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Dentin Hypersensitivity: A Literature Review

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Abstract: Dentin hypersensitivity is characterized by brief, intense pain triggered by external stimuli that affect exposed dentin with open tubules. The condition is primarily caused by two factors: the exposure and loss of dentin and the opening of dentin tubules, which allows sensory signals from the pulp to be transmitted in response to stimulation of the dentin surface. A precise diagnosis is crucial before initiating treatment and begins with a thorough patient medical history and clinical examination. Various treatment options have been developed to manage sensitivity, focusing primarily on nerve desensitization and the closure of open dentinal tubules. Developing a better understanding of the causes and mechanisms of dentin hypersensitivity is essential for effective prevention and treatment strategies for patients suffering from this condition. This review aims to provide dental professionals with comprehensive knowledge about dentin hypersensitivity, including its causes, diagnostic procedures, clinical management techniques, and innovative methods for reducing sensitivity.

Keywords: Dentin hypersensitivity; Exposed dentin; External stimuli

Introduction

Dentin hypersensitivity (DHS) is an exaggerated response to stimuli that typically do not provoke a response in healthy teeth or an overreaction to non-harmful stimuli. These non-harmful stimuli include thermal, tactile, osmotic (Barcellos et al., 2012), evaporative, chemical (Mantzourani et al., 2013), and mechanical stimuli such as tooth brushing, sweet or acidic foods, as well as hot and cold water (Yu et al., 2014). When these stimuli come into contact with exposed dentin, they can cause pain without any pathological changes in the dentin-pulp complex.

Dentin hypersensitivity is a global clinical problem, especially among adults in their thirties and forties (Ishihata et al., 2012). Clinically, DHS is characterized by brief, sharp, and distinct pain with a rapid onset. The pain can be localized or generalized, affecting one or several tooth surfaces simultaneously. The definition of DHS has two aspects: the first describes the clinical presentation, while the second identifies the condition by excluding other pathological conditions (Mantzourani et al., 2013). Dentin hypersensitivity was first reported by Blum in 1530 (N. West et al., 2013).

Dentin hypersensitivity is reported to affect 8-30% of the adult population, with the most common age of patients being around 20 to 30 years. The first premolar is the most frequently affected tooth in more than half of cases, and the most commonly affected region is the cervical area on the buccal surface (Barcellos et al., 2012). According to Yu et al. (2014), the order of preference for the occurrence of DHS is from canine teeth, first premolars, incisors, second premolars, to molars. The prevalence of DHS in the mandibular premolars and canines is higher. The probable cause of this sensitivity is the position of the teeth in the jaw, which exposes the teeth to tooth brushing with more force; therefore, the probability of gingival recession and the loss of hard tissue covering the teeth is higher in these teeth (Mahdisiar et al., 2019). Recently reported prevalence

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rates of DHS are 34.5% in China, 46% in Brazil, 32% in India, and 41.9% in seven countries in Europe (Mehta et al., 2015). Understanding the etiology, morphology of dentinal tubules, mechanisms of dentin hypersensitivity, and diagnosis aids in the management of dentin hypersensitivity.

Method

The literature search was conducted across several primary electronic databases, including PubMed, Scopus, and Google Scholar. These sources were selected due to their extensive coverage and high relevance to the fields of medicine and dentistry. Articles included in this review met the following criteria: published in English or Indonesian, directly related to dentin hypersensitivity, including its etiology, pathophysiology, diagnosis, and management, original studies, systematic reviews, meta-analyses, and relevant case reports. Exclusion criteria encompassed: opinion pieces, editorials, or letters to the editor, studies that were inaccessible in full text or did not meet adequate methodological standards. The literature search employed a combination of keywords such as "dentin hypersensitivity", "etiology of dentin hypersensitivity", "treatment of dentin hypersensitivity", and "diagnosis of dentin hypersensitivity". The search strategy followed the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines to ensure transparency and reproducibility. After initial screening of titles and abstracts, full-text articles were reviewed to confirm eligibility based on the inclusion and exclusion criteria.

Result and Discussion

Etiology of Dentin Hypersensitivity

Current knowledge of the etiology of dentin hypersensitivity (DHS) is still limited, and the factors that initiate the exposure of dentin are still uncertain. Dentin in a normal condition is covered by enamel and cementum and does not show sensitivity to external stimuli. Dentin will begin to show sensitivity only when it is exposed and comes into contact with the oral cavity environment (Barcellos et al., 2012).

Dentin exposure can be caused by physical, chemical, pathological, biological challenges and/or developmental abnormalities that result in dental and/or periodontal damage or defects (Liu et al., 2020). Various clinical conditions thought to play a role in the development of DHS include enamel attrition and erosion, abrasion and abfraction (Mantzouri et al., 2013). Periodontal tissue loss or gingival recession is another major predisposing factor since this leads to exposure of cervical and root dentin (N. X. West et al., 2013). The loss of soft tissue (gingival recession) is caused by the anatomy of the buccal alveolar bone, poor oral hygiene, and orthodontic movements (Dalmolin et al., 2023).

