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Dentine Hypersensitivity

This project is submitted to Al-Zahrawi University College, Department of Dentistry, as part of the requirements for obtaining a Bachelor of Dental Surgery degree, and it focuses on the study of Dentine Hypersensitivity and the effectiveness of the applied treatment methods.

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بسم الله الرحمن الرحيم

(يَا أَيُّهَا النَّاسُ قَدْ جَاءَتْكُمْ مَوْعِظَةٌ مِنْ رَبِّكُمْ وَشِفَاءٌ لِمَا فِي الصُّدُورِ وَهُدًى وَرَحْمَةٌ لِلْمُؤْمِنِينَ)

صدق الله العلي العظيم

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Certification of the Supervisor

I certify that this project entitled 'Dentine Hypersensitivity' was prepared by the fifth-year student [Ali Khalaf Hashem , Rasheed Mohammad Hussein ,Sajjad Kazim Hussain, Sura Moaid Ahmed] under my supervision at Al-Zahrawi University College in partial fulfillment of the graduation requirements for the Bachelor Degree in Dentistry."

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Dedication

I dedicate this research to Allah, the Almighty, who has granted me health, wisdom, and the ability to pursue knowledge. Through His grace and guidance, I was able to complete this research with patience and diligence. I pray that this work is accepted and that it serves as a step toward greater success in both my academic and professional journey.

Acknowledgment

In the Name of Allah, the Most Merciful, the Most Compassionate, all praise be to Allah, the Lord of the worlds; and prayers and peace be upon Mohamed, His servant and messenger.

First and foremost, I must acknowledge my limitless thanks to Allah, the Ever-Magnificent; the Ever-Thankful, for His help and blessings.

I owe a deep debt of gratitude to our university for giving us the opportunity to complete this work.

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Introduction:

Dentin hypersensitivity DH is characterized by brief and intense pain, occurring in teeth with exposed cervical dentin. This pain is triggered by different external stimuli including thermal changes, cold air evaporation, tactile pressures e.g. probing, electrical sensations, osmotic influences, or chemical exposures. Importantly, this pain cannot be attributed to any other dental pathology, defect, or disease **(Nnaji et al., 2021)**. A widely accepted theory that explains the sensation of pain resulting from external stimuli is the hydrodynamic theory, initially proposed by Brannstrom **(Brannstrom 1992).**

This theory suggests that the movement of fluids within dentin tubules is responsible for the sensation of pain **(Dionysopoulos et al., 2023)**. To induce DH, two surface changes must occur on the tooth the exposure and removal of dentin due to receding gums, coupled with the loss of cementum, and the enlargement of dentin tubules **(Dionysopoulos et al., 2023)**. Gum recession and dentin exposure can arise from factors such as excessive or incorrect brushing techniques, as well as periodontal disease **(Niemczyk et al., 2024).**

Accurately diagnosing DH requires a substantial amount of time, as it involves obtaining a comprehensive dental history and relies on the process of eliminating other comparable conditions. When symptoms of DH are present, it is imperative to conduct a differential diagnosis in order to distinguish it from other forms of orofacial pain, such as pulp inflammation, periodontal discomfort, tooth cracking, microleakage, and atypical odontalgia, among others **(Gillam et al., 2013).**

Although DH is most frequently observed in premolars and canines, all teeth and surfaces are susceptible to experiencing

hypersensitivity. The prevalence of DH has been documented to vary significantly across various studies, even reaching up to 100 percent. This variability arises from the diverse population groups studied and variations in assessment methodologies **(de Castro et al., 2021).**

Despite the numerous methods proposed for treating DH, a definitive solution to the issue has yet to be identified. Clinical studies evaluating the efficacy of different treatment approaches yield contradictory results **(Trushkowsky 2011) (Bak 2023) (Jang 2023).** This discrepancy stems from the fact that all these studies assess pain intensity, a parameter that proves notably challenging to evaluate due to its inherently subjective and intricate nature **(Arshad 2021).**

Therefore, the objective of this literature review was to furnish dental practitioners with comprehensive information concerning DH. This includes outlining its etiology, diagnostic methods, and treatment options, while also addressing the effectiveness of the approaches employed to manage DH.

