Review

Successful management of dentin hypersensitivity: A narrative review

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Abstract

The prevalence of dentin hypersensitivity (DH) is increasing around the world. At least one in 10 individuals in the general population has been diagnosed with DH. It is a diagnosis that has significant negative effects on a person's oral health-related quality of life. This condition, which is characterized by sharp, short tooth pain in response to thermal, chemical, tactile, and evaporative stimuli, is more commonly seen in adults. DH has a tremendous impact on the social and financial aspects of patients and society at large. It is essential to recognize the factors that can contribute to a successful treatment outcome to guarantee the overall well-being of DH patients.

The aim of this narrative review was to highlight strategies that can lead to successful DH treatment outcomes, along with current updates on DH mechanisms, treatment options, and the latest management approaches. A positive treatment outcome for DH requires a concerted effort from both the patient and the dental practitioner. Highly motivated patients and dental practitioners with sound knowledge of DH diagnosis and available treatment options will ensure successful long-term improvement of DH symptoms.

Keywords: therapy, treatment outcome, pain, dentin hypersensitivity

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Introduction

Tooth sensitivity, which is clinically referred to as dentin hypersensitivity (DH), is becoming increasingly more common in dental practice.[1–3] DH is described as short, sharp pain arising from the teeth in response to hot and cold temperatures and evaporative, tactile, osmotic, and chemical stimuli. It cannot be attributed to any dental defects or disease.[4] At least one in 10 individuals in the general population suffers from DH, which causes discomfort and pain and compromises quality of life.[3,5–7] The prevalence of DH ranges from as low as 1.3% to as high as 92.1%, depending on sociodemographic characteristics, recruitment strategies, and number of study sites involved.[1] DH is more prevalent in adults and imposes significant impacts on social and financial aspects for patients and society at large.[1,8,9] Understanding DH and identifying the factors that contribute to successful treatment outcomes are essential to ensure not only improvement of DH patients' oral health condition but their overall well-being. Thus, the aim of this narrative review was to highlight strategies for successful DH treatment outcomes, along with current updates on DH mechanisms, treatment options, and the latest management protocols.

Material and methods

A computerized literature search on DH management was performed using the PubMed and Google Scholar databases. The keywords searched included "dentin hypersensitivity management", "dentin hypersensitivity treatment", "dentin hypersensitivity prevention", "dental pain", "therapy for dentin hypersensitivity", and "dentin hypersensitivity and treatment outcome". The articles included in this review were from 2011 to 2021. Moreover, some earlier references were also cited according to their relevance to current research progress on the management of DH. All relevant original articles, review articles, and case reports available in full text and published in English were included.

Pathophysiology and mechanisms of pain in dentin hypersensitivity

Dentin is the middle layer of the tooth that makes up the bulk of the tooth's structure. It is composed of closely packed dentinal tubules that are aligned throughout its thickness and contain odontoblastic processes. The odontoblast cell bodies that synthesize dentin are aligned at the inner dentinal layer, which indirectly forms the boundary of the dentin-pulp complex. Under normal conditions, dentin is protected from the external oral environment by the outermost mineralized enamel layer (for coronal dentin) or cementum (for cervical dentin). The innermost layer is composed of pulpal tissue where the primary dental afferents are found. Dentin loses its protection when the enamel layer disappears or when gingival tissue recedes, exposing the dentin layer and the dentinal tubules within. These structural changes can result from acidic food intake, overzealous dental hygiene practices, and parafunctional habits.[2,10] The discomfort and pain associated with DH develop as a result of these activities; however, as pain signaling requires stimulation of the free nerve endings on the primary afferent neurons and that dentin is lacking neuronal fibers,[11] the exact mechanism to explain this common intraoral condition remains elusive.

Three theories have been proposed to explain the mechanism of pain in DH: the hydrodynamic, direct innervation, and odontoblast transducer theories (Fig. 1). The hydrodynamic theory proposed by Braennstroem and Astroem[12] remains the most widely accepted mechanism.

