

Dentin Hypersensitivity: A Comprehensive Review

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Abstract: Dentin hypersensitivity (DH) is a common clinical condition characterized by sharp, transient pain arising from exposed dentin in response to various external stimuli. Its multifactorial etiology involves dentin exposure due to enamel or cementum loss and open dentinal tubules that permit fluid movement, activating pulpal nerves. The hydrodynamic theory remains the most accepted explanation. Management strategies include preventive care, desensitizing agents, restorative materials, and advanced techniques such as laser therapy and biomimetic materials. This review provides a comprehensive understanding of the etiology, pathophysiology, diagnostic methods, and therapeutic options for dentin hypersensitivity, emphasizing individualized patient-centered care for long-term success.

Keywords: Dentin Hypersensitivity, Hydrodynamic Theory, Desensitizing Agents, Fluoride Varnish, Laser Therapy, Dentin Occlusion.

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I. INTRODUCTION

Dentin hypersensitivity (DH) is a prevalent oral health condition that affects millions of adults globally. As advances in preventive dentistry allow more individuals to retain their natural dentition longer, the incidence of DH is increasing. Clinically, DH is characterized by transient, sharp pain originating from exposed dentin in response to external stimuli such as thermal, mechanical, osmotic, and tactile triggers. Severe cases can persist for months, significantly affecting quality of life and sometimes progressing to neuropathic pain.

The pathophysiology of DH involves increased dentin permeability and nerve excitability, contributing to amplified pain perception. Treatment aims to interrupt these mechanisms, with a focus on dentinal tubule occlusion and nerve desensitization. However, individual variability in symptom intensity highlights the complex interplay between peripheral and central mechanisms.

With increasing life expectancy and reduced tooth loss, the prevalence of DH is expected to rise. Modern dentistry offers numerous treatment options, ranging from in-office procedures to commercially available desensitizing agents. Nevertheless, the heterogeneous nature of DH necessitates an individualized approach tailored to each patient's condition. Understanding factors such as acid erosion, gingival recession, and microleakage is essential for effective management and long-term symptom control.

II. DEFINITION AND TERMINOLOGIES

Dentin hypersensitivity (DH) has been described using various terms, including cervical sensitivity, root hypersensitivity, dentine sensitivity, and cemental sensitivity. While these terms are often used interchangeably, dentin hypersensitivity remains the most widely accepted term, describing a painful condition arising from exposed dentin that cannot be attributed to any other dental pathology (Holland et al., 1997).

➤ Commonly Used Terms for Dentin Hypersensitivity:

- Dentine Sensitivity
- Dentine Hypersensitivity
- Dentinal Hypersensitivity
- Cervical Hypersensitivity/Sensitivity
- Root Hypersensitivity/Sensitivity
- Cemental Hypersensitivity/Sensitivity

According to the Canadian Consensus Document (2003), DH is defined as “pain derived from exposed dentin in response to chemical, thermal, tactile, or osmotic stimuli that cannot be explained by any other dental defect or disease.” The Canadian Advisory Board on Dentine Hypersensitivity later suggested replacing ‘pathology’ with ‘disease’ to emphasize DH as a distinct clinical entity.

➤ *Evolution and Site Descriptors*

While terms such as cemental hypersensitivity and root sensitivity have been used, these site descriptors may be inappropriate, as DH can occur on any tooth surface, with cervical and root areas being the most commonly affected. In 2002, the European Federation of Periodontology (EFP) proposed using 'root sensitivity' for sensitivity associated with periodontal disease before and after non-surgical or surgical therapy.

Despite the debate over terminology, dentin hypersensitivity remains the preferred term due to its consistent usage and acceptance over the years, distinguishing it from other types of dentinal pain with different etiologies.

III. ETIOPATHOGENESIS

A. *Anatomy of the Dentin-Pulp Complex*

Dentin is a vital tissue that responds to both physiological and pathological stimuli. It is covered by enamel in the crown and a thin layer of cementum in the root. Despite histological differences, dentin and pulp share the same ectomesenchymal origin and form a dentin-pulp complex, where odontoblastic processes extend into the dentinal tubules. These tubules, filled with dentinal fluid, contribute to dentin's sensitivity.