Aims of Review

- to provide a brief overview of the diagnosis, etiology and clinical management of dentin hypersensitivity
- to discuss technical approaches to relieve sensitivity

Chapter One

Review of literature

1.1 Etiology of Dentin Hypersensitivity

Its etiology is multifactorial, involving both the initiation and progression of dentin exposure and the subsequent neural response. The initial phase often begins with the loss of enamel**.(Gillam, D., 2022)**

Dentin hypersensitivity typically occurs when external stimuli are applied to the surface of exposed dentin with open tubules. Different stimuli lead to rapid fluid movement within the tubules, creating pressure changes that activate pressure receptors located close to the pulp. This activation results in immediate acute pain **(Dionysopoulos et al., 2023).** Unlike other stimuli, heat causes a gentle movement of the dentinal tubule fluid toward the pulp, leading to a milder activation of the pressure receptors and consequently causing less pain **(JOHAR et al., 2021).**

It has been discovered that clinically hypersensitive teeth exhibit an exposed dentin tubule count approximately eight times higher and a dentin tubule diameter around two times larger in comparison to non-sensitive teeth **(Pranati et al., 2022).** This discrepancy in dentin tubule diameter between hypersensitive and non-sensitive teeth appears to hold clinical significance **(Grover et al., 2022).**

Two prerequisites are necessary for DH to occur on a tooth's surface. Firstly, there must be exposure and removal of dentin, which can result from enamel loss or a combination of receding gums and the loss of root cementum. Secondly, the dentinal tubules must become accessible, allowing external stimuli to activate the sensory mechanism present on the dentin surface **(Dionysopoulos et al., 2023).**

Gum recession and dentin exposure can be attributed to excessive brushing, improper brushing techniques, and periodontal disease, often

arising after either surgical or non-surgical treatment. Sixteen percent of patients develop DH following non-surgical periodontal treatment which peaks during the first week **(Kanarakis et al., 2022)** and then often subsides within few weeks.While regular brushing typically does not lead to substantial enamel loss, the combination of acid erosion from acidic foods and beverages with brushing can result in significant tooth tissue loss across all tooth surfaces, with a particular impact on cervical regions **(Schneider , 2023)**.

In addition to local etiologic factors, systemic conditions such as gastroesophageal reflux disease (GERD), eating disorders (e.g., bulimia nervosa), and even iatrogenic dental procedures like crown preparations, or bleaching treatments may contribute to dentin exposure and tubule patency. Anatomic variations such as failure of meeting between enamel and cementum at CEJ could be a contributing factor for DH. **(Asnaashari M, Moeini M., 2020)**

During the removal of tartar from root surfaces, it is common for scalers to inadvertently eliminate a thin layer of cementum (20–40 μm), giving rise to the creation of a smear layer containing blood and saliva that covers the underlying dentin **(Pashley & Tay, 2012).** Pashley and Tay (2012) demonstrated that dentin tubule fluid gradually permeates the smear layer and moistens the dentin surface. However, the smear layer presents significant resistance (86%) to the outward flow of fluids through the dentin **(Dionysopoulos et al., 2023).** Generally, the presence of the smear layer offers patients protection against dentin sensitivity, as it effectively seals the dentin tubules, even more efficiently than the resin infiltrations formed by bonding agents **(Carrilho et al., 2007; Pashley, 2013).**

This smear layer, however, does not persist on the dentin surface for an extended period. Over approximately 7–10 days, microbes accumulate on it, forming a biofilm that produces acids capable of dissolving the smear layer **(Mokeem et al., 2023).** The loss of the smear layer leads to increased dentin permeability, resulting in the emergence of dentin sensitivity. In some instances, the level of sensitivity can escalate further, progressing to the state of hypersensitivity from the prior condition of simple dentin sensitivity.