The neural theory was among the earliest DH theories brought forward. Indeed, the involvement of nerve fibers in pain signaling is an ideal hypothesis to explain DH, as it has already served as a well-established

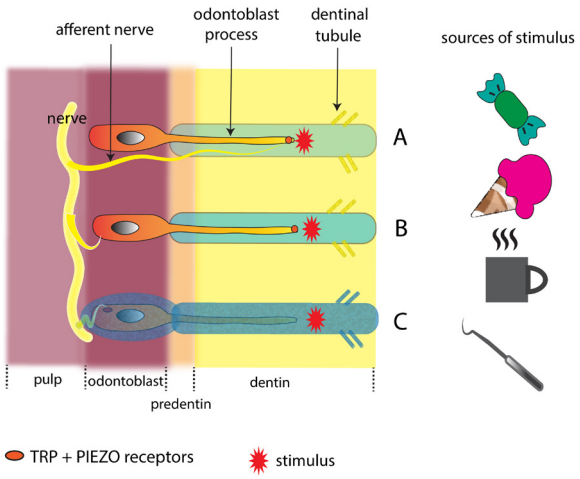

Fig. 1. Schematic diagram on theories of dentin hypersensitivity (DH)
Three theories have been proposed to explain the mechanism of pain in DH: A – The neural theory assuming the involvement of nerve fibers in pain signaling. This theory proposed that nerve fibers from the dentin–pulp junction project to the inner dentinal layer, which enables them to detect external stimuli; B – The odontoblast transducer theory by Rapp et al. (1968)[15] proposed that at the dentin–pulp junction, odontoblasts serve as sensory cells that are activated by increased intratubular fluid movement. They convert this movement into electrical signals, which are passed to synapses with primary dental afferent nerve endings; C – The hydrodynamic theory by Braennstroem and Astroem (1964)[12] is the most widely accepted mechanism. The dentinal pain is associated with fluid movement in dentinal tubules; this dentinal fluid movement excites both thermosensitive and mechanosensitive C- and Aδ-nociceptors of the primary dental afferents present at the dentin–pulp junction, activating the dental pain signaling pathway.

explanation for pain mechanisms in other parts of the body. However, there are conflicting reports about the presence of nerve fibers in dentin and dentinal tubules; more studies support the absence of nerve fibers within dentin and dentinal tubules.[11,13,14] In this regard, state-of-the-art 3D imaging techniques – CLARITY and light sheet microscopy – have enabled imaging of intact tissues with high resolution and preserved anatomical structures.[14] Using fluorescent dyes, the initial findings of this technique revealed pronounced nerve bundles and their branches running along the longitudinal axis of the root from the apical foramina toward the coronal pulp with terminal endings close to the dental pulp periphery.[14] However, as this study only involved imaging of the pulp tissue, extension of the peripheral terminals (whether or not they reach the periphery of the dentin) has yet to be determined. Future studies using CLARITY and light sheet microscopy that preserve the dentin-pulp complex could resolve the conflicting findings on the extension and presence of nerve terminals into the dentin.

Another theory, the odontoblast transducer theory, was proposed by Rapp et al.[15] They hypothesized that odontoblasts act as sensory cells that are triggered by increased intratubular fluid movement, transducing this movement into electrical signals and synapsing with nearby primary dental afferent nerve endings at the dentin-pulp junction. This posited role of odontoblasts as sensory cells probably stems from their embryogenic origin; they are derived from the ectomesenchyme cells of the neural crest,[16] similar to neurons.[17] Recent research has revealed that mechanosensitive and thermosensitive receptor channels (Piezo channels) and several members of the transient receptor potential superfamily ion channels are expressed in odontoblasts,[18,19] which further supports the odontoblast transducer theory.

The concept of the hydrodynamic theory was proposed by Gysi, when he explained that dentinal pain is associated with fluid movement in the dentinal tubules.[20] However, it was only in the 1960s that Braennstroem and Astroem were able to demonstrate that when mechanical stimulation, cold and hot temperatures, and osmotic pressure (caused by sugar or salt intake) were introduced at the site of exposed dentin, there was an increase in fluid movement into the dentinal tubules; this led to DH.[12] Brännström proposed this mechanism as the hydrodynamic theory.[21] Further studies have demonstrated that this increased fluid movement into the dentinal tubules excites both C- and Aδ-fibers in the dentin–pulp junction, which in turn activates the dental pain signaling pathway.[22,23] The expression of thermosensitive and mechanosensitive receptors, as well as voltage-gated ion channels involved in pain signaling in the primary dental afferent neurons,[19] further supports the hydrodynamic theory.

