IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 21, Issue 2 Ser.5 (February. 2022), PP 01-07
www.iosrjournals.org

Dentine Hypersensitivity: An Overview

Ruchi Gupta[1], Anil K Tomer[2], Akhil Thomas[3]
1 Professor, Dept of Conservative Dentistry and Endodontics, Divya Jyoti College of Dental Sciences and Research, Modinagar, Ghaziabad
2 Professor and Head, Dept of Conservative Dentistry and Endodontics, Divya Jyoti College of Dental Sciences and Research, Modinagar, Ghaziabad
3 PG Student, Dept of Conservative Dentistry and Endodontics, Divya Jyoti College of Dental Sciences and Research, Modinagar, Ghaziabad
Corresponding Author:
Dr. Ruchi Gupta
Professor, Dept of Conservative Dentistry and Endodontics, Divya Jyoti College of Dental Sciences and Research, Modinagar, Ghaziabad

Abstract
The ever-changing profiles of human diseases in mankind's history have not left dentistry untouched. The improving oral health status of populations, people keeping teeth for longer has brought impressive benefits, but at the same time has created awareness of other oral and dental health problems. Following the decline of dental caries, the management of periodontal diseases gained priority, and other, painful dental problems, such as dentin hypersensitivity stepped forward. Little attention has been paid to scientific research and practical management of this condition. Dentin hypersensitivity has been discussed in the dental literature to explain 'the sensitiveness of dentin and described fluid movement in the dentinal tubules. This has brought a change in the attitudes of dental researchers and practitioners concerning dentin hypersensitivity.

Key words: *Dentine, Hypersensitivity, Desensitization, Pain, Dentinal fluid, Laser*
Clinical Significance: *Dentin hypersensitivity can be controlled by various desensitizing agents.*

Date of Submission: 28-01-2022 Date of Acceptance: 09-02-2022

Dentine Hypersensitivity: An Overview

Dentine hypersensitivity is characterized by short, sharp pain arising from exposed dentine in response to stimuli typically thermal, evaporative, tactile, osmotic or chemical and which cannot be ascribed to any other form of dental defect of pathology.[1] A definition for dentine hypersensitivity was suggested in 1983 and, with minor amendment was adopted in 1997 by an international workshop on the design and conduct of clinical trials for treatments of the condition. The Canadian Advisory Board on Dentine Hypersensitivity in 2002 suggested that it would be more correct to substitute 'disease' for 'pathology'. Other causes of the typically short, sharp, dentinal pain include caries, chipped teeth, fractured restorations, marginal leakage around restorations, some restorative materials, cracked tooth syndrome and palato-gingival grooves. Such conditions clearly require treatment options that are usually quite different from those used for dentine hypersensitivity. Other names such as sensitive dentine, cervical dentinal sensitivity, cemental hypersensitivity and root sensitivity have been applied for dentin hypersensitivity.

Actually exposed dentin is sensitive because it is innervated tissue. Hypersensitivity implies that the dentin is more sensitive than normal. Normally, dentin is sealed peripherally by enamel or cementum and hence is not very sensitive.[2] When it is suddenly exposed, as occurs in tooth fracture or periodontal surgery, the patient becomes acutely aware that the dentin is sensitive, but regards it as hypersensitive relative to their previous experience. Similarly, patients with sensitive root surfaces can become more sensitive if those surfaces are acid-etched. Scientists have suspected that bacterial products or endogenous mediators of inflammation might lower the threshold of pulpal nerves, making the dentin truly hypersensitive. There is little published evidence to support that idea as occurring commonly in most cases of cervical dentin sensitivity. Cementum is not innervated and hence can not be sensitive. Thus, the old term, 'hypersensitive cementum' is a misnomer, which should be discarded. In fact, the presence of sensitive root surfaces indicates that the cementum is not present and that the underlying dentin has become exposed.

