

“COMPARATIVE EVALUATION OF EFFECT ON DENTINAL TUBULE OCCLUSION BY THREE DIFFERENT DESENSITIZING DENTIFICES: A SEM STUDY”

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Abstract

Introduction: Dentine hypersensitivity (DH) occurs on exposed dentine and is dependent on the patency of dentinal tubules. This study compared the effectiveness of toothpaste containing Strontium Chloride, Novamine, Pro-Arginine in occluding dentine tubules along with comparison of those.

Materials and Method: 45 freshly extracted teeth were randomly divided (15 each) into 3 groups:

Group 1 15 Teeth treated with toothpaste containing Strontium Chloride.

Group 2 15 Teeth treated with toothpaste containing Novamine

Group 3 15 Teeth treated with toothpaste containing Pro-Arginine.

Each specimen was brushed with the dentifrice slurries and examined under SCM.

Result: The mean of occluded dentinal tubules on the dentin surface after brushing with three dentifrices were significant among them Pro-arginine group showed maximum occlusion.

Conclusion: The present *in vitro* SEM study results revealed that: All the experimental agents– Strontium chloride, novamin, Pro- Arginine were effective in occluding dentinal tubules & the percentage (%) of occluded tubules was found to be highest for Pro- Arginine as compared to the other groups.

Keywords: SCM, DH

Introduction:

Dentine hypersensitivity is a relatively common, painful dental condition. Typically, the pain is short and sharp and occurs in response to certain stimuli applied to exposed dentine. At a macroscopic level, dentine exhibiting hypersensitivity appears no different from nonsensitive dentine.

Dentin hypersensitivity (DH) is neither a recent problem nor a rare one; nonetheless, this clinical condition remains poorly understood with no effective or permanent treatment available.¹ Dentine hypersensitivity 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

It is clinically described as a painful response to thermal, chemical, mechanical, evaporative, or osmotic stimuli applied to opened dentinal tubules, which cannot be ascribed to any other form of dental defect or pathology.¹ The hydrodynamic theory best explains the mechanism of dentin hypersensitivity, and this is widely accepted as how dentin hypersensitivity occurs. The two main approaches to treating dentin hypersensitivity are interference of nerve transmission and to occlude the open dentin tubules.⁴

Toothpastes are the most widely used dentifrices for delivering over-the-counter desensitizing agents. Desensitising toothpastes should utilise both or at least one of these two mechanisms of action to relieve dentine hypersensitivity. If daily brushing with desensitising toothpastes occlude open dentinal tubules, it should be an effective method to treat this common disease.

Currently no universally accepted product that completely relieves the symptoms of DH appears to be available although there are a number of products that have been formulated for the treatment and management of DH which have demonstrated varying degrees of effectiveness.

Strontium chloride: It is a salt of strontium and chloride. It is prepared by treating strontium hydroxide with hydrochloric acid.

SrCl₂ is useful in reducing tooth sensitivity by forming a barrier over microscopic tubules in the dentin containing nerve endings that have become exposed by gum recession. Known in the these products are called "strontium chloride toothpastes", although most now use **potassium nitrate** instead which works as a nerve calming agent rather than a barrier

Pro- arginine: Arginine bicarbonate is a salt of naturally derived arginine which has oral health benefits.

When the desensitizing paste is applied to exposed dentin, Arginine (positively charged) and calcium carbonate, found in saliva naturally, work together to accelerate the natural mechanisms of occlusion by binding to the negatively charged dentine surface to deposit a dentin-like mineral, as a plug within the dentin tubules and a protective layer on the dentin surface. This consists of arginine, calcium carbonate and phosphate and salivary glycoproteins. It is resistant to normal pulpal pressures and to challenge by acids in oral cavity. It is also effective in reducing dentin fluid flow thereby relieving hypersensitivity.

Novamine: composed of sodium calcium phosphosilicate. It increases salivary pH and supercharges the saliva with calcium and phosphate ions..

It is indicated that when NovaMin comes in contact with saliva or any aqueous media, its active ingredient, inorganic chemical C-SPS, binds to the tooth surface to initiate the remineralization process on the tooth enamel. This is performed by providing silica, calcium, phosphorous, and sodium ions to the tooth structure. A localized transient increase in pH occurs during the initial exposure of the mineral due to the release of sodium. This rise in pH helps the calcium and phosphate to form the NovaMin particles, followed by calcium and phosphorous found in saliva to form a Ca-P layer. As the particles' reaction continues and deposition of Ca-P phosphate complex takes place, this layer crystallizes into a calcium hydroxyl apatite and also known as hydroxyl carbonate apatite.

