



INTERNATIONAL JOURNAL OF CREATIVE RESEARCH THOUGHTS (IJCRT)

An International Open Access, Peer-reviewed, Refereed Journal

DENTIN HYPERSENSITIVITY: A REVIEW

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Abstract: Dentine sensitivity (DS) or dentin hypersensitivity (DH) is a sequel to dentin exposure. It is the most frequently reported dental problem in daily practices. It occurs due to dentin exposure to various etiological factors leading to discomfort and deterioration to the quality of life of affected individual as it causes striking pain, difficulty in food consumption as well as esthetic appearance. The most neglected phase during DH management is identification and treatment of causative factor. Dentist should not underestimate the role causative factor plays in localizing and initiating hypersensitive lesions. The objective of this article is to highlight the etiopathogenesis, mechanism, diagnosis and clinical management of DH.

Keywords: Dentin Hypersensitivity, dentin exposure, dentine sensitivity, stimuli, desensitizing.

I. Introduction:

Dentin hypersensitivity is defined as a sharp pain arising from the exposed dentin as a result of various stimuli like heat, cold, chemicals, or due to bacteria^[1] and this occur when exposed dentin gets affected by thermal, evaporative, tactile, osmotic, or chemical stimuli.^[2] It is commonly related to the loss of enamel due to abrasion, abfraction or erosion, gingival recession, or the loss of cementum due to periodontal treatment.^[3] DH is increasingly recognized as an important issue to be addressed from both diagnostic and problem-management perspectives because of the improved success of caries prevention and periodontal disease management measures have resulted in improved oral health status and dentition function throughout life.^[4] DH is commonly seen in adults due to various etiological factors and in various individual group and less commonly seen in children. Caleb Shitsuka et al (2015) reported that dentin hypersensitivity in children is associated with dietary habits, developmental disturbance of teeth and salivary flow.^[5]

II. Prevalence:

DH commonly affects 8-57% of the population with an increased prevalence of 72-98% in periodontal patients aged between 30-40 years, mostly women.^[6-8] These variations were related to the difference in population, habits, dietaries & methods of investigations (patient questionnaires or clinical examination).^[9-11] Teeth involved are canines and premolars of both the arches. Also it was reported that buccal aspect of cervical area is the commonly affected site.^[12]

III. Etiopathogenesis:

It has been postulated that DHS evolves in two phases: localization of lesions and initiation of lesions.^[13]

In the first phase, due to loss of enamels dentin are exposed by attrition, abrasion, erosion, and abfraction, along with the loss of cementum on the root surface. As compared to non-sensitive dentin, their smear layer is thin which leads to increase in fluid movement and therefore pain response.^[14-16]

In the second phase, the tubular plug and smear layer is removed from the exposed dentin, in consequence of which dentinal tubules and pulp are exposed to external stimuli causing dentinal hypersensitivity.^[17]

According to various studies, dentin exposure occurs due to one or more processes;^[18-19]

1. Improper tooth brushing
2. Premature contacts
3. Gingival recession
4. Exogenous or endogenous acids (erosive agents)
5. Poor oral hygiene

