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Tooth Hypersensitivity

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By

Nabaa Abdul Rida Hammood

Supervised by

Dr. Bashair Abdul Sahib

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Certification of the Supervisor

I certify that this project entitled " **Tooth Hypersensitivity**"

was prepared by the fifth-year student **Nabaa Abdul Rida Hammood** under my supervision

at the College of Dentistry/University of Baghdad in partial fulfilment of the graduation

requirements for the Bachelor Degree in Dentistry.

Supervisor's name

Dr. Bashair Abdul Sahib

Date

Dedication

This review is dedicated to my supporting family and loving friends who were with me through thick and thin.

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This review was not possible if it weren't for the support and guidance of my supervisor **Dr. Bashair Abdul Sahib.**

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Abbreviations	Meaning
DH	dentin hypersensitivity
SEM	scanning electron microscope
DI	direct innervation
OR	odontoblast receptor
DEJ	dentinoenamel junction
CDS	cervical dentinal sensitivity
RDA	relative dentin abrasivity
OTC	over-the-counter
CPP-ACP	Casein phosphopeptide-amorphous calcium phosphate
CO2	carbon dioxide

Introduction

Different terms have been used to describe dentin hypersensitivity. These terms are used based on the place of occurrence of hypersensitivity and include: cervical, root, dentine, cemental, and the terms sensitivity, and hypersensitivity. All of these terms convey the same clinical conception and can be used interchangeably. **(Davari et al, 2013)**

Dentin hypersensitivity can be defined as a short, sharp pain that arises from exposed dentin in response to stimuli (typically thermal, evaporative, tactile, osmotic or chemical) and that cannot be ascribed to any other form of dental defect or pathology.

Conditions that should be ruled out include dental caries, pulpitis, fractured teeth, fractured restorations, post-restorative sensitivity, marginal leakage, chipped teeth and gingival inflammation. Dentin hypersensitivity is, therefore, a diagnosis of exclusion.

The problem appeared to be intermittent, affecting most participants “occasionally” and few participants “always”. **(Cunha-Cruz et al, 2013)**

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

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1.1. Etiology

Patients have reported that pain was initiated mainly by cold drinks but also by hot drinks, toothbrushing and sweet foods.

Dentin tubules may become exposed as a result of enamel loss from attrition, abrasion, erosion (acid dissolution) or abfraction (cervical stress lesion), but dentin exposure often may be a result of gingival recession and cementum loss from root surfaces, most frequently in canines and premolars. The enamel and cementum loss may be visible clinically as non-carious cervical lesions. A diet rich in acidic liquids and foods, occupational exposure to acids, use of tooth-whitening agents and gastric reflux have been implicated as causes of dental erosion. Aggressive or frequent toothbrushing and periodontal treatment (such as scaling and root planing) may contribute to gingival recession, cementum loss and subsequent dentin exposure. **(Cunha-Cruz et al, 2013)**

DH is multifactorial, wherein more than one causative factor always plays a role, either directly or indirectly. **(Haneet et al, 2016)**

Investigators in studies conducted in general dental practices have reported that the prevalence were 52 percent, 42.4 percent, 40.3 percent, 15 percent, 25 percent, 4.1 percent, 3.8 percent and 1.3 percent. The reason for this wide range of prevalence might be explained by how dentin hypersensitivity was estimated, by means of self-reports or questionnaires, which can provide a higher prevalence than that estimated by means of a specific clinical examination.

Patients tried at-home treatments for dentin hypersensitivity, but most reported that the treatment had no effect or that relief lasted less than six weeks.

At-home and in-office treatments are more often than not ineffective in eliminating pain caused by this condition in the long term. **(Cunha-Cruz et al, 2013)**

Periodontal disease can be considered as a risk factor or a cause of dentinal hypersensitivity as it involves gingival recession and therefore is associated with dentin exposure. A study performed on an adult and elderly population in Brazil stated that a reduction in the prevalence of tooth sensitivity may be accomplished by periodontal health improvements. **(Costa et al, 2014)**

Improper tooth brushing includes using hard- or thick-bristle tooth brushes, brushing teeth with excessive pressure, excessive scrubbing at cervical areas or even missing to brush cervical areas. **(Davari et al, 2013)**