A high diet in acidic liquids and foods, acid exposure, the use of bleaching agents, and gastric reflux can be implicated as causes of dental erosion (Farooq et al., 2013). Aggressive and frequent tooth brushing and periodontal treatments such as scaling and root planing can also contribute to gingival recession (Farooq et al., 2013), loss of cementum, and dentin exposure (Cunha-Cruz et al., 2013). Harmful habits (parafunctional habits) and periodontal surgical procedures can cause dentin exposure; furthermore, the anatomy of the teeth also affects the onset of DHS (Guentsch et al., 2012). Other factors, such as aging, soft tissue dehiscence, including aggressive brushing, can also cause apical displacement of the gingival margins thereby leading to exposure of dentin that can ultimately lead to the development of DHS (N. X. West et al., 2013).

Predisposing factors for dentin hypersensitivity include the exposure of dentin due to gingival recession and loss of cementum or enamel. In normal conditions, dentin is protected by enamel or cementum, and dentin hypersensitivity can occur only at the peripheral terminations of dentin tubules. Loss of enamel can occur due to abrasion or erosion, followed by acidic dietary actions on existing tubules, which are also risk factors for dentin hypersensitivity (Cunha-Cruz et al., 2013). For example, toothbrush abrasion, followed by abrasive components, can lead to the loss of enamel structure. Continuous exposure to the same area leads to abrasion, and dietary changes have been known to show a gradual increase in the remaining tooth structure. Further damage can occur even from slight pressure from brushing. Gingival recession, which can occur due to aging, chronic periodontitis, or harmful destructive habits, can also contribute as a predisposing factor for dentin hypersensitivity (West et al., 2013). In general, patients with periodontitis have a higher prevalence of dentin hypersensitivity due to greater risk and wider exposure of the roots as a result of periodontal tissue destruction (Shiau, 2012). Hypersensitive dentin has a close relationship with occlusal stress, as occlusal force initiates an increase in intrapulpal pressure, leading to an increased movement of dentinal fluid (Barcellos et al., 2012).

Morphology of Dentinal Tubules

Dentin structure plays a crucial role in determining the tooth's mechanical characteristics. It is primarily made up of 75% inorganic components, 20% organic materials, and approximately 5% water along with various other substances. Although dentin is

significantly softer than enamel—about one-fifth of its hardness—it reaches its maximum hardness levels near the junction between enamel and cementum. The modulus of elasticity for dentin is approximately 1.67 x 10^6 PSI, which provides essential support to the more brittle enamel layers. Dentin is produced and shaped by odontoblasts, featuring prominently the dentinal tubules as its key structural element (Karteva et al., 2019). Dentin contains dentin tubules, which are canals that radiate outward from the pulp cavity in the dentine. Each dentin tubule contains various Tomes fibers and odontoblasts that are connected with the pulp. The dentin tubules have two kinds of nerve fibers, unmyelinated (C-fibers) and myelinated (A-fibers). The A-fibers are accountable for the sensation of dentin hypersensitivity, which is perceived as pain (Dam et al., 2022).