Management of dentin hypersensitivity

Preventive measures for dentin hypersensitivity

There is a well-known phrase: Prevention is better than cure. The search for advances toward better management of DH is ongoing, and better modes of treatment are becoming available. However, to date, the gold standard for DH that provides long-lasting and sustained improvement of the hypersensitivity condition has yet to be discovered. Among the many predisposing factors and etiologies for DH, incorrect tooth brushing technique is cited as the most common cause of DH.[6,24] Thus, dental education is essential to prevent DH and provide better oral health and overall well-being of the patient. Because DH may result from inappropriate/incorrect oral hygiene practices, educating the public on correct dental hygiene techniques may help prevent or improve DH. In addition, recommending healthier dietary habits to reduce or avoid acidic foods and beverages should be emphasized. Furthermore, destructive parafunctional habits, such as tooth grinding, which may result in erosive tooth structure loss, should be discouraged.

Diagnosis of dentin hypersensitivity

The diagnosis of DH is very subjective and difficult to establish despite several DH guidelines already published and available online.[4,24,25] The difficulty in diagnosing DH may partially be due to the numerous dental conditions that can present with symptoms similar to those of DH.[24] For this reason, DH is considered a diagnosis of exclusion—only when the signs and symptoms fail to meet the criteria of any other oral conditions or diseases can the diagnosis of DH be made. The sharp, sudden pain experienced upon consuming hot, cold, and sweet foods and beverages is a common complaint for both DH and reversible pulpitis. To a lesser extent, the symptoms of DH may also mimic those associated with tooth crack syndrome involving enamel or dentin, dental caries, post-bleaching/whitening dental hypersensitivity, and periodontal disease and its treatment.[26] As some patients may confuse the pain of DH with other types of pain, making an accurate diagnosis of DH requires a significant amount of time to take a proper and thorough history regarding the chief complaints and to conduct a complete intraoral clinical examination. Certain tests might also be required to exclude the other aforementioned oral conditions that mimic the symptoms of DH. Thus, a complete history and clinical examination are vitally important to obtain a definitive diagnosis of DH.

Since several common oral conditions can cause dental pain, distinguishing these conditions from DH is essential before a diagnosis of DH can be made. Some of the techniques that are commonly used to exclude the diagnosis of DH include eliciting a pain response upon percussion of the affected teeth (which would indicate periodontal or pulpal involvement), pain upon biting a tongue depressor, and the use of transilluminating lights or dyes (which would indicate tooth fractures). The presence of recent defective restorations may also be the source of pain. However, DH usually occurs at the area of gingival recession that is associated with loss of cementum on the root surface.[27]

After these common dental conditions that cause pain have been excluded, the diagnosis of DH can be confirmed by means of exposing the dentinal tubules to stimuli that mimic those that trigger DH. Some of the diagnostic tools that are commonly used for this purpose include the use of an air or cold water jet, thermal testing, electrical devices, a dental explorer, a periodontal probe, radiographs, caries diagnostic devices, percussion testing, assessment of occlusion, and the bite stress test.[6,28]

Acknowledging a patient's complaints is the first step toward making a successful diagnosis and managing DH. Despite the high prevalence of DH, dental practitioners do not routinely assess their patients for this condition and rely more on the patient to self-report it.[6] Surprisingly, some dental practitioners even have a lack of confidence in diagnosing DH,[4] which could explain the low prevalence of DH in certain regions.

Soares et al. assessed the quality of life of people diagnosed with DH and concluded that DH was associated with a significantly high oral health impact.[3] In particular, the oral health impact was highest in terms of psychosocial discomfort, followed by physical pain, psychological disability, physical disability, social disability, and functional limitations. Thus, managing DH not only resolves patients' complaints but, more importantly, improves the physical and psychosocial aspects of their lives.

Treatment of dentin hypersensitivity

Since DH is caused by exposed dentinal tubules, occluding these tubules is the foundation of any therapeutic agents developed for DH treatment. Several treatment modalities are available for DH. They are classified by their mechanism of action – physical and chemical occlusion of the dentinal tubules, nerve desensitization, and photobiomodulation.

