Dentine hypersensitivity

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Introduction

Dentine hypersensitivity (DH) is defined as a 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 dental defect or pathology.

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. This 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. The prevalence distribution and appearance of the disease have been reported differently in different studies. Studies in the adult population have been reported that DH could affect as many as 1 in 7 of patients attending for dental treatment. Clinical studies and questionnaires on DH indicate a prevalence of 4% to 74% and the incidence ranging between 10-30%. DH could affect any age group; however, it is more prevalent in the patient with the age range of 30-40 (third decay) and more prevalent in female individuals. Regarding the type of teeth involved, canines and premolars of both the arches are the most affected teeth. Buccal aspect of cervical area is the commonly affected site.

Although DH can be triggered by various stimuli, however cold in the most common stimuli. Pain may also occur by chemical stimuli such as acidic foods (mainly fruit), sweets and rarely with salty foods. Mechanical stimulus frequently occurs when the patient rubs the sensitive area with a fingernail, or toothbrush bristles during brushing, setting off pain. The atmospheric air during mouth breathing, particularly in winter, which is associated with cold, or the air of a triple syringe by dehydration also causes pain.

Etiology

Dentin is covered and protected by hard tissues such as enamel or cementum. Dentin itself is a vital tissue, consisting of dentinal tubules, and is naturally sensitive because of extensions of odontoblasts and formation of dentine–pulp complex. Exposure of dentin could be either to removal of enamel covering the crown of the tooth or denudation of the root surface by loss of cementum & overlying periodontal tissues.

1. Enamel loss may result from (Fig 1)

a. Attrition relating to occlusal abnormalities. Attrition is defined as the wearing of the teeth surfaces due to normal or abnormal function.

b. Tooth brush abrasion which is wearing of the teeth substance through an abnormal mechanical process as incorrect brushing which leave a deep V-shaped cervical lesion.

c. Dietary erosion which is a chemical process (as acids) manifested as a localized progressive destruction of enamel & dentine. The defects vary in shape from saucer-like depressions to deep wedge-like grooves.

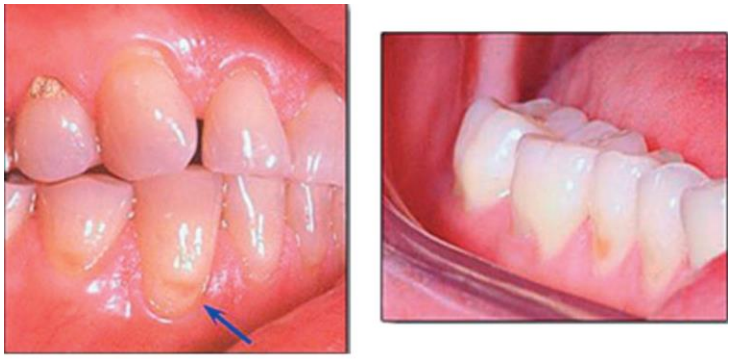

Figure 1: (A) Tooth abrasion, (B) tooth erosion. (Courtesy, Dr. Beatrice Gandara, University of Washington, School of Dentistry)

Exposure to non-bacterial acids in the diet, chemical products, medication, drugs or endogenous acids from reflux or regurgitation of stomach acid; that is, substances with low pH lead to the loss of dental structure by chemical dissolution without bacterial involvement. This process, produces a more softened enamel zone. In the cervical area, the thinner enamel can be gradually dissolved

and dentin becomes exposed to the oral environment. The acid environment can also open the dentinal tubules even further, leading to greater sensitivity.

d. Habits as grasping things between teeth.

2. Cementum loss could be due to:

a. Gingival recession which increase in severity with advancing age. Gingival Recession may be caused by:

- Mechanical trauma: hard brush, vigorous technique
- Predisposing anatomic factors
  - Thin gingiva
  - Prominent roots
  - Dehiscences
  - Fenestrations
  - Frenum pulls
  - Roots moved outside alveolar housing by orthodontic appliances.
- Faulty crown or restoration margins
- Periodontal disease
- Occlusal trauma
- Trauma from teeth in opposing jaw
- Oral habits (tobacco smoking & chewing)
- Poorly designed partial dentures
- Tooth position
- Healing response following periodontal surgery

b. Chronic periodontal disease as the root surface may become exposed as part of the disease process & the overlying cementum layer is thin & more easily removed.

c. Following periodontal therapy as scaling and root planing & periodontal surgery.