B. *Neuroanatomy of the Pulp-Dentin Complex*

The pulp contains both myelinated (A-fibers) and unmyelinated (C-fibers) nerve fibers, primarily entering through the apical foramen. A-delta fibers transmit sharp pain in response to stimuli, while C-fibers convey dull, throbbing pain. These fibers form the subodontoblastic plexus of Raschkow, with terminal branches extending toward dentin and terminating as free nerve endings. When stimulated, these endings elicit pain, making dentin hypersensitivity (DH) a distinct clinical condition.

C. *Pathogenesis*

DH develops in two stages:

➤ *Lesion Localization*

Dentin exposure occurs due to enamel or cementum loss caused by attrition, abrasion, erosion, or abfraction. Gingival recession can also expose dentin, especially on buccal surfaces of canines and premolars.

➤ *Lesion Initiation*

For hypersensitivity to develop, the smear layer and tubular plugs must be removed, exposing patent dentinal tubules to the external environment. Increased fluid movement within these tubules generates pain, contributing to DH.

D. *Multifactorial Etiologies of D:*

Several factors contribute to dentin exposure and tubular opening:

- **Periodontal Disease and Treatment:** Recession induced by periodontal disease or treatment exposes root dentin, making it susceptible to DH.

- **Tooth Wear:** Pathological wear through erosion, attrition, abrasion, and abfraction accelerates dentin exposure, leading to DH. Erosion, caused by intrinsic (gastric reflux) or extrinsic acids (dietary factors), removes the smear layer and opens tubules. Abrasion from aggressive tooth brushing and abrasive dentifrices can exacerbate this process.
- **Tooth Whitening:** Whitening agents such as carbamide peroxide dehydrate the tooth, increasing sensitivity.
- **Developmental Lesions:** Conditions like amelogenesis or dentinogenetic imperfecta can alter surface morphology and expose dentin.
- **Dietary and Lifestyle Factors:** Acidic diets, occupational exposure to acidic environments, and parafunctional habits contribute to enamel loss, enhancing DH.

DH results from multifactorial processes involving both physical and chemical insults that expose dentin and alter tubular dynamics. Understanding these mechanisms helps in implementing preventive and therapeutic strategies to manage DH effectively.

IV. THEORIES

Several theories have been proposed over the years to explain the mechanism involved in the etiology of dentin hypersensitivity (DH), with three major theories being widely recognized:

A. *Neural Theory (Direct Innervation Theory)*

This theory suggests that thermal or mechanical stimuli directly affect nerve endings within the dentinal tubules, which communicate with pulp nerve fibers. The stimuli excite nerve endings in the tubules, transmitting signals to the brain via afferent fibers.

➤ *Limitations:*

- Nerves in dentinal tubules are rarely present, and if they are, they do not extend beyond the inner dentin.
- Topical anesthetics do not consistently eliminate sensitivity.
- Newly erupted teeth exhibit sensitivity even before the nerve plexus is established.

B. *Odontoblastic Transduction Theory*

- Proposed by Rapp et al., this theory suggests that odontoblasts act as receptor cells, mediating changes in membrane potential through synaptic junctions with nerves. Odontoblastic processes are believed to transmit impulses to the brain in response to stimuli.

Limitations:

- No evidence of neurotransmitters has been found in odontoblastic processes.
- Membrane potential of odontoblasts is too low to facilitate transduction.
- Sensitivity persists even after destruction of the odontoblastic layer, invalidating this mechanism.

C. Hydrodynamic Theory

This is the most widely accepted theory proposed by Gysi and later refined by Brännström et al. in 1964. It suggests that when exposed dentin is subjected to thermal, chemical, tactile, or evaporative stimuli, fluid movement within the dentinal tubules occurs. This fluid movement either inward or outward activates mechanoreceptors in A-delta fibers, resulting in pain.