Hypersensitive dentine affects between 10-20 % of the population. The prevalence appears to be fairly similar in different parts of the world, although there are some regional differences. The prevalence of dentin sensitivity ranges from 8% to 30%. This wide range is due, in part, to widely different methods used to diagnose

the condition. Most clinicians use a 1- second air blast, while others ask the patient to fill the mouth with ice-cold water. Hypersensitive dentine may affect any tooth, but most studies agree that it is most common in canines and first premolars, and is almost exclusively found on the vestibular surfaces.[3] Hypersensitive dentine may also be present on other surfaces, including cuspal and incisal edges, and on lingual or palatal surfaces; in the latter case, it is usually indicative of acid regurgitation. However, not all exposed dentinal surfaces are sensitive, and not all regions of hypersensitive dentine are the same: they vary in extent, and also in sensitivity to different stimuli. For example, it is often found that hypersensitive teeth are sensitive to one form of stimulus e. g. cold, but not to another, e. g. probing. The reasons for these differences require further investigation.

Age seems to be a factor with most complaints of dentin sensitivity peaking at 25-30 years of age (range 20-40). The incidence of exposed root surfaces rises with age from 21% in 16 to 24 year -olds to 81% in 34 to 44 year- olds, and to 98% in 55 to 64 year -olds. The decline in the degree of sensitivity with age, even in the face of increased gingival recession and root surface decay, may be due to sclerosis of dentin and/or the formation of reparative dentin.

Patients may develop dentin sensitivity when they begin a grapefruit diet regiment, which then disappears when they stop eating **acidic foods**. Root sensitivity commonly occurs following **oral prophylaxis or root planing**, but this slowly resolves over the next week or weeks, similarly, in restorative dentistry, dentin sensitivity often follows cavity or crown preparation and insertion of restorative material, but disappears over time. Dentin sensitivity is often observed on the buccal cervical areas of canines and premolars, especially on the left side of right handed individuals. Most cervical dentin sensitivity is caused by **improper tooth brushing**, and is seldom seen on the lingual surfaces of teeth, except in bulimic patients. The sensitive teeth are often absolutely free of bacterial plaque because they are brushed 3-4 times a day. Clinicians should observe the patient's brushing technique if they suspect obsessive or compulsive habits.[4]

Excessive loss of tooth structure such as occurs in **bulimic patients**, leaving smooth but sensitive dentin surfaces exposed, is another problem. The exposed coronal dentin is such more difficult to treat than cervical dentin because of its higher permeability and innervation density. **Females** tend to have more sensitivity than males. This has been attributed to their practicing better oral hygiene

INNERVATION OF THE TEETH: PAIN SENSORY PATHWAYS

The role of Adelta and C fibers in dental **pain** perception was studied while sudden cold stimuli were applied to the teeth (Jyväsjärvi and Kniffki, 1987). When the mean rating of the subjective **pain** vs. time was plotted, it was correlated very closely with the time-course of firing of the Ad fibers. The C fiber discharge was much slower and uncorrelated with the **pain** from cooling. This suggested a strong role for Ad fibers in transmission of **pain** induced by cold stimulation. Pulpal C fibers responded to thermal, mechanical, and chemical stimulation. Thus, they appeared to be polymodal nociceptive fibers.

CENTRAL PATHWAY OF DENTAL PAIN

Afferents from the mandibular and maxillary divisions of the trigeminal nerve relay in the spinal sensory nucleus of V. From this region fibers cross the pons and many relay in the pontine reticular formation; ultimately they project to the intralaminar and ventroposterior thalamic nuclei, and thence diffusely to the cortex.

The projections of sensory axons innervating a tooth may be traced with the use of horseradish peroxidase.HRP is injected into a pulp cavity, from where it is transported in sensory axons to their terminations. Following a 1-2 day period for transport to occur, label was found in the trigeminal ganglion. Later studies (e.g Arvidsson and Gobel, 1981) used this technique to show that a single pulp nerve projected to the dorsomedial parts of the main sensory nucleus of V as well as the subnuclei oralis and interpolaris.

