Annals of International Medical and Dental Research
E-ISSN: 2395-2822 | P-ISSN: 2395-2814
Vol-7, Issue-3 | May-June, 2021
Page no- 540-553 | Section- Research Article (Dentistry)

Dentin Hypersensitivity: A Review of its Treatment Modalities

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Received: February 2021
Accepted: February 2021

Abstract

Background: An important clinical issue faced in routine dental practice is dentin hypersensitivity (DH). Managing this condition requires a clear understanding of the complexity of the issue, as well as the variety of currently available treatment modalities.In order to assess the evidence supporting the current treatment options, the purpose of this article was to review treatment modalities for DH with various mechanisms of action and delivery modes. **Methods**: A comprehensive literature review was carried out using electronic databases. **Results**: Treatment with nerve desensitizers and dentinal tubule occlusion agents will minimize the short-term symptoms of DH, but the long-term outcome of such therapies is unclear. There is also a need to provide DH patients with more effective relief. **Conclusion**: It is evident from the currently available scientific literature that there is no recognized ideal desensitizing product that offers both fast-acting and long-lasting protection against DH-related pain.

Keywords*:* Dentin Sensitivity, Dentin Desensitizing Agents, Oral Health, Dental Care.

INTRODUCTION

Dentin hypersensitivity (DH) can have similar clinical symptoms to other dental conditions, such as caries, deficient restorations, cracked-tooth syndrome, and pulpit. Therefore, all potential causes should be removed before the diagnosis of DH is made. It is necessary to begin with patient evaluation and instruction on dietary habits, oral hygiene and evaluation of DH over time in order to exclude any etiological factors.[1]

The etiology of DH was accompanied by three theories: hydrodynamic theory (movement of the fluid within the dentinal tubules),[2,3] direct innervation theory (direct stimulation of the nerve ends) and odontoblastic transducer theory.[4,5] However, evidence does not support the second and third hypotheses, and hydrodynamic theory remains the most commonly accepted theory to describe the pain associated with DH.[6]

Numerous treatment modalities are available for the management of DH,

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but DH can be difficult and inconsistent, considering the range of treatment options available.

The aim of this article was to comprehensively review treatment modalities for DH with different mechanism of action and mode of delivery in order to determine the evidence supporting the available treatment options.

Treatment Modalities for Dentin Hypersensitivity

The currently available treatment modalities for DH are almost based on two main pathways: 1) the reduction of neural transmission (nerve desensitization) and/or 2) the physical occlusion of dentinal tubules (block/occlude the dentinal tubules).[7] In addition, DH therapy has been categorized on the basis of the mode of delivery into (professional or in-office) and (over-the-counter or home-use) therapies. Over-the-counter desensitizing agents are usually based on formulations of the same active ingredients as in-office desensitizing agents, but with a lower dosage that enables safe use, appears to be affordable and can simultaneously treat generalized DH affecting many teeth.[7]

Below are the most commonly used active agents, depending on the mode of action for DH treatment.

1. Nerve desensitization (neural blocker)

❖ 1.1. Potassium salts

The ability of potassium salts, such as potassium nitrate, potassium chloride, and potassium citrate, to decrease the transmission of neural pain. Extracellular potassium ions penetrate the dentinal tubules and disrupt the neural (pulp) response, thereby preventing the nerve from repolarizing.[8,9] It is, therefore, necessary that the high level of extracellular potassium ions be sustained to have the required effect, which may take a longer period of time.[10]

Various potassium salts have been formulated into toothpaste, and the most widely evaluated desensitizing agent has been the 5% potassium nitrate formulations (e.g., SensodyneProenamel toothpaste; GlaxoSmithKline, Weybridge, UK).[11] Early studies indicated that when used for two weeks, potassium-containing toothpaste was beneficial for DH relief.[12–14] On the other hand, other research found no substantial difference in pain relief compared to control or negative control toothpaste when potassium-containing toothpaste were used.[15,16]

