

## DENTINAL HYPERSENSITIVITY – A REVIEW

Dr. Surabhi Parakh<sup>1</sup>, Dr. Vijaykumar Shiraguppi<sup>2</sup>, Dr. Bharat Deosarkar<sup>3</sup>, Dr. Maneesha Das<sup>4</sup>,  
Dr. Godavari Nagargoje<sup>5</sup>, Dr. Kiran Wanve<sup>6</sup>

Post graduate student<sup>1</sup>, Prof & HOD<sup>2</sup>, Reader<sup>3,4</sup> Sr. Lecturer<sup>5</sup>, Post graduate student<sup>6</sup>

Department of Conservative Dentistry and Endodontics, Saraswati Dhanwantari Dental College and Hospital & Post-Graduate Research Institute, Parbhani, Maharashtra, India.

### Abstract

Tooth sensitivity is a very common clinical presentation which can cause considerable concern for patients. This condition is frequently encountered by periodontists, dentists, hygienists and dental therapists. The management of this condition requires a good understanding of the complexity of the problem, as well as the variety of treatments available. This review considers the etiology, incidence and management of dentinal hypersensitivity.

**Key words:** Dentinal hypersensitivity, desensitizing agents.

### INTRODUCTION

Dentinal Hypersensitivity has been defined as “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”.<sup>1</sup> It is brief, sharp pain arising from exposed dentin. It occurs typically in response to chemical, thermal, evaporative or osmotic stimuli and cannot be explained as arising from other dental defects or pathology. The primary cause of dentin hypersensitivity is loss of enamel on the tooth crown and gum recession exposing the tooth root, with subsequent loss of cementum.<sup>2</sup>

Dentin hypersensitivity is a prevalent oral problem affecting more than 40% of the adults. Patients with periodontal diseases are at particularly high risk, and some studies have found that over 70% of patients with periodontal disease have experienced dentin hypersensitivity. Several studies have reported non-carious cervical lesions (NCCLs) and DH in adult populations, with prevalence rates ranging from 5% to

85% and 2-8% to 74%, respectively.<sup>3,4</sup> prevalent in the patient with the age range of 30-40 and more prevalent in female individuals that would probably be related to their dental hygiene and dietary.<sup>5-6, 7-8, 9</sup>

### ETIOLOGY<sup>4</sup>

The primary cause of dentin hypersensitivity is loss of enamel on the tooth crown and gum recession exposing the tooth root. The most common clinical cause for exposed dentinal tubules is gingival recession which in turn may be caused by:

1. Inadequate attached gingiva
2. Prominent roots
3. Toothbrush abrasion
4. Pocket reduction periodontal surgery
5. Oral habits resulting in gingival laceration, i.e., traumatic tooth picking, eating hard foods
6. Excessive tooth cleaning
7. Excessive flossing
8. Gingival loss secondary to specific diseases, i.e., NUG, periodontitis
9. Crown preparation

### Other causes include:

1. Loss of enamel
2. Denudation of cementum
3. Attrition
4. Abrasion
5. Abfraction
6. Erosion (intrinsic and extrinsic)
7. Thinning, fenestration, absent buccal alveolar bone plate
8. Bleaching
9. Scaling and root planning: Removes 20-50µm of cementum and expose the dentinal tubules to a variety of stimuli.



**Enamel loss exposing dentine of tooth**



**Loss of gingival tissues exposing dentin of tooth roots**



**Dental erosion due to frequent intake of acidic beverages**

### Theories For Dental Hypersensitivity<sup>5</sup>

Three major mechanisms of dentinal sensitivity have been proposed in the literature:

#### Odontoblastic transduction theory

#### Neural theory

#### Hydrodynamic theory

#### Odontoblastic transduction theory

The odontoblast transducer theory proposed by **Rapp et al.** postulated 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. However, evidence for the odontoblast transducer mechanism theory is deficient and unconvincing. This is because the majority of studies have shown that odontoblasts are matrix forming cells and they are not considered to be excitable cells, and no synapses have been revealed between odontoblasts and nerve terminals<sup>3</sup>

#### Neural theory

This theory advocated that thermal, or mechanical stimuli, directly affect nerve endings within the dentine tubules through direct communication with the pulpal nerve endings. Although this theory has been reinforced by the presence of unmediated nerve fibers in the outer layer of root dentine and the presence of putative neurogenic polypeptides, it is still considered theoretical with lack of solid evidences to support it.<sup>3</sup>