Scaling & root planing may lead to removal of the thin cementum layer during periodontal scraping & expose the dentinal tubules which induce the hypersensitivity which is transitory it reaches the peak in the first week after treatment & subside or disappear within few weeks.

Also, after periodontal surgery in which large root surface area often exposed so leading to hyper sensitivity. However, occasionally the condition may become a chronic pain problem and may persist for months or years. Patients appear to be especially at risk after periodontal surgery.

The increase in pain intensity after periodontal therapy may have one or both of the following two explanations.

Firstly, the smear layer formed on the root surface by the scaling procedure will be dissolved within a few days. This in turn will increase the hydraulic conductance of the involved dentinal tubules and thus decrease the peripheral resistance to fluid flow across dentin. Thereby pain sensations are more readily evoked.

Secondly, open dentinal tubules serve as pathways for diffusive transport of bacterial elements in the oral cavity to the pulp, which is likely to cause a localized inflammatory pulpal response.

The fact that root dentin hypersensitivity often disappears a few weeks after the scaling procedure is best explained by the development of a natural occlusion of the exposed dentinal tubules by mineral deposits.

d. Physiological causes. The increase in the number of teeth with root exposure is evident, as age advances. Dental extrusion, in the absence of an antagonist tooth, results in root exposure, which may lead to DH.

e. Anatomic variations: failure of meeting between enamel and cementum at CEJ.

The main symptoms of dentine hypersensitivity (D.H) is sharp pain of rapid onset & short duration provoked by different stimuli & usually resolves immediately after withdrawal of the stimulus. In more severe, long-standing cases, shorter or longer periods of lingering, dull or aching pain symptoms may be provoked.

Dental caries with pulpal changes has the same symptoms but in the absence of other dental pathology, these symptoms refers to DH.

Theories of DH

Three main mechanisms of dentin sensitivity are proposed (Fig 2):

A. Direct Innervation (DI) theory

B. Odontoblast Receptor (OR) theory

C. Fluid Movement/Hydrodynamic theory

According to DI theory, nerve endings penetrate dentine and extend to the dentino-enamel junction. Direct mechanical stimulation of these nerves will initiate an action potential. There are many shortcomings of this theory. There is lack of evidence that outer dentin, which is usually the most sensitive part, is innervated. Moreover, pain inducers such as bradykinin fail to induce pain when applied to dentine, and bathing dentine with local anesthetic solutions does not prevent pain, which does so when applied to skin.

OR theory states that odontoblasts act as receptors by themselves and relay the signal to a nerve terminal. But majority of studies have shown that odontoblasts are matrix forming cells and hence they are not considered to be excitable cells, and no synapses have been demonstrated between odontoblasts and nerve terminals

Brannstrom (1964) has proposed that dentinal pain is due to hydrodynamic mechanism, i.e., fluid force. Scanning electron microscopic (SEM) analysis of “hypersensitive” dentin shows the presence of widely open dentinal tubules. The presence of wide tubules in hypersensitive dentin is consistent with the hydrodynamic theory. This theory is based on the presence and movement of fluid inside the dentinal tubules. This centrifugal fluid movement, in turn, activates the nerve endings at the end of dentinal tubules or at the pulp–dentine complex. This is similar to the activation of nerve fibers surrounding the hair by touching or applying pressure to the hair. It has been noted that stimuli which tend to move the fluid away from the pulp–denitin complex produce more pain. These stimuli include cooling, drying, evaporation and application of

hypertonic chemical substances. Approximately, 75% of patients with DH complain of pain with application of cold stimuli. This theory is the most accepted one related to the explanation of DH.

In general, the “hypersensitive” dentin has more widely open tubules and thin/under calcified smear layer as compared with “non-sensitive” dentine. The wider tubules increase the fluid movement and thus the pain response.

Diagnosis of DH

Like any other clinical condition, an accurate diagnosis is important before starting the management of DH. Diagnosis of DH starts with a thorough clinical history and examination. The other causes of dental pain should be excluded before a definite diagnosis of DH is made. Some of these techniques include pain response upon the pressure of tapping teeth (to indicate pulpitis/periodontal involvement), pain on biting a stick (suggests fracture), use of transilluminating light or dyes (to diagnose fractures), and pain associated with recent restorations.