➤ Key Points:

- A higher number and larger diameter of open dentinal tubules increase fluid flow, intensifying pain.
- Cold stimuli trigger rapid outward fluid movement, leading to quicker and more intense pain compared to heat stimuli, which create slower inward movement.
- Hyperosmolar solutions, such as sugar or salt, draw fluid out of the tubules, causing painful sensations.
- Desiccation from air exposure causes outward fluid movement, activating nerve fibers and eliciting pain.

D. Supporting Evidence

- SEM studies show that hypersensitive dentin has more open and wider dentinal tubules than non-sensitive dentin.
- Patients with DH frequently report sensitivity to cold stimuli, aligning with the rapid outward fluid movement described by the hydrodynamic theory.

➤ Clinical Implication

Since the hydrodynamic theory is the most accepted, many treatment modalities target occlusion of dentinal tubules to prevent fluid movement, thereby reducing sensitivity.

➤ Clinical Manifestations

Dentin hypersensitivity (DH) typically presents with the following clinical features:

- More sensitivity to cold stimuli than hot.
- Patients tend to avoid stimuli that provoke pain.
- No tenderness on percussion is observed.
- Radiographic examination shows no periapical changes.

➤ Conditions Mimicking Dentin Hypersensitivity

Several conditions can present with symptoms similar to DH but require different management approaches. These include:

- Dental caries
- Post-surgical/post-nonsurgical periodontal sensitivity
- Sensitivity after restoration or fractured restorations
- Cracked tooth syndrome
- Palatogingival grooves

V. DIAGNOSIS

Accurate diagnosis of dentin hypersensitivity (DHS) is essential to ensure effective treatment, often achieved through the exclusion of other conditions requiring different management approaches. The diagnostic process for pulp diseases heavily relies on pulp sensibility tests, which reproduce the patient's reported symptoms to identify the affected tooth and determine the disease state.

Pulp tests are broadly classified into sensibility and vitality tests. Sensibility tests, including thermal tests (heat and cold stimuli), electric pulp tests (EPTs), and test cavities, assess the nerve fibers' ability to respond to external stimuli. In contrast, vitality tests such as laser Doppler flowmetry (LDF), pulse oximetry, and tooth temperature measurement assess the blood supply within the pulp tissue.

However, these tests have inherent limitations. Sensibility tests, though commonly used, do not assess pulp health or vascular integrity and may yield unreliable results in cases of trauma, immature teeth, or following orthognathic surgery. Thermal tests are highly subjective, relying solely on the patient's perception, whereas EPTs provide more quantifiable results that can be compared over time. Despite this, no single test can definitively establish a diagnosis, making it essential to combine sensibility and vitality tests with other clinical assessments such as periodontal probing, percussion, palpation, mobility tests, transillumination, and anesthetic tests.

Clarifying terminology is crucial for accurate diagnosis and treatment. Sensibility refers to the pulp's response to stimuli, while sensitivity denotes heightened responsiveness that varies among individuals. Vitality, however, is defined by the presence of blood supply and can only be assessed through true vitality tests like LDF and pulse oximetry.

To enhance diagnostic accuracy, testing should follow a structured sequence: contralateral, opposing, asymptomatic teeth, and finally, the most suspicious tooth. This approach familiarizes the patient with the process and minimizes false responses. Repeating the test after a one-minute recovery period further improves reliability.

The rationale behind pulp sensibility testing lies in its protective function, alerting the patient to potential damage. Sharp, non-lingering pain upon thermal stimulation is a normal response, while exaggerated reactions may indicate lowered thresholds. Since A-delta fibers, which require less stimulus, are primarily activated during pulp testing, their response is often used to assess pulp status.

A. Thermal Tests

Sensory response to thermal stimuli occurs before there is a temperature change in the pulpo-dentinal junction (PDJ) area, where sensory nerve endings are located. Thus, it appears that the sensory response is not initiated by temperature changes in the receptors. Instead, thermal tests activate hydrodynamic movement of fluid within dentinal tubules, which excites the A-d fibres. The C fibres are not activated by these tests unless they produce injury to the pulp.