Traumatic toothbrushing in an otherwise healthy dentition is often undiagnosed in adolescents and young adults. Subclinical soft- and hard-tissue abrasion lesions are probably a precursor of gingival recession and tooth wear, and thus DH. **(Pashley, 2008)**

Chrysanthakopoulos reported that the horizontal brushing method, performed once daily with a toothbrush with medium-hardness bristles, is associated with gingival recession, one of the etiological factors for DH. **(Chrysanthakopoulos, 2011)**

The most brushed teeth with the lowest plaque scores exhibit the most gingival recession. This has led to the description of gingival recession/DH as ‘toothbrush disease’. **(Pashley, 2008)**

In a study published by Fukumoto *et al*., teeth devoid of plaque were more hypersensitive than teeth with plaque accumulation. This statement was disputed by another work that found a significant association between high plaque accumulation and gingival recession. **(Fukumoto et al, 2014) (Toker et al, 2009)**

Plaque accumulation on tooth surfaces may lead to demineralization of tooth structures, which could be associated with patency of dentinal tubule orifices. **(Kawasaki et al, 2001)**

Facial piercings are strongly correlated with the prevalence of recession. **(Clark et al, 2016)**

Individuals who may be at risk for dentin hypersensitivity such as: **(Davari et al, 2013)**

- Overenthusiastic brushers
- Periodontal treated patients
- Bulimics
- People with xerostomia
- High-acid food/drink consumers
- Older people exhibiting gingival recession

- Chewing ‘smokeless’ or ‘snuff’ tobacco

Figure 1.1 Patient with exposed cervical dentin surfaces and signs of erosion. **(Abuzinadah et al, 2021)**

Periodontal surgery is not a risk-free procedure and should not serve as the first line of treatment for tooth hypersensitivity. Periodontal surgery should be the last resort for the resolution of tooth hypersensitivity and used only when other, less invasive, methods were unsuccessful as long as there is no other indication for tooth coverage. Only after all the above non-invasive and less-invasive methods have failed to reduce the symptoms should the root-coverage option be considered. **(Clark et al, 2016)**

There is insufficient research to establish whether scaling and root planing procedures had any impact on tooth hypersensitivity and, as a result, more research needs to be performed before making recommendations

specific to the correlation of periodontal disease and dentinal hypersensitivity. **(Draenert et al, 2013)**

1.2. Pathogenesis

As it is known, dentin is covered by enamel in the crown surface and by a thin layer of cementum in the root surface of the tooth. Dentin is sensitive to stimuli due to the lesion extension of odontoblastic process and formation of dentin-pulp complex. **(Davari et al, 2013)**

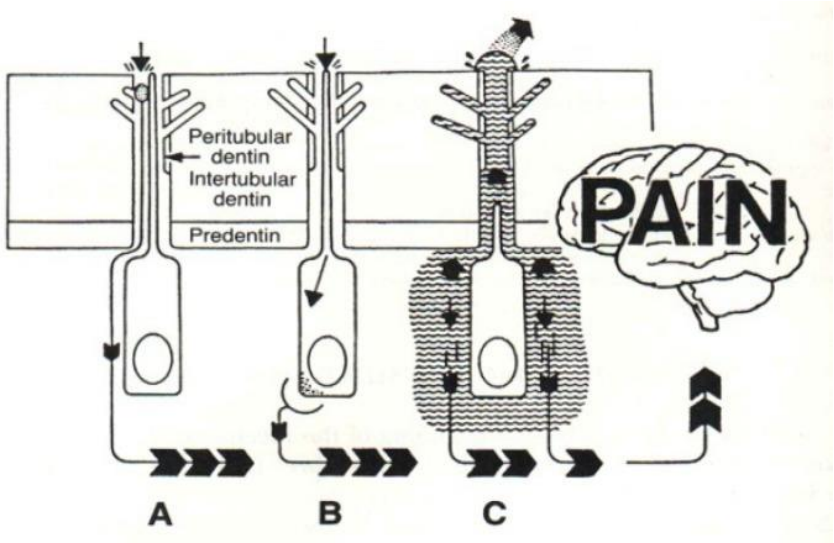
Dentin and pulp are histologically different. However, they have the same embryonic origin; ecto-mesenchymal origin. The formation of dentin-pulp causes dentin to be affected by pulp and vice versa. Dentin has very minute tubules which are filled with odontoblastic process. The processes are also surrounded by dentinal fluid which forms about 22% of the total volume of dentin. The fluid is completely filtrated and originates from the blood vessels of the pulp.