In addition, several studies have shown that when other agents for the treatment of DH were compared with potassium salts, some of them were more effective. For example, potassium was found to provide inferior pain relief in randomized clinical trials when directly compared to positive controls, such as calcium sodium phosphosilicate (CSPS),[16,17] or Arginine.[15,18]

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A systematic review and meta-analysis of six randomized control trials (RCTs), which aimed to evaluate the efficacy of potassium-containing toothpaste in reducing DH compared to non-potassium containing toothpaste, failed to show a significant effect.[19]

In addition, another systematic review of the effectiveness of potassium salts showed weaker evidence for potassium salts to relieve DH when compared to arginine and CSPS over long-term assessment.[20] The mode of action of Arginine and CSPS is occluding dentinal tubules rather than desensitizing the nerve as in potassium salts. In other words, the evidence for desensitization by potassium only tends to be marginal. The addition of an occluding agent leads to substantial reductions in sensitivity, indicating the individual effect of potassium is limited.
The addition of other ions such as stannous fluoride, and silica in toothpaste containing potassium, resulted in more effectiveness in reducing DH pain than a potassium-only toothpaste.[12,21]

❖ **1.2. Iontophoresis**

In 1984, the use of iontophoresis was first applied in dentistry. For the treatment of DH, iontophoresis of fluoride has been used; however, its real value is debated. Two mechanisms for its function have been suggested. The first and key mechanism for reducing DH is the use of electrical current to discharge fluoride ions into the dentinal tubules, reducing the sensory nerve's permeability or paresthesia.[22] By way of clarification, the penetration of fluoride ions into dentinal tubules would occlude the tubules and reduce the conduction of stimuli.[23] The second suggested mechanism is that it may desensitize hypersensitive dentin by formation of a secondary dentin as a consequence of the electrical current applied.

However, the effects of iontophoresis for dental practice may be impractical since molecules must be charged in order to repulse them. Long-term randomized clinical trials of efficacy or the absence of side effects are still not available.[2,3]

2.<u>Dentinal tubules occlusion</u>

The most direct approach to dentin desensitization is the alteration of the flow of fluid in the dentinal tubules by the occlusion of the dentinal tubules. The efficacy of tubular occluding agents will rely on their resistance to removal, particularly in the acidic environment. There are numerous and dynamic techniques in which various agents and products could potentially work to partially or completely occlude tubules.[14,24]

❖ **2.1. Strontium**

Strontium has been investigated as a treatment for DH since 1956. It was introduced as a solution of 25% strontium/water and a paste of 75% glycerin.[20] For decades, toothpastes containing strontium salts (strontium chloride and strontium acetate) have been available.

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Strontium salt has been found to decrease DH by utilizing its metal compound to occlude the dentinal tubules.[9] The mode of action of strontium is occlusion of tubular through replacing calcium in hydroxyapatite, thus forming strontium apatite.[25,26]

Davies et al,[27] have shown with in-vitro experiments that strontium-containing toothpaste was more durable than arginine-containing toothpaste against acids. However, controversial studies exist regarding the therapeutic effectiveness of strontium. Several clinical trials have reported a decline in DH pain when patients used strontium-containing pastes.[28,29] Although in-vitro studies have shown creation of small crystalline deposits on dentin surface by using strontium acetate or strontium chloride, this layer was easily washed away by water,[30,31] and acid.[32]

A systematic review of RCTs by Bae et al,[33] supported the use of potassium, stannous fluoride, calcium sodium phosphosilicate and arginine-containing toothpaste for the treatment of DH. However, in a systematic review by West et al,[20] the literature revealed that both strontium acetate and arginine have an unclear effect in terms of their ability to reduce DH.
One recent study, however, found that artificial silica abrasive (an element in these dentifrices) was actually responsible for occlusion of the dentinal tubules. Without further investigation of strontium, the clinical efficacy of this product remains uncertain.[34]