Physical occlusion of dentinal tubules

Physical occlusion of dentinal tubules is achieved by incorporating particles or nanoparticles into the dentinal tubules. Sources of particles that are commonly used for the physical occlusion of dentinal tubules include hydroxyapatite and bioactive glass. Hydroxyapatite composes approximately 70% of dentin's structure; thus, its use as a bone substitute and for tooth remineralization has shown promising results due to its biocompatibility and because it is nontoxic and harmless to human biological tissues.[29,30] Hydroxyapatite has been incorporated in desensitizing toothpaste as an agent to promote the repair of exposed dentinal tubules. Research is ongoing to further improve the effectiveness of hydroxyapatite in the management of DH.[31–33] Unlike hydroxyapatite, bioactive glass is an exogenous material that releases calcium and phosphate ions upon contact with fluids, such as saliva and water during drinking. These ions subsequently crystalize into hydroxycarbonate apatite to occlude the dentinal tubules.[34–36] Similar to hydroxyapatite, bioactive glass shows promising results as a bone substitute and for dentin remineralization, particularly due to its improved durability.[36–39]

Chemical occlusion of dentinal tubules

It has been widely reported that exposed dentinal tubules can also be treated with chemical agents, such as fluoride. Fluoride has long been known to play a vital role in preventing dental caries. Fluoride ions bind to calcium and phosphate by replacing the hydroxyl ion in the hydroxyapatite crystal structure, thereby forming a stronger enamel structure. In the management of DH, the use of desensitizing fluoride toothpaste has been recommended as the first-line noninvasive treatment.[4] Fluoride relieves DH by forming calcium-phosphate precipitates, calcium fluoride, and fluorapatite within the dentinal tubules, which prevents the intratubular fluid movement that would otherwise be triggered by the external stimuli that cause DH.[40–42] Other agents that regenerate hydroxyapatite similar to fluoride include oxalate, which is also commonly used in toothpaste and mouthwash for indirect occlusion of dentinal tubules.[43–45] Although chemical occlusion of dentinal tubules using fluoride, nitrate, and oxalate is common, its effects are not immediate and less efficient in withstanding acid challenges and mechanical stress.[32,46,47]

Nerve desensitization

Considering the proposed theory of nerve fiber involvement, nerve desensitization by means of applying potassium salts has become another treatment option for DH. Different formulations of potassium salts have been widely used in the management of DH. The use of potassium salts in DH was first introduced by Hodosh, whose clinical trial demonstrated that the use of 1–15% potassium nitrate (KNO3) was able to mitigate DH symptoms.[48] The mechanism of action of potassium salts in reducing

DH symptoms in vitro and in human subjects has been discussed in depth elsewhere.[49,50] Briefly, increasing the concentration of potassium ions in the extracellular fluid around the peripheral dental afferent neurons depolarizes the nerve terminals and subsequently blocks conduction of an action potential.[50] Consequently, less pain is perceived in the brain, which improves the symptoms of DH. A systematic review and follow-up analysis on the effectiveness of different treatments for DH showed that the use of potassium nitrate desensitizing agents resulted in a significant long-term reduction in DH symptoms.[51]

Photobiomodulation

The concept of photobiomodulation for the treatment of DH was introduced by Matsumoto in the 1980s, who originally used neodymium-doped yttrium aluminum garnet (Nd:YAG) laser to treat cervical hypersensitivity.[52] This method is currently the most advanced technique for the treatment of DH. Nd:YAG is delivered by directing lasers of different wavelengths (ranging from ~600–800 nm) and laser powers (35–100 mW)[53] onto the exposed dentin.

In general, there are two types of photobiomodulation treatment depending on the laser power used for the treatment: low-level and high-level laser power. Low-power lasers such as gallium-aluminum-arsenide laser (GaAlAs) offer pain control in DH by increasing the production of mitochondrial ATP and increasing the threshold of the free nerve endings.[54] High-power lasers, such as carbon dioxide (CO_2), Nd:YAG, erbium-doped yttrium aluminum garnet (Er:YAG), and erbium, chromium-doped yttrium scandium gallium garnet (Er,Cr:YSGG), obliterate the dentinal tubules by inducing the formation of secondary and tertiary dentin produced by odontoblasts.[53,55] Studies have shown that when applied correctly, photobiomodulation with both low- and high-power lasers provides therapeutic relief for the treatment of DH without any detrimental effects to the pulp and dentin.[53,56]

Other treatment options

Depending on the clinical evaluation and assessment, DH can be managed with restoration when there is loss of tooth structure, such as due to abrasion. An appropriate choice of restorative materials should be used, or a root coverage procedure for DH caused by gingival recession can be performed. A systematic review of the effectiveness of surgical root coverage found reduced cervical DH following surgery[57]; however, more clinical studies are still required before surgical intervention is recommended for the management of cervical DH. In this regard, more recent clinical studies have demonstrated promising outcomes using a gingival flap combined with connective tissue grafts to augment and promote root coverage.[58–60]

Management of dentin hypersensitivity: How can successful outcomes be achieved?

