Dentinal Hypersensitivity

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Abstract: Dentinal Hypersensitivity is a common phenomenon affecting several patients worldwide. Due to clinical symptoms closely resembling other conditions such as Cracked tooth syndrome, Reversible pulpitis etc... it is, unfortunately, a largely under reported and misdiagnosed condition. It is characterized by sharp and transient tooth pain precipitated by exposure of dentin to external oral environment. The clinical cause of dentin hypersensitivity is exposed dentinal tubules, resulting from gingival recession and subsequent loss of cementum on root surfaces. (⁶⁾ Various exogenous stimuli induce this response, including introduction of hot or cold food and drinks into the mouth, sweet food items, and acidic beverages (eg: Pepsi, Coca Cola and other soft drinks), brushing of teeth etc. . Clinical procedures such as tooth bleaching and whitening treatments are also reported to invoke hypersensitivity.⁽⁸⁾ The treatment of dentinal hypersensitivity involves 3 general principal strategies. The exposed tubules can be covered through application of dental restorative techniques or gingival grafts. The tubules can be plugged or blocked using compounds that precipitate together into a sufficiently large mass to occlude the tubules. ⁽⁶⁾ The nerve tissue may also be desensitized through use of desensitizing agents. Hypersensitivity is influenced by the health and characteristics of the teeth and periodontium, as well as the oral environment and external factors. Predisposition to hypersensitivity is multifactorial. Risk factors are numerous, often acting synergistically and always influenced by individual susceptibility. Susceptible individuals are those with poor oral hygiene, bruxism and other such detrimental oral habits, periodontitis, gingival recession, and patients undergoing periodontal treatments of any kind. Treatment options involve pain management and gradual cessation of adverse symptoms. Treatment procedures can be carried out in - office or at - home, as a wide selection of over the counter medication and products are available to patients at pharmacies and drug stores.

Keywords: Dentinal hypersensitivity, clinical symptoms, exposed dentinal tubules, gingival recession, cementum loss, treatment, pain management, desensitizing agents, predisposing factors

1. Introduction

If not 'Dentinal Hypersensitivity', 'sensitive teeth' is a term the general public is familiar with. With a plethora of products, such as Hydent Pro, Sensodyne, Colgate Sensitivity Toothpaste, and Crest etc.... available in the market, meant to facilitate therapeutic management and treatment of Dentin Hypersensitivity. In accordance, the majority of the population would have come across the aforementioned products, or perhaps, a commercial or an advertisement, and would almost certainly recognize 'Tooth Sensitivity', the layman's term corresponding to Dentinal Hypersensitivity.

Dentin Hypersensitivity is a fairly common phenomenon, with statistics sourced from a number of reliable source material, i. e. – Clinical Trials, Review Articles reporting that ⁽¹⁾ 57% of our population experiences this condition. In particular, it is shown to peak between the ages of 20 - 40. This condition is usually reported by patients after they experience a sharp pain in the region of their tooth/teeth following exposure to various stimuli. These include: Osmotic, Thermal, Chemical, Mechanical, Pressure, Electrical and evaporative stimuli, to name a few. ⁽⁶⁾

The above mention etiological factors will be later discussed in this paper more extensively, under the same heading. Regardless, we can see that a majority of patients seek treatment after experiencing pain/sensitivity/discomfort during the consumption of very hot or cold food and beverages, or acidic or sweet food, eg: soft drinks, chocolate, candy....

Despite failure to reach a definite consensus on what causes hypersensitivity, several theories have been put forth over the years, of which the most widely accepted one is the Brannstrom Hydroynamic Theory.

2. Definition

- 1) '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' ⁽¹⁾
- ~ Canadian Advisory Board, Dentin Hypersensitivity, 2003
- 2) A short, sharp pain arising from exposed dentin in response to stimuli typically thermal, evaporative, tactile, osmotic or chemical and which cannot be ascribed to any other dental defect or pathology (2)

~ Dowell and Addy, 1983

It is defined as an exaggerated response to a usually non harmful stimulus that does not usually incite an adverse response in healthy and sound teeth. These may include: thermal, tactile, or osmotic stimuli that, when applied on exposed dentin, evoke pain despite the absence of pathological alterations or damage to the dental tissues.