❖ **2.2. Fluoride**

Fluoride compounds (e.g., stannous fluoride and sodium fluoride) reduce the permeability of dentin by its ability to react with hydroxyapatite and form fluorapatite, which is considered less susceptible to acid dissolution than hydroxyapatite.[35]

In addition, fluoride induces calcium fluoride (CaF_2) to precipitate inside the dentinal tubules, thus occluding tubules and reducing the flow of fluid and subsequent nerve stimulation.[36,37] The in-vitro experiments, which showed that stannous fluoride leaves a deposit on the dentin surface that occludes the tubules have also investigated the mode of action for stannous fluoride. Importantly, it was shown that this deposit was water and acid-resistant.[30,38–41]

In a RCT, topical fluoride (1.23% sodium fluoride) has been shown to reduce postoperative sensitivity after bleaching procedures.[42]

The efficient treatment of DH using stannous fluoride products has been clinically demonstrated by RCTs.[43] The effectiveness of toothpaste-containing stannous fluoride (0.454%) in DH pain relief was investigated byHe et al,[44] Compared to the negative control (1000 ppm fluoride as sodium mono-fluoro-phosphate), they considered it to be superior and offered substantially better pain relief for sensitivity at four and eight-week time points.[44,45]

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E-ISSN: 2395-2822 | P-ISSN: 2395-2814
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When a stannous fluoride-containing toothpaste was compared to a positive control toothpaste containing arginine, the stannous toothpaste showed a substantial superior reduction in DH pain in both short- and long-term tests.[46] When stannous toothpaste-containing fluoride and arginine calcium carbonate were compared in both short-term and long-term studies, it was shown that stannous toothpaste fluoride had a significantly superior pain relief effect in the treatment of DH.[46,47]

When combined with other agents, Stannous seems to perform well. It has been shown that the efficacy of both stannous and potassium-containing toothpaste is often higher than potassium-only or negative control toothpaste.[12,21,48] However, the systematic review by West et al,[20] found that compared to conventional fluoride toothpaste, the quality of evidence supporting the effectiveness of stannous fluoride alone or in combination with potassium in reducing DH pain was moderate. A recent systematic review by Hu et al,[49] reported that toothpaste containing stannous fluoride alone or in combination with potassium are effective in reducing DH pain compared to negative control.

The major disadvantage of use of a stannous fluoride dentifrice is that it stains the teeth, the tongue,[50] and ceramic materials,[51] and that it takes a long time to be effective.

❖ 2.3. Oxalate

Oxalate products were first introduced for the treatment of DH in the late 1970s. They form calcium-oxalate crystals within the tubules, thus serving as desensitizing agents by occluding the tubules and decreasing the hydrodynamic fluid flow.[52] Oxalate products are relatively insoluble in acid, which makes them more resistant to dissolution.[53]
BisBlockTM (Bisco, Inc., Schaumburg, IL, USA) is an example of oxalate dentin desensitizer products available in the market for professional use. The product kit includes a phosphoric acid etchant to be used before the oxalate agent, and a dentin bonding agent to be applied following the oxalate agent to provide a seal over the crystals that are formed within the tubules and enhance durability.[54] Some oxalate products for home-use come as toothpaste or mouthwash.

The evidence showed that oxalates demonstrated inconsistent efficacy and ability to decrease DH compared to other agents. A mouthwash containing potassium oxalate has been shown to be efficient for occluding dentinal tubules in a study by Sharma et al.[55]
Oxalate decreased pain to an equal degree when compared to controls in some studies,[56–58] and less than the controls in other studies.[59,60] Oxalate has also been reported to be inferior to sodium chloride solutions in reducing DH.[61] A systematic review showed that oxalates have not been proven to be more effective than placebos in the treatment of DH.[62]

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However, new evidence of a self-administered oxalate product in the form of a gel strip is emerging. The use of these adhesive strips based on oxalate results in a substantial and durable crystalline deposition into the dentinal tubules. The use of oxalate in a gel strip has resulted in a 70% reduction in hydraulic conductance of fluid through dentin samples, which was still evident after 30 days.[63]