Dentin's sensitivity to stimuli does not lead to any problem while it is covered with protective tissues; enamel and cementum. The results of scanning electron microscope (SEM) indicate that the number of tubules in sensitive dentin is eight times more than the number of tubules in non-sensitive dentin. Furthermore, tubules of sensitive dentin are thicker than those in non-sensitive dentin.

It is worth noticing that not all the exposed dentins are sensitive. However, their calcified smear layer, as compared to non-sensitive dentin, is thin and this leads to an increase in the fluid movement and consequently the pain response.

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. Plug and smear layer on the surface of exposed dentine are composed of elements of protein and sediments which are derived from salivary calcium phosphates and seal the dentinal tubules inconsistently and transiently.

It seems that Microbial plaque is not a significant factor in triggering DH. First, as mentioned previously, the canines and first premolars have the greatest recession and sensitivity. The same teeth also reveal the lowest buccal plaque scores. Secondly, teeth with DH are cleaned extremely by patients suffering from the condition. This would suggest that plaque does not produce dentin hypersensitivity itself nor does it act as a stimulus for



pain. **(Davari et al, 2013)**

Figure 2 The schematic picture of the propped theories on DH

A. Direct Innervation (DI) Theory

B. Odontoblast Receptor (OR) Theory

C. Fluid Movement/Hydrodynamic Theory **(Davari et al, 2013)**

Regarding the first theory; DI, it has been reported that the nerve's endings enter dentin through pulp and extends to DEJ and the mechanical stimuli directly transmit the pain. However, there is little evidence to prove this theory; firstly, because there is little evidence that can support the existence of nerve in the superficial dentin; where dentin has the most sensitivity; and secondly because the plexus of Rashkov do not become mature until complete tooth eruption. However, the newly developed teeth can be sensitive too.

In the OR theory, odontoblasts act as receptors of pain and transmit signals to the pulpal nerves. But this theory has also been rejected since the cellular matrix of odontoblasts is not capable of exciting and producing neural impulses. Furthermore, no synopsis has been found between odontoblasts and pulpal nerves.

Hydrodynamic Theory for sensitive dentine is the most widely accepted theory for DH & has proposed based on the movement of the fluid inside the dentinal tubules. The movement of fluid stimulates a baroreceptor and leads to neural discharge. This process is similar to activating the neural fibers around the hair by touching or pressing the hair. The movement of fluid can be toward the inside of the pulp or the outside of dentin. Cooling, drying, evaporation, and hypertonic chemical stimuli cause the dentinal fluid to flow away from the dentin-pulp complex and lead to an increase in pain. Heating causes the fluid to flow toward the pulp. **(Davari et al, 2013)**

1.3. **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. **(Cunha-Cruz et al, 2013)**

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. **(Cunha-Cruz et al, 2013)**

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)**

he 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)**

1.4. Effects

Dentin hypersensitivity can lead to both physical and psychological problems for the patient. Furthermore, it can have a negative effect on the

quality of a person's life, especially with regards to dietary selection, maintaining optimal dental hygiene, and beauty aspects.

It has been observed that some people with DH do not pursue treatment of the disease. However, they may report it in a clinical visit to the dentist. This is perhaps due to the fact that they do not consider DH as a (specific) disease. **(Davari et al, 2013)**

1.5. Diagnosis

The often forgotten or neglected phase in the treatment of DH is the diagnosis and eliminating or treating the main routs of DH.

There are two common methods to determine the intensity of DH. One of them is through asking some questions from the patient and the other is through clinical examination. The prevalence distribution of DH in the first method is usually estimated higher than that of the second method.

The diagnosis of the disease starts through investigating the medical history of the patient and examination. In investigating the medical history some questions are asked about the time of the start of DH, the intensity of the pain, the stability of the pain and the factors that reduce or increase the intensification of the disease.