A simple clinical method of diagnosing DH includes a jet of air or using an exploratory probe on the exposed dentin, in a mesio-distal direction, examining all the teeth in the area in which the patient complains of pain. The severity or degree of pain can be quantified either according to categorical scale (i.e., slight, moderate or severe pain) or according to using a visual analogue scale (VAS).

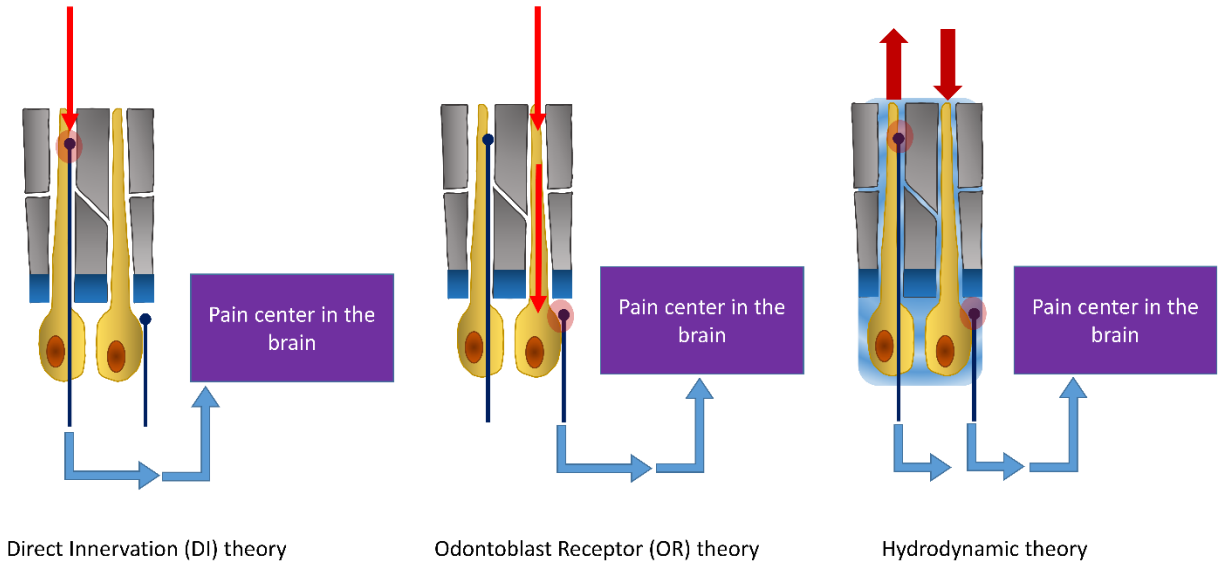

Figure 2: Theories of DH

Differential diagnosis

DH has features which are similar to other conditions (Table 1)

Dental-related reasons	**Non-Odontogenic origin**
Cracked tooth syndrome	Musculoskeletal
Fractured restoration	Neuropathic
Chipped teeth	Neurovascular
Dental caries	Inflammatory (sinusitis)
Periodontal disease	Systemic (cardiac, herpes zoster, sickle cell anemia, neoplasm)
Post-restorative sensitivity	Psychogenic
Pulpitis	Referred pain
Palato-gingival groove	
Bleaching sensitivity	
Marginal leakage	

Table 1: Potential reasons resembling DH

Taking these factors into consideration, it is necessary to exclude other forms of pain or dental sensitivity.

Methods of measuring DH

Although DH is a subjective sensation that varies greatly from one individual to another and in the same individual at different times. However, some methods of measuring pain arising from DH were introduced.

A Visual Analogue Scale (VAS) is a measurement instrument that tries to measure a characteristic or attitude that is believed to range across a continuum of values and cannot easily be directly measured. Operationally a VAS is usually a horizontal line, 100 mm in length, anchored by word descriptors at each end. The patient marks on the line the point that they feel represents their perception of their current state. The VAS score is determined by measuring in millimeters from the left-hand end of the line to the point that the patient marks (Fig 3).

