Comparative Effectiveness of Two Desensitizing Products in Dental Cervical Erosions

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The aim of this clinical study was to investigate the comparative effectiveness of two dental desensitisers based on 2-hydroxyethyl methacrylate (Gluma desensitizer and Shield activ desensitizer), in decreasing the dentinal painful sensitivity caused by cervical erosions.The clinical trial consisted of 63 patients (323 teeth), with minimum three teeth which presented cervical erosion with dentinal hypersensitivity. The sensitive teeth were treated with Gluma Desensitizer-Heraeus Kulzer (first batch of teeth), Shield activ desensitiser (second batch) and distilled water (third batch of teeth=control group). We effectuated in total three desensitisation sessions, at 5 days difference. The level of cervical dentinal hypersensitivity (CDH) was determined in six sessions, after the patient's response to air-blast stimuli. We used the Visual Analog Scale (VAS) to assess the CDH. The results proved the effectiveness of used desensitizing agents in treating CDH in the first and the second batch of teeth, in comparison with the control group teeth. We noticed that after the second application of Gluma and Shield products significant differences, in comparison with the baseline, appeared. After 3 month follow-up, we ascertained that both products induced the decreasing of CDH. No significant differences were recorded between the two products. A placebo effect in reduction of CDH was observed in the group of teeth treated with distilled water. Both dentin desensitizing agents have proven effective in reducing CDH.

Key words: cervical erosion, dentin hypersensitivity, Gluma desensitiser, Shield activ desensitiser

Modern lifestyle has induced the apparition of various dental diseases, other than dental caries, which determined the loss or the deterioration of the enamel and this enamel loss conduces to Dentinal Hypersensitivity (DH) [1].

DH manifests through dental abrasion, dental erosion, occlusal trauma, etc. [2,3].

Cervical erosion is the damage of dental hard tissues, situated in cervical area of dental crowns, which is not produced by oral bacteria [4]. Once enamel is lost and cement or dentine is exposed, the dental under-layers are subjected to massive erosion, as a consequence of lower inorganic mineral content [5]. When the dentine is exposed, and the dentinal tubules are opened, external stimuli (as thermal, mechanical, chemical, tactile, dehydrative or osmotic stimuli), can cause excessive pain response in the pulp. [6,7]. The described clinical condition, which cannot be attributed to another disease or tooth defect, has been defined as dentin hypersensitivity [8-10].

The dentin and the dental pulp are considered as a biologic entity named dentin-pulp complex [11,12]. The sensory function of the pulp-dentin complex is one of his many functions. The acute localised pain, decreased after the cessation of the stimulus action is called dentinal pain/ dentinal hypersensitivity, which was described as one of the most painful affection of teeth [13,14]. According to the Canadian consensus document [15], dentinal hypersensitivity (DH) has been defined as pain derived from exposed dentin in response to chemical, thermal tactile or osmotic stimuli which cannot be explained as arising from any other dental defect or disease.

Because organs and tissues located in the oral cavity are well supplied with blood and nerve endings, all pathological processes in the region cause high-intensity pain [16]. The perception of pain originating in the mouth is perceived as disproportionately severe in relation to the actual cause of the pain, as compared to the rest of the body [17]. Three main mechanisms of dentin sensitivity are proposed: direct innervations' theory, the odontoblastic-receptor theory and fluid movement/hydrodynamic theory (fig. 1) [18].