#### Hydrodynamic theory

The currently accepted mechanism of dentine hypersensitivity is the hydrodynamic theory which has been proposed by **Brannstrom** in 1964. According to this theory, when the exposed dentin surface is subjected to thermal, chemical, tactile or evaporative stimuli, the fluid flow within the dentine tubules will be increased. This fluid movement within the dentine tubules causes

an alteration in pressure and excites pressure-sensitive nerve receptors across the dentine. So the response of the excited pulpal nerves, mainly in intradentine fibers, will be depended upon the intensity of stimuli in pain production. Scanning electron microscopic (SEM) examination of hypersensitive dentin surface reveals the presence of widely open dentine tubules which is considered consistent with the hydrodynamic theory. Accordingly, the number and the diameter of the dentine tubules are considered important factors in initiating pain from DHS. Hence, the higher the number and greater the diameter of the open dentine tubules the more intense will be the pain from DHS. It has been noted that triggers such as cold stimuli stimulate fluid to flow away from the pulp creating more rapid and rigorous neural responses than heat stimuli, which cause somewhat sluggish fluid flow towards the pulp. This is aligned with the observation that dentine hypersensitivity patients are more frequently complain of pain in response to cold stimuli than to heat.<sup>3</sup>

#### **Treatment strategies for dentinal hypersensitivity<sup>4</sup>**

##### 1. Nerve desensitization

Potassium nitrate

##### 2. Anti-inflammatory agents

Corticosteroids

##### 3. Cover or plugging dentinal tubules

###### a. Plugging (sclerosing) dentinal tubules

Ions/salts

Calcium hydroxide

Ferrous oxide

Potassium oxalate

Sodium monofluorophosphate

Sodium fluoride

Sodium fluoride/stannous fluoride combination

Stannous fluoride

Strontium chloride

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

resin-modified GIC

Clinpro™ XT

conventional GIC Vidrion R

c. Periodontal soft tissue grafting

d. Crown placement/restorative material

e. Laser

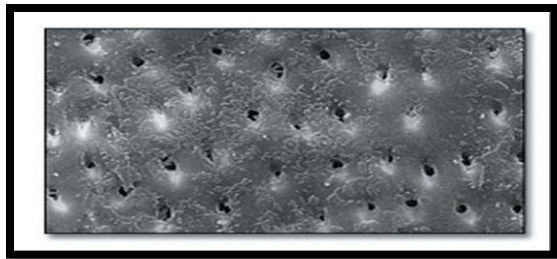
f. Casein phosphopeptide–amorphous calcium phosphate

g. Bioglass

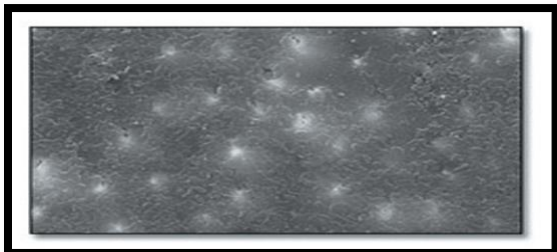
#### **Recently introduced material-**

##### a. Carbonated hydroxyapatite nanocrystals

More recently, the toothpastes containing carbonated hydroxyapatite nanocrystals are being studied. These have high reactivity by which they bind to enamel and dentine apatite producing a biomimetic coating on enamel, contrasting plaque formation. They also prevent tooth from decay, rebuild and revitalize the teeth and seal dentinal tubules, annulling hypersensitivity. In near future new products of this kind will be a breakthrough in the treatment of dentinal hypersensitivity.<sup>5</sup>



Open tubules following treatment  
Tubules following treatment



Closed With non-sensitivity fluoride  
Toothpaste. With SnF2 dentifrice

**Recommended actions for preventing dentine hypersensitivity (adapted from Drisko, 2002)**

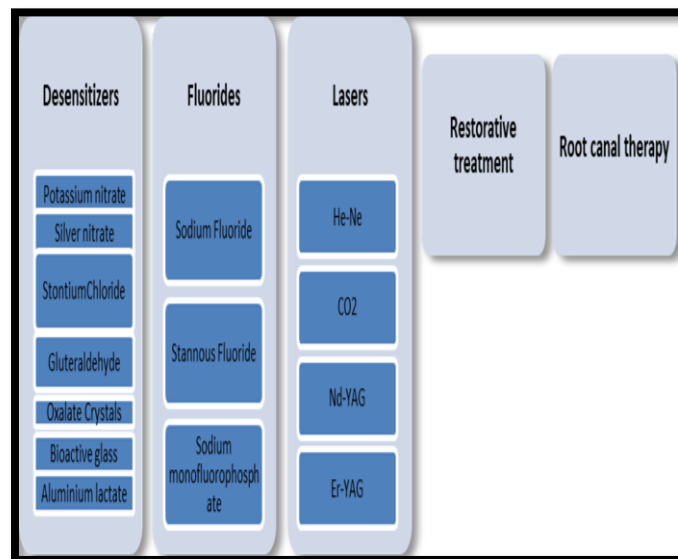
**Suggestions for patients**

- Avoid using large amounts of dentifrice or reapplying it during brushing
- Avoid medium- or hard-bristle toothbrushes
- Avoid brushing teeth immediately after ingesting acidic foods
- Avoid brushing teeth with excessive pressure or for an extended period of time
- Avoid excessive flossing or improper use of other interproximal cleaning devices
- Avoid “picking” or scratching at the gumline or using toothpicks inappropriately.<sup>6</sup>