The aim of this SEM study was to evaluate the effects of three different desensitizing dentifrices i.e Strontium Fluoride, arginine and Novamine in occluding dentinal tubule by **Scanning Electron Microscopy (SEM)** & Inter group comparison of different desensitizing dentifrices i.e Strontium Fluoride, arginine and Novamine.

study.

Materials & method:

The present in vitro study was conducted in the Department of Periodontology and Implantology, D.J. College of Dental College and Research, Modinagar (U.P.) in collaboration with the Birbal Sahani Institute of Paleobotany, Lucknow (U.P.). Freshly extracted 45 molars were collected from the Department of Oral and maxillofacial surgery, D.J. College of Dental Sciences and Research, Modinagar and stored in 10% formalin. All experiments were conducted within 2 month of extraction. Periodontally compromised molar teeth, Impacted 3rd molars which need to be extracted were selected. Carious teeth, Restored teeth, Fractured teeth, RCT treated teeth, Teeth with developmental anomalies, Teeth that

demonstrated the presence of any wasting diseases were excluded.

To prepare the Dentin specimen extracted molar teeth were scaled with ultrasonic scaler and thoroughly cleaned with normal saline and stored in 10% formalin at room temperature for no longer than two months prior to their use. The teeth will be sectioned mesio-distally using a double-sided diamond disc. One block will be obtained from each flat cervical dentine surface measuring 5x5x3mm (L x B x H) by transverse sectioning of the tooth. The specimens were ultrasonicated in distilled water for 12 mins to remove residual smear layer and to open dentinal tubules.

Samples were ultrasonicated in a ultrasonic cleaner with distilled water for 12 min for the removal of smear layer and expose the dentinal tubules to simulate hypersensitive dentin. Subsequently the sections were copiously rinsed with distilled water for a period of 30 seconds.

After the selection of teeth, they were cleaned by irrigating with saline to get rid of blood and saliva then teeth were washed with distilled water and stored in 10% Formalin at room temperature.

The specimens were randomly divided (15 each) into 3 groups:

Group 1 15 Teeth treated with toothpaste containing Strontium Chloride.

Group 2 15 Teeth treated with toothpaste containing Novamine

Group 3 15 Teeth treated with toothpaste containing Pro-Arginine.

Each specimen from 1,2 & 3 group 1 were brushed with the dentifrice slurries which were prepared by diluting 2gm of the dentifrice in 6ml of distilled water.

The Scanning Electron Microscopic analysis was done at Birbal Sahani Institute of Palaeobotany, Lucknow U.P. Treated specimens were mounted on aluminum stubs with a double sided adhesive tape. Mounted samples were then placed in the sputter coater for gold-palladium coating which were done on the each sample.

After that the specimens were examined under scanning electron microscope at magnification of 1000X and a representative photomicrograph of the each specimen were randomly taken.

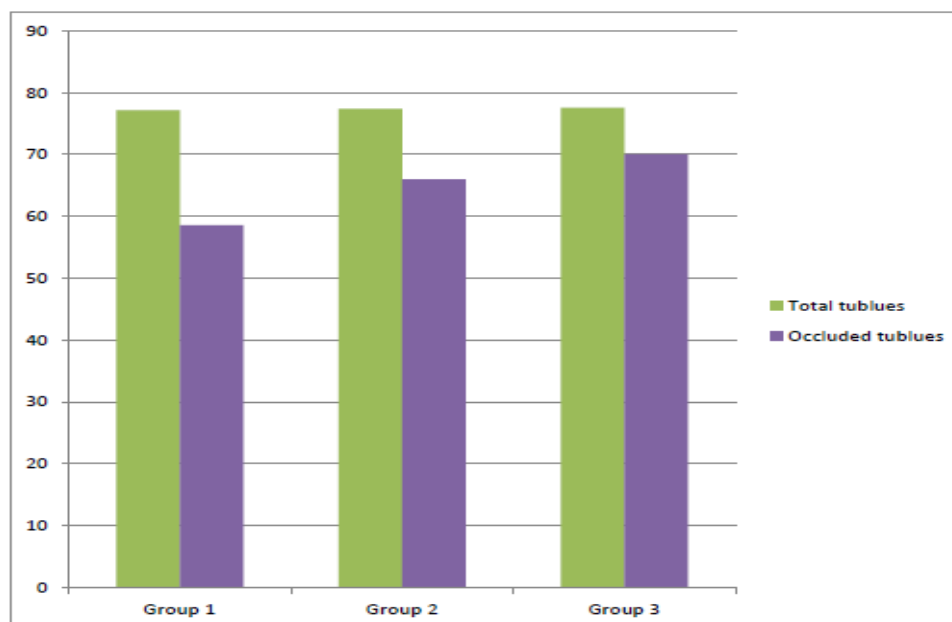
Result:

The mean of occluded dentinal tubules on the dentin surface after brushing with three dentifrices was significant.