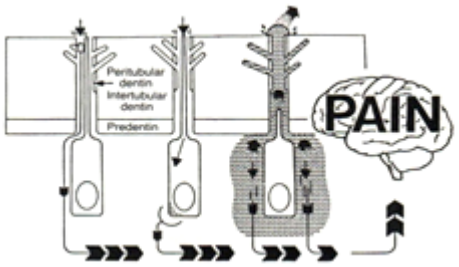

Fig. 1. Schematic aspect of the theories on DH

In conformity with the direct innervations' theory, the nerve's endings form dental pulp enters in the dentinal tubules and extends to dentin-enamel junction, and so, all mechanical stimuli directly transmit the pain [19]. In the odontoblastic-receptor theory, odontoblasts act as receptors of pain and transmit signals to the pulpal nerves, but no synopsis has been found between odontoblasts and pulpal nerves [18]. Hydrodynamic theory for sensitive dentine was first proposed by Brännstorm [20] and is the most widely accepted theory for dentinal hypersensitivity. According to this theory, the stimuli acting on the exposed

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dentine surface cause movement of the fluid in the dentinal tubules, stimulating pulp nerve receptors and this stimulation transmitted to the central nervous system, is experienced as pain [9, 21-23]. The movement of fluid can be toward the inside of the pulp or the outside of dentin. Cooling, drying, evaporation, and hypertonic chemical stimuli cause the dentinal fluid to flow away from the dentin-pulp complex and lead to an increase in pain [18,24-26]. The intensity of the pain varies from strong with high intensity, to mild with moderate intensity.

The incidence of the disease in the adult population ranges from 8% to more than 50% [7,9].

There are various methods available for the treat-ment of dentin hypersensitivity, all with the aim of oblit-erating the dentinal tubules. Dentinal tubule sealing can be secured with the use of restorations, dental adhe-sives or the formation of a smeared dentin surface. Fluoride varnishes were introduced on the market to increase the efficiency and permanence of fluoride when in contact with the tooth surface, in order to allow a slow and continuous release of fluoride [27].

2-Hydroxyethyl Methacrylate is a hydroxyl-ester compound and a resin monomer, used in desensitizing dentin. By applying 2-hydroxyethyl methacrylate locally to sensitive teeth, sensitive areas in the teeth get sealed and block the dentinal tubules at the dentin surface from stimuli that cause pain. This prevents excitation of the tooth nerve and relieves pain caused by tooth hypersensitivity [28].

Hydroxyethyl-methacrylate is the monomer that is used to make the polymer polyhydroxyethyl-methacrylate. The polymer is hydrophobic, and when the polymer is subjected to water it will swell due to the molecule's hydrophilic pendant group. Depending on the physical and chemical structure of the polymer, it is capable of absorbing from 10 to 600% water relative to the dry weight [29].

The aim of this study was thus to assess the effec-tiveness of two desensitizing agents in reducing dentin hypersensitivity in a randomized clinical trial.

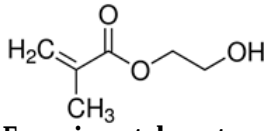


Fig. 2. Chemical structure of hydroxyethyl-methacrylate

Experimental part

Materials and method

The requirements for an acceptable dental material are many, but one of the most important is the biocompatibility [30].

Gluma desensitizer (Heraeus Kulzer) is a combination of glutaraldehyde and hydroxyl-ethyl-methacrylate (HEMA). Glutaraldehyde causes the coagulation of amino acids and proteins in dentin tubules and is effective in sealing dentinal tubules, being an efficient disinfectant too [31-34]. Gluma Desensitizer as desensitizing agent is an aqueous solution of 5% glutaraldehyde (GA) and 35% hydroxyethyl-methacrylate (HEMA) and 60% water. The mechanism of sealing is the precipitation of plasma protein (produced by GA and not by HEMA), in the dentinal fluid in order to occlude the dentinal tubules [35,36].

Shield activ dentin desensitizer (produced by Prevest Denpro Ltd, India), is an aqueous solution, which contain 2-hydroxiethil-methacrylate, benzalkonium chloride, sodium fluoride, potassium nitrate and excipiens [37].

The researches were conducted in the Dental Medicine Faculties of Bucharest, Tirgu-Mures, Oradea and Craiova Universities.

The patients were selected after a detailed anamnesis and were attended only by those that have expressed their desire to be part in the research.