Failure to treat its symptoms significantly affects the quality of life of patients suffering from DH. As prolonged hypersensitivity is manifested as pain, it may disturb an individual's daily activities, eating, drinking, and socializing. The resulting disruption of the patient's normal lifestyle may lead to the decision to remove the tooth causing the problem. Tooth loss may lead to even more functional impairment, as well as esthetic problems, depending on the location of the tooth. Depending on the type of materials used, tooth replacement can be a financial burden; thus, the vicious cycle of financial and psychosocial burdens continues. Identifying the factors that may lead to DH management failure may save a tooth from being removed and liberate the patient from a poor quality of life. Failure of DH management can be attributed to several factors, including dental practitioner factors,[4,24,61] patient factors,[62,63] and the methods and materials used.[51,64,65]

Dental practitioner

Making the correct diagnosis

Failure to identify the causative factor may lead to unsuccessful treatment of DH. Some dental practitioners overlook DH and manage their patients' complaints with invasive procedures. Mismanagement and misdiagnosis of DH cases may lead to irreversible loss of tooth structure and pulp vitality, or even tooth loss,[66] which could be prevented if an accurate diagnosis is made and the correct management performed. Dental practitioners should take a thorough history and perform a complete intraoral examination. This step is essential to establish the correct diagnosis, which must be achieved before treatment is initiated in patients.

Identifying factors that contribute to dentin hypersensitivity

It is now known that oral hygiene practices and dietary habits are significant etiologic factors that predispose individuals to DH. In addition to these well-accepted and well-known scientifically proven causes, an emerging association of DH with medication use has been reported. A recently published case report suggested an association between DH and steroid use. In this case report, a 47-year-old woman complained of symptoms similar to DH, i.e., generalized pain from all teeth, which was triggered by hot, cold and sweet foods, and beverages. The patient was on steroid treatment for her medical condition, and tapering and discontinuing the steroids improved her DH-like pain symptoms,[67] thus indicating that the DH was related

to the steroids. Prior to this case report, a survey of 220 subjects was conducted to study the association between steroid use and DH. Surprisingly, almost 20% of the subjects reported DH-like symptoms similar to those in the aforementioned case report; the pain symptoms subsided following tapering and discontinuing of the steroids.[68]

In another study conducted by Farag and Awooda, 40 asthmatic patients who presented to an emergency department and were diagnosed with an acute asthmatic attack were monitored for DH and dental erosions.[69] The authors found significantly more DH in the asthmatic patients compared to the non-asthmatic patients. Studies have shown that the use of inhalers in asthmatic patients decreases salivary flow and salivary composition, which in turn reduces the buffering effect of saliva that protects tooth structures from erosion.[70–72] In this regard, the medications used to treat asthma, which include inhaled Beta2 agonists and inhaled steroids, may play a role in these salivary changes. For example, Beta2 agonists have been shown to impair saliva production[73] and lower esophageal sphincter function, which leads to gastroesophageal reflux.[74,75] Several studies, including a recent meta-analysis of clinical studies from 2012 to 2020, have suggested that gastroesophageal reflux is a risk factor for dental erosions.[76] The low pH of most inhalers also contributes to dental erosions and DH in asthmatic patients.[73] There is still limited literature about the association between DH and medical conditions and the use of certain medications. More studies are required to confirm this association and the possible mechanism that could link DH to medication use and/or other diseases.