1.2 Epidemiology

Patients with hypersensitivity were more likely to be younger, to be female and to have a high prevalence of gingival recession and at-home tooth whitening **(Mohammed et al., 2017).**

Higher prevalence in females would probably be related to their dental hygiene and dietary habits **(Davari et al., 2013).**

The decrease in dentin hypersensitivity with increased age might be explained by the continued deposition of dentin and subsequent pulp atrophy of the teeth during the lifetime or even by tooth loss in people in the older age group. The average participant had multiple teeth that were sensitive, with sensitivity occurring more frequently in molars, premolars and incisors than in canines **(Carvalho & Lussi, 2017).**

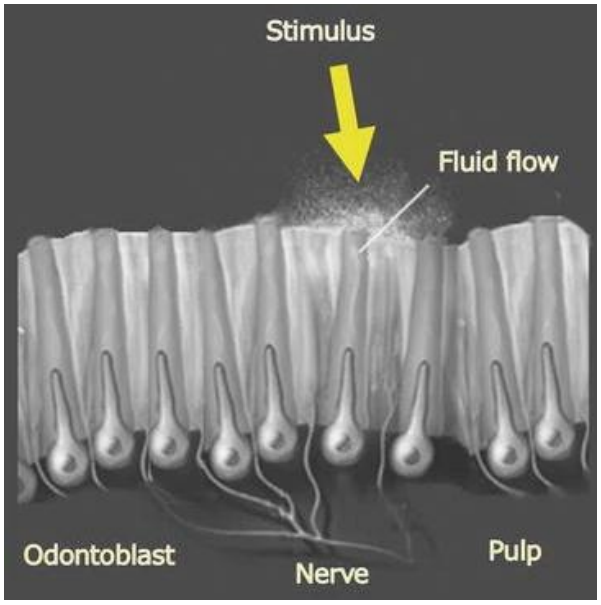
Other studies reported that the occurrence of DH in canines and premolars is more than other teeth.

The buccal surface of the teeth has been reported to be more involved with the disease than other places **(Davari et al., 2013).**

The quality of life and enjoyment of daily activities are both impacted by dentin hypersensitivity, and many patients are unaware that it is a treatable illness **(Salam et al., 2023).**

1.3 Pain Mechanism of Dentin Hypersensitivity

While considerable knowledge has been acquired regarding DH since its initial documentation, there remains a deficiency in clinical evidence-based research, particularly concerning the underlying pain mechanisms, which remain incompletely understood **(West et al., 2013).** The theory that continues to hold the most widespread acceptance posits that sensitive dentin arises from the stimulus-triggered fluid flow within the dentin tubules, leading to the subsequent activation of nociceptors at the border between the pulp and dentin **(Dionysopoulos et al., 2023).** It is believed that intradental myelinated A-β fibers and some A-δ fibers respond to stimuli that displace the fluid within the dentin tubules, culminating in the distinct, brief, and sharp pain experienced in cases of DH **(Matthews et al., 2000).**



Figure(1) : Brännström's hydrodynamic theory
Adapted from Kanehira et al. (2015)

Despite the fact that the inner diameter of dentin tubules measures about 1 µm, their hydrodynamic behavior, influenced by collagen fibers and calcium phosphate salts within them, resembles that of tubules with a functional diameter of less than 0.1 µm **(Yu et al.,, 2024).** Consequently, while microbial penetration through the dentin tubules into the pulp is unlikely, microbial byproducts, including endotoxins and exotoxins, can readily traverse the tubules and reach the pulp **(JOHAR et al., 2021).** In cases where toxins induce localized inflammation of the pulp, an escalation in pulpal pressure occurs, consequently increasing fluid outflow. This process ultimately leads to increased sensitivity of the pulp nerves beyond normal levels. Furthermore, localized pulp inflammation can prompt the proliferation of nerve endings located close to the dentin tubules, intensifying the overall sensitivity of the pulpo–dental complex **(Pashley, 2013; Kerns et al., 1991).**