- Cold Stimulus: Stimulates the fast-conducting A-d fibres, which produce a sharp localized pain.
- Heat Stimulus: Stimulates the slower conducting C fibres, located deeper in the pulp, resulting in dull pain of longer duration.

B. Electric Pulp Test (EPT)

Electric pulp tests deliver a current sufficient to overcome the resistance of enamel and dentine and stimulate the A-d fibres. The non-myelinated C fibres do not respond to the conventional EPTs because significantly more current is needed to stimulate them.

➤ Interpretation of Test Results When Making a Diagnosis:

When performing a test, the clinician should evaluate the immediacy, intensity, and duration of the response. Importantly, it is the duration of the response, compared to the baseline that has been established by testing other teeth in the same patient, which may be the most helpful aid to diagnosis, especially when testing a tooth for pulpitis.

➤ Pulp Status Based on Test Results

- Clinically Normal Pulp: Mild to moderate transient response to cold and EPT. No response to heat tests.
- Reversible Pulpitis: Sharp pain from thermal stimuli subsides quickly.
- Irreversible Pulpitis: Sharp pain followed by prolonged dull ache lasting up to an hour.
- Pulp Necrosis: No response to EPT and thermal tests.
- Pulp Necrobiosis: Partial necrosis with infected pulp, showing mixed test results.
- Acute Apical Periodontitis: Pulp sensibility tests required alongside radiographs.
- Acute Apical Abscess vs. Lateral Periodontal Abscess: Differentiation through pulp sensibility tests and radiographs.
- Chronic Apical Periodontitis: Sequel to infected root canal system, confirmed using pulp sensibility tests and radiographs.

C. False Responses

False Negative Responses: More common and misleading, may occur due to:

- Calcified pulp space
- Pre-medication with analgesics or sedatives
- Extensive restorations or pulp-protecting bases
- High pain threshold
- Orthodontic appliance activation

➤ False Positive Responses: Less Common but Possible due to:

- Conduction of current to adjacent tissues
- Moisture in canal or inflamed tissue
- Breakdown products capable of conducting current
- Adjacent metallic restorations
- Multi-rooted teeth with partially necrotic pulp

While EPTs and thermal tests are simple non-invasive tests, they are not completely reliable. Heat tests have high sensitivity but low specificity, making cold tests more accurate than EPTs.

D. Thermal Pulp Tests

Thermal pulp sensibility tests are among the oldest and most common methods used to assess the health of the pulp and its ability to respond to external stimuli. These tests are

often incorrectly referred to as 'vitality tests' since they cannot determine whether a tooth has a viable blood supply. Thermal sensitivity is a clinical indicator of pulp inflammation, making these tests a standard approach for pulp assessment.

E. Types of Thermal Tests:

➤ Cold Tests

Cold tests are generally considered more accurate than heat tests for assessing the state of the pulp, particularly for identifying pulp necrosis. Cold tests stimulate the A-delta (A-d) fibers, which transmit a sharp, localized pain response. However, they are not always reliable in cases where the pulp is calcified or where secondary and reactionary dentin acts as an insulating barrier.

➤ Methods of Cold Testing

- *Ice Sticks*
 - ✓ Ice sticks are formed by freezing water in hypodermic needle covers or sterilized local anesthetic cartridges.
 - ✓ Applied to the cervical or middle third of the buccal or lingual crown.
 - ✓ Application should last for 5 seconds or until the patient experiences pain.
 - ✓ Major disadvantage: Ice sticks are often not cold enough to provide conclusive results.
- *Refrigerant Sprays*
 - ✓ Most convenient and widely used method.
 - ✓ Common refrigerant sprays include 1,1,1,2-tetrafluoroethane (TFE), available as Green Endo-Ice (-26.2°C).
 - ✓ Application provides reliable and reproducible results.
- *Carbon Dioxide Snow (CO₂ or Dry Ice)*
 - ✓ Produces a temperature of approximately -78°C, but clinically, the applied temperature may be around -56°C.
 - ✓ Rapid sensory response, usually within 2 seconds.
- *Ethyl Chloride*
 - ✓ Chloroethane with a temperature of -12.3°C.
 - ✓ Available as a compressed spray and used commonly as a skin refrigerant.