The clinical research has been carried out on 63 patients (29 males and 34 females), with minimum three teeth with cervical hypersensitivity in their oral cavity, resulting in a total number of 323 teeth included in the study. Majority of the patients were female (53.96%). The age range of the patients was similar, between 48-53 years, with a median age of 50.5 years and a mean of 50.5 ± 2.5 years (fig. 3).

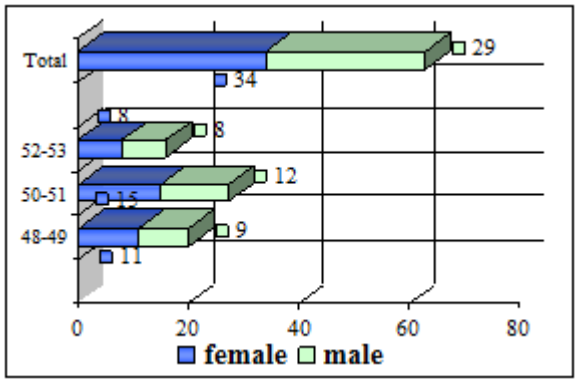


Fig. 3. Distribution of patients after age and gender

The established exclusion criteria were: the pres-ence of untreated caries/occlusal pits/ fissures, incorrect restorations, ongoing orthodontic or periodontal treatment at the CDH site; patients which presented systemic diseases; pregnant women or lactating mothers; patients with unshielded cardiac pace maker; patients with CDH who has subjected in the previous 3 months exogenous dental bleaching; patients with poor oral hygiene; uncooperative patients.

The established inclusion criteria were: patients who presented teeth with CDH to mechanical and chemical stimuli (including air stimulus), cooperative patients with good oral hygiene. The presence of gingival recession and/ or non-carious cervical lesions was considered acceptable. The written agreement for participation in the research was signed by the patients.

The sensitive teeth were treated with Gluma desensitizer-Heraeus Kulzer (first batch of teeth), Shield activ desensitiser (second batch of teeth) and distilled water (third batch of teeth=control group).

Age (years)	Female 34 patients, 172 teeth			Male (29 patients, 151 teeth)		
	Gluma desensitizer	Shield desensitizer	Distilled water	Gluma desensitizer	Shield desensitizer	Distilled water
48-49	21	21	20	17	17	17
50-51	17	17	18	16	17	16
52-53	19	20	19	17	17	17
Total	57	58	57	50	51	50

Table 1
THE DISTRIBUTION OF TEETH (323) IN RESEARCH, AFTER THE AGE AND GENDER OF PATIENTS

In table 1 is presented the distribution of teeth (323) in research, after the age and gender of patients. Male (29) patients presented 151 teeth with CDH, which represent 5.20 teeth with CDH /patient, and the female (34) patients presented 172 teeth with CDH, which represent 5.05 teeth/ patient.

We have effectuated in total three desensitisation sessions, at 5 days difference. The hypersensitivity level was determined before the first treatment, immediately after every desensitising session (3), at one month and at 3 months post-treatment. In total, we conducted six sessions of determining CDH.

The deposition of the desensitizing substances used in the study was in according to the manufacturers' recommendations. The method of deposition the both desensitizers and of distilled water was as follows: we cleaned the dentine by rinsing with water, we dried the treated surface, we applied smallest amount of desensitizer/distilled water on the eroded cervical dentinal surface with a pellet, in thin layer, and leaved for 30 s; we dried the eroded area by applying a stream of compressed air; we repeated the applications of agents for three times in each session; we reapplied the substances at five days interval. The patients did not know which type of treatment cor-responded to each tooth. After the first session of desensitization, we distributed to all patients and participants in research Colgate Sensitive Complete Protection toothpaste (which contain active potassium nitrate, active sodium fluoride, water, glycerine, hydrated silica, sorbitol, PEG-12, sodium lauryl sulphate, flavor, poloxamer 407, tetrapotassium pyrophosphate, PVM/MA copolymer, xanthan gum, cellulose gum, etc.) and two Colgate 360° Enamel Health™ Sensitive Toothbrush (fig. 4).