The proliferation of nerve endings is triggered by inflammation mediators, such as histamine, bradykinin, prostaglandins, neuropeptides, and others. These mediators induce the proliferation of fibroblasts, aiming to restore the damaged collagen within the pulp's connective tissue due to inflammation **(Pohl et al., 2024).** Furthermore, the cell membrane receptors on the newly formed nerve branches identify microbial antigens and inflammatory byproducts. This recognition activates mechanisms for generating new protein factors that subsequently stimulate sodium channels. As a result, these channels are further stimulated, leading to an increased sensitivity of the pulp **(Renton et al., 2005).**

It has been noted that DH can, in certain instances, gradually subside without external therapeutic intervention, often following a period of exacerbation **(Dionysopoulos et al., 2023).** Experimental

studies conducted in vivo by Pashley et al. (1983) offered insight into this phenomenon. When cavities were created in dog teeth and dentin permeability was measured hourly over an 8 h span, a decline of 15% in permeability occurred each hour in living teeth, in contrast to pulpless teeth **(Pashley et al., 1983).** The researchers deduced that this decrease in permeability resulted from the release of plasma proteins from the pulp into the fluid of the dentin tubules, leading to a reduction in tubule diameter. The significance of fibrinogen, the largest plasma protein, in reducing dentin permeability after cavity preparation was also experimentally confirmed in dogs **(Jiang et al., 2022).** Additionally, other researchers discovered that immunoglobulins, such as IgG, can induce a gradual reduction in dentin permeability over time **(Chen et al., 2017).**

Consequently, microbial toxins originating from saliva and plaque have the capability to infiltrate the pulp, prompting pulp irritation, elevating pulp pressure, stimulating new neuron growth, and increasing nerve sensitivity **(Galler et al., 2021).** These toxins are believed to underlie the sustained presence of DH within a subset of patients who initially experienced it.

It is plausible that this persistence of hypersensitivity is linked to multiple episodes of localized pulpitis in these patients, which eventually resolves through pulpal eschar formation **(Pashley, 2013).** The escharated tissue lacks vasculature, potentially hindering the delivery of sizable proteins into the dentin tubules, unlike the norm for a healthy pulp. Furthermore, the reaction of the pulp to microbial toxins might not be uniform across all patients **(Pashley, 2013).**

1.4 Effects of Dentin Hypersensitivity

Dentin hypersensitivity can cause both physical and psychological issues for individuals, impacting their quality of life. This condition can affect dietary choices, dental hygiene practices, and aspects of personal appearance. Some individuals may not seek treatment for dentin hypersensitivity, even though they may mention it during dental appointments, possibly because they do not perceive it as a distinct disease **(Davari et al., 2013).**

1.5 Diagnosis of Dentin Hypersensitivity

A multitude of studies on dentin hypersensitivity (DH) exist; however, the majority of dental practitioners remain uncertain about its origin, diagnosis, and causative factors **(Porto et al., 2009).**

Achieving a conclusive diagnosis of DH requires a comprehensive clinical history, thorough clinical and radiographic examinations, and professional inquiries. Factors such as the nature of pain (sharp, dull, or throbbing), the number and location of affected teeth, the specific tooth component causing pain, and the severity of pain must all be considered **(Trushkowsky and Oquendo, 2011).**

Diagnosis of DH is established when symptoms are linked to exposed dentin, although teeth with vital pulps may exhibit similar symptoms if there is a specific etiological factor causing sensitivity, such as dental caries, fractures, leaking restorations, or recent restorative treatment. The overlap of clinical factors for reversible pulpitis with visible dentin can further complicate the diagnosis. Hypersensitive teeth and inflammatory pulps often manifest similar symptoms, such as sensitivity to heat, cold, and air **(Cohen, 2002).**