IV. Theories:

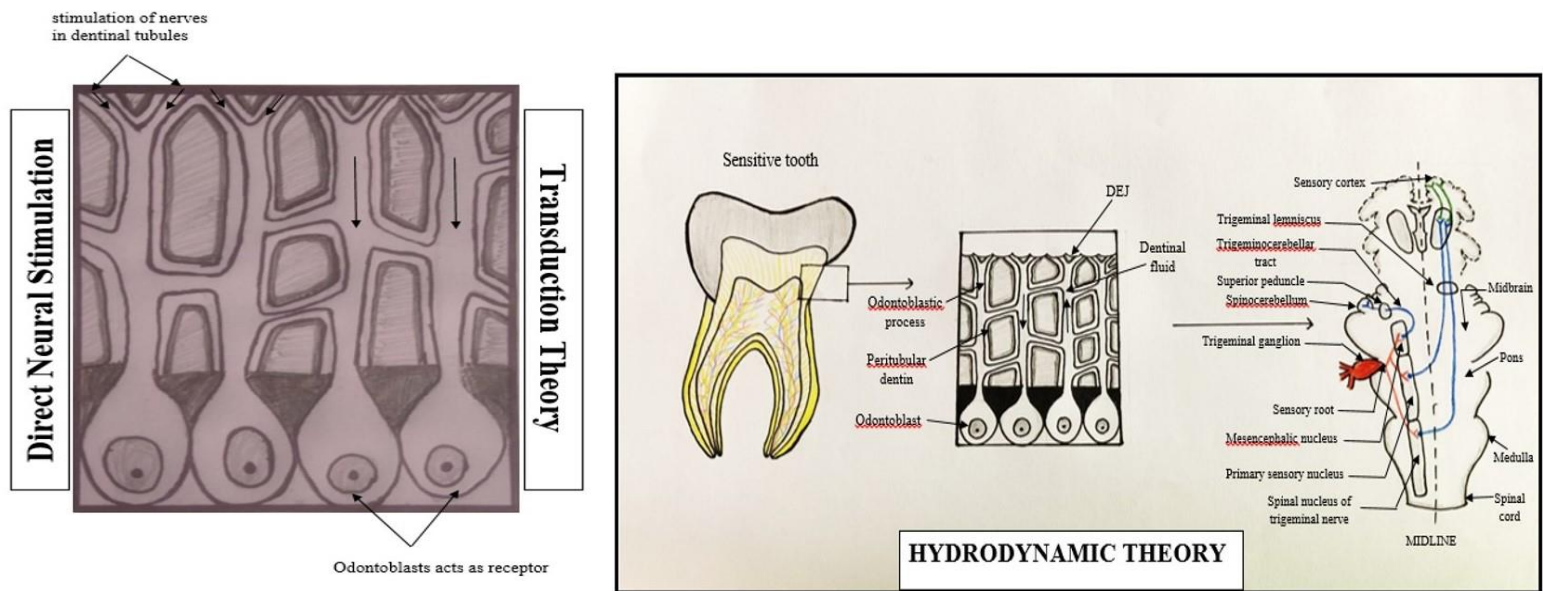


Fig.1 Theories of Dentin Hypersensitivity

Direct innervation theory

According to direct innervation theory, nerve endings penetrate dentine and extend to the dentino-enamel junction,^[20] direct mechanical stimulation of these nerves will initiate an action potential. This theory fails due to short comings and lack of evidence.

Hydrodynamic theory

Hydrodynamic Theory for dentine sensitivity was first proposed by Brannstrom and is the most widely accepted theory.^[21] The theory is based on the movement of the fluid inside the dentinal tubules. This theory states that tubules are open between dentine surface which is exposed to the environment and pulp.^[21-22] It is believed that dentin hypersensitivity occurs due to fluid movement inside the dentinal tubule when the dentin is exposed to the external stimuli such as thermal, physical or chemical changes or as a result of formation of osmotic stimuli near the exposed dentin. This fluid movement inside the tubule stimulates the baroreceptor and leads to neural discharges. This process is therefore known as hydrodynamic theory of pain.^[23]

Transduction theory

The transduction theory was proposed by Rapp et al. The theory states that odontoblasts act as receptor cells, and transmit impulses via synaptic junctions to the nerve terminals causing the sensation of pain from the nerve endings located in the pulpodentine border.^[24] However, the theory was unconvincing and lack evidences.

V. Diagnosis:

The clinical examination should include identification and assessment of all sensitive teeth and confirming it will clinical signs such as dental erosion, gingival recession, and exposed cervical dentin.^[25-26] It should include different trigger factors^[27,18] such as:

1. Thermal and evaporative stimuli
2. Mechanical/tactile stimuli
3. Chemical (osmotic) stimuli
4. Electrical stimulation

VI. Management of Dentin Hypersensitivity:

1. Removing etiologic factors and preventing DH

First and foremost management of dentin hypersensitivity is removal of causative factor and preventing it from further damage. It is important to note from the literature that there are individuals who are at risk of DH; they are ^[18]

- Overenthusiastic brushers
- Periodontal treated patients
- Bulimics
- People with xerostomia
- High-acid food/drink consumers
- Older people exhibiting gingival recession
- Chewing 'smokeless' or 'snuff' tobacco

2. Use of desensitizing agents

Classification of desensitizing agents ^[28,9]

I. Mode of administration

- At home desensitizing agents
- In-office treatment

II. On the basis of 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 ($\text{SiO}_2\text{-P}_2\text{O}_5\text{-CaO-Na}_2\text{O}$)

Dentine adhesive sealers

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



Lasers

- Neodymium:yttrium aluminum garnet (Nd-YAG) laser
- GaAlAs (gallium-aluminium-arsenide laser)
- Erbium-YAG laser

Homeopathic medication

- Propolis

III. On the basis of manufacturer

- Sensodyne
- Colgate
- Aloe Dent
- Crest
- Pepsodent
- Himalaya
- Fresh Mint
- Emoform-R
- Advanced Sensitive
- Aquafresh
- Thermoseal
- Elimex
- Biorepair
- Dontodent

IV. On the basis of key elements

- Natural elements
- Chemical elements

VII. Conclusion:

Though, there have been change in lifestyle from the last few decades which shows current consumption of acidic food and beverages is extremely high, especially in children. Chemical stimulus is one of the causative factor for causing sensitivity, thus discomfort to patients. Enamel erosion due to developmental disturbance also involves for exposing dentinal tubules to the external stimuli. Along with this, salivary factor that is associated is salivary rate flow. Lack of salivary flow hinder the defense mechanism and various other functions of saliva. In conclusion, dietary habits, developmental disturbance, salivary flow contributes to dentinal sensitivity associated in children. Further investigations are required for pediatric dentist to understand and manage DH in children.

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