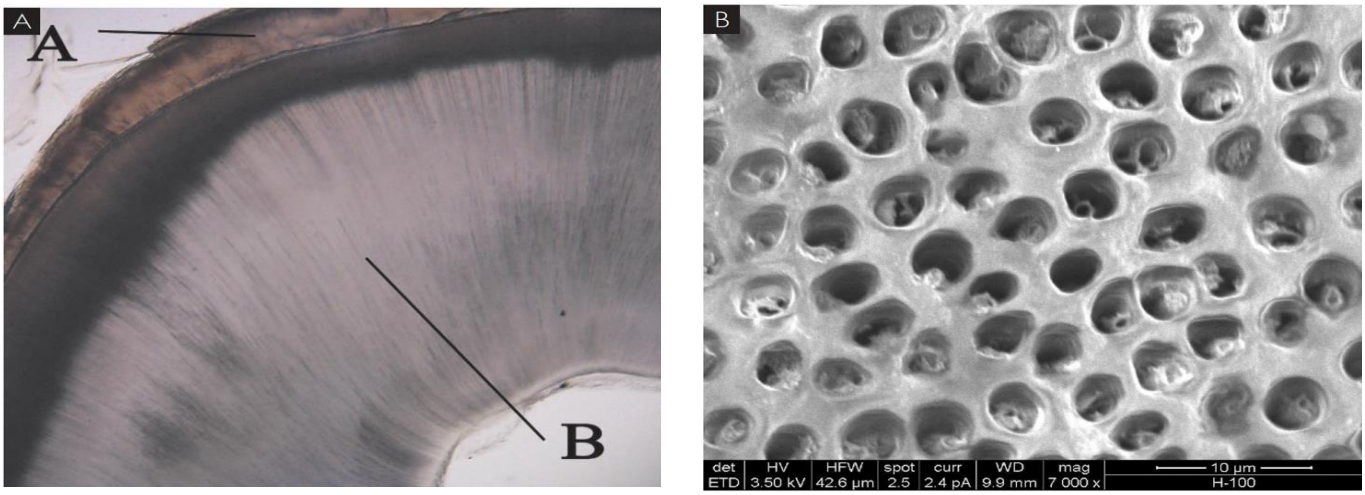

Figure 1. (A) Histologic cross-section of tooth showing enamel, labeled A, and dentin, labeled B. (B) With smear layer removed the ultrastructure of open dentin tubules are visualized clearly (Shiau, 2012)

The number of tubules per area near the pulp surface compared to the outer regions is 4:1. Therefore, superficial dentin is less permeable than dentin in deeper areas. The diameter of dentinal tubules decreases progressively from the pulp to the enamel in the crown dentin. The diameter of the tubules in the deeper dentin region is about 3-4 micrometers, while in peripheral dentin, it is about 1 micrometer. Microscopic studies have shown that areas experiencing dentin hypersensitivity exhibit larger and more numerous tubules compared to nonsensitive areas. This indicates that the transmission of stimuli through dentin in hypersensitive teeth is mediated by hydrodynamic mechanisms (Barcellos et al., 2012).

According to the hydrodynamic theory of dentin sensitivity, various stimuli such as thermal, osmotic, tactile (from probing), and evaporative effects cause fluid movement within the dentin tubules, which transmit sensations to the pulp nerves; this is especially problematic in cases of dentin hypersensitivity, where exposed dentin with open tubules responds to external pressures and leads to acute pain through the pressure changes that stimulate nearby receptors in the pulp (Dionysopoulos et al., 2023). When this theory is accepted as an explanation for the transmission of stimulus through dentin, it must be presumed that dentinal tubules are open at the exposed dentin surfaces and extend clearly towards the pulp. This helps explain why patients with exposed cervical dentin experience hypersensitive dentin while others do not. Open tubules are not uniformly distributed along the dentin surface. This can be observed by probing areas where only regions with open tubules respond with pain (Barcellos et al., 2012).

Dentin hypersensitivity is more commonly seen in young adults who experience rapid exposure of root surfaces. In contrast, older individuals, even when exhibiting exposure at the roots, often do not show sensitivity due to the following factors: mineral deposition within the tubules (dentin sclerosis), decreased number of tubules, reduction in pulp chamber size due to increased reparative dentin, and decreased cellularity, vascularity, and nerve fibers in the pulp (Zeola et al., 2019).

Borges et al. (2012) propose that the pressure in the pulp is greater than atmospheric pressure, which explains why fluid continually flows through the exposed permeable dentin, albeit at a very low rate, to activate the mechanoreceptors in the pulp. If there is exposure that increases the pressure gradient, it will enhance fluid movement, resulting in pain. Dentin hypersensitivity will be triggered by thermal, evaporative, tactile/mechanical, osmotic stimuli (Barcellos et al., 2012) or chemical (Arora et al., 2021),

and electrical (Trushkowsky et al., 2014). Thermal, pain occurs due to temperature variations causing fluid contraction when cold, and expansion when hot, leading to fluid movement. Evaporative, application of a cold spray on the tooth can cause evaporation of fluid in the open dentinal tubules. Tactile, a simple device for testing hypersensitivity involves moving a sharp probe along the cementum-enamel junction. This device triggers fluid movement, potentially due to vibrational mechanisms. Osmotic, pain is triggered by the presence of sweet substances. Due to its hypotonic nature, this creates osmotic pressure in the dentin, causing the dentinal fluid to move towards acidic substances (Barcellos et al., 2012). Electrical stimulation can also be used, but the results may be modified due to current loss through the periodontium and possible stimulation of periodontal nerves (Barcellos et al., 2012).

Mechanism of Dentin Hypersensitivity

The mechanism underlying dentin hypersensitivity is closely related to the anatomy and histology of the dentin-pulp complex. Odontoblasts synthesize the collagen matrix of dentin (primarily type 1) and play a role in the mineralization process; they are also involved in the formation and repair of dentin. The macrostructure of dentin consists of tubule units, which are surrounded by hypermineralized tissue known as peritubular dentin. Dentin tubules contain fluid resembling serum and odontoblast cell processes. Some studies have shown a physical proximity between sensory nerve fibers to the odontoblast (both the processes and cell body) (Shiau, 2012).

Several theories regarding the mechanisms of pain from dentin hypersensitivity are recognized. The dentinal receptor mechanism theory explains how direct stimulation of sensory nerve endings in the dentin occurs. However, experimental research has microscopically concluded that it is impossible for neural cells to be present on the outer portions of dentin (Dam et al., 2022). In the odontoblastic transduction theory, the function of odontoblasts is to mediate changes in membrane potential through synaptic junctions with pulp innervation. In this theory, odontoblastic processes are exposed on the surface of dentin and therefore can be easily excited by chemical and mechanical stimuli. However, there is no scientific background supporting this odontoblastic transduction theory (Shiau, 2012).