3. Prevalence

The prevalence, distribution, and epidemiology of dentinal hypersensitivity has been reported with vast variations in different studies. These are probably owing to the differences between populations, and corresponding lifestyle, including dietary habits, as well as the methods of investigation used to obtain relevant information. There are a reported 40 million cases annually in the United States alone. (⁴⁾

Dentinal Hypersensitivity is prevalent amongst patients within the age range of 20 - 50 years. However, it is most probable to occur in patients who are 30 - 40 years old.

It reportedly has higher occurrences in female individuals.

It generally involves the facio - cervical and bucco - cervical areas of teeth, and are found most often in canines and premolars. $^{(2)}$

Patients undergoing periodontal treatment are particularly susceptible, due to loss of cementum and gingival recession following periodontal surgery.

In addition, bad brushing habits and periodontal disease also contribute to gingival recession, which in turn, instigates Dentinal Hypersensitivity.

Severe cases can last more than 6 months and become a consistent irritation, inducing psychological and emotional disturbance, which may trigger the development of chronic dental pain condition known as neuropathic pain.⁽¹⁴⁾

4. Etiology

(a) Anatomy of the tooth and dentin pulp complex. Dentin is considered as a vital tissue and has the capacity to respond to physiological and pathological stimulus. As it's 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 extension of Odontoblastic process a formation of dentin pulp complex. (21)

Dentin and pulp are historically different. They have the same ectomesenchyme origin. Pulp causes dentin to be affected by the pulp and vice versa. Dentin has very minimal tubular which are filled with odontoblastic process. (25)

The processor also surrounded by dentinal fluid which forms about 22 percentage of the total volume of the dentin. The fluid is completely filtered and originated from the blood vessels of the pulp. (22)

5. Pathogenesis

In medical literature, it has been stated that dentinal hypersensitivity develops in 2 phases:

- 1) Lesion localization
- 2) Lesion Initiation
- Lesion Localization: It occurs by loss of protective covering over the dentin (enamel, cementum) which leads to exposure of dentin to the external oral environment. ⁽²⁾ This loss of the outer covering may be induced by attrition, erosion, abrasion etc. Another cause of lesion localization is gingival recession which can be due to tooth brush abrasion, pocket reduction surgery, tooth preparation for crown, excessive flossing, or as a consequence of periodontal disease, eg: gingivitis
- *Lesion Initiation:* It occurs when the protective covering of the smear layer is removed, leading to exposure and opening of the dentinal tubules. ⁽²⁾ The removal of the protective smear layer causes patency of the tubules. In order for dentinal hypersensitivity to occur, a number of dentinal tubules in close proximity to each other must be patent from the pulp to the oral environment. ⁽⁹⁾ There is a continuous flow of fluid from the dentinal tubules, and upon application of stimuli, the rate of flow is increased, causing excitation of nerves and, consequently, a sensation of pain.

6. Mechanism

The intradentinal nerve is confined to the predentin, and the innnermost part of dentin, which is adjacent to the pulp. This supports the likelihood of an indirect stimulatory mechanism evoking the pain.

According to a study conducted and published on Pubmed. ncbi, we have 5 mechanisms describing pathogenesis of dentinal hypersensitivity:

- a) Classic Hydrodynamic Theory
- b) Direct innervation of dentinal tubules
- c) Neuroplasticity and sensitization of nociceptors
- d) Odontoblasts serving as sensory receptors
- e) Algoneurons⁽⁷⁾

It is important to note that these theories are not mutually exclusive, and so, it is possible that more than just one of them behave as contributing factors to dentinal sensitivity.