They concluded that the stimuli must have been directly exciting nerves that traveled to the dentin surface.[4] However, histological studies using special stains for nerves failed to identify such pathways at either the DEJ or cemento-enamel junction (CEJ). Rather, their distribution was limited to the pulp or, at most, extended only 0.1 mm into the dentinal tubules. Furthermore, topical application of local anesthetics to peripheral dentin did not produce the desired effect. Similarly, topical application of agents that normally activated nerve fibers (potassium salts acetylcholine) did not produce pain. Thus, the notion that dentin sensitivity was due to direct stimulation of dentinal nerves had to be rejected.

In the 1960's a new hypothesis was developed, suggesting that dentin sensitivity was due to stimulation of odontoblast process in the exposed dentin. This theory was based on the idea that odontoblasts could serve as receptors and that there must be synapses between pulpal nerves and odontoblasts. Most authorities now believe that there are no synaptic junctions between odontoblasts and pulpal nerves.

Circumstantial and direct evidence disproved the theory of 'innervation of dentine' and 'odontoblast transducer' mechanisms. This left the hydrodynamic hypothesis first proposed by Gysi in 1900, and for which significant evidence accrued in the 1950s and 1960s, as the most widely accepted theory to date. Brannstrom and his colleagues, by combining clinical and laboratory experiments, developed support for what is now called

the hydrodynamic theory of dentin sensitivity. In essence, they observed that in extracted teeth a wide variety of pain -producing stimuli induced fluid movement, in both inward and outward directions, through dentin. They reasoned that this fluid movement through dentin excited mechanoreceptors nerves near and pulp. A corollary to this theory is that anything that interferes with fluid movement through dentinal tubules, or which lowers nerve excitability, would decrease dentin sensitivity. This theory can also explain most causes of sensitivity under restorations.

The hydrodynamic theory postulates that most pain evoking stimuli increase the outward flow of fluid in the tubules. This increased flow, in turn, causes a pressure change across the dentine, which activates A - δ intradental nerves at the pulp dentine border or within the dentinal tubules[1]. The stimulation is thought to occur via a mechanoreceptor response, which occurs when gentle pressure is applied to skin hair. In Addition, when fluid moves in tubules, an electrical discharge known as steaming potential occurs; this is directly proportional to pressure. Whether this discharge reaches levels sufficient to stimulate nerves has not been established, although it is theoretically possible. In vivo studies (Linden and Brannstrom, 1967; Pashley et al., 1981a; 1981b; Maita et al., 1991) have reported that dentinal fluid can slowly seep to exposed dentin; surfaces as it flows down a hydrostatic pressure gradient from the pulp.[5] Apparently, this spontaneous rate of fluid movement is too slow to activate mechanoreceptors which may be more responsive to the rate of change of fluid movement rather than the absolute rate.

Mechanism creating hypersensitive dentin

1.	Increases in the hydraulic conductance of dentin
a.	Dissolution of smear layer
b.	Loss of smear plugs
c.	Loss of mineralized plaque
Decreases in A delta nerve threshold (i.e. nerve hypersensitivity)	
a.	Elevations in local pulpal pressure due to inflammation
b.	Direct effect of neurogenic peptides on local tissues pressure and/or neural membranes
c.	Direct effect of bacterial products on the conductance channels

DIAGNOSTIC CONSIDERATIONS

Clinician has to evaluate the abuse of modern life style drinks (which often contain high amounts of titratable acid), or diets high in vegetables may contribute to erosive effects removing dentine and/or dentinal smear layer, thereby opening the tubules. Moreover, exploring the medical history very cautiously can provide valuable information on intrinsic (e.g. vomiting, regurgitation, rumination; eating disorders like anorexia and bulimia nervosa) and extrinsic erosive factors (e.g. acidic occupational/environmental reasons or the excessive use of acidic medicaments such as vitamin C or aspirin as fluids). Clinicians have focused on physical factors like tooth brushing abrasion when trying to eliminate extrinsic factors of DH. Indeed. Mechanical tooth wear can be caused by abrasive dentifrices when used with a brush excessively.[71] However, evidence from the literature seems to be inconsistent, since toothpastes can block the dentinal tubules by producing a smear layer or are able to occlude the orifices with some of their (primarily abrasive) ingredients, depending on the type of action. Therefore, it seems to be reasonable to suppose a combination of erosive and abrasive influence on DH; the effects of intrinsic/extrinsic acids will be particularly enhanced by tooth brushing with abrasives. This erosion-abrasive effect will be responsible for cervical enamel loss; moreover, with respect to dentine, these insults will cause a ready opening of the dentinal tubules, accompanied by an accelerated dentin loss.[6]