❖ 2.4. Lasers

Laser therapy was implemented as a possible option for treating DH. The nerve is assumed to be depolarized by lasers with low output-power (i.e., diode lasers and helium-neon lasers). Medium output-power lasers (i.e., Nd:YAG, CO2, and Er-YAG) are thought to minimize DH by narrowing dentinal tubules.[64]

Moreover, lasers work by coagulation of proteins in the dentinal fluid, thereby reducing permeability.[65] They are also believed to create an amorphous sealed layer at the dentin surface, which tends to be due to partial meltdown of the surface.[66]

Nevertheless, due to inadequate knowledge related to the irradiation standards and instructions, laser effectiveness and mechanism of action are questioned.[64]

However, the effectiveness of lasers has ranged from 5% to 100%.[67] In a systematic review, three RCTs demonstrated that the reduction was not significantly different from using a placebo.[64] Laser-induced pulp damage could also be a problem, although it has not been thoroughly investigated.[64]

A meta-analysis has reported that lasers Er:YAG and Nd:YAG are proving to be a successful alternative for treatment, but diversity in the data suggests the need for further studies.[68] Recently, it has been shown that diode laser application occluded 68.9% of all tubules surveyed, demonstrating its potential value in the treatment of DH.[69]

Further studies are needed to determine the mechanism by which the lasers minimize DH and the possible side effects associated with them.

❖ 2.5. Nanoparticles

Managing DH with nanoparticles is a new field of investigation.[70] This was proposed by the use of nanoparticles, such as calcium phosphate,[71] Silica,[72] and nano-hydroxyapatite.[73] The nanoparticles may penetrate dentinal tubules, thus, blocking fluid movement within the tubules.

In a RCT, a new dentifrice containing zinc-carbonate hydroxyapatite (CHA) nanocrystal was shown to significantly reduce DH after 4 and 8 weeks when compared to a commercially available potassium nitrate/fluoride dentifrice.[74] An in-vitro experiment, a mixture of nano-sized calcium oxide particles containing a mesoporous silica biomaterial (NCMS) and 30% phosphoric acid was placed on dentin. Significantly, NCMS demonstrated a reduction in dentin permeability under simulated pulpal pressure.[75] Applying nano-hydroxyapatite tooth

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Annals of International Medical and Dental Research
E-ISSN: 2395-2822 | P-ISSN: 2395-2814
Vol-7, Issue-3 | May-June, 2021
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bleaching reduced the number of days of sensitivity by half compared to the placebo group.[76] Nano-carbonate apatite toothpaste has been shown to be effective in occluding dentinal tubules compared to its control and strontium chloride.[77] The occlusion effect of nano-carbonate apatite in another in-vitro study was higher by 87% compared with its control group, which was 33% higher than the Er:YAG laser irradiation group.[78]

However, there are no sufficient RCTs and long-term trials to verify the capability of nanoparticles in the management of DH.

❖ 2.6. Polymer-based sealing materials

Professional application of polymer-based materials, such as adhesive resin bonding, primers, sealants, varnishes, and glass-ionomer cement, have been suggested for the treatment of DH by that mechanism of tubular occlusion.[79]

Fundamentally, it facilitates micro-mechanical interlocking through a process of hybridization. In addition to the presence of functional monomers, they have the ability to interact chemically with the residual hydroxyapatite calcium ions that remain present in the submicron hybrid layer.[80]

Several investigators confirmed the use of dentin adhesive products to relieve DH.[81,82] They have been reported to demonstrate varying degrees of efficacy for the DH treatment, with some evidence supporting their application,[57,83] and others suggesting that they are ineffective.[81,84]

A dentin bonding agent (Seal&Protect® by Dentsply Sirona) is a self-etching UDMA-based resin material produced specifically for sealing dentinal tubules. Some of its essential components are nano-fillers for reinforcement and improved abrasion resistance and Triclosan for an anti-bacterial effect.[85]

Acidity in the oral environment continues to be a major challenge that hinders the long-term dentin desensitizing effect.