The currently most accepted theory regarding pain transmission is the hydrodynamic theory, wherein pain transmission occurs through rapid movement of fluid within the dentinal tubules (Sivakumar et al., 2014). Since many tubules contain mechanoreceptor nerve fibers close to the pulp, slight fluid movement within the tubules due to cutting, drying, pressure changes, osmotic changes, or temperature changes can lead to pain transmission. Sensitivity can occur when odontoblasts and their processes are stimulated during operative procedures, although pain receptor mechanisms appear to be located near the dentinal tubules that are adjacent to the pulp (Heymann et al., 2011). Hyperesthesia is a result of fluid within the dentinal tubules being disrupted by temperature, physical stimuli, or osmotic changes. This fluid changes or moves, thereby stimulating baroreceptors and generating neural signals. Air movement when drying the dentin surface leads to fluid flow from the dentin toward the dehydrated surface. This fluid movement excites the nerves and causes pain sensation. Thermal stimuli cause contractions in the dentinal tubules, resulting in changes in the flow of dentinal fluid and exciting the nerves (Rotpenpian, 2020). Osmotic stimuli, such as acids, salts, and sugars, can also cause fluid movement (Shiau, 2012).

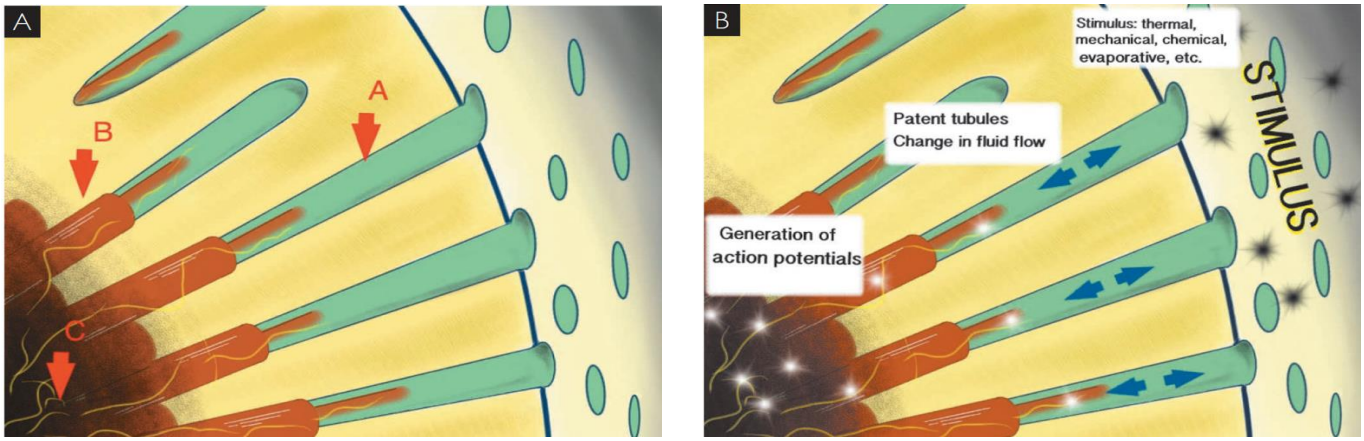

Figure 2. (A) A simplified diagram of the dentinopulpal complex. A, Dentin tubule; B, odontoblast cell and extending process, note proximally associated nerves (yellow); C, nerve/nerve plexus in pulp region. (B) Upon challenge by a stimulus (thermal, mechanical, evaporative, chemical), the exposed dentin and open tubules permit a change in rate of fluid flow in the dentin tubule. An action potential is generated involving the trigeminal sensory nerves (yellow) (Shiau, 2012)

The dentinal tubules contain dentinal fluid, which is a transudate of plasma. When enamel or cementum is removed during tooth preparation, the external covering of the dentin is lost, allowing tubular fluid to move toward the cutting surface. Pulp fluid has a slight positive pressure that forces the fluid out toward the cutting on the external layer. Dentin permeability is not uniform throughout the entire tooth. Coronal dentin is much more permeable than dentin in the root of the tooth. There are also variations within coronal dentin. Dentin permeability initially depends on the thickness of the remaining dentin (such as the length of the dentinal tubules) and the diameter of the tubules. Because the dentinal tubules are shorter, more numerous, and have a larger diameter, and are closely located to the pulp, the inner dentin acts as a less effective pulp barrier compared to the superficial dentin (Heymann et al., 2011).

