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DENTIN HYPERSENSITIVITY-A REVIEW

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ABSTRACT

The aim of this review is to provide dentists with comprehensive information regarding dentin hypersensitivity. This includes presenting its etiology, outlining the process of diagnosis, discussing clinical management strategies, and exploring technical approaches aimed at alleviating sensitivity. Dentin hypersensitivity is characterized by distinctive short, sharp pain arising from exposed cervical dentin in response to various external stimuli. The etiological factors cause two specific changes in teeth. First, the dentin surface must be exposed and denuded, which requires the loss of enamel or gingival recession combined with the loss of cementum. The second condition is the opening of the dentin tubules to allow the sensory mechanisms in the pulpal area following stimulation of the dentin surface. The accurate diagnosis of dentin hypersensitivity before receiving therapies is critical for successful treatment. The diagnosis of the disease starts through investigating the medical history of the patient and examination.Generally, the primary approach in addressing dentin hypersensitivity involves the utilization of toothpaste containing potassium salts and fluoride. Newly introduced materials and in-office methods for treating dentin hypersensitivity include bioactive glasses, iontophoresis, , and lasers.

INTRODUCTION

"Dentin hyperpersensitivity is an enigma being frequently encountered yet ill understood," a statement made by Johnson and colleagues in 1982 .It's been used for many years to characterize a prevalent painful dental ailment. According to the Canadian consensus document[1] , dentin hypersensitivity has been defined as “pain derived from exposed dentin in response to chemical, thermal tactile or osmotic stimuli which cannot be explained as arising from any other dental defect or disease”.

Dentin hypersensitivity is a prevalent oral problem affecting more than 40% of the adults. Patients with periodontal diseases are at particularly high risk, and some studies have found that over 70% of patients with periodontal disease have experienced dentin hypersensitivity.

The incidence can vary considerably depending on the cohort being studied with periodontal patients, patients with gingival recession and smokers with periodontitis showing the highest incidence of diagnosed dentinal hypersensitivity.[2]

Several studies have reported noncarious cervical lesions (NCCLs) and dentin hypersensitivity in adult populations, with prevalence rates ranging from 5% to 85% and 2-8% to 74%, respectively.[3]It is also known that the oral health related quality of life in patients with DHS can be improved after DHS has been treated successfully.[4]

The teeth most commonly affected by dentinal hypersensitivity are the upper premolars followed by the upper first molars with the incisors being the least sensitive teeth. It has been reported that there is a slightly higher incidence of dentine hypersensitivity in females compared to males .[5]

HISTORICAL BACKGROUND

Dentin hypersensitivity is mainly a cause of exposed dentin or tooth wear as a result of loss of enamel. Tooth wear, either natural or artificial, has proved a fascination to mankind since the dawn of history. Attrition in ancient material can be excessive not only due to massive muscle attachments, but also to the unrefined nature of the diet. Wear patterns on the enamel surface were studied usually by scanning electron microscope and they were usually due to silica phytoliths and recently it has been suggested that these can be classified and related to individual plant stems.[6]

ANATOMY OF DENTINAL TUBULES

Odontoblast processes, similar to osteocyte processes, run in canaliculi that traverse the dentin layer and are referred to as dentinal tubules .[7]