The differential diagnosis of DH includes cracked tooth syndrome, defined as a fracture plane of unknown depth originating from the crown, passing through the tooth structure, extending subgingivally, and potentially connecting with the pulp space and/or periodontal ligament. Cracked tooth syndrome poses a diagnostic challenge due to its variable and atypical clinical signs and symptoms, even for experienced dental practitioners **(Torabinejad and Walton, 2009; TÜRP and Gobetti, 1996).**

Early diagnosis is crucial for successful restorative management and prognosis. However, several dental conditions can mimic cracked tooth syndrome, leading to misdiagnosis. These conditions include acute periodontal diseases, reversible pulpitis, dentinal hypersensitivity, galvanic pain from silver amalgam restorations, sensitivity due to microleakage from recently placed composite resin restorations, hyperocclusion from dental restorations, occlusal trauma from parafunctional habits, orofacial pain from conditions like trigeminal neuralgia, and psychiatric disorders like atypical facial pain **(De Laat, 2020)**.

Pulpal pain is typically described as more prolonged, dull, aching, and poorly localized, lasting longer than the stimulus that initiated it **(Närhi et al., 2016).**

1.6 Management

Dentin hypersensitivity poses a common clinical challenge due to the unpredictable nature of treatment outcomes. Current treatment methods are often transient, and their effectiveness can vary. **(Liu et al., 2020).**

Grossman outlined criteria for an ideal treatment, emphasizing the need for fast, long-lasting, easily applicable, non-irritating, painless, non-staining, and consistently effective interventions. **(Grossman, 1935)**

Traditionally, two approaches were used to treat cervical dentinal sensitivity (CDS): blocking dentinal tubules and modulating mechanoreceptor responses. **(Aminoshariae & Kulild, 2021).**

Patients should be educated on proper toothbrushing techniques, avoiding abrasive toothpaste, and refraining from brushing for at least an hour after consuming acidic foods or drinks to prevent the synergistic effects of acidic erosion and toothbrush abrasion. Aggressive toothbrushing, defined as excessive force with a hard-bristled toothbrush, is a common factor in dentinal hypersensitivity **(Davari et al., 2013).**

Research indicates that many patients with dentinal hypersensitivity use hard toothbrushes, highlighting the importance of educating patients on proper oral hygiene practices **(Vijaya et al., 2013)**.

In some cases, correcting occlusion or using an occlusal splint can effectively resolve the issue. For gingival recession, consulting a periodontist is advisable, and treatments like grafts or positioning flaps may be considered. Monitoring the patient's diet is important, particularly regarding the quality and frequency of acidic food consumption, to provide appropriate recommendations. Patients with gastroesophageal

regurgitation and eating disorders should be referred to their physicians for management of the underlying conditions.. **(Davari et al., 2013)**

Classification of desensitizing agents:

- Classification of desensitizing agents based on mode of administration

- Classification of desensitizing agents based on mechanism of action

Classifying desensitizing agents used in treating dentin hypersensitivity (DH) poses a challenge due to their abundance and the unclear mechanisms of action. These agents can be broadly categorized based on their mode of administration, with one category being at-home treatments, which are convenient and effective for multiple teeth **(Liu et al., 2020)**.