The cervical region of the tooth is particularly prone to wear, producing an angular lesion (horizontal to the dentine and acute at the enamel margin), this kind of class V cavity is localized very frequently at the facial surfaces of the upper canines and premolars. Abfraction could weaken the tooth by forming stress concentrations near to the gingival margin; subsequently, this would render the apatite crystals more susceptible to chemical attack (erosion) or further mechanical deterioration of both. Again this phenomenon would result in opened dentinal tubules being responsible for DH.

In notch-shaped cervical lesions, a wider diameter of the tapered dentinal tubules can be found near the pulp; at the same time, more tubules per unit area are present. While in the coronal dentine the tubule density increases four-fold (in superficial dentine the area occupied by tubule lumina is approximately 1 pr cent of the total surface area, and their value will increase up to 22 per cent at the pulp), in the root dentine this consequently means that with a preceding cervical lesion the extent of the discomfort is likely to increase.[75]

DESENSITIZING DENTIFRICES

The most popular dentin desensitizing dentifrices include 5% potassium nitrate as the active ingredient. The success of dentifrices depends on the frequency of use (morning and evening use preferred), length of time of use and on whether they brush the sensitive areas. Although fluorides may be marginally

effective at reducing dentin sensitivity as well. Those dentifrices containing potassium salts seem to provide more relief than other desensitizing dentifrices.[7]

The improvement of dentin sensitivity over time (the waning phase) may be due to remineralization phenomenon and even calculus formation. As most anti tartar dentifrices are designed to interfere with crystal growth, which is important in remineralization of dentin, patients may find their dentin sensitivity increases when they use such products.

TOPICAL DESENSITIZING AGENTS

The topical use of calcium hydroxide, sodium or stannous fluoride solutions, gels, varnishes, potassium oxalate, ferric oxalate, (table 3). These are all designed to occlude the orifices of the dentinal tubules and thereby block hydrodynamic reactions from causing pain. None of these agents are permanent and they may require reapplication. Potassium and ferric oxalate seems to be more effective immediately but such clinical studies are always complicated by significant placebo effects. However, these agents may provide temporary relief to dentin sensitivity and can be applied by dental hygienists as well as by dentists. It is worth mentioning that patients with moderate to severe sensitivity scores should be given special consideration during such treatment. Whatever solutions are used should be warmed between 34-37°C to avoid thermally induced pain with room temperature solutions. A topical solution of potassium oxalate (Protect, John O. Butler Co.), or Sensodyne Sealant, (Block Drug Co. Inc.), an acidic solution of ferric oxalate can be used.

Dentin Desensitizing agents

Active Ingredient	**Brand name**	**Source**
A. Over-the-counter remedies		
Potassium Nitrate (5%)	Denquel	Procter & Gamble Co
Strontium Chloride (10%)	Promise	Block drug company Inc
Citrate/Pluronic Gel	Sensodyne-F	Jersy City, NJ
(0.5% citric acid, 1.5% sodium citrate, 20% pluronic F-127)	Sensodyne	Block Drug Company, Inc
	Protect	Jersey City, NJ
		John O. Butler Company
		Chicago, IL
B. Professional Products		
Potassium Oxalate	Protect Dentin	John O. Butler Company
Ferric Oxalate	Desensitizer	Chicago, IL
	Senosodyne Sealant	Block Drug Company Inc.
Topical Fluorides	Omni-Gel	Jersy City, NJ
Stannous Fluoride (0.4%)	Gel-kam	Dunhall Pharmaceuticals Inc.
Neutral NaF (1.1%)	Luride	Stratford, TX
Acidulate NaF (1.2%)	Luride	Scherer Laboratories, Inc,
Sodium Fluoride Pase	Dentin Desensitizing Paste	Dallas, TX
(331/3% NaF)		Lorvic Corporation
		St. Louis, MO
		Colagate-Hoyt Laboratories
		Needham, MA
		Sultan Dental Products,
		Engle wood, NJ