**Suggestions for dental professionals**

- Avoid over-instrumenting the root surfaces during scaling and root planing, particularly in the cervical area of the tooth
- Avoid over-polishing exposed dentine during stain removal during in-office bleaching, and advise patients to be careful when using home bleaching products

during in-office bleaching, and advise patients to be careful when using home bleaching products.<sup>6</sup>



**TREATMENT OPTIONS FOR DENTINAL HYPERSENSITIVITY<sup>3</sup>**

**Conclusion**

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”. Clinically, Dentinal hypersensitivity is a relatively common and significant dental problem for which patients look for treatment and visit dental clinics. There are many treatment modalities for Dentinal hypersensitivity which the clinician may find successful in relieving the pain of Dentinal hypersensitivity. The dental practitioner should first identify the causative or predisposing factor after taking a thorough history before the treatment plan is designed. The treatment strategy of the Dentinal hypersensitivity should be begun with prevention, selfcare management and later may be supplemented with professional interventions depending on the severity of the case.<sup>8</sup>

## REFERENCES

- 1) Davari AR, Ataei E, Assarzadeh H. Dentin hypersensitivity: etiology, diagnosis and treatment; a literature review. *Journal of Dentistry*. 2013 Sep;14(3):136.
- 2) Bartold PM. Dentinal hypersensitivity: a review. *Australian dental journal*. 2006 Sep;51(3):212-8.
- 3) Bartlett DW, Shah P. A critical review of non-carious cervical (wear) lesions and the role of abfraction, erosion, and abrasion. *J Dent Res* 2006; 85: 306-312.
- 4) Que K, Guo B, Jia Z, Chen Z, Yang J, Gao P. A crosssectional study: non-carious cervical lesions, cervical dentine hypersensitivity and related risk factors. *J Oral Rehabil* 2013; 40: 24-32.
- 5) Porto IC, Andrade AK, Montes MA. Diagnosis and treatment of dentinal hypersensitivity. *J Oral Sci* 2009; 51: 323-332.
- 6) Miglani S, Aggarwal V, Ahuja B. Dentin hypersensitivity: Recent trends in management. *J Conserv Dent* 2010; 13: 218-224.
- 7) Aranha AC, Pimenta LA, Marchi GM. Clinical evaluation of desensitizing treatments for cervical dentin Hypersensitivity. *Braz Oral Res* 2009; 23: 333-339.
- 8) Chu CH, Lo ECM. Dentin hypersensitivity: a review. *Hong Kong Dent J* 2010; 7: 15-22.
- 9) Cummins D. Recent advances in dentin hypersensitivity: clinically proven treatments for instant and lasting sensitivity relief. *Am J Dent* 2010; 23 Spec No A:3A- 13A.
- 10) Suchetha A, Prasad K, Apoorva SM, Lakshmi P. Dentinal Hypersensitivity-A Review. *Indian Journal of Dental Sciences*. 2013 Jun 1;5(2).
- 11) Najat Bubteina and Sufyan Garoushi. Dentine Hypersensitivity: A Review; 2015; 5(9)
- 12) Somya Khetawat and Surendra Lodha, Nanotechnology (Nanohydroxyapatite Crystals): Recent Advancement in Treatment of Dentinal Hypersensitivity. *JBR Journal of Interdisciplinary Medicine and Dental Science*. 2015, 3:3
- 13) Chun-Hung Chu, Edward Chin-Man Lo. Dentin hypersensitivity: a review *Hong Kong Dent J* 2010; 7:15-22
- 14) Holland GR, Narhi MN, Addy M, Gangarosa L, Orchardson R. Guidelines for the design and conduct of clinical trials on dentine hypersensitivity. *J Clin Periodontol* 1997;24:808-13.

### Corresponding Author Details:

Dr. Surabhi Parakh, PG students, Department of Conservative Dentistry Saraswati Dhanwantari Dental College & Hospital & Post Graduate Research Institute, Parbhani, Maharashtra, India.