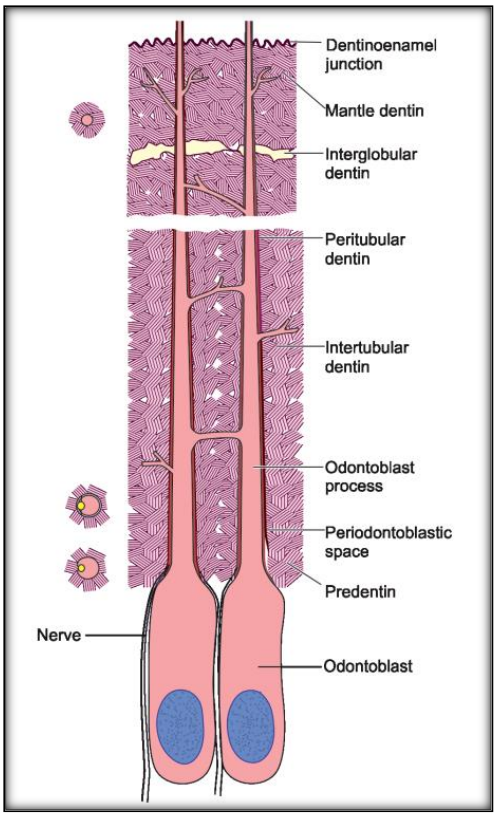

Figure 1 :Diagram of the odontoblast and its process in the dental tubule.[8]

SYNONYMS FOR DENTIN HYPERSENSITIVITY

Dentin hypersensitivity/sensitivity , Dentinal hypersensitivity/sensitivity,Cemental hypersensitivity/sensitivity,Root hypersensitivity/sensitivity,Cervical hypersensitivity/sensitivity[9]

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PATHOPHYSIOLOGY OF DENTIN HYPERSENSITIVITY

Three mechanisms, all involving an understanding of the structure of dentin and pulp, have been proposed to explain dentin sensitivity:

(1) The dentin contains nerve endings that respond when it is stimulated;

(2) The odontoblasts serve as receptors and are coupled to nerves in the pulp; and

(3) The tubular nature of dentin permits fluid movement to occur within the tubule when a stimulus is applied, a movement registered by pulpal free nerve endings close to the odontoblasts.[7]

THEORIES OF DENTIN HYPERSENSITIVITY

Five theories that have been proposed are Direct innervation theory ,Odontoblast Receptor Theory ,Transducer Theory ,Modulation Theory ,Gate Control Theory and Hydrodynamic Theory.The most accepted theory is the hydrodynamic theory which was first hypothesized btGysi.

HYDRODYNAMIC THEORY

Fish, referring to the interstitial fluid of the dentin as dental lymph, stated that pressure variation in the surrounding can bring about change in the fluid flow.[15]This change in fluid flow can cause dentinalgia according to Brannstrom and Astrom, which subsequently brings about deformation in the odontoblast, or its process and elicits a response perceived as pain by the stimulation of associated "mechanoreceptor like" nerve ending.[10,11]

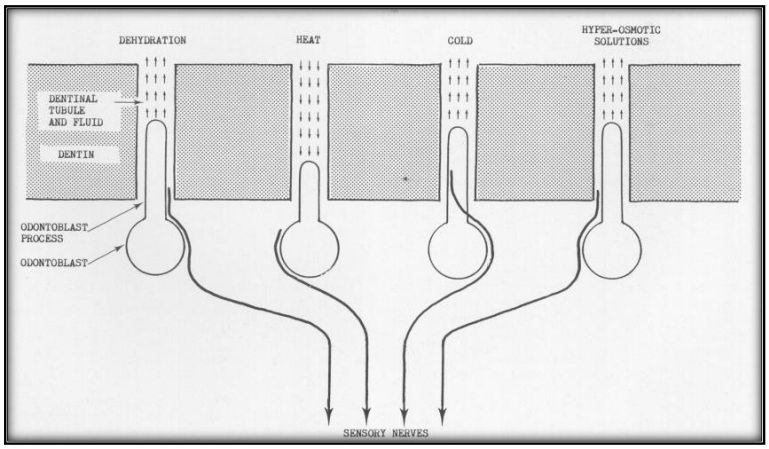

Figure 2:Diagramatic depiction of dentinal components. With the application of various stimuli (dehydration, heat, cold, or hyper-osmotic solutions), there will be a subsequent movement ofthedentinalfluid within the tubules, and consequential agitation oftheodontoblast, its process and associated sensory nerves. This is the basis ofthe Hydrodynamic Theory ofdentinal sensation, as described by Brannstrom[11]