Appropriate management

Similar to the management of other diseases, managing DH requires a holistic approach. Patients need to be motivated to change the habits that contribute to DH, and dental practitioners should consult with patients and develop appropriate and effective treatment plans. To ensure treatment success, both patients and dental practitioners must work together to eliminate the causative factors of DH and improve the discomfort and pain associated with this condition. Lack of commitment from either party—patient or dental practitioner—will lead to further tooth structure damage, for which some dental practitioners resort to a root canal.[77] Dental practitioners should keep themselves aware and updated about current advances in DH treatment. The choice of treatment options should consider the patient's conditions. Patients need to be evaluated with respect to their desire to improve their health and their motivation to achieve their treatment goals in order to ensure successful treatment. The initial management of DH often begins with patient education about the etiology and contributing factors, followed by in-office treatment in which chemical agents are professionally applied. Continuation with in-home treatment, which is cheaper and more convenient for the patient, is highly recommended to maintain the reduction of DH symptoms. However, when there is no improvement in symptoms, more advanced treatments, such as tooth restoration, root canal therapy, or even surgery, may be required. Dental practitioners need to assess and evaluate the most effective treatment methods and whether they should be provided by themselves or in combination with other treatments. Studies have shown that all types of treatment (physical occlusion, chemical occlusion, nerve desensitization, and photobiomodulation) significantly improved pain symptoms in DH patients compared to placebo group patients. However, there is no consensus in the literature regarding the effectiveness of one method compared to the others;[64,65,78–81] this divergence may be attributed to confounding factors, such as the type of study (in vivo or in vitro), study population, and study methods.

Patient habits and motivation

There are several risk factors for DH, including loss of tooth structure by erosion, attrition, abrasion or abfraction, bruxism, gingival recession, occlusal trauma, and abnormal tooth position.[82] These risk factors, in turn, are closely linked to the patient's dietary habits, such as frequent consumption of fruit juice, soft drinks, and alcohol, and tooth brushing habits, which include method of tooth brushing, type of toothbrush and toothpaste, and frequency of tooth brushing.[2]

In this regard, compliance and adherence to instructions are of paramount importance to ensure the success of DH treatment. Compliance and adherence are two different terms that are sometimes confused as having the same meaning. Compliance is defined as the consistency and accuracy with which a patient follows any regimen prescribed by a dentist, physician, or other healthcare professional.[83] Compliance does not reflect a stable state of the patient abiding by the healthcare provider's instructions. Therefore, compliance is complemented by adherence, which is defined as the extent to which the patient continues with the agreed-upon mode of treatment under limited supervision when faced with conflicting demands.[84] Thus, patients play a principal role in the treatment and management of their sensitive teeth, so they need to take control of their own efforts to modify unhealthy behaviors in order to reduce or eliminate DH symptoms. In this regard, dental professionals play a vital role in educating their patients and emphasizing the importance of compliance and adherence in changing behaviors and achieving a successful treatment outcome.

A patient's lack of motivation to comply and adhere to instructions provided by clinicians during therapy has been recognized as one of the main problems that prevent successful therapy. In their book chapter "Advances in the management of the patient with dentine hypersensitivity: Motivation and prevention", Gillam and Ramseier

explain the challenges of modifying health behaviors in the management of dental conditions and outline their recommendations for managing DH using a motivational interviewing technique.[62] Motivational interviewing is defined as "a client (patient)-centered, directive method for enhancing intrinsic motivation to change by exploring and resolving ambivalence".[85] This technique helps patients recognize the importance of changing the detrimental habits that contribute to their DH and take ownership and responsibility of their problems through improvement of oral hygiene and dietary habits, i.e., by reducing acidic food and beverage consumption.[62]

While it may be unrealistic to expect patients to understand the professional advice on DH management, this can be accomplished by a few visits to a dental clinic. Although frequent dental visits may be inconvenient for patients, establishing good rapport during the first visit may help develop an effective dental practitioner-patient relationship and improve the patient's willingness to attend dental appointments.

Method chosen for the management of dentin hypersensitivity

A meta-analysis of the use of three DH management methods – physical occlusion, chemical occlusion, and nerve desensitization – for in-office and at-home treatment showed that only chemical occlusion and nerve desensitization were effective for at-home treatment. For in-office treatment, all three methods were very effective in managing DH compared to a placebo group.[80] A systematic review and network meta-analysis of in-office treatments for DH concluded that all methods of treatment were effective in reducing DH symptoms. The author reported no significant difference in the efficacy of the methods used to manage DH.[78]

Materials used for the management of dentin hypersensitivity

Regardless of the type of materials used to improve DH symptoms—by occlusion of dentinal tubules, nerve desensitization, or photobiomodulation—several factors need to be considered to ensure treatment success. These factors include, but are not limited to, physical properties, potential for microleakage, and durability of the materials. These materials should be able to withstand acidic environments (diet) and mechanical removal (tooth brushing) as well as avoid salivary clearance (dissolution in saliva).