The mean of occluded dentinal tubules on the dentin surface for three different groups

Group 1: 58.60 , Group2: 66.00 , Group3: 70.00

Which was greatest for group 3(Pro-Arginine)



Graph :1 Mean of total and occluded dentinal tubules on the dentin surface after brushing with dentifrices and without dentifrice.

Discussion:

Dentine hypersensitivity (DH) is a painful response of the tooth to different stimuli such as brushing, low pH beverages, occlusal overload, dental caries and thermal changes⁵. DH is characterized by a rapid onset of sharp burst of pain, lasting for seconds or minutes. The desired goal for treatment of dentin hypersensitivity is attainment of immediate as well as lasting relief from discomfort. This is achieved by application of a desensitizing agent used alone or as an adjunct to another dental procedure. Till date no such treatment has been discovered and there is no 'gold standard' by which one can assess the efficacy of the agent use⁶.

Dentinal hypersensitivity satisfies all the criteria to be classified as a true pain syndrome. It mostly affects individuals at the end of their third decade of life, causing great discomfort. In severe cases, it may lead to emotional alterations and behaviour changes. It is mostly found in permanent canines and premolars in both dental arches. The cervical region of the vestibular face of teeth is the most affected region.

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 literature⁷ - Direct innervation theory, Odontoblast receptor, Fluid movement/hydrodynamic theory

According to direct innervation theory, nerve endings penetrate dentine and extend to the dentinoenamel junction. Direct mechanical stimulation of these nerves will initiate an action potential. 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.⁸

Odontoblast receptor theory states that odontoblasts acts as receptors by themselves and relay the signal to a nerve terminal. 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.⁷

Brannstrom⁹ has proposed that dentinal pain is due to hydrodynamic mechanism, i.e., fluid force. Scanning electron microscopic (SEM) analysis of "hypersensitive" dentin shows the presence of widely open dentinal

tubules.⁵⁰ 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. 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.¹⁰

Etiological and predisposing factors¹¹ for DH may be Loss of enamel, Denudation of cementum, Gingival recession, Attrition, Abrasion, Abfraction, Erosion (intrinsic and extrinsic), Tooth malposition, Thinning, fenestration, absent buccal alveolar bone plate, Periodontal disease and its treatment, Periodontal surgery, Patient habits.

The pain arising from DH/RDS is extremely variable in character, ranging in intensity from mild discomfort to extreme severity. The degree of pain varies in different teeth and in different persons. It is related to the patient's pain tolerance as well as to emotional and physical factors. It may emanate from one tooth or several teeth and it is sometimes felt in all quadrants of the jaws. Most patients describe the pain arising from DH as being rapid in onset, sharp in character, and of short duration.¹² Patients also report a wide variety of pain-producing conditions and a large combination of stimuli has been recorded in the literature. The external stimuli eliciting dentinal pain can be thermal, osmotic, chemical physical, or mechanical in nature. The thermal stimuli include hot and cold food and beverages and warm or cold blasts of air entering the oral cavity. Osmotic stimuli include sweet food and beverages. Acid stimuli include grapefruit, lemon, acid beverages, and medicines. Common mechanical stimuli are toothbrushes, eating utensils, and dental instruments. The use of cold air blasts from a dental air syringe, cold water, and suction from a dental aspirator tip (physical) may also cause discomfort.

On the basis of mechanism of action Desensitizing agents are Nerve desensitization, Potassium nitrate, Protein precipitation, Gluteraldehyde, Silver nitrate, Zinc chloride, Strontium chloride hexahydrate, Plugging dentinal tubules,

Sodium fluoride, Stannous fluoride, Strontium chloride, Potassium oxalate, Calcium phosphate, Calcium carbonate, Bio active glasses (SiO_2 P_2O_5 CaO Na_2O), Dentine adhesive sealers, Fluoride varnishes, Oxalic acid and resin, Glass ionomer cements, Composites, Dentin bonding agents, Lasers, Propolis.