The cellular and molecular mechanisms of dental nociception are described through the following 3 hypotheses:

- 1) Transduction Theory: Supported by functional expression of temperature sensitive transient receptor potential channels, emphasizes the direct transduction of noxious temperatures by dental primary afferent neurons.
- 2) Hydrodynamic Theory, Brannstrom, 1966: Attributes dental pain to fluid movement within dentinal tubules. According to Brannstrom, upon application of an appropriate stimulus to the outer dentin surface, there is displacement of the fluid of dentin tubules, which gives rise to the mechanical stimulation of pain at the pulpodentinal border⁽⁵⁾

Volume 11 Issue 6, June 2022

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- 3) Modulation Theory: States that the nerve impulses in the pulp are modulated through release of polypeptides from injured odontoblasts located in the dentin. These substances (polypeptides) selectively alter the permeability of the odontoblastic cell membrane through the process of hyperpolarization. This has the following effect on pulpal neurons - they are more prone to discharge upon receiving subsequent stimuli, contributing to overall increased sensitivity. Aside from these theories, the lesser accepted Gate control and Vibration theories have been cited as well.
- 4) Gate control theory: This theory suggests that the spinal cord contains a neurological 'gate' that either blocks pain signals or allows them to continue on to the brain. According to this, any non painful stimulus/input closes the nerve gates to painful input, which prevents any painful sensations from traveling to the CNS.

7. Clinical Features

Patients suffering from hypersensitivity typically present with complaints of pain/discomfort/sensitivity in their teeth. The pain described by most patients is sharp and transient and usually occurs upon application of stimuli. For example, while consuming acidic beverages, like soft drinks. Some patients have been known to report difficulty in brushing teeth due to pain. The degree of pain varies in character, ranging in intensity from mild discomfort to extreme/severe pain which may emanate from a single tooth or multiple teeth. Clinical studies show that individuals with less than adequate plaque control experience more root hypersensitivity to air stimuli than others with better oral hygiene. Therefore, we can deduce that microbial plaque has an effect on root sensitivity. ^(2, 3)

8. Diagnosis

As in the case of arriving at a medical diagnosis of any other disease, a complete and thorough examination and investigation by the clinician is imperative. The diagnostic procedures involved include: elicitation of patient's medical history, holistic assessment of patient, conducting appropriate medical tests to gauge level of pain experienced, and finally, ruling out differential diagnoses, allowing the clinician to arrive at the correct conclusion.

Initially, an examination of the patient's general health and oral cavity, including assessment of the condition of the involved tooth/teeth is conducted. In the case of those presenting with complaints of pain, the history of the patient's pain is the first clinical data the clinician is expected to collect and consider. Much can be deduced through the characteristics of pain described, eg: lingering, transient, pulsating, severe, sharp/dull, localized/generalized etc...

Generally, Dentine hypersensitivity is underdiagnosed and underreported by the general public, causing lack of efficient diagnosis. This is due to the large number of conditions in which similar clinical presentation of symptoms and pain occur. These include fractured enamel/dentine, chipped tooth, abfraction, pain due to reversible pulpitis, caries, postdental bleaching sensitivity, cracked tooth syndrome, palate gingival grooves, marginal leakage around restorations etc...⁽²⁾

Prior to making a definite diagnosis, other possible causes of dental pain must be excluded.

Some techniques to affirm this are assessment of the pain response upon tapping teeth, to indicate pulpitis or periodontal involvement, pain on biting a stick which suggests fracture, use of transilluminating dyes or light, to diagnose fracture, and even pain associated with recently done restorations.

The standard method of diagnosing Dentinal hypersensitivity is a jet of cold air, or using an exploratory probe on exposed dentin in a mesio distal direction.

The severity of pain experienced by the patient can be quantified according to categorical scales or visual analogue scale (VAS)

DOI: 10.21275/SR22517104014

International Journal of Science and Research (IJSR)

ISSN: 2319-7064 SJIF (2022): 7.942



Figure 1: Diagnostic criteria and assessment methods for dentinal hypersensitivity (15)

I. Treatment Aim

Most available treatments seek to reverse the patency of dentinal tubules, or occlude them in order to minimize and eventually prevent the painful symptoms experienced by patients.