The diameter, the status of narrowing (patency), and the number of open tubules are factors that differentiate teeth with dentin hypersensitivity from non-sensitive teeth. Teeth experiencing dentin hypersensitivity have a significantly higher number of dentin tubules, nearly 8 times per unit area more than non-sensitive teeth. The diameter of dentin tubules is also larger in teeth with dentin hypersensitivity compared to non-sensitive teeth (Agarwal, 2019). This indicates that patency (or lack thereof) is related to acid demineralization and the inability to remineralize. Exposed dentin tubules can become closed as a result of reactive sclerosis and deposition of secondary and tertiary dentin. Hypermineralization of peritubular dentin, where dentin is adjacent to odontoblast processes, as well as precipitation of minerals from saliva or fluid within the tubules, results in reactive sclerosis. Scanning electron microscopic evaluation of hypersensitive dentin confirms the presence of widely open dentin tubules (Shiau, 2012).

Diagnosis

Dentin hypersensitivity can be diagnosed through the pain experienced by the patient. Conditions that can lead to dentin hypersensitivity include dental caries, pulpitis, tooth fractures, fractured restorations, post-restoration sensitivity, marginal leakage, teeth experiencing attrition, and gingival inflammation (Cummins, 2009). The diagnosis may also involve a subjective evaluation of how this hypersensitivity affects the patient's daily life (Cunha-Cruz et al., 2013). According to Shiau (2012), an accurate diagnosis of dentin hypersensitivity (DHS) is an important component in determining treatment.

Clinical conditions that resemble DHS and can serve as differential diagnoses include: postoperative pain after filling procedures, cracked tooth syndrome, sensitivity due to bleaching procedures, fractured restorations, dental caries, pulpitis (Shiau, 2012), gingival inflammation, cusp fractures, and chipped teeth (Liu et al., 2020; Mantzourani et al., 2013), enamel invaginations, and teeth in acute hyperfunction (Mrinalini et al., 2021). A clinical examination directly related to dentin hypersensitivity should be performed, with exclusion of all differential diagnoses, including the following: cracked tooth syndrome, fractured restoration, restoration in traumatic occlusion, chipped teeth, dental or root caries, postoperative sensitivity, pulpal response to restorative treatment or material, marginal leakage of restoration, pulpitis (pulpal status), gingival inflammation, vital bleaching procedures, and atypical odontalgia (Liu et al., 2020; Trushkowsky et al., 2014).

Therefore, accurate information regarding dietary history, oral hygiene, pain history, clinical examinations, radiological evaluations, and the use of diagnostic tests such as percussion, palpation, and pulp vitality tests are necessary to help confirm the diagnosis of DHS while ruling out other conditions. Borges et al. (2012) emphasize the importance of obtaining information during the health history regarding tooth-cleaning habits, including frequency and duration, as well as the type of toothbrush, brushing technique, intervals between brushing, and the pressure applied while brushing. It is also important to be aware of habits that may cause trauma to the teeth and gums.

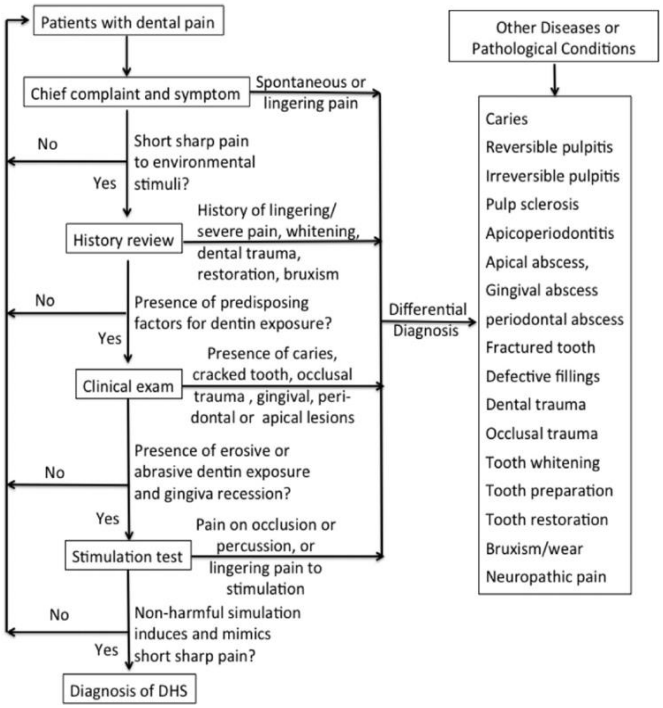

Figure 3. Flow chart for differential diagnosis of dentin hypersensitivity (Liu et al., 2020)

Clinical examination techniques for diagnosing DHS include the use of air spray from a triple syringe or exploring with a probe on exposed dentin. The severity or degree of pain can be qualitatively categorized based on a pain scale (mild, moderate, severe) or using a Visual Analog Scale (VAS) (Bandeca et al., 2017; Shiau, 2012). Inquiries about contact history with erosive substances need to consider environmental factors, work-related exposures, medications, diseases, and the most frequent factor, diet (Barcellos et al., 2012).