They can be easily classified into two groups based on the mode of their administration:

1. At home: this mode is simple and reasonable and can be used in treatment of many teeth. **(Davari et al., 2013)**

a. Tooth dentifrice and tooth pastes: Toothpaste is a common over-the-counter (OTC) option for desensitization. Toothpaste containing potassium salts can block axonal action in dentinal tubules, reducing tooth excitability. These toothpastes should be used with soft-bristled brushes and minimal water to maximize effectiveness. Remineralizing toothpaste with sodium fluoride and calcium phosphates has shown significant reduction in DH. Casein phosphopeptide amorphous calcium phosphate (CPP-ACP) is claimed to remineralize early enamel lesions and is considered effective in both preventing and treating DH. **(Cummins, 2009)**

b. Mouthwashes and chewing gums

2. In office treatment of (DH) : is a complex and costly procedure suitable for a limited number of teeth. While theoretically, it should provide immediate pain relief, this may not always be the case in practice. **(Miglani et al., 2010)**

Several methods are used for in-office DH therapy:

a. Potassium nitrate is available in aqueous solution and adhesive gel forms. It reduces the number of potassium ions in dentinal tubules, thus decreasing nerve excitability and pain transmission. **(Miglani et al., 2010)**

b. Fluorides, particularly sodium fluoride at a 2% concentration, precipitate calcium fluoride crystals inside dentinal tubules, reducing dentinal permeability. The crystals are nearly insoluble and can be removed by saliva or mechanical scrubbing. Acidulated sodium fluoride is used to enhance precipitation **(Escalante-Otárola et al., 2021).**

c. Oxalates can occlude dentinal tubules and reduce dentin permeability by up to 98%. However, the reduction in dentin hypersensitivity induced by oxalate is short-lived. Surface etching of the tooth can enhance oxalate effectiveness. **(Pillon et al., 2004, Vieira et al., 2009)**

The application of 28% potassium oxalate can lead to the formation of calcium oxalate in the depth of dentinal tubules. However, findings have indicated that the reduction of dentin hypersensitivity induced by oxalate, remains for a short time. To increase the effectiveness of oxalate, the surface of the tooth can be etched**.(Sauro et al., 2006)**

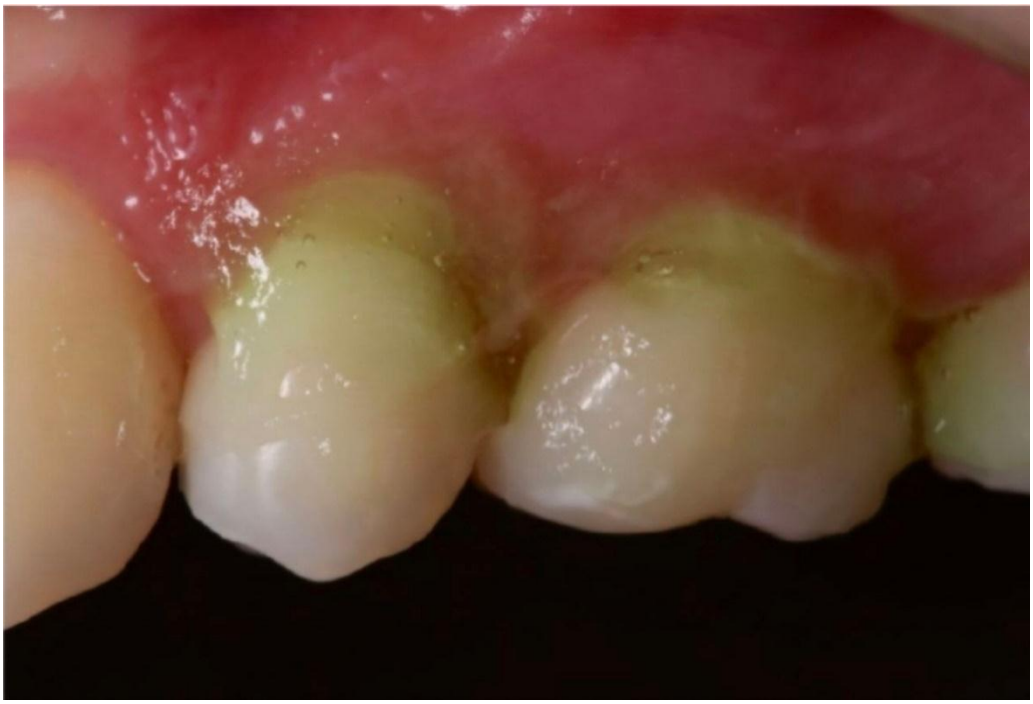


figure (2) pplication of an in-offce potassium oxalate gel on sensitive dentine. As recommended by the manufacturer, the product was kept on the exposed dentine for 3 min and removed without rinsing. (Images provided by Dr. Victor Mosquim and Dr. Mariele Vertuan)