Currently there is no proven therapy that can always reduce the pain at satisfactory levels, even with the combination of different protocols. The treatment of chronic dentinal hypersensitivity is based on the concept of reducing fluid movement inside the dentin tubules by narrowing or occluding of tubule openings.

The occlusion of dentin tubules leads to the reduction of dentin permeability to decrease the feeling of pain from Chronic Dentinal hypersensitivity. According to the hydrodynamic theory, the effectiveness of dentin desensitizing agents is directly related to their capacity of promoting the sealing of the dentin canaliculi.

Conventional therapies for the treatment of Dentinal hypersensitivity comprehend the topical use of desensitizing agents, either professionally or at home such as nerve desensitizers (potassium nitrate), protein precipitators (glutaldehyde, silver nitrate, zinc chloride, strontium chloride, dentinal tubule pluggers (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.

Since dentinal hypersensitivity can wax and wane over time even in the same individual one must be able to decisively diagnose and recognize the predisposing factors to formulate a treatment plan for effectively managing dentinal hypersensitivity. The goal of the present study was to find out the most efficacious agent amongst Strontium chloride, Potassium nitrate and Sodium monofluorophosphate containing dentifrices in the dentinal tubule occlusion.

In the present *in vitro* study the dentin blocks were divided into three groups **Group 1** (Strontium chloride); **Group 2** (Novamine); **Group 3** (Pro-Arginine) and were evaluated using a SEM to determine the amount of dentinal tubular occlusion.

Each specimen from group 1 group 2 & 3 were brushed with the dentifrice slurry.

The Scanning Electron Microscopic analysis was done and the results were as follows:

All the experimental agents—were effective in occluding dentinal tubules as compared to the control group & The percentage (%) of occluded tubules was found to be highest for Pro-Arginine as compared to the other groups.

Conclusion:

The present *in vitro* SEM study results revealed that: All the experimental agents– Strontium chloride (Sensodyne original), novamin, Pro- Arginine were effective in occluding dentinal tubules as compared to the control group. The percentage (%) of occluded tubules was found to be highest for Pro- Arginine as compared to the other groups. The occlusion of the tubules by Novamin, and Strontium chloride showed comparable results.

However further long term *clinical* and *in-vitro* studies are required to establish the role of these agents in reducing dentin hypersensitivity.

References

1. Seltzer S, Bender I B, Ziantz M. The dynamics of pulp inflammation: Correlation between diagnostic data and actual histological findings in the pulp. *Oral Surg Oral Med Oral Pathol* 1963 16: 846-969.
2. Passley D H. Mechanisms of dentine sensitivity. *Dental Clinics of North America* 1990 34: 449-474.
3. Addy M. Etiology and clinical implications of dentine hypersensitivity. *Dental Clinics of North America* 1990 34: 503.
4. Flynn J, Galloway R, Orchardson R. The incidence of 'hypersensitive' teeth in the West of Scotland. *J Dent* 1985 13: 230-236.
5. Addy M, Uguhart E. Dentine hypersensitivity: its prevalence, aetiology and clinical management. *Dent Update* 1992; 19(10):410-412.
6. R H Dababneh, A Khouri, M Addy. Dentine hypersensitivity-an enigma? A review of terminology, mechanism, aetiology and management. *British Dental Journal* 1999; 187:606-611.
7. Miglani S., Aggarwal V., Ahuja B. Dentine Hypersensitivity: Recent Trends in Management. *Journal of Conservative Dentistry* 2010; 13: 218-224.
8. Chu CH, Lo ECM. Dentine hypersensitivity: a review. *Hong Kong Dent J*. 2010;7:15-22.
9. Braennstrom M, Astrom A. A study on the mechanism of pain elicited from the dentin. *J Dent Res*. 1964;43:619-625.
10. Shah S, Shivakumar Tumkur A, Khot O. Efficacy of NovaMin- and Pro-Argin-Containing Desensitizing Dentifrices on Occlusion of Dentinal Tubules: Dental Hypotheses 2017;8-4.
11. Khijmatgara S, Reddy B U, John S. Is there evidence for Novamin application in remineralization?: A Systematic review. *Journal of Oral Biology and Craniofacial Research* 10 (2020) 87-92.
12. Gillam DG, Orchardson R. Advances in the treatment of root dentine sensitivity: mechanisms and treatment principles. *Endodontic Topics* 2006;13:13-33.