The soluble oxalate salts react with calcium in dentinal fluid to form microscopic crystals of insoluble calcium oxalate in the orifices of the tubules. Normal remineralization mechanisms should restore these surfaces within a few days. Topical fluorides form crystals that are smaller than those of the oxalates, and therefore do not occlude dentinal tubules as quickly. However, fluoride treatment reduces the acid solubility of dentin, promotes remineralization, and has antibacterial effects. Thus, there is no contraindication to the use of stannous fluoride, for example, immediately after topical oxalate treatments. Additionally fluoride treatment may contribute to tubule occlusion and may make the dentin less susceptible to future sensitivity caused bye erosion of root structure. The maintenance of dentin desensitization can be promoted by such fluoride treatments at home. Following topical application, these treatments should offer significant reduction in dentin sensitivity for several weeks to months, until the natural desensitizing mechanisms express their full potential. These topical treatments should be done on unanesthetized patients.[8] If they are not effective immediately, the treatment should be repeated.

If the patient has many sensitive teeth, an alternate approach would be the fabrication of a vinyl mouthguard that extends over the sensitive surfaces. These devices can be used by the patients to hold materials such as calcium hydroxide paste, 1.23% neutral sodium fluoride gel or 0.5% dexamethasone ophthalmic ointment on the sensitive surfaces overnight.

POTENTIAL TREATMENT MODALITIES FOR DENTINE HYPERSENSITIVITY: HOME USE PRODUCTS

The incidence of dentine hypersensitivity has been reported to range from 8 to 35% in 'normal' populations. The products include agents such as potassium salts, strontium salts and fluoride salts in toothpaste, mouthwash and gel formulations.

These agents are believed to reduce the symptoms of dentine hypersensitivity by both occluding dentine tubules and thus blocking the neural stimulus and response. and/or intercepting the neural response by chemical intervention. Acid foods and drinks have been shown to soften dentin and may remove deposits on the dentine surface. Brushing has been shown to exacerbate the removal of any surface deposits. These deposits may be performing the desirable function of blocking tubules and reducing dentine hypersensitivity. The effectiveness of these self-treatment products that occlude dentine tubules could perhaps be improved by counseling patients on their diet and brushing habits.

Strontium salts

Zappa examined the effect of toothpastes containing strontium salts, either as the chloride or the acetate, on patients with dentine hypersensitivity.

Potassium salts

Potassium salts either potassium nitrate or potassium chloride are commonly used agents incorporated into toothpastes and mouthwashes for the self-applied treatment of dentine hypersensitivity. The effect of the product increased with time.[9]

Other agents

The effects of different fluorides and of a mouthrinse containing aluminium lactate on dentine hypersensitivity have been reported.

POTENTIAL TREATMENT MODALITIES FOR DENTINE HYPERSENSITIVITY: IN OFFICE PRODUCTS

In office treatments for hypersensitive dentine

I. Treatment agents that do not polymerize
- A. Varnishes/precipitants
  1. Shellacs
  2. 5% sodium fluoride varnish
  3. 1% NaF, 0.4% SnF2, 0.14% HF solutions
  4. 3% mono potassium –monohydrogen oxalate
  5. 6% acidic ferric oxalate
  6. Calcium phosphate preparations
  7. Calcium hydroxide
- B. Primers containing HEMA
  1. 5% glutaraldehyde, 35% HEMA in water
  2. 35% HEMA in water

Treatment agents that undergo setting or polymerization reactions
- A. Conventional glass ionomer cement
- B. Resin reinforced glass ionomer/compomers
- C. Adhesive resin primers
- D. Adhesive resin bonding systems

I. Use of mouthguards
V. Iontophoresis
V. Lasers

I. Treatment agents that do not polymerize

A. Varnishes, precipitants

The use of 5% sodium fluoride (NaF) in a thick varnish as a dentine desensitizer has been reported by Clark et al. (1985). The varnish does temporarily occlude dentinal tubules but the material is readily lost over time. For instance, burnishing sensitive root surfaces with a paste made up of 33% NaF, 33% kaolin and 33% glycerine has been used for over 50 years.