Some techniques such as pure air, pure water, and sounds are used in order to reconstruct the stimulating factors and to determine the degree of pain of the patient. Other diagnostic tests are as follows: palpitation for diagnosing pulpitis or periodontal involvement, pushing a wood stick or transillumination for diagnosing a fracture or cracked tooth. **(Davari et al, 2013)**

1.6. Management

Although DH is a prevalent disorder and one of the most annoying diseases, the treatments which have been suggested for it are not sufficient and very successful. **(Davari et al, 2013)**

Until recently, two approaches were used to cure cervical dentinal sensitivity (CDS). The first is blocking the dentinal tubules, and the second is intervening in the response of the mechanoreceptors. **(Lin et al, 2013)**

The patient should be taught the correct method of tooth brushing, avoid the use of abrasive tooth pastes and avoid brushing at least for one hour after consuming acid drinks or foods (due to agonist effect of acidic erosion on tooth brush abrasion). **(Davari et al, 2013)**

Aggressive toothbrushing is considered to be the use of excessive force with a hard-bristled toothbrush. A study published in 2013 indicated that most cases of dentinal hypersensitivity studied involved patients who were currently using a hard toothbrush. **(Vijaya et al, 2013)**

Sometimes through correction of occlusion or the use of an occlusal splint, the problem can be easily resolved.

For gingival recession, the patient should see a periodontist for consultation. Moreover, treatments such as graft or positioning flap might be adopted.

The patient’s diet should be monitored for a while, concerning the quality and the frequency of consumption of acidic foods so that the necessary recommendations can be offered to the patient.

Patients with gastro-esophageal regurgitation and eating disorders are recommended to be referred to their doctors for the underlying diseases. **(Davari et al, 2013)**

Relative dentin abrasivity (RDA) is a method for measuring the abrasiveness of certain ingredients in toothpastes on the dentin surface. An *in situ* randomised trial published in 2012 determined that RDA was directly related to dentin loss and concluded that patients with dentin hypersensitivity should opt for a toothpaste with lower RDA. **(West et al, 2012)**

Classification of desensitizing agents:

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: tooth pastes are amongst the most common *over-the-counter* (OTC) materials in desensitizing. Potassium salts, present in toothpaste, move along the dentinal tubules and through blocking the axonic action of the intra-dental nerve fibers decrease the excitability of the tooth. These tooth pastes should be used with soft-bristled tooth brushes and the minimum amount of water so that the tooth pastes would have their maximum positive effects. Remineralizing tooth pastes which contained sodium fluoride and calcium phosphates could reduce DH dramatically.
    b. Mouthwashes and chewing gums
2. In office: This is a complicated and expensive mode which can be used in treatment of a limited number of teeth. Theoretically, in-office

therapy of DH should lead to immediate relief of the pain. However, practically, this might not be the case.

a. Potassium nitrate is available in two forms of aqueous solution and adhesive gel. The number of potassium ions decrease when they enter dentinal tubules and decrease the excitability of nerves that transmit pain.
b. Fluorides precipitate calcium fluoride crystals inside dentinal tubules, and thus decrease dentinal permeability. These crystals are almost insoluble. Sodium fluoride with a 2 % concentration is used in the office. The precipitate which is formed by sodium fluoride can be removed by the saliva or mechanical scrubbing. Therefore, acid has been added to the formula so that the resultant acidulated sodium fluoride can form precipitates deep in the tubules.
c. Oxalates can occlude dentinal tubules and reduce permeability of dentine. 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. Potassium oxalate can lead to some digestive disorders so it should not be used for a long term.
d. The composites can effectively seal dentinal tubules through forming a hybrid layer. The old adhesives formed the hybrid layer through removing the smear layer and etching the dentinal surface so that deep resin tags could be formed. The new

adhesives, however, act in a way that the smear layer will be modified and incorporated into the hybrid layer.