A meta-analysis of the use of toothpaste as a desensitizing agent for DH found that incorporating potassium, stannous fluoride, potassium and strontium, potassium and stannous fluoride, calcium sodium phosphosilicate, arginine, and nanohydroxyapatite is effective in reducing sensitivity and improving symptoms compared to patients who used toothpaste without active ingredients.[86] However, the results may be biased in terms of the effectiveness of these active ingredients, as the studies had variable degrees of bias; some of them were funded by the manufacturers of the toothpastes studied.

Managing DH with photobiomodulation has gained considerable attention and is becoming more popular among dental practitioners. Many studies comparing lasers with other DH treatments support the use of lasers as being as effective as other techniques, including nerve desensitization and chemical and physical occlusion.[56,64,87,88] Several other studies have reported that combining laser therapy with any other method provides better outcomes for successful DH treatment.[76,79,89]

Suri et al. compared physical occlusion using 5% sodium fluoride with 980 nm diode laser treatment at one-week, one-month, and two-month intervals.[79] They determined that both methods improved DH symptoms, with no significant difference between methods. However, the combined use of 5% sodium fluoride and laser improved DH symptoms more than sodium fluoride or laser alone.[79]

Praveen et al. studied the application of glutaraldehyde topical desensitizer and compared it with low-level GaAlAs laser.[64] They concluded that both methods reduced DH immediately and after one week, but only GaAlAs laser improved symptoms after both one week and three months.[64] Interestingly, a clinical study conducted by Sgreccia et al. comparing potassium oxalate with low-level GaAlA laser treatment concluded that occluding dentinal tubules with potassium oxalate provided relief of DH symptoms immediately after the first application and after three weeks (with four applications), while laser was only effective in reducing cervical DH after three weeks.[90] These conflicting results may be due to the shorter duration of study by Sgreccia et al. compared to the other studies.

Lopes et al. assessed different DH treatment protocols that included low-level laser (with different doses), high-level laser, and desensitizing agents.[91] They studied 32 patients with 117 lesions who underwent one of the following DH protocols: low-power laser alone, high-power laser alone, or low-power laser combined with a desensitizing agent (Gluma desensitizer). The level of sensitivity was measured using a visual analog scale (VAS) for pain with a triple syringe and an exploration probe. The assessments were performed at intervals of five minutes, 12 months, and 18 months. At the end of their study, they found that all treatment protocols significantly reduced DH but that there was no significant difference in VAS scores between the different protocols used. Thus, they concluded that low-power laser, high-power laser, and low-power laser combined with a desensitizing agent were equally effective in reducing DH symptoms.

The photobiomodulation method used in the treatment of DH does not cause damage to the pulp tissue despite a slight rise in intrapulpal temperature, as reported in the systemic review of photobiomodulation effectiveness on

DH by Machado et al.[53] Photobiomodulation has further been proven to improve the oral health-related quality of life of DH patients.[92]

Although a large body of evidence supports the use of photobiomodulation as superior to other DH treatments, given alone or in combination with other methods, many confounding factors affect its clinical success. A recent systematic review and meta-analysis of photobiomodulation parameters determined that the clinical outcome depends on variations in anatomy, site location, clinical condition, and subject individuality.[54] To achieve a better prognosis and successful treatment outcome, it is essential that these factors, combined with sound knowledge and understanding of laser parameters, tissue volume, and target depth to deliver an adequate dose, are considered during treatment planning.[54]

Photobiomodulation is known to be a safe nonsurgical therapy for DH that does not cause significant tissue hyperthermia. A recent randomized clinical trial reported a slight rise in intrapulpal temperature (<5°C) following two diode laser protocols—1 W diode laser for 10 seconds or 0.5 W diode laser for 60 seconds.[81] Despite only a small rise in temperature, dental practitioners should be cautious when performing photobiomodulation for DH as any damage to pulp tissue can compromise tooth vitality and increase pain instead of relieve it.

Conclusions

DH is highly prevalent, especially in females. DH significantly affects the quality of life of many patients, which can impact their psychological and social well-being. Management of DH with a sound treatment plan is crucial to ensure successful treatment outcomes, which will improve the patient's oral health condition and quality of life. This review provides an update on the mechanisms of DH and its management and emphasizes the factors that determine the success of DH treatment. Pharmacological management of DH using desensitizing agents will only be successful if there is a concerted effort from both the dental practitioner and the patient.

Ethics approval and consent to participate

Not applicable.

Data availability

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Consent for publication

Not applicable.

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