B. Primers containing HEMA
The primer, composed of 5% glutaralde-hyde and 35% HEMA in water, was very effective in reducing dentine sensitivity both in the presence or absence of the smear layer

II. Treatment agents that undergo setting or polymerization reactions

A. Conventional glass ionomer cements
The cervical lesions were etched with 50% citric acid for 30- 45 s, then rinsed and dried prior to placement of the glass ionomer cement. The author reported complete loss of hypersensitivity in 89.7% of all patients by Low (1981).

B. Resin- reinforced glass ionomers
Hansen (1992) obtained a 1- year success rate of 79% using a resin – reinforced glass ionomer to treat hypersensitive dentine.

C. Adhesive resin primers
Theoretically, the use of Adhesive primers to occlude the open tubules of hypersensitive dentine looks very thin residual film thickness. The long-term effectiveness of this resin product may be limited by the inability of the resin tags to bond the walls of the peritubular dentine matrix lining most dentinal tubules. Only if the peritubular dentine is removed by acid etching to expose the collagen fibrils of the surrounding intertubular dentine matrix can liquid resin infiltrate into the demineralized matrix and hybridize with it.

D. Adhesive resin bonding systems
Jensen and Doering (1987) used a light- cured system to treat root surface hypersensitivity. Other materials that have good potential for treating dentine hypersensitivity are the self -etching/ self- priming bonding systems. The disadvantage of the Adhesive systems is that their polymerization is inhibited by atmospheric oxygen to a depth of 10-15µ m. If they are over thinned with an air cure even though Adequate light irradiation is done. This is normally not a problem in conservative dental treatment where these thin Adhesive layers are covered with resin composites that exclude atmospheric oxygen and provide additional free radicals for polymerization. Although the latest generation of Adhesive bonding systems are hydrophilic and provide better bonds in wet environments, the wetness should be water, not blood or protein-rich ceveicular fluid which may lower bond strengths.[9]

III. Use of mouthguards
The use of a mouth guard-type appliance to deliver potassium nitrate desensitizing agent was first reported by Reinhart et al. (1990).

IV. Iontophoresis
The in- office use of iontophoresis of NaF to treat hypersensitive dentine has been used. Reports of lack of efficacy (Brough et al. 1985) may be due to inadvertent passage of current through adjacent gingival tissue rather than through cervical dentine.

Principles: Iontophoresis is the process of introducing ionic drugs in to body surfaces for therapeutic purposes, and is highly suited to therapy of conditions at or near body surface. High concentrations of drugs can be placed precisely where they are needed, rather than depending upon diffusion or systemic Administration.[10] Iontophoresis requires that: (a) a charged drug be delivered the electrode of the same polarity, (b) the condition or disease under treatment be delivered at the electrode of the same polarity, (b) the condition or disease under treatment be at or near the surface and (c) a modern, sophisticated source of direct current, with appropriate means of application, be used. This current source must have features that make it not only effective but also safe (Gangarosa and Jeske, 1992)

Medical and dental uses: Iontophoresis has a long history of use, having been suggested for various therapies for many years in medicine, physical therapy and dentistry.

Use of iontophoresis in desensitization: Iontophoresis with sodium fluoride solutions was proposed as a method of treating hypersensitive dentine.

V. Lasers
Another in office management for hypersensitive dentine is the use of lasers Renton –Harper and Midda (1992) evaluated an Nd:YAG laser to treat patients with cervical sensitivity to cold air. Treated roots were lased for 2 min at 10 pulses/s at increasing power levels until the patient detected the laser energy or until a maximum of 100mJ was reached. The presumed mechanism of action is the coagulation and precipitation of plasma proteins in dentinal fluid. It is also possible for the thermal energy to alter intradental nerve activity.

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Ruchi Gupta, et. al. "Dentine Hypersensitivity: An Overview." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 21(02), 2022, pp. 01-07.