• Cold Water Bath

A group of teeth can be isolated using a rubber dam, and iced water can be syringed onto the tooth.

➤ Heat Tests

Heat tests are used less frequently but are beneficial in cases where cold tests provide inconclusive results. Heat stimulates A-d fibers initially and C fibers with prolonged exposure, causing a delayed and longer-lasting pain response.

• Methods of Heat Testing

✓ Heated Gutta-Percha (Grossman's Method)

Gutta-percha is heated to 120–140°C and applied to a dry tooth protected with petroleum jelly.

- Warmed Hand Instruments

- Ball burnishers or other instruments are heated and brought close to the buccal surface of the crown to transfer radiant heat.

✓ *Electrical Heat Sources*

Devices such as Touch'N Heat and System B provide a hot stimulus to teeth.

✓ *Frictional Heat*

Generated using a rubber cup rotated on the buccal aspect of a tooth without prophylactic paste.

✓ *Hot Water Bath*

Rubber dam isolates the tooth, and hot water is syringed onto it for 5 seconds.

- Clinical Implications:
- *Cold Sensitivity*: Indicates early pulp inflammation.
- *Heat Sensitivity*: Suggests advanced pulp deterioration or irreversible pulpitis.
- *Delayed or Absent Response*: May indicate pulp necrosis.

Thermal pulp tests, when combined with EPT and radiographic analysis, provide a comprehensive evaluation of pulp health and help guide accurate diagnosis and treatment planning.

VI. MANAGEMENT

The management of dentin hypersensitivity (DH) involves a systematic approach that aims to reduce pain, address underlying causes, and prevent recurrence. Successful management requires identifying etiological and predisposing factors, implementing preventive measures, and selecting appropriate desensitizing agents.

The therapeutic management of dentin hypersensitivity (DH) is categorized into *at-home treatments* and *in-office treatments*, depending on the severity of the condition and the patient's response to initial measures. These approaches aim to alleviate sensitivity by either disrupting nerve transmission or occluding the dentinal tubules to minimize fluid movement.

A. At-Home Therapeutic Treatments:

At-home treatments are primarily aimed at managing mild to moderate DH and include various desensitizing agents available in toothpaste, mouthwashes, and gels. These products act through different mechanisms to provide relief by altering nerve excitability, precipitating proteins, or occluding dentinal tubules.

➤ *Potassium Salts*

Potassium salts, such as potassium nitrate, potassium chloride, and potassium citrate, are widely used in desensitizing toothpastes and mouthwashes. These agents reduce pain by altering the nerve potential, preventing repolarization after depolarization, and reducing nerve excitability.

➤ *Mechanism of Action:*

- Alteration of Nerve Potential: Potassium ions diffuse through dentinal tubules, decreasing the excitability of nerve fibers.
- Protein Precipitation: Precipitates formed by potassium salts occlude the dentinal tubules.
- Tubule Occlusion: Potassium ions block dentinal tubules by forming potassium precipitates.

➤ *Sodium Fluoride*

Sodium fluoride (NaF) plays a dual role in DH management by remineralizing the dentin surface and occluding exposed dentinal tubules. The fluoride ions interact with calcium and phosphate ions to form a protective mineral layer, thereby reducing permeability and alleviating pain.

➤ *Stannous Fluoride*

Stannous fluoride (SnF₂) is highly effective in sealing dentinal tubules due to its strong affinity to tooth surfaces. It forms a protective layer that penetrates dentinal tubules up to 20 µm deep, providing extended relief. However, it may cause staining and a metallic taste, which can reduce patient compliance.