Generally, from the related history, patients express agreement with several aspects including: pain caused by thermal stimuli (cold and/or hot food, air during speaking or breathing), chemical stimuli (sweet and acidic foods), tactile stimuli (brushing and probing), localized acute pain of short duration that disappears when the stimulus is removed, and spontaneous periods of recession (Agarwal, 2019). Pain associated with DHS differs from that experienced in inflamed pulp. When a stimulus is applied to a hypersensitive tooth, the patient can immediately indicate the source of pain and discomfort, whereas pulp pain may last longer, be intermittent, and pulsating (Abraham et al., 2022). When determining the diagnosis of DHS, other pathological conditions and dental damage in the affected area must be eliminated to rule out other pain causes (Barcellos et al., 2012).

Management of Dentin Hypersensitivity

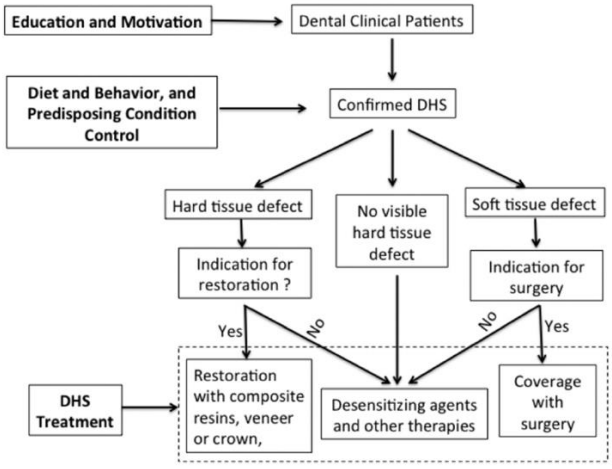

Figure 4. Strategies for managements of dentin hypersensitivity (Liu et al., 2020)

Knowledge of the etiology and the establishment of accurate diagnosis is crucial in determining the appropriate treatment plan for dentin hypersensitivity (DHS). According to Mantzourani et al. (2013), the mechanisms of action in DHS treatments are grouped into two main categories: nerve stabilization/desensitization and occlusion of the exposed dentinal tubules. The strategy of occluding or blocking open dentinal tubules is widely used as a treatment modality, ranging from the use of ions/salts/proteins to close dentinal tubules, application of restorative materials (dentin sealers) designed for physical blockage, use of periodontal grafts, and currently the use of lasers (Cummins, 2009).

Nerve Stabilization/Desensitization.

Nerve stabilization/desensitization in the management of dentin hypersensitivity (DHS) is accomplished by inhibiting or reducing nerve transmission (Shiau, 2012). The following desensitizing agents are very effective in the treatment of dentin hypersensitivity, strontium chloride, oxalate desensitizers (e.g., Calcium Oxalate), Potassium Nitrate, Varnishes and ADHSesive systems (e.g., Composite resins, GIC), Calcium hydroxide, Fluoride compounds (e.g., Stannous fluoride, 5% Sodium fluoride varnish) and others (Arua et al., 2021).

Potassium salts are known as nerve numbing agents and were used to reduce pain before anesthetics were developed. In 1974, Hodosh reported that potassium nitrate (KNO3) is effective in reducing DHS. A reduction in DHS was observed with the application of a KNO3 solution at concentrations of 1-15% and with pastes containing 10% KNO3 (Shiau, 2012). Several other potassium salts are also effective, such as potassium chloride, potassium citrate, and potassium bicarbonate. Potassium salts have the advantage of being compatible with fluoride, which is commonly added to toothpaste to prevent caries (Borges et al., 2012).

The mechanism of action of KNO3 is not fully understood. It was previously stated that nitrate ions were considered the active component because of their desensitizing ability when compared to silver nitrate. Further research has specifically identified potassium as an important component. The current hypothesis suggests that potassium and strontium have a direct action on the nerves present in the pulp. Potassium ions must pass through the dentinal tubules to reach the pulp nerves, and it takes 4-8 weeks before the pain subsides. A concentration of potassium of 8 mM is required to deactivate the intradental nerves at the pulp endings in the dentinal tubules (Mantzourani et al., 2013).

Potassium nitrate is the most common agent when discussing strategies for nerve desensitization (Bandeca et al., 2017). The mechanism of potassium nitrate is associated with its ability to increase the concentration of extracellular potassium ions, leading to nerve depolarization and preventing nerve repolarization. Generally, patients report favorable outcomes after using potassium nitrate for the treatment of DHS symptoms they experience. In previous studies,

toothpaste containing potassium nitrate (5% potassium nitrate) was shown to be safe for the pulp, and with regular daily use, it can effectively desensitize teeth for up to 4 weeks. However, the latest Cochrane review on toothpaste containing potassium nitrate, which included 6 meta-analysis studies, did not provide strong support for the use of potassium salts in the management of DHS (Shiau, 2012). Nevertheless, due to the affordability and ease of use of topical desensitising agents like Gluma, clinicians are recommended to use them as a routine treatment for DHS and, lasers can be used in cases where topical desensitising agents are ineffective (Naghsh et al., 2024).

