Review Article

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Dentin Hypersensitivity: An Enigma

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ABSTRACT

Background: Dentinal hypersensitivity is a common clinical condition usually associated with the exposed dentinal surface. It can affect patients of any age group and most commonly affects canine and premolars. This paper reviews the mechanism and clinical management of the dentinal hypersensitivity. By removing the etiological factors, the condition can be even prevented from occurring or recurring. There are various treatment modalities available. The desensitising agents include toothpaste, mouthwashes and chewing gums and they act by either occluding the dentinal tubules or blocking the neural transmission. The recent treatment option like bioglass, Portland cements, lasers and casein phosphopeptides are discussed.

Keywords: Hypersensitivity, desensitizing agents, toothpaste, bioglass.

INTRODUCTION

Dentinal hypersensitivity is a significant clinical problem. It is defined as "pain arising from exposed dentine typically in response to thermal, chemical, tactile or osmotic stimuli".[1] These stimuli are produced by the ingestion of hot or cold beverages, by contact with acidic foods, or by tooth brushing. Pain may be localized or generalized, affecting one or more tooth surfaces concomitantly, and generally ceases immediately after removing the pain stimulus.[2]

The dentinal hypersensitivity has been shown to have a prevalence range of 4 to 74% and is reported to increase with advancing age.[3] 4% to 74% prevalence variations is due to the difference in populations and methods of investigations. The prevalence range of root sensitivity due to only periodontal disease conditions are 85-95%.[16] The methods employed are usually patient questionnaires or clinical examinations. Interestingly, the incidence of dentinal hypersensitivity is much higher inpatient questionnaire studies than in clinical studies which quote an incidence of a mere 15%. [4]

A slightly higher incidence of dentinal hypersensitivity was reported in females than in males; while dentinal hypersensitivity can affect the patient of any age, most affected patients are in the age group of 20–50 years, with a peak between 30 and 40 years of age. Regarding the type of teeth involved, canines and premolars of both the arches are the most affected teeth. The buccal aspect of the cervical area is the commonly affected site.[4]

Various means of dentine exposure was reported in the past, which include attrition, abrasion or erosion. Alternatively, in some individuals, the cementum and enamel which normally cover the dentine do not meet and result in dentine exposure as a result of a developmental anomaly.[1]

The most common aetiology for radicular dentin exposure is "recession" of the gingival marginal tissues. This process is characterised by the displacement of the gingival margin apical to the

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cementoenamel junction thereby exposing visible cementum of the root surface, which is then rapidly lost.[5]

Plaque accumulation on root surfaces may lead to demineralization of tooth structures which was associated with patency of dentinal tubule orifices, leading to dentinal hypersensitivity.[1] The establishment of healthy periodontal conditions by non-surgical periodontal treatment may result in a number of undesired side effects, such as, the gingival recession that leaves the root surface exposed to the oral environment, and the iatrogenic denudation of root – dentine due to removal of the cementum layer by scaling and root planning procedures. A large number of dentinal tubules will thus be exposed leading to direct avenues to the pulp for bacteria and bacterial elements present in the oral environment. As a result, the patient may experience increased sensitivity of the exposed dentine.[6]

Dentin consists of microscopic channels, called dentinal tubules, which radiate outward through the dentin from the pulp to the exterior cementum or enamel border.[7] The dentinal tubules extend from the dentinoenamel junction (DEJ) in the crown area, or dentinocemental junction (DCJ) in the root area, to the outer wall of the pulp.[8] From the outer surface of the dentin to the area nearest the pulp, these tubules follow an S-shaped path. The diameter and density of the tubules are greatest near the pulp, its diameter at dentino- enamel junction is 0.06 µm, and 3.0 µm at the pulpal wall and most of these tubules are filled with fluid, and odontoblast process, collagen, and occasional non-myelinated pulpal nerves.[9] These nerve fibres are in contact with the odontoblasts and act as mechanical receptors that cause pain. (Ten Cate 1998).[6]

Several theories have been put forth related to the aetiology of dentine sensitivity; the most widely accepted is the hydrodynamic theory that is based on the excitability threshold changes of intrapulpal nerve fibres. The hydrodynamic theory was first proposed by Gysi (1900)[6] and reported that fluid distortion on the cavity floor caused sharp pain. Later Brannstrom, (1966, 1986 and 1996)[6] demonstrated that dentine sensitivity is the result of rapid fluid movement in the tubule complex, aggravated by aggressive air drying.

Dentine sensitivity can be treated in different ways, which can be used at home or maybe professionally applied. The "at home" desensitizing agents include toothpaste, mouthwashes or chewing gums and professionally by applying over the teeth. They act by either occluding the dentinal tubules or blocking the neural transmission.; and also first by reducing the dentinal tubules hypo conduction by occluding them; second, by reducing the nerve fibres excitability and by a combination of these two approaches also can be done.(Gillam & Orchardson, 2006).[10]

Substances capable of forming a crystalline precipitate that occludes the dentinal tubules are found in solutions and dentifrices or can be applied through iontophoresis as stannous fluoride and strontium chloride (Rees 2000).[11] Potassium nitrate, potassium bicarbonate and potassium chloride are active agents that can reduce nerve excitability (Gillam &Orchardson, 2006), [10]while the combination treatments as the use of bipotassium oxalate that has an obstructive mechanism and also has a direct action on pain receptors.