➤ *Arginine*

Arginine-based formulations contain 8% arginine and calcium carbonate, which interact to create a calcium-rich layer that occludes dentinal tubules. Arginine also maintains an alkaline environment that facilitates remineralization and provides long-lasting relief.

➤ *Nano-Hydroxyapatite (n-HA)*

Nano-hydroxyapatite, structurally similar to natural hydroxyapatite, integrates into the dentin surface and replenishes lost mineral content. It occludes dentinal tubules by mimicking the natural mineral composition of tooth tissues, thereby reducing DH.

➤ *Strontium Chloride*

Strontium chloride (SrCl₂), commonly available at a 10% concentration, substitutes calcium ions in the hydroxyapatite structure, leading to the formation of strontium apatite complexes. These complexes effectively occlude dentinal tubules and stimulate dentinogenesis, contributing to long-term DH reduction.

B. Occlusive Approach – Aiming at Dentinal Tubular Blocking/Plugging:

The occlusive approach for managing dentin hypersensitivity (DH) involves the application of agents that physically or chemically block dentinal tubules, reducing the movement of fluids within them and subsequently minimizing pain. The agents employed in this approach can be broadly classified into two categories:

➤ *Varnishes*

Early formulations of copal varnishes formed a protective layer over dentinal tubules, blocking the passage of external stimuli and reducing sensitivity. However, these varnishes had limited efficacy due to their short duration of action and the need for frequent reapplication. Modern

fluoride varnishes, on the other hand, provide prolonged exposure to fluoride ions, enhancing their efficacy and safety. Fluoride varnishes set rapidly upon application, minimizing the risk of ingestion and ensuring extended contact with the tooth surface, allowing slow and sustained fluoride release.

➤ *Sodium Fluoride*

Sodium fluoride (NaF) is one of the most commonly used fluoride agents in managing DH. It reduces dentin permeability by forming insoluble calcium fluoride (CaF₂) crystals within dentinal tubules. However, NaF-based varnishes have limited longevity, as the formed crystals can be dislodged by saliva and brushing. To improve the stability of the formed precipitates, acidulated phosphate fluoride (APF) with 1.23% fluoride (12,300 ppm, pH 3.2) is preferred, as it facilitates the formation of fluoroapatite, which exhibits better resistance against saliva, brushing, and dietary acids.

➤ *Silver Diamine Fluoride (SDF)*

Silver diamine fluoride (SDF) is another effective agent for DH management, known for its ability to occlude dentinal tubules. Upon contact with hydroxyapatite, SDF forms silver fluoride (AgF) and silver chloride (AgCl) compounds, which create a barrier against external stimuli, reducing sensitivity. However, SDF may cause temporary discoloration of the treated area due to the formation of silver compounds. To mitigate this, potassium iodide (KI) is often applied alongside SDF to reduce staining.

➤ *Resins*

Composite resins, composed of organic and inorganic components, are widely used in restorative dentistry and can be modified to include desensitizing agents. They provide effective tubular sealing by forming a physical barrier and also release fluoride, reducing the risk of acid attacks. Resins with incorporated fluoride components offer dual benefits—providing mechanical strength and sustained fluoride release for long-term DH management.

➤ *Dentin Bonding Agents (DBAs)*

Dentin bonding agents (DBAs) achieve tubular sealing through micromechanical interlocking and chemical bonding with hydroxyapatite crystals in dentin. These agents penetrate dentinal tubules, replacing dissolved hydroxyapatite and creating a hybrid layer that prevents fluid movement. Common monomers used in DBAs include hydroxyethyl methacrylate (HEMA) and bisphenol-glycidyl methacrylate (bis-GMA), which exhibit amphiphilic properties that facilitate better interaction with both hydrophilic and hydrophobic environments.

➤ *Glass Ionomers*

Glass ionomers, composed of polyacrylic acid, alkenoic copolymers, and glass-filler particles, bond chemically to dentin through a dual mechanism—micromechanical hybridization and ionic bonding between polyalkenoic acid and calcium in hydroxyapatite. Resin-modified glass ionomers (RMGIs) provide better adhesion, fluoride release, and enhanced durability.