Closure of Open Dentinal Tubules

The closure of open dentinal tubules is an effective method to reduce dentin hypersensitivity (DHS) (Shiau, 2012). There are several mechanisms by which open dentinal tubules can be closed to reduce DHS. The natural formation of a smear layer through the mechanical polishing (burnishing) of dentin can induce tubule closure. The introduction of compounds that are topically applied in the form of materials that precipitate and are insoluble on the surface and within the dentinal tubules or facilitate the formation of natural biological minerals is also effective. These compounds include abrasive particles, strontium or stannous salts, calcium phosphate, soluble oxalates, and bioactive glasses. Most of these materials work by physically blocking the dentinal tubules (Mantzourani et al., 2013).

Strontium was introduced for the treatment of dentin hypersensitivity (DHS) in 1956 when Pawlowska reported the benefits of 25% strontium in aqueous solution and 75% glycerin paste. Strontium chloride was the active ingredient in Sensodyne, first commercially introduced over 50 years ago. Strontium has several possible mechanisms of action to address DHS, although there is still limited research supporting these mechanisms. One possible mechanism is the deposition of particles on the dentin surface that can prevent the movement of fluid in the dentinal tubules. Strontium can replace calcium in hydroxyapatite due to its chemically similar form, thus reinforcing dentin that has undergone demineralization. Strontium has been shown to integrate into bone, enamel, and dentin, and also has a stabilizing effect on nerve cell membranes (Mantzourani et al., 2013).

The deposition of insoluble metal compounds from strontium salts on tooth surfaces can occlude or partially cover open dentinal tubules (Shiau, 2012). Other literature mentions that strontium has a nerve depolarization effect or that it can replace calcium in hydroxyapatite scaffolds to strengthen demineralized dentin. An example of toothpaste known to contain strontium salts is Sensodyne Rapid Relief, which contains 8% strontium acetate in a silica base and sodium fluoride. Recent research emphasizes that toothpaste containing strontium has the ability to close tubules, but it is suggested that the abrasive capacity of the silica in this toothpaste may be the responsible agent for desensitization (Agarwal, 2019).

As a biomaterial, bioactive glass has been used in the field of dentistry for over four decades, developed as a material for bone regeneration and repair in orthopedic, maxillofacial, and periodontal fields. The mechanism of action of bioactive glass in managing dentin hypersensitivity (DHS) is by covering open dentinal tubules. This material deposits on the hydroxicarbonate apatite layer, thereby blocking the dentinal tubules. Marketed under the trade name Novamin (Dentsply, USA), this component is available in the form of toothpaste and prophylactic paste used in clinics (Shiau, 2012).

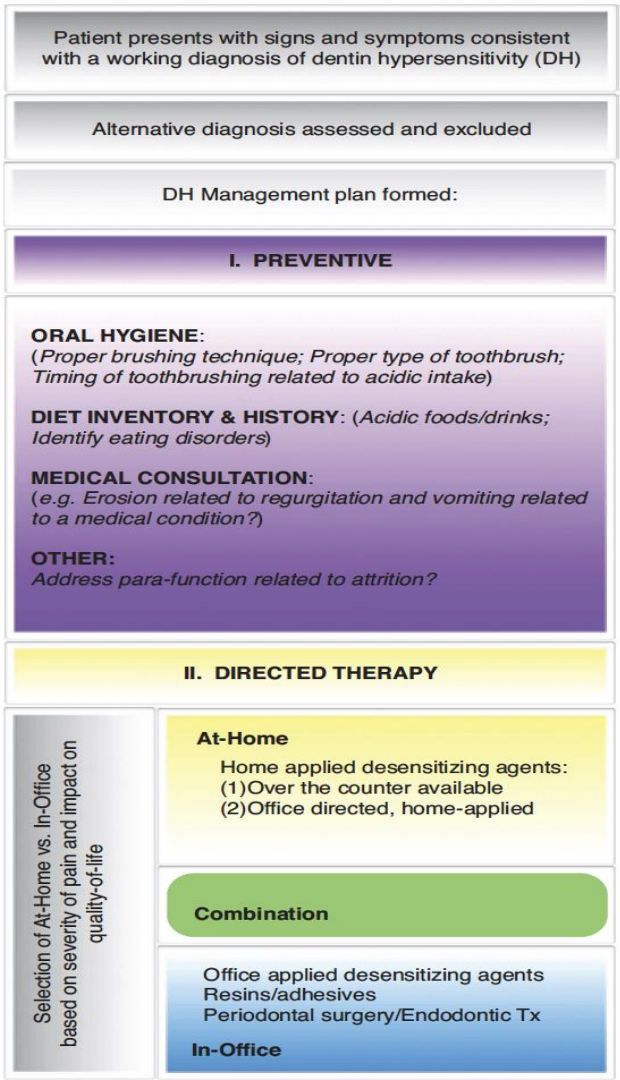

Figure 5. The managements of dentin hypersensitivity (Shiau, 2012)