d. Composites effectively seal dentinal tubules by forming a hybrid layer. Old adhesives created this layer by removing the smear layer and etching the dentinal surface to form deep resin tags. Newer adhesives modify the smear layer and incorporate it into the hybrid layer. Self-etch bonding systems, which contain acidic ingredients that condition the dentin, and two-step systems are commonly used for this purpose. They reduce

hypersensitivity significantly over a 4-week period, with two-step systems being more durable and less permeable. Pashley et al, 1978)**(Pashley, 1990)**

e. Bioglass, originally developed to stimulate bone formation, is used in periodontal surgery to fill osseous defects. Its application results in the formation of an apatite layer, which aids in the occlusion of dentinal tubules **(Nicholson, 2022).**

f. The effect of laser therapy: Lasers increase the temperature of dentinal tubules, causing them to fuse and obliterate the tubules **(Lee et al., 2015; Pourshahidi et al., 2019)**. Low-intensity lasers provide analgesic effects by stimulating mitochondrial ATP production and increasing the threshold to excite free nerve fibers' excitability **(Gerschman et al., 1994).** The analgesic effect of lasers may be due to the alteration of sensory axons nerve endings blocking C and Aβ fibers **(Orchardson et al., 1997).** Laser therapy with CO2, Er:YAG, and Nd:YAG lasers is thought to have a clinical advantage over topical medicament in treating DH. The effectiveness of laser treatments ranges from 50 to 94.5% at different follow-up periods from 1 to 3 months **(Asnaashari and Moeini, 2013; Corona et al., 2003; Zhang et al., 1998).**

g. Calcium sodium phospho-silicate: This is formulated to promote enamel remineralization and dentinal tubule occlusion. In the oral cavity, sodium ions exchange with hydrogen ions, releasing calcium and phosphate from the dentifrice. These minerals deposit within dentinal tubules, eventually leading to tubule occlusion **(Alharkan, 2021).**

h. Dentin adhesive system (Gluma desensitizer): It is an aqueous solution containing 5% glutaraldehyde (GA) and 35% hydroxyethyl methacrylate (HEMA). It prevents hypersensitivity by clotting proteins in the dentinal

tubules, reducing permeability and fluid flow **(Arrais et al., 2004).** It is a non-irritating, painless, easy-to-apply procedure with immediate and long-lasting effects. Clinical trials have shown GLUMA to have success rates ranging from 92 to 100% at 1–6 months posttreatment **(Dondi dall'Orologio et al., 2002; Patil et al., 2015)**. However, proximal tooth areas with poor access for adhesive application may remain vulnerable to sensitivity **(Vora et al., 2012).** GLUMA desensitizers are more effective than oxalates or placebo applications **(Orchardson and Collins, 1987; Rosaiah and Aruna, 2011).**

Chapter Two
Discussion and Conclusion

Discussion and Conclusion of the Literature Review on Dentin Hypersensitivity (DH)

Dentin hypersensitivity (DH) is a common yet complex clinical condition characterized by sharp, transient pain in response to various external stimuli such as thermal changes, osmotic influences, and tactile pressures **(Gillam, 2025).** Despite extensive research, the underlying mechanisms of DH remain incompletely understood, and treatment outcomes are often unpredictable. This review synthesizes current knowledge on DH, encompassing its etiology, pain mechanisms, epidemiology, diagnostic challenges, and management strategies, while highlighting gaps in clinical evidence and areas for future research.