➤ *Glutaraldehyde-Based Agents*

Glutaraldehyde-based agents, such as Gluma (Heraeus Kulzer), are composed of 5% glutaraldehyde and 35% hydroxyethyl methacrylate (HEMA). These agents coagulate plasma proteins within dentinal tubules, creating a multilayered protein barrier that reduces osmotic fluid exchange and alleviates DH.

➤ *Portland Cement*

White Portland cement, enriched with calcium silicates, shows potential in occluding dentinal tubules through dentin remineralization. It releases calcium and hydroxide ions upon contact with moisture, contributing to dentin remineralization and improving structural integrity. However, its application in DH management is not yet a standardized protocol in dental practice.

➤ *Oxalate Salts*

Oxalate salts, such as potassium oxalate and ferric oxalate, occlude dentinal tubules by forming insoluble calcium oxalate crystals, effectively blocking the tubules and reducing dentin permeability by up to 98%. Etching the dentin surface before applying oxalates enhances their effectiveness, although they may cause tooth discoloration or form insoluble deposits.

VII. RESTORATIVE APPROACH

Restorative interventions offer a durable solution by physically covering exposed dentin, which is often necessary due to the limitations of sealers and varnishes that may wear off over time. The decision to restore is based on the extent of tooth loss, esthetic concerns, and the patient's oral hygiene and maintenance capabilities.

➤ *Restorative Materials and Techniques*

- **Glass Ionomer Cement (GIC):** Chemically bonds to dentin and releases fluoride, offering reasonable adhesion and moisture tolerance. However, its poor esthetics and susceptibility to fracture limit its use.
- **Resin-Modified Glass Ionomer Cement (RMGIC):** An improved version of GIC with better esthetic and physical properties, preferred for cervical restorations despite its higher cost and maintenance challenges.
- **Microfilled/Flowable Resin Composite:** Offers better esthetics and wear resistance while flexing with the tooth to reduce marginal breakdown. It ensures long-lasting occlusion of dentinal tubules.
- **GIC/RMGIC Liner Laminated with Resin Composite:** Combines the benefits of fluoride release from GIC with the superior bonding and esthetic properties of composite, ensuring prolonged tubular occlusion.

➤ *Indications for Restorative Treatment-*

Restorative approaches are indicated in cases involving cervical erosion, structural loss due to attrition or parafunctional habits, and severe gingival recession with persistent sensitivity.

VIII. SURGICAL AND ENDODONTIC

➤ *Surgical Correction for Soft Tissue Defects*

Periodontal Surgical Procedures: Includes guided tissue regeneration (GTR), coronally advanced flap (CAF), connective tissue grafting (CTG), and free gingival grafts (FGG) to cover exposed dentin and manage gingival recession. However, long-term stability remains debatable, and restorative management may be required in persistent cases.

➤ *Endodontic Therapy*

Considered a last resort when DH persists despite occlusive and restorative measures. Root canal therapy removes inflamed pulp tissue and seals the root canal system to prevent further irritation.

❖ *Tooth Whitening Considerations for DH Patients*

➤ *Risks and Precautions*

Bleaching agents increase dentin permeability, risking transient pain. Pre-treatment measures, such as using desensitizing toothpaste, fluoride varnish, and sealants, can reduce sensitivity before whitening. However, these interventions should not interfere with bleaching efficacy.

IX. RECENT TRENDS IN DENTIN HYPERSENSITIVITY (DH) MANAGEMENT

➤ *Casein Phosphopeptide-Amorphous Calcium Phosphate (CPP-ACP)*

CPP-ACP, derived from milk casein proteins and marketed as GC Tooth Mousse (GC Asia Pty. Ltd., Japan), acts by buffering free calcium and phosphate ions, promoting remineralization, and forming a protective crystalline layer that reduces dentin permeability. While effective, it requires regular application and may not create a uniform tubule covering.