ETIOLOGY

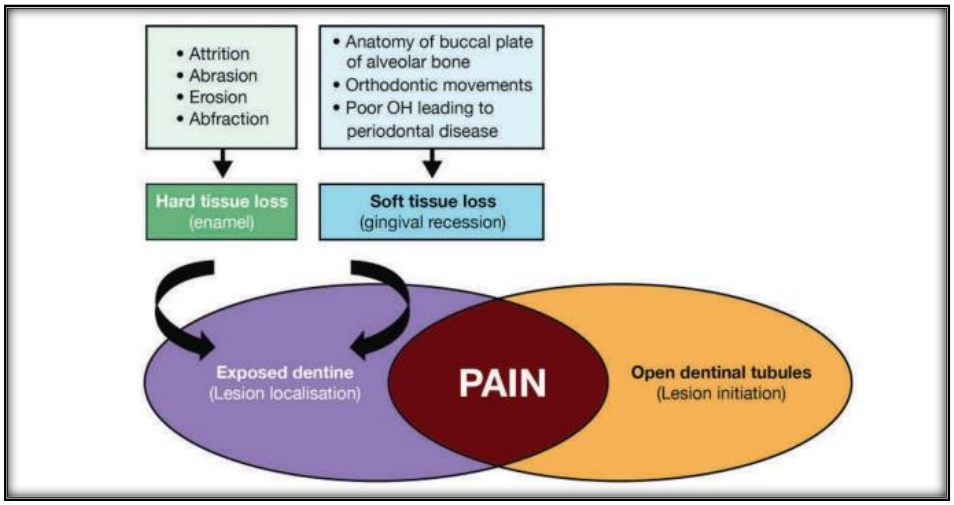

Figure 3: Prerequisites for dentine sensitivity. OH, oral hygiene.[12]

An understanding of the etiology of any disease or condition is essential for effective prevention and treatment. The current knowledge of the etiology of dentinal hypersensitivity is still limited and the factors that lead to a dentinal exposure are still uncertain.[24]Dentin in normal conditions is coated by enamel and cementum, not presenting sensitivity to external stimuli.Thus, the dentin starts presenting hypersensitivity only when exposed to the mouth environment, after the wear of the protection structures. The enamel layer can be removed by attrition from occlusal wear and parafunctional habits, toothbrushing abrasion, erosion from acids, coronal fracture, abfractions or defective fillings. Gingival recession, periodontal disease, periodontal surgery and incorrect toothbrushing can expose root surfaces.

Reasons for continued dentinal exposure are poor plaque control, i.e., acidic bacterial byproducts,excess oral acids, i.e., sodas, fruit juice, swimming pool chlorine, bulimia,cervical decay·toothbrush abrasionand gingival recession.[13] Gingival recession is one of the most common reason for dentin hypersensitivity.

Reasons for gingival recessionare inadequate attached gingiva,prominent roots ,low-level and long-lasting trauma ,chronic inflammatory periodontal disease ,periodontal treatment ,occlusal trauma,etc.[14]

PHASES OF DEVELOPMENT OF DENTIN HYPERSENSITIVITY

- Lesion localization- In this phase, dentinal tubules, due to loss of enamels, are exposed by attrition, abrasion, erosion, and abfraction.
- Lesion initiation - In this phase, for the exposed dentin to be sensitized, the tubular plugs and the smear layer are removed and consequently, dentinal tubular and pulp are exposed to the external environment .[15,16]

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DIAGNOSTIC METHODS AND INSTRUMENTS FOR DENTIN HYPERSENSITIVITY

In order to determine an appropriate and effective treatment, it is critical to arrive at a correct diagnosis. A thorough patient history and clinical examination are required.

Diagnosis of dentin hypersensitivity includes:

Case History : Includes nature of pain, intensity, frequency, duration, and stimuli causing pain.During the appointment, it is therefore essential that clinicians obtain a thorough medical history,dental history and history of pain from patients to enable them to take into account all relevant information prior to formulating a treatment plan.

Chief complaint and illness history : The chief complaint provides primary subjective information for the diagnosis of dentin hypersensitivity, but it can be misleading for inexperienced practitioners..[17]

Clinical examination: Various tests like radiographic examination, percussion test, and vitality test to rule out pulpal involvement are done.