Oxalate is used for the management of DHS as it has the ability to form precipitates within the dentin tubules, thereby blocking the flow of dentin fluid (Dam et al., 2022). Oxalates such as 3% monohydrogen monopotassium oxalate have additional advantages, such as their relatively low solubility in acid, making them resistant to dissolution during treatment. Existing studies have found that 3% monohydrogen monopotassium oxalate is effective in reducing DHS, but further studies are needed (Shiau, 2012).

Fluoride products, such as sodium fluoride and stannous fluoride, are known to show positive effects in closing dentinal tubules and can reduce dentin sensitivity. Topical application of fluoride can form a barrier through the precipitation of CaF2 on the surface of dentin (Petersson, 2013). Research has shown the effectiveness of 2% and 5% sodium fluoride varnish measured with VAS responses to air and cold testing over 24 weeks. A topical fluoride gel (1.23% sodium fluoride) has been recommended for use in reducing sensitivity following procedures related to tooth bleaching. Stannous fluoride, often administered in aqueous solution or with carboxymethylcellulose, is effective in managing DHS. Stannous fluoride can form specific precipitates on the dentin surface and can block tubules (Shiau, 2012).

A combination of products consisting of a 5% glutaraldehyde solution and 35% hydroxyethyl methacrylate (Gluma Desensitizer, Heraeus, Germany) has been known to be an effective desensitizing agent for up to 7-9 months. Glutaraldehyde blocks the dentinal tubules, inhibiting the hydrodynamic mechanism that causes dentin hypersensitivity (DHS). A reported decrease in DHS cases of 5% to 27% has been noted (Naghsh et al., 2024). The mechanism of glutaraldehyde involves a reaction with serum albumin in dentin fluid, leading to the formation of precipitates and blocking of the tubules. Studies using scanning electron microscopy and confocal laser scanning microscopy have verified the existence of intratubular blocking with the formation of protein coagulation (Shiau, 2012).

The basis for using resins and aDHSesives is to seal dentinal tubules and thus prevent the transmission of hydrodynamic stimuli to the pulp nerve complex. The deposition of a thin film coating using polymer-based materials, such as resins and dentin-bonding agents, forms an artificial smear layer to close the open tubules (Canali et al., 2017). Dentin-bonding system products are not specifically designed for treating DHS, but efficacy in reducing sensitivity has been demonstrated (Shiau, 2012).

Arginine and calcium carbonate formulations have been developed for the treatment of DHS based on the biological process of tubule occlusion by salivary glycoproteins (Halappa et al., 2015). Saliva carries calcium and phosphate, which interact with the dentinal tubules to induce occlusion and the formation of protective salivary glycoproteins with calcium and phosphate—a process that resembles alkaline pH conditions. Studies conducted to evaluate fluid movement through hydraulic conductivity have shown the hydrodynamic mechanism. Research evaluating the efficacy of an 8% arginine-calcium carbonate-fluoride toothpaste in reducing DHS symptoms has shown superiority over a 2% potassium ion-containing toothpaste over an 8-week period. Toothpaste formulations containing arginine have the ability to quickly address hypersensitive dentin through topical application (Shiau, 2012).

The He-Ne laser or diode-type laser has been recognized as a prospective treatment for dentin hypersensitivity (DHS). Early publications on the use of the He-Ne laser applied with low output at 6 mW, both in continuous wave and pulsed mode. Low-output diode lasers play a role in mediating analgesic effects by reducing nerve transmission. High-output lasers, such as Nd:YAG, can induce blockage or contraction of dentinal tubules and/or provide analgesic effects (Tabibzadeh et al., 2018). Meanwhile, CO_2 lasers work by closing or constricting dentinal tubules. Research has shown the safety of using these lasers as they do not cause thermal damage to the pulp (Shiau, 2012).

Mucogingival surgery aims to close the roots to minimize the area of exposed dentin. Clinical periodontal research on root coverage procedures has shown satisfactory results with the physical changes that occur. However, the authors note that the predictability of treatment outcomes remains questionable, necessitating further clinical studies (Shiau, 2012).

Conclusion

Dentin hypersensitivity is a prevalent issue among dental patients, requiring appropriate evaluation and management to relieve symptoms. Those experiencing this condition should receive tailored treatments and recommendations, including effective tooth brushing methods, dietary modifications, and regular dental check-ups, to minimize the risk of developing further sensitivity. Additionally, sensitizing toothpaste serves as an effective initial strategy for managing mild hypersensitivity and can be supplemented with one or both in-office treatments, if necessary.

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Author Contributions
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