Also, protein denaturing substances such as formaldehyde, glutaraldehyde, zinc chloride, zinc iodide, phenols, concentrated alcoholic solutions and strong or weak acids, act directly on the nerves and cause precipitation of dentinal fluid proteins that can occlude the dentinal tubules (Bruce 2001).[6]

Recently, Lasers have been used to treat dentinal hypersensitivity; Nd: YAG laser can induce occlusion or narrowing the dentinal tubules (Lan and Liu 1996)[12] as well as direct nerve analgesia (Whitters et al. 1995)[13]. According to Pashley (2000)[14], it may be through the coagulation and precipitation of plasma proteins in dentinal fluid or through alteration of the interdental nerve activity. McCarthy et al. (1997) also suggested that reduction of root-dentine hypersensitivity may be as a result of creating an altered surface layer on the root physically occluding the tubules

(smear layer creation). The combination use of Laser irradiation with chemical agents such as sodium fluoride and stannous fluoride (Moritz et al. 1998)[15] can enhance treatment effectiveness.

Dentinal hypersensitivity is a common clinical condition; its management requires a good understanding of its complexity, as well as its various treatment modalities. Hence in this present library dissertation, an attempt is being made to review dentinal hypersensitivity and its etiopathogenesis, prevalence and various treatment modalities and recent trends in the treatment.

DEFINITION

Dentine hypersensitivity has been 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 disease.[16]

AETIOLOGY

Dentin hypersensitivity develops in two phases: lesion localization and lesion initiation.

Lesion localization occurs by exposure of dentin, either by loss of enamel or by gingival recession. Loss of enamel can occur via attrition, abrasion, erosion or abfraction[17]. Gingival recession can be due to toothbrush abrasion, pocket reduction surgery, tooth preparation for crown, excessive flossing or secondary to periodontal diseases[18].

Lesion initiation occurs when the smear layer or tubular plugs are removed, which opens the outer end of the dentinal tubules. These occur in cases of Tooth brushing with abrasive toothpaste[19]. Most soft drinks, some alcoholic beverages and yoghurt readily remove the dentin smear layer Excessive flossing, Traumatic tooth picking, Eating hard foods, Inhalation of toxic fumes, Regurgitation of stomach acid (GERD)

EPIDEMIOLOGY:

The prevalence of dentin hypersensitivity varies from 4 to 74 %[20]. Mostly occur between age group of 20- 40 years[21]. It can affect any tooth, but mostly canines and premolars[22-24].

Table 1. Dentinal hypersensitivity epidemiological studies[20]

Authors	Country	Setting	Study type	N	Prevalence (%)
Jensen, 1964	USA	University	Clinical	3000	30
Graf and Glase, 1977	Switzerland	Practice	Clinical	351	15
Flynn et al, 1992	UK	University	Clinical	369	18
Orchardson and Collins, 1987	UK	University	Clinical	109	74
Fisher et al, 1992	Brazil	University	Clinical	635	17
Murray and Roberts, 1994	Indonesia	Not stated	Questionnaire	1000	27
Murray and Roberts, 1994	USA	Not stated	Questionnaire	1000	18
Murray and Roberts, 1994	Japan	Not stated	Questionnaire	1000	16
Murray and Roberts, 1994	France	Not stated	Questionnaire	1000	14
Murray and Roberts, 1994	Germany	Not stated	Questionnaire	1000	13
Murray and Roberts,	Australia	Not stated	Questionnaire	1000	13

1994					
Chabanski et al., 1997	UK	University	Clinical	51	73
Irwin and McCusker, 1997	UK	Practice	Questionnaire	250	57
Liu et al., 1998	Taiwan	University	Clinical	780	32
Rees, 2000	Uk	Practice	Clinical	3593	4
Taani and Awartani, 2002	Saudi Arabia	University	Clinical	295	42-60
Clayton et al., 2002	UK	Air force	Questionnaire	228	50
Rees and Addy 2002	UK	Practice	Clinical	4841	4.1
Rees et al., 2003	Hongkong	Hospital	Clinical	226	67.6

MECHANISM OF DENTIN HYPERSENSITIVITY:

Three theories were put forward to explain the mechanism of dentin hypersensitivity[25]:

- Direct Neural Stimulation theory
- Transduction theory
- Hydrodynamic theory

Direct Neural Stimulation theory: According to this theory the stimuli reaches the nerve endings in the inner dentin[26]. But how it reaches the nerve endings could not be explained. Due to little scientific proof it is not accepted now[27].