- Tactile or mechanical stimulus: This is done using No. 23 explorer with a force of 5 - 10 gm.
- Thermal stimulus: Using cold-water of varying temperature and heat test (Guttapercha, hot water) or blowing air from a distance of 1 cm.
- Osmotic test: Cotton applicator saturated with sucrose solution is applied for a period of 10 seconds.[18]
- Scratch device: Made by Smith and Asc in 1964. In this, stainless steel wire moves along the highest arc of curvature on the facial surface of the sensitive tooth. Scratching force could be increased by a screw. A tooth that fails to respond at 80 centinewtons is said to be nonsensitive.
- Yeaple probe: It is a pen-like device that was introduced in 1990. It contains a handpiece with a probe tip and electronic control unit that allows the force of 0.05 - 1 N. [19]

ASSESSMENT METHODS OF DENTIN HYPERSENSITIVITY

Dentine sensitivity may be evaluated either in terms of the stimulus intensity required to evoke pain (stimulus-based assessment), or as the subjective evaluation of the pain produced by a stimulus (response-based assessment).[20]

Stimulus based assessment:

Stimulus-based methods usually involve the measurement of a pain threshold whwreas response-based methods involve the estimation of pain severity. In stimulus-based investigations of dentin sensitivity, the stimulus is adjusted with varying levels of intensity. One frequently used approach in this category involves utilizing a probe that is adjusted to exert forces on the tooth in increments of 10 g, employing an electronic device for control . In this method, the patient is directed to indicate the point at which they initially experience pain subsequent to the probe stimulation, and the applied force is recorded. While thermal and electrical devices have been previously used to generate increasing stimulus intensities, concerns have arisen regarding their efficacy .[21]

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Response based assessment:

1)Visual Analogue Scale(VAS)

During this method, adjacent teeth are isolated using the examiner's fingers, and a stream of air with a pressure of 60 ± 5 psi and a temperature of 21 ± 1 °C is directed perpendicularly to the tooth under examination from a distance of 1 cm for a duration of 1 s. Immediately following the stimulus, the patient rates the pain by visually recording it on an analog table (visual analogue scale—VAS), which features gradations spanning from no pain to the most intense pain.[22]

2)Schiff's scale

Schiff's scale of sensitivity to cold air, frequently introduced, comprises four distinct gradations as follows :

0 'subject does not respond to air stimulus',
1 'subject responds to air stimulus but does not request discontinuation of stimulus'
2 'subject responds to air stimulus and requests discontinuation or moves from stimulus'
3 'subject responds to the air stimulus, considers stimulus to be painful and requests discontinuation of the stimulus'

3)Verbal Descriptor scale(VDS)[22]

Keele (i948) described a 4-point scale grading pain as slight, moderate, severe and agonising. This simple descriptive pain scale has been modified and a typical VDS may look like the following:Scoring criteria for VDS is as follows :

0 'no discomfort'
1 'mild discomfort'
2 'marked discomfort'
3 'marked discomfort that lasted more than 10 s'

DIFFERENTIAL DIAGNOSIS OF DENTIN HYPERSENSITIVITY

Flowchart 1: Flow chart for differential diagnosis of dentin hypersensitivity (DHS). The diagnostic process for DHS includes reviewing chief compliant, illness history and predisposing factors, and clinical exam, stimulation test, and radiographic examinations, if necessary.[23]

