatic decline in DH at ten minutes after application and **one-month** after therapy than did the other treatments. Authors deduced that **Gluma** was the optimum in-office treatment for DH (32).

Also in the same year, Hajizadeh et al. evaluated the effectiveness of Gluma desensitizer against a single-bottle bonding agent, and Clearfil S3 Bond in lowering DH. After a periodontal procedure, the teeth were treated with one of these agents and the differences were assessed in contrasted with a water control group (placebo). **Resembling the findings of** our study, CDH was significantly reduced in all groups at starting point; however, at the 1st and 4th weeks of follow-up, the one-bottle self-etching bond failed against Gluma which rendered a significant lowering in CDH by one month of follow-up (33).

For further substantiation of bonding agent efficacy in DH treatment, in 2019, Ghosh et al tested the **effectiveness** of non-carious CDH treatments by fluoride varnish, a **bonding agent** and Er, Cr: YSGG laser in a randomized clinical trial. They found that all treatments were effective in reducing pain after 4 weeks, and hypersensitivity recurrences did not return to pretreatment levels and were more in those who exhibited high pretreatment sensitivity. They suggested that **bonding agent** desensitization effect occurred due to the **high wettability** of the material to the tooth structure which allows good penetration into DTs thus sealing them after curing (34).

Moreover, in the same year, a study conducted by Askari and Yazdani assessed the efficacy of propolis extract desensitizing agents in 2 concentrations in contrast with **Single-Bond Universal** along with water as a control (placebo). Using VAS, the severity of DH was assessed based on the patients' responses to air-blast and tactile stimuli, and the intensity of pain was measured prior to treatment as well as at the 1st day, one week, two weeks, 30 days, and 90 days following therapy. The outcomes showed that all treatments were significantly effective in decreasing DH at 60 and 90 days, yet the **adhesive** displayed total effectiveness at all times. All of the therapies alleviated DH more than the control group. Authors explained that Single-Bond Universal caused rapid alleviation of DH in comparison with the other 2 tested materials. Therefore, **Dentin bonding agent** may be a preferable option when **immediate** results are required. They inferred that dentin adhesive proved successful in prolonged alleviation of hypersensitive dentin, and employing **Single-Bond Universal** promotes DH quick resolution (35).

Meanwhile, Kannan and Gowri tested **Gluma** desensitizer against Duraphat desensitizer on subjects experiencing DH. VAS was used to quantify DH levels after five minutes as well as after seven days of follow-up, where no significant differences between the outcomes of the two groups were discovered. Nonetheless, in contrast to Duraphat, Gluma demonstrated a considerable decline in VAS ratings at one week. They determined that both desensitizers were successful in minimizing DH, with **Gluma** reducing DH more dramatically than Duraphat at one week following application (36).

Furthermore, in 2021, another randomized clinical trial was carried out by Abuzinadah et al. to compare the effectiveness of **Gluma** to self-etch Tetric-N-Bond and fluoride varnish in alleviating DH measured instantly and for thirty days subsequent to one application. **Gluma** exhibited statistically significant reductions in the Schiff-Cold-Air Sensitivity Scale and VAS ratings of CDH both directly following application of the agents and at the second-week of follow-up. Additionally, a nonsignificant higher drop in VAS ratings was found in Gluma measures at one month of follow-up (18).

On the other hand, other studies examined the length of service of Gluma in DT occlusion and found that the effect was reversible and after sometime the DTs become exposed again (20). Chen et al in his study of the effectiveness of biomimetic strategies elaborated that Gluma caused partial occlusion of the superficial

layers of DTs by forming an emboli that was merely attached to dentin. Therefore, this emboli or globule was easily removed from the tubule orifice under different conditions causing failure of long-term repair (37).

The results of our study can be confirmed and elaborated by a previous in-vitro study conducted by Jain et al, where All-Bond DS, dentin bonding agent without etching, showed the greatest reduction in permeability when compared to Gluma, Synsodyne and other treatment modalities involving etching of dentin (38). The data indicated that applying primers without etching hypersensitive dentin produced superior reduction in dentin permeability than did any other method, thus only in case of composite placement over the bond may a sensitive dentin be etched. Also the SEM micrographs results of the study revealed that **Gluma**® presented a dramatic rise in solubility and leakage when immersed in saliva, which explains the slower effect it had on CDH reduction between different sessions when compared to Scotchbond universal adhesive, with the latter showing more resistance to salivary dissolution (38).

Investigators have delineated a number of other ways for participants of clinical studies to achieve pain alleviation other than from desensitizing agents. The effect of suggestion by Placebo and/or self-healing ability by secondary and reparative dentin formation may impact hypersensitivity reduction over time. Pain alleviation is a combination of physiological and psychological interactions greatly influenced by doctor-patient relationship (39).

The lack of comparison of the tested agent to a placebo (water) limits the applicability of our study, where instead we contrasted Gluma, a real desensitizing agent, with Scotchbond, a universal adhesive, both of which are claimed to reduce dentin sensitivity.

CONCLUSION

In conclusion, the findings of the present clinical study demonstrated that both desensitizing materials significantly reduced CDH. Through the course of the trial, neither group experienced any negative effects. Gluma® has shown more decline in VAS ratings on the first day as compared to the scotchbond® for airblast hypersensitivity test. Yet, during the six months follow-up for both tests, nearly equal VAS ratings were found between the two groups. Scotchbond showed higher long term accumulated effect than did Gluma with a significant pain reduction difference between both agents for tactile stimulus and a non-significant difference in pain reduction for airblast stimulus.

CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest.

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