➤ *Propolis*

Propolis, a resin produced by bees, contains flavonoids, amino acids, and bioactive compounds that provide antibacterial, antifungal, antiviral, anti-inflammatory, and anesthetic effects. It promotes tissue repair and occludes dentinal tubules, reducing hypersensitivity.

➤ *Topical Guanethidine*

Guanethidine reduces dentinal hypersensitivity by inhibiting nerve transmission. While preliminary research suggests potential benefits, further clinical trials are needed to validate its efficacy and establish standardized treatment protocols.

➤ *Bioglass*

Bioglass, or calcium sodium-phospho-silicate glass (NovaMin®), releases calcium and phosphate ions, forming a hydroxycarbonate apatite (HCA) layer that mimics hydroxyapatite. This occludes dentinal tubules, reduces fluid movement, and promotes long-term remineralization. Air abrasion with bioactive glass particles enhances tubule occlusion and remineralization through chemical reactions.

➤ *Bleaching Agent with Amorphous Calcium Phosphate (ACP)*

ACP-enriched bleaching gels reduce hypersensitivity during and after whitening by preventing demineralization and facilitating enamel and dentin remineralization. This maintains enamel integrity, reducing post-treatment discomfort.

➤ *Laser Therapy*

➤ *Low-Power Lasers (LLLT or Cold Lasers)*

These lasers, such as GaAlAs diode lasers (685–980 nm), are cost-effective and promote biomodulation of the inflammatory response by stimulating mitochondrial activity and altering Na⁺/K⁺ pump polarity, blocking painful stimuli.

➤ *High-Power Lasers*

High-power lasers, including Nd:YAG (1064 nm), Er,Cr:YSGG (2780 nm), and Er:YAG (2940 nm), melt and resolidify dentin, forming a glazed, non-porous layer that resists acidic challenges. Despite their effectiveness, limitations include high costs, challenges in treating inaccessible areas, and potential pulp damage due to increased temperature.

➤ *Combined Protocols*

Combination protocols, such as laser therapy paired with desensitizing agents, offer dual benefits by physically blocking tubules and providing neural desensitization. This approach results in longer-lasting effects and improved patient satisfaction.

X. RECALL/MAINTENANCE FOR DENTIN HYPERSENSITIVITY MANAGEMENT

Despite extensive research, dentin hypersensitivity (DH) remains a clinical challenge with unpredictable outcomes. To ensure long-term success, a systematic recall program, known as the “3C’s Approach”, has been proposed to customize patient care plans.

- *Continue* is recommended when mild or persistent DH remains despite improvement, requiring continued use of the desensitizing agent and reinforcement of oral hygiene practices.
- *Change* is suggested when DH persists despite active therapies, requiring a switch to an alternate agent, supplementation with another, or transitioning from home-care to in-office procedures.
- *Cease* is applied when DH is in complete remission, where active treatment is discontinued, and focus shifts to maintaining oral hygiene and behavior modification to prevent relapse.

By following this approach, clinicians can effectively manage DH, ensuring prolonged relief and improved patient outcomes.

XI. CONCLUSION

Dentin hypersensitivity (DH) is a common but often underdiagnosed condition that affects the quality of life due to its multifactorial etiology, including gingival recession, enamel wear, and occlusal stress. Accurate diagnosis requires a thorough patient history and clinical examination to distinguish DH from similar conditions like caries, pulpitis, and periodontal disease. Initial management involves desensitizing toothpaste containing potassium nitrate, strontium chloride, or arginine, which provides effective relief for most patients. However, persistent cases may require in-office treatments such as fluoride varnishes, dentin-bonding agents, or laser therapies, with combination protocols offering superior outcomes.

Long-term success in managing DH relies on patient education and regular follow-ups to prevent recurrence. Educating patients about oral hygiene practices, dietary modifications, and adherence to treatment helps minimize relapse. Advancements in dental materials and technologies hold promise for more predictable and effective management of DH. Ultimately, a comprehensive, patient-centered approach that combines preventive strategies, targeted treatment protocols, and consistent follow-up ensures better oral health and overall well-being for affected individuals.

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