❖ 2.7. Arginine

One of the challenges for most desensitizing agents using the occlusion technique with dentinal tubules is the inability to endure chemical and mechanical challenges in the oral environment. Therefore, they demand the development of new technologies focused on the stimulation of biological mineral development, i.e., induction of natural mineral formation and salivary-based products.[24,86]

An example is Pro-Argin technology, which is claimed to act through its binding to exposed dentinal tubules to initiate the formation of biological minerals.[24] Pro-Argin is an arginine-bicarbonate-calcium carbonate complex that simulates the natural process of DH reduction.[86]

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Arginine is a simple amino acid, naturally found in the free form of saliva, as well as in the salivary peptides and proteins that are known to be its primary source.[87] It acts in combination with calcium carbonate and phosphate to create a plug in dentinal tubules with low solubility.[18,24,86]

Arginine has a well-established efficacy in raising the surrounding pH by being metabolized by alkali-producing oral bacteria. This results in a favorable environment for the mineralization of the tooth structure, particularly if it is assisted by the availability of calcium.[86]

The suggested mechanism of action claims that arginine is attracted to dentin and that alkaline pH facilitates the deposition of calcium, phosphate, arginine and carbonate on the dentin surface and within the dentinal tubules.[88]

Clinical trials have shown the efficacy of desensitizing arginine-containing products. Multiple clinical trials evaluating the use of arginine and calcium carbonate toothpaste with fluoride (1450 ppm) over a one-week span have shown good treatment results that give immediate pain relief following treatment compared to 2% potassium-containing toothpaste.[89–91]
Arginine has also been shown to be substantially and reliably more effective in reducing sensitivity in medium term trials, up to 8 weeks.[18,92] In addition, a study showed that the use of arginine/calcium carbonate-containing paste substantially decreased DH within one to two weeks of starting treatment and continued to reduce DH after the 11thweek of treatment.[93]

A study found that a single professional application of 8.0% arginine-containing paste and calcium carbonate showed an instant sensitivity relief that lasted for 28 days and was significantly better than the control of pumice prophylaxis paste.[94] Moreover, systematic reviews have indicated that arginine/calcium carbonate is an important agent for the reduction of DH and can be considered a gold standard for the treatment of DH.[24,95,96]

❖ 2.8. Calcium sodium phosphor-silicate (CSPS)

Incorporation of calcium sodium phosphor-silicate (CSPS) into toothpastes have also been reported to be effective in the DH treatment. The in-vitro investigations have shown tubule occlusion created by the use of calcium, phosphate, and silica together. Also, they tend to be water and acid-resistant. CSPS precipitates onto dentin collagen as calcium phosphate and silicate, forming deposits both on the dentin surface and within the dentinal tubules.[95,97]
A RCT that compared the effectiveness of CSPS, potassium nitrate and stannous fluoride in the treatment of DH revealed that while all three agents provided clinical efficacy, a significant and substantial improvement in pain relief was demonstrated by CSPS.[17]

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Furthermore, several RCTs have shown a statistically significant benefit of using CSPS when compared to a negative control.[16,98] A more recent study provided evidence that CSPS containing toothpaste may both mineralize dentin and occlude dentinal tubules and that these blockages are capable of adapting dietary acid challenges.[99]

CONCLUSION

- Several current methods have demonstrated their potential for treating or controlling DH, but their clinical effectiveness is still uncertain in terms of durability and safety. Thus, there is no "gold standard" treatment for DH.
- Most of the treatment modalities of DH are not satisfactory in the long-term, and re-treatment may be needed.
- This requires a well-designed comparative efficacy study and a long-term practice review to provide dentists and patients with reliable scientific evidence to assess the effectiveness and safety of alternative treatment methods in the management of DH among various available treatments.

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Source of Support: Nil, Conflict of Interest: None declared

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