Fig. 4. The used toothpaste and toothbrush

The determinations of CDH were realised after the patient's response to air-blast stimuli. The air blast was applied with the air-water syringe of the dental unit, for 15 seconds, at a distance of 1 cm from the tooth surface, to avoid desic-cating the area with cervical dentinal erosion. The neighbouring vital teeth with cervical erosion were protected with dental gauze and the operator's hand.

We used the Visual Analog Scale (VAS) to assess the CDH, scored as follows: 0=no discomfort; 1=discomfort but no severe pain; 2=severe pain during stimulation; 3=severe pain during and after stimulation.

After recording the baseline scores, the teeth with CDH of patients were randomly and blindly assigned to one of the treatment groups, according to the applied desensitizing agent, Gluma desensitizer (n=107) or Shield activ desensitizer (n=109), or to the placebo group of teeth (n=107) (table 1).

Results and discussions

The distribution of assessment scores after the application of desensitizing agents/distilled water on the teeth with CDH is presented in table 2.

The results of our clinical study showed the effectiveness in treating cervical sensitivity in the first and the second batch of teeth, in comparison with the control group teeth. After the second application of Gluma and Shield products, significant differences were observed, compared with the baseline.

After 3 month follow-up, we ascertained that both desensitizing agents induced the decreasing of CDH level.

No significant differences were recorded between the two products. We must notice that for both tested desensitizers, a lower level of CDH was founded in the 6-th assessment (after 3 months) compared with the baseline CDH score.

After the assessments, we can conclude that both desensitizing agents decreased CDH, in comparison with the placebo group of teeth.

Placebo effect was observed in the treated teeth with distilled water, but the decreasing level of CDH appeared only in the second and third recording sessions.

Dentine hypersensitivity (DHS) remains a worldwide under-reported and under-managed problem, despite making some dental treatments more stressful than necessary and having a negative impact on the patient's quality of life [38].

After the studies of Davari et al [39], the number of tubules in sensitive dentin is eight times more than the number of tubules in non sensitive dentin and the dentinal tubules of sensitive dentin are wider than those in non sensitive dentin.

Many therapeutic agents to be used by patients (at home) and professional ones (to be used in the dental office) are in present on market [6, 14, 40-43].

After the researches of Jumanca et al [44], the treatment of cervical caries and even root caries can be achieved successfully by combining minimal surgical procedures with restorative therapy.

Contemporary reports show that the treatment of dentin hypersensitivity involves interruption of the neural response to pain stimulus by topical application of preparations containing potassium salts, because potassium ions cause

Table 2

THE EVALUATION OF DENTINAL SENSITIVITY IN TEETH WITH CDH BEFORE AND AFTER DESENZITIZATION

Determination / Responses	Sensibility determinations											
	Gluma desensitizer 107 teeth with CDH				*Shield activ desensitizer 109 teeth with CDH*				*Distilled water 107 teeth with CDH*			
	0	1	2	3	0	1	2	3	0	1	2	3
First determination	-	-	74	33	-	-	74	35	-	-	73	34
Second determination	6	24	60	17	5	24	61	19	-	21	62	24
Third determination	11	55	35	6	11	57	35	6	-	6	71	30
Fourth determination	15	59	33	-	15	62	32	-	-	-	74	33
Fifth determination	16	61	30	-	16	62	31	-	-	-	73	34
Sixth determination	16	61	30	-	16	62	31	-	-	-	72	35

depolarization of nerve receptors and reduce the conductivity. A second approach is by mechanical occlusion of the lumen of open dentinal tubules and then, the formation of insoluble precipitates with the participation of calcium, fluoride, strontium, arginine, the adhesive resins or lasers [9,21,25,40,42-48].

Dentinal tubules can be obliterated on the surface and/or occluded within the tubule orifices. The superficial occlusion of tubules can be removed by daily tooth brushing, dissolution of the precipitate promoted by saliva or consumption of acidic beverages, leading to short-term desensitizing effects [49].