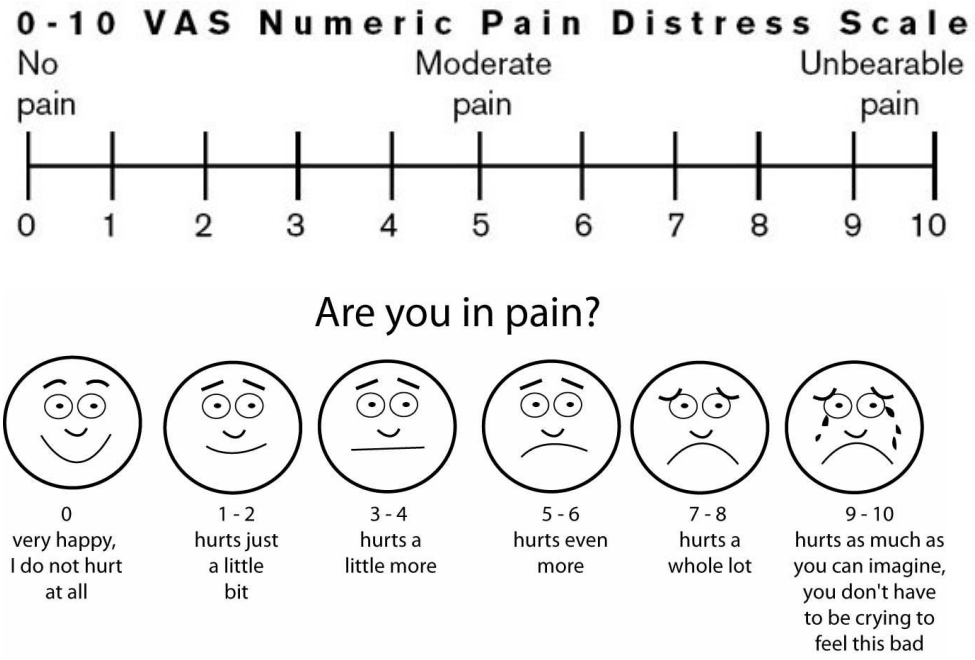

Figure 3: Visual analogue scale (VAS)

Treatment of DH

Spontaneous cure may occur by the natural remineralization process in the mouth, which promotes natural tubular occlusion of dentin, and pain may return because of the smear layer removal by food and acidic drinks thus explaining the cyclic characteristic of DH.

After observing the severity and number of teeth involved, an active approach to DH can begin in the cases of generalized DH, by a home method followed by in-office treatment when the first option is not successful.

This principle of treatment is based on using different types of desensitizing agents that either occlude or reduce the diameter of dentinal tubules, which minimize the fluid movement in these tubules, and hence reducing the pain. Desensitizing agents need to have certain requirements that include:

1. Non-irritant to the pulp.
2. Relatively painless on application.
3. Easily applied.
4. Rapid in action.
5. Effective for a long time without staining effects.

Clinical management of DH is the identification and treatment of the causative factors of DH. By removing the etiological factors, the condition can be even prevented from occurring or recurring. **In severe cases**, where no remedy is achieved with any advice or treatment approach, pulpectomy and root filling or even extraction may be the last resort.

Hypersensitivity may prevent proper plaque control, therefore treatment of hypersensitivity with plaque control measures may be created. Plaque control is an important integral part of the prevention and treatment of root dentin hypersensitivity. It has been observed clinically that, with time, teeth in patients with excellent oral hygiene habits develop hard, smooth and insensitive root surfaces.

However, when severe symptoms of root hypersensitivity have emerged it is difficult to motivate the patient to maintain the degree of plaque control that is necessary to allow for a natural occlusion of the dentinal tubules. In such situations an agent may be beneficial which has a reasonable capacity to block the tubular openings, at least temporarily, so that proper oral hygiene measures can be reinforced.

CLASSIFICATION OF DESENSITIZING AGENTS

1- <u>Mode of administration</u>

A- At home desensitizing therapy

These "at home" desensitizing agents include toothpastes, mouthwashes and chewing gums. Toothpastes are widely indicated, particularly because of their low cost, ease of use and home application. They are effective but it often takes four to eight weeks to achieve pain relief in addition to the need of patient's compliance.

B- In-office desensitizing agents

Dental professionals can deliver a wider range of more complex and more potent desensitizing treatment with immediate relief from pain of DH. A variety of office applied agents are currently available, which include cavity varnishes, calcium compounds, oxalates, resins and adhesives, restorative materials, laser treatment and an aqueous solution of glutaraldehyde and hydroxyethyl methacrylate.