II. Treatment Protocol

Classification of desensitizing agent

- 1. Mode of administration
- At home desensitizing agents
- In office treatment
- 2. On the bases of mechanism of action
- Nerve desensitization e. g., potassium nitrate
- Cover or plugging dentinal tubule
- Ions/salts
- Aluminum
- Ammonium hexafluoro silicate
- Calcium hydroxide
- Calcium phosphate
- Calcium carbonate
- Calcium silicate
- Sodium citrate dibasic
- Fluorosilicate
- Potassium oxalate
- Silicate
- Sodium Mono fluorophosphate
- Sodium fluoride
- Sodium fluoride/stannous fluoride combination
- stannous fluoride
- strontium acetate with fluoride
- strontium chloride

- Protein precipitants
- formaldehyde
- glutaraldehyde
- silver nitrate
- strontium chloride hexahydrate
- Zinc chloride
- · Phyto complexes
- Rhubarb Rhaponicum
- Spinaciaoleracea
- Fluoride iontophoresis
- (B) Dentin Sealers
- Glass ionomer cements
- Composites
- Dentinal adhesives
- Resinous dentinal desensitizers
- Varnishes
- Sealants
- Methyl Methacrylate

(C) Periodontal soft tissue grafting

(D) Lasers

(E) Homeopathic medications• proplisAt home Desensitizing Agent

The advantage of this mode is that it is simple and can be used in number of teeth. The disadvantage is that about 2 - 4 week of tie symptom may reoccur. Some of the desensitizing agents that can be used at hoe are tooth paste, mouth wash, chewing gums, containing potassium nitrate, sodium fluoride or potassium citrate can be recommended.

Volume 11 Issue 6, June 2022

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Recent Advances

Arginine, an amino acid occurring naturally in saliva and calcium carbonate provide relief for DH. Pro - Argin contains 8% arginine and calcium carbonate seal dentinal tubules which are exposed. it has been suggested that Pro Argin mouthwash or paste provide relief for dentin hypersensitivity. (18, 20)

NovaMin contains15%Calcium Sodium PhosphoSilicate (CSPS) is based on bio glass technology. The CSPS releases sodium ions resulting in rapid formation and precipitation of calcium hydroxyapatite mineral layer on the dentin surface in aqueous environment. It is effective only as an in office treatment. (22)

Recent advance in nanotechnology uses three layered guided tissue regeneration for DH with localized gingival recession. (21)

9. Discussion

Dentin hypersensitivity as a chronic disease is increasingly prevalent among adults and some researches has been done on determining etiologic factors in the causation of the disease, its diagnosis, and its treatment. This disorder usually occurs as the result of the loss of enamel and cementum or exposure of dentinal tubules. There have been many methods and materials to reduce or remove sensitivity. They include the use of tubes of toothpaste containing potassium salts, fluoride composites, resins, laser; bio glass, and so on. They exert their effects by sealing dentinal tubules or by disturbing the transmission of nerve impulses. Etiologic factors have been underestimated by dentists or specialists in the treatment of DH. Based on studies, different theories have been proposed on the dentin hypersensitivity, in which direct innervation theory and odontoblast receptor theory had some challenges. However, Hydrodynamic theory by Brannstorm deals with the flow of fluid inside the dentinal tubules is readily accepted. Still, the most common therapy and usually the first therapy in treating dentin hypersensitivity is the use of kinds of toothpaste containing potassium salts and fluoride. The new offered materials and methods, such as Bio glasses, CPP -ACP, laser, iontophoresis, and homeopathy for the treatment of DH have been tested through studies and the obtained results have been different.

10. Conclusion

To conclude, Dentin hypersensitivity is a common dental complaint, and prior to treatment a differential diagnosis is critical. Identification of the various risk factors should be ascertained and a determination should be made of whether the pain is localized or generalized. Unfortunately, most currently available tests are subjective. Ideally, a more objective technique is required in order to adequately quantify patients' response. It is clear that some products appear to be more effective than others. For those products developed for self application at home, potassium nitrate, fluoride, fluoride, sodium stannous sodium monofluorophosphate and strontium chloride have all been extensively studied and shown to be not only safe to use but of benefit to individuals suffering fro dentinal hypersensitivity

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