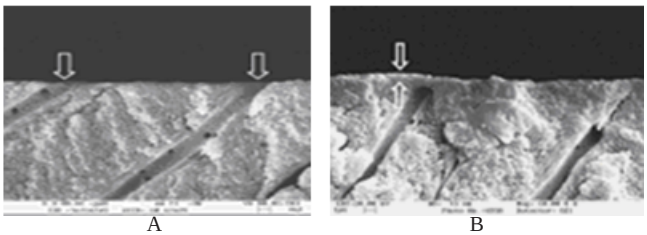
Effective treatment with long-term results has been related to intratubular deposition, which reduces the fluid flow rate or totally seals the tubule lumen [50].

The exact mechanism of the desensitization is not yet clearly understood. The use of bonding resin or adhesive material for sealing dentinal tubules has been suggested. One such product, HEMA, is said to have desensitizing property. It blocks the tubules by coagulation of dentinal fluid proteins within the tubules, thereby counteracting the hydrodynamic theory [51].

HEMA is soluble in water allowing it to penetrate deep into tubules, however, the effect is reversible and HEMA gradually loses its effect allowing the dentinal tubules to again become exposed [52].

Gluma desensitiser penetrate exposed dentinal tubuli up to 200μm. The result is the formation of multiple layers of protein septa, that prevent intertubular fluid movements due to osmotic changes. In the same time, Gluma desensitizer provides a hermetic seal that acts as a microbial barrier, inhibiting bacterial growth and resurrects collapsed collagenous fibres, improving the bond strength of many adhesives [53].

Figure 5A present the SEM micrograph of untreated dentin where the open dentinal tubules are indicated (x 10,000) and figure 5B, the image of HEMA glutaraldehyde-treated dentin, where is visible a thin layer that occlude the entrance of dentinal tubules (x 10,000) [54].



A B

Fig. 5. SEM micrograph of untreated dentin (A: open dentinal tubules are indicated) and of HEMA glutaraldehyde-treated dentin (B: a thin layer occlude the entrance of dentinal tubules) (x 10,000)

Both desensitizers used in our researches are one step chair side treatment for dentinal hypersensitivity caused due to cervical erosions/exposed roots, acts to seal dentinal tubules and inhibits the growth of bacteria.

The researches of Galuscan and al [5] on three desensitizing dental materials concluded that Gluma Desensitizer (Hereus Kulzer) leads to the most homogeneous hybrid layer on the dental surface, followed by Fluor Protector (Ivoclar) and Tooth Mouse (GC).

The study of Bechir et al [57] have underlined that prolonged desensitization effect in teeth with abfraction of Geristore hybrid ionomers-composite dental material, in comparison with the simple coating with Gluma desensitizer.

The prevalence in distribution and appearance of CDH have been reported differently in different studies. These differences are due to the differences in populations, habits, dietaries, and methods of investigation [18]. Several studies have reported non-carious cervical lesions (NCCLs) and DH in adult populations, with prevalence rates ranging from 5% to 85% [55] and 2-8% to 74%, respectively [56].

A large number of studies were published regarding the ideal product for reducing the dentinal hypersensitivity, but it has not been possible to reach a consensus about the product that represents the gold standard in the treatment of this affection [58].

Biocompatibility of dental materials is an important consideration for the patient, clinician, laboratory technician, and manufacturer. Ideally, a dental material that is to be used in the oral cavity should be harmless to all oral tissues, gingiva, mucosa, pulp, and bone [59].

The requirements for an acceptable dental material are many, but one of the most important is the biocompatibility. Furthermore, it should contain no toxic, leachable, or diffusible substances that can be absorbed into the circulatory system, causing systemic responses, including teratogenic or carcinogenic effects [60].

Conclusions

Within the limitations of this study, the following conclusions can be drawn:

- both desensitizing agents were capable to reduce the teeth CDH to different physical and chemical agents;
- distilled water produced a placebo effect in the witness group of teeth with CDH;
- clinical procedures for the application of desensitizing agents are very simple.

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