2- <u>On the basis of mechanism of action (Table 2)</u>

1. Nerve desensitization
   - Potassium nitrate
2. Anti-inflammatory agents
   - Corticosteroids
3. Cover or plugging dentinal tubules
   a. Plugging (sclerosing) dentinal tubules
      i- Ions/salts
   - Calcium hydroxide

- Ferrous oxide
- Potassium oxalate
- Sodium monofluorophosphate
- Sodium fluoride
- Sodium fluoride/stannous fluoride combination
- Stannous fluoride
- Strontium chloride

ii- Protein precipitants

- Formaldehyde/Glutaraldehyde
- Silver nitrate
- Strontium chloride hexahydrate
- Casein phosphopeptides
- Burnishing
- Fluoride iontophoresis

b. Dentine sealers

- Glass ionomer cements
- Composites
- Resins
- Varnishes
- Sealants
- Methyl methacrylate

c. Periodontal soft tissue grafting

d. Crown placement/restorative material

e. Lasers

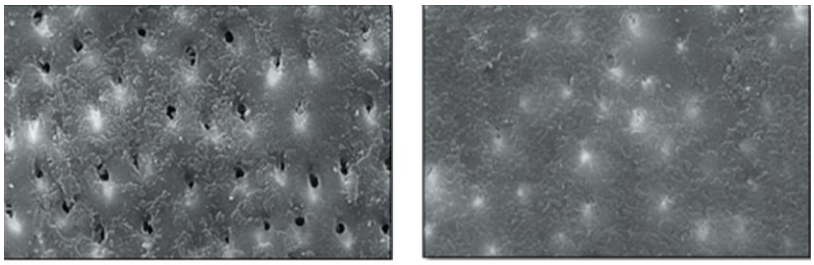

Figure 4: Open tubules (A), (B) Closed tubules following treatment with SnF2 dentifrice.

Dentinal hypersensitivity is a relatively common and significant dental problem which can be successfully managed by a very wide variety of procedures, agents and formulations applied locally, either “in office” or “at home”. It is clear that some products appear to be more effective than others.

Mode	Desensitizing agent	Mechanism	Vehicle or mode of application
Nerve desensitization	Pottasium nitrate	Reduces the excitability of the nerve transmitting pain	5-10% concentration in toothpaste, commercially known as EMOFORM
Covering or plugging dentinal tubules (ions/salts)	Calcium hydroxide	- Binding of calcium ions to the protein in the exposed dentinal tubules - Increases mineralization of exposed dentine	In-office application (requires multiple applications to maintain its effect)
	Sodium fluoride, Sodium monofluorophospha te, Stannous fluoride	- Precipitation to the exposed dentinal tubules by forming insoluble precipitates - Increases resistance of dentine to acid decalcification	- Fluoride-containing toothpaste - Professionally applied
	Oxalates	Reacts with the calcium ions of dentine and forms calcium oxalate crystals inside the dentinal tubules as well as on the dentinal surface	Topical application of 3% potassium oxalate. Action can be improved by acid etching of the dentinal surface, thus increasing the penetration of calcium oxalate crystals deep into the dentinal tubules
	Strontium chloride	Reduces fluid movement in the dentin tubules by occluding or sclerosing the tubules.	Toothpaste, commercially known as SENSODYNE
Covering or plugging dentinal tubules (Protein precipitation)	Formaldehyde or glutaraldehyde	Precipitate salivary proteins in dentinal tubules. They should be used with extreme caution because they are strong tissue fixatives	Toothpaste

Dentine sealers	Resins and adhesives	Seal the dentinal tubules effectively by forming a hybrid layer	Professionally applied ex: Gluma and Copal. This technique is generally reserved for localized rather than generalized dentinal pain
	Varnishes	Enhance therapeutic action of other agents such as fluoride and chlorhexidine. Re-application is required	Professionally applied
	Restorative materials	Glass ionomers and composites	Occlude dentinal tubules and restore tooth contours Indicated in case of significant loss of cervical tooth structure or in case of failure of other less invasive desensitizing protocols
Lasers	Nd:YAG and CO_2 lasers	- Coagulate protein inside dentinal tubules, thus preventing fluid movement - Enhance the action of other desensitising agents such as sodium fluoride and stannous fluoride	Professionally applied
Periodontal surgery	Lateral sliding grafts, free gingival grafts, connective tissue grafts and coronally repositioned flaps	Cover exposed dentinal tubules	In general, soft tissue grafting for the management of sensitivity is not regarded as a very predictable treatment strategy

Table 2: Classification of desensitizing agents according to the mode of action

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