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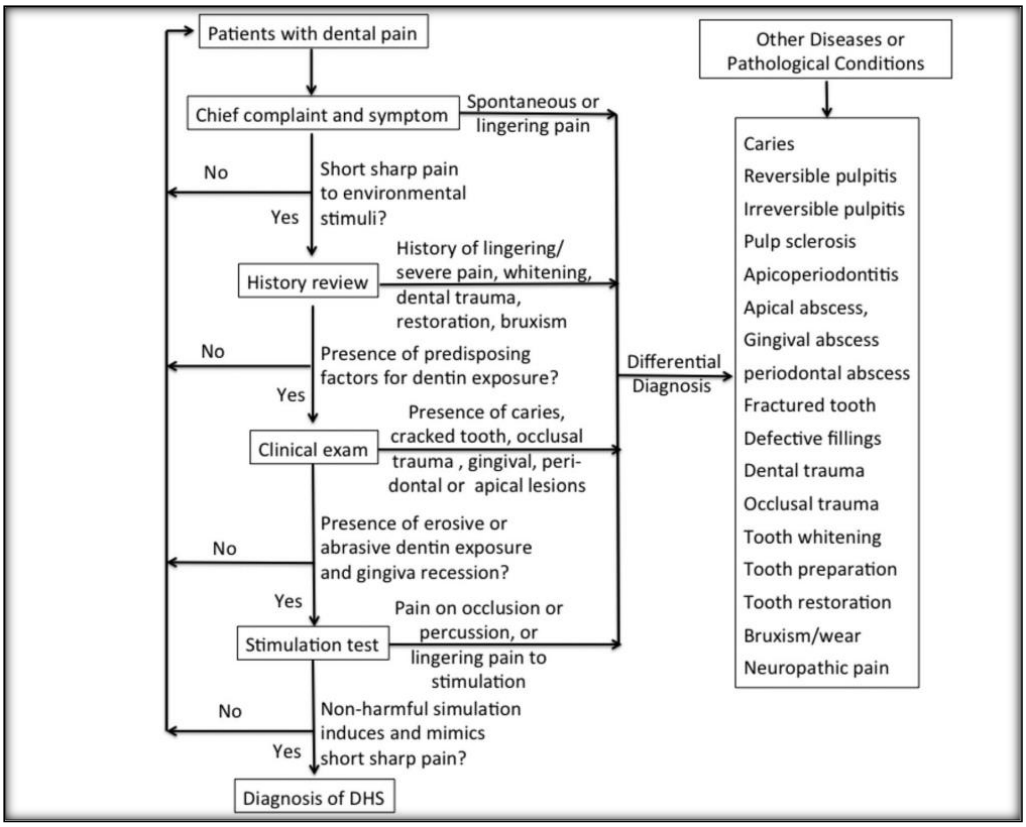
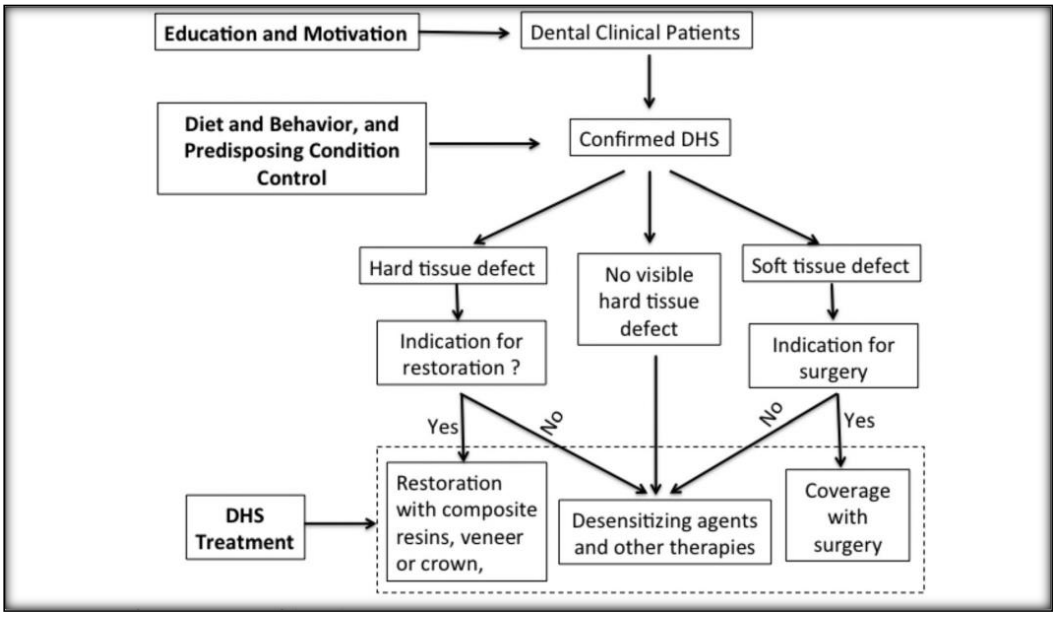