e. Bioglass has been produced to stimulate bone formation. It is employed to fill the osseous defects during periodontal surgery. The application of bioglass causes the formation of an apatite layer which further leads to the occlusion of dentinal tubules.
f. CPP-ACP remineralizes the early lesions of enamel subsurface. The manufacturing factory has claimed that the product can be effective in prevention and treatment of DH.
g. The effect of laser on the treatment of DH is different and is based on the type of laser and therapeutic parameters such as the laser's length of beam; the amount of time spent on the use of laser; and the intensity of laser. Mechanisms of action include: occlusion through coagulation of the proteins of the fluid inside the dentinal tubules, occlusion of tubules through partial sub-melting, and discharging of internal tubular nerve. This type of therapy is highly acceptable to patients because its proper usage has no negative impacts. (Davari et al, 2013)Middle-output lasers, such as Nd:YAG, CO_2 and Er:YAG, work by occluding dentinal tubules (Sgolastra et al, 2011) and lower level output lasers, such as He-Ne and GaAlAs, affect nerve activity.
h. Calcium Sodium Phospho-silicate is designed to stimulate the remineralization of enamel and simultaneously it occludes dentinal tubules. (Clark et al, 2016) When in the oral cavity, sodium ions exchange with hydrogen ions, allowing the release

of calcium and phosphate from the dentifrice. These minerals deposit within the dentinal tubules until occlusion occurs. **(Chen et al, 2015)**

i. Bonding agents are used for a variety of dental applications, one of which is restorative dentistry. Bonding agents etch tooth surfaces in order to provide an adhesive layer for the application of a desired material. Another use for dentin bonding agents, however, can be to treat hypersensitivity. Self-etch bonding systems typically contain acidic ingredients that condition the dentin, as well as monomers that combine on the dentin, forming a hybrid layer. This layer provides a coating over the dentin and significantly reduces hypersensitivity over a 4-week period. Two-step systems are thought to be even more effective as they are proven to be less permeable and more durable. **(Pashley et al, 1978)**

Other methods of management include

A. Cervical restorations: Covering exposed dentin seals tubules, thus eliminating hypersensitivity symptoms. A study performed by Laybovich *et al*. compared the treatment of a tissue graft *versus* a Class V restoration in treating dentin hypersensitivity. Their results indicated no significant difference in the reduction of sensitivity; however, they found that patients preferred the tissue graft for aesthetic reasons. **(Leybovich et al, 2014)** Glass ionomer material is commonly used for cervical restorations as a result of its ability to bond with the dentin and enamel whilst simultaneously releasing fluoride. A study focusing on the longevity of glass ionomer

restorations over a period of 13 years revealed that the restorations sustained satisfactory qualities. Therefore, and because of its well-known advantages, glass ionomer might be the restorative material preferred for this treatment option for tooth hypersensitivity. **(Gordan et al, 2014)**

B. Root canal treatment: endodontic treatment that involves the removal of pulp and its replacement with gutta percha eliminates all sensory feeling associated with that tooth. Dentinal hypersensitivity is not, and should not be a reason or indication for, root canal treatment. More often this procedure is performed to treat irreversible pulpitis and pulp necrosis. (Veitz-Keenan et al, 2013) Although root canal therapy would not be a first-line treatment, it is an option that might be considered in extreme cases when no other option can relieve the hypersensitivity. **(Clark et al, 2016)**

C. Tissue graft: Regarding sensitivity caused by exposed root surfaces, a gingival graft may be desired to cover the exposed dentinal tubules. A study performed in 2013 demonstrated a statistically significant reduction of cervical dentinal hypersensitivity after treatment with a coronally positioned flap and connective tissue graft. **(Douglas de Oliveira et al, 2013)**

Chapter Two: Conclusion and Suggestions

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A question specifically about sensitive teeth may generate more positive responses than may a general question about ill effects of daily activities such as drinking cold water.

With vague prevalence comes uncertainty in diagnosis, the appropriate time to treat and how aggressive the treatment should be.

The treatment of dental hypersensitivity should be on a regular basis and initiate with at-home therapy and then continue with complementary therapies. It is recommended that follow-up visits should be organized for all the patients after undergoing periodic treatments.

A systematic review focusing on the effectiveness of lasers in treating dentinal hypersensitivity concluded that although lasers are effective, the evidence is considered weak because of the strong placebo effect.

To treat the condition properly, consider the patient's risk factors and the initial cause of the sensitivity. When all the factors are considered, the dental professional and patient can agree on a treatment plan based on the desired outcome. As in any condition, begin by eliminating the causes and then select the least invasive option that is believed to provide the desired result.

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