The hydrodynamic theory, first proposed by **(Brännström in 1992),** continues to be the most widely accepted explanation for DH. This theory posits that external stimuli cause rapid fluid movement within dentinal tubules, generating pressure changes that activate mechanoreceptors near the pulp-dentin junction, resulting in sharp, acute pain. Supporting this theory, studies have demonstrated that hypersensitive teeth exhibit approximately eight times more exposed dentinal tubules and twice the tubule diameter compared to non-sensitive teeth **(Behniafar et al.,2024).** The presence of a smear layer initially provides protection by sealing dentinal tubules, but its dissolution by bacterial biofilm acids increases dentin permeability, contributing to hypersensitivity. Additionally, microbial toxins from plaque can infiltrate the pulp, inducing localized inflammation, elevating pulp pressure, and increasing nerve sensitivity, which may explain why some patients experience persistent DH **(Mall et al., 2024).**

Chapter Two……...…… Discussion and Conclusion

Epidemiological studies reveal that DH is more prevalent among younger individuals, females, and those with gingival recession or a history of at-home tooth whitening. The condition most frequently affects premolars and canines, though all teeth can be involved. Contributing factors include improper brushing techniques, dietary acid consumption, and periodontal treatments that remove cementum. Interestingly, DH prevalence decreases with age, likely due to natural dentin deposition and pulp atrophy over time. However, the condition significantly impacts patients' quality of life, affecting dietary choices, oral hygiene practices, and overall well-being **(Nnaji et al., 2021).**

Diagnosing DH remains challenging due to the need for careful differential diagnosis to exclude other conditions such as cracked tooth syndrome, reversible pulpitis, and atypical odontalgia. A thorough clinical history, combined with tactile and evaporative stimuli tests, is essential for accurate diagnosis. Radiographic and clinical examinations are also necessary to rule out other dental pathologies that may mimic DH symptoms **(Gillam, 2024).**

Management of DH involves both at-home and in-office treatments, each with varying degrees of effectiveness. At-home approaches include desensitizing toothpastes containing potassium salts or remineralizing agents like fluoride and casein phosphopeptide-amorphous calcium phosphate (CPP-ACP), which work by occluding dentinal tubules or reducing nerve excitability **(Hamlin et al., 2012).** In-office treatments range from topical applications of fluoride varnishes and oxalates to more advanced interventions such as laser therapy and restorative procedures. For instance, Gluma desensitizer, which contains glutaraldehyde and hydroxyethyl methacrylate (HEMA), coagulates proteins within tubules, providing long-lasting relief. Laser therapy,

particularly with Nd:YAG or CO_2 lasers, has shown promise in occluding tubules and reducing nerve sensitivity, with success rates ranging from 50% to 94.5% in various studies **(Asnaashari et al., 2013; Patil et al., 2015).**

Despite the array of available treatments, no single method guarantees permanent resolution of DH. The effectiveness of desensitizing agents varies, with some providing immediate but short-term relief, while others require prolonged use for noticeable improvement. Patient compliance and proper oral hygiene practices play a crucial role in treatment success **(Liu et al., 2020).**

Moreover, combination therapies, such as using fluoride varnishes alongside adhesive systems, may enhance durability and efficacy **(Petersson, 2013).**

In conclusion, DH remains a significant clinical challenge due to its multifactorial etiology and subjective pain assessment. While current treatments offer varying degrees of relief, further research is needed to develop more effective and long-lasting solutions. Future studies should focus on long-term clinical trials comparing different desensitizing agents, exploring novel biomaterials for tubule occlusion, and improving diagnostic tools to differentiate DH from other orofacial pain conditions. By integrating evidence-based approaches with patient-centered care, dental practitioners can better manage DH and improve patients' quality of life. Ultimately, a comprehensive understanding of DH's underlying mechanisms and individualized treatment plans will be key to addressing this pervasive dental concern.

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