TREATMENT OF DENTIN HYPERSENSITIVITY

The treatment of dentine hypersensitivity is challenging .There are certain strategies for the management of DHS which include[23]

Flowchart 2:Strategies for managements of dentin hypersensitivity



Treatments can be categorized based on whether they can be applied by the patient (over-the-counter) or require professional application(in – office treatment)[24]

Classification of desensitizing agents based on:[25]

MODE OF ADMINISTRATION

- At home treatment (Over the counter products)
- In-office treatment

MECHANISM OF ACTION

Nerve desensitization

- Potassium nitrate

Protein precipitation

- Gluteraldehyde
- Silver nitrate
- Zinc chloride
- Strontium chloride hexahydrate

Plugging dentinal tubules

- Sodium fluoride
- Stannous fluoride
- Strontium chloride
- Potassium oxalate
- Calcium phosphate
- Calcium carbonate
- Bio active glasses (SiO_2–P_2O_5–CaO– Na_2O)

Dentine adhesive sealers

- Fluoride varnishes
- Oxalic acid and resin
- Glass ionomer cements
- Composites
- Dentin bonding agents

Lasers

- Neodymium:yttriumaluminum garnet (Nd-YAG) laser
- GaAlAs (galium-aluminium-arsenide laser)
- Erbium-YAG laser

Homeopathic medication

- Propolis

PREVENTIVE MEASURES

Table 1:Prevention involves the measures taken to eliminate the etiological or predisposing factors causing dentinal hypersensitivity .

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Preventive Recommendations for Dentin Hypersensitivity

Suggestions for patients	Suggestions for clinicians
1. Use of soft to medium bristle toothbrushes.	1. Proper instrumentation on the root surfaces during scaling and root planing.
2. Avoid overbrushing with excessive pressure or for an extended period.	2. Avoid over-polishing exposed dentin during stain removal.
3. Avoid brushing teeth immediately after ingesting acidic foods.	3. Avoid violating the biologic width during restoration, as this may cause recession.
4. Avoid improper use of other interproximal cleaning devices.	4. Avoid burning the gingival tissues during bleaching and advise patients to be careful when using home bleaching products.
5. Avoid picking or scratching at the gumline or using toothpicks inappropriately.	

FUTURE PROSPECTS

Newer agents for management of dentin hypersensitivity are as follows [26]

1) CASEIN-PHOSPHOPEPTIDE-AMORPHOUS CALCIUM PHOSPHATE (CPP) -(ACP)

   A minimum of 6 weeks of topical application is recommended. It prevents the dissociation of calcium and phosphate ions and maintains their availability.

2) PRO-ARGININE TECHNOLOGY

   Arginine being positive charge binds to negatively charged tubule. It plugs and seals the tubule and makes them resistant to acid. Usually, the effect lasts for a minimum of 28 days.

3) NANOMATERIALS IN THE MANAGEMENT OF DENTIN HYPERSENSITIVITY

   It includes material with a dimension less than 100 nm. This works by occluding the tubule and lasts a minimum of 7 days. The agents from this group include nanostructure bioactive glass, novamin, nanohydroxyapatite.

4) OTHERS

   Portland cement, Propolis, etc. However, many times these agents fail to provide relief. In such scenarios, root canal treatment is considered as the last resort for relief of pain.

CONCLUSION

Proper diagnosis of dentin hypersensitivity is important for the management of dentin sensitivity. Various treatments for dentin sensitivity include professional in-office, at-home, and over-the-counter treatments. Desensitizing toothpaste is the least invasive and the most cost-effective treatment. The treatment for dentin hypersensitivity should not only focus on relieving pain but should also focus on curing the etiology and dental problems. Management of dentinal hypersensitivity should be done with more conservative strategies first, followed by irreversible dental interventions.

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