Transduction theory: According to this theory, the Odontoblastic processes are excited by the stimulus and transmit the impulses to the nerve endings[26]. This theory is also of little importance as it has been noticed that there are no neurotransmitters in the dentin.

Hydrodynamic theory: This theory is also known as 'Fluid theory'[28]. This is the most widely accepted theory, according to this theory the harmful stimuli such as heat, cold, air blast, mechanical pressure etc causes some changes in the fluid movement in the dentinal tubules either inwards or outwards which causes some mechanical disturbances due to mechanoreceptors present at the nerve endings, which stimulates the pain mechanism[29].

DIAGNOSIS

Clinical recommendation for valid diagnosis of dentin hypersensitivity modified on base of the algorithm published by the Canadian Advisory Board (Fig. 1) on Dentin Hypersensitivity in 2003[16].

TREATMENT:

Home Care Management

A. Tooth Brushing

Improper tooth brushing is major etiology for dentinal hypersensitivity; instruction for proper tooth brushing can prevent abrasion and thus dentinal hypersensitivity[30, 31].

B. Dentrifices

New desensitizing toothpaste is available that contains the following:

1) 5% Potassium Nitrate
2) 0.7% Sodium monoflourophosphate
3) 0.3% Triclosan
4) Fluorides

The efficacy of 5% potassium nitrate dentifrice used daily for 4 weeks for the alleviation of the pain of tooth hypersensitivity[32]. It occludes the tubules through crystallization and causes no pulpal changes. Sodium monoflourophosphate acts by occluding open tubules & thus preventing fluid shift in dentinal tubules, thus reducing sensitivity. Triclosan inhibits plaque formation as it is an antimicrobial agent[33]. Fluorides can be used in various forms as home care treatment like sodium fluoride, stannous fluoride etc.

C. GC Tooth Mousse[34]

Tooth mousse is a unique combination containing amorphous calcium phosphate (ACP) and casein phosphopeptide (CPP) obtained from milk casein. This distinct complex make strong bonding with biofilm on teeth restoring the mineral balance by strengthening the hard tissues. This process has been shown to significantly decrease dentine permeability by creating precipitates on the dentine surface that reduce the diameter of dentinal tubules thereby reducing dentinal sensitivity (Fig. 2).

Fig 2: GC Tooth Mousse

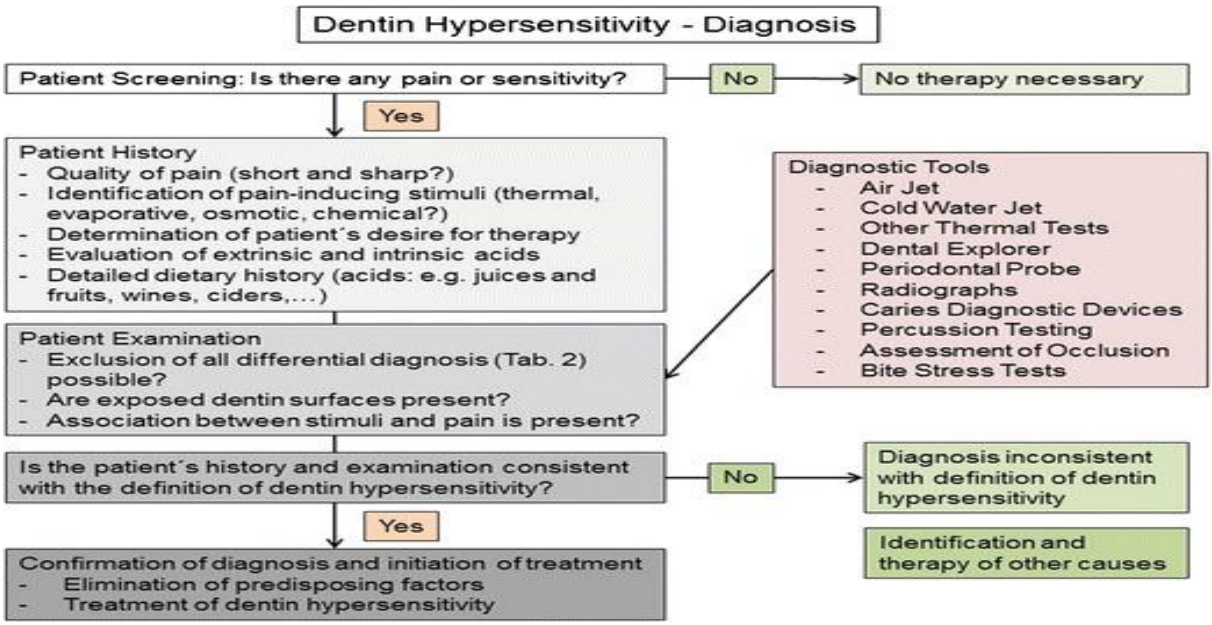

Fig 1: Diagnosis of Dentin Hypersensitivity.

Mouthwashes

Mouthwashes containing potassium nitrate & Sodium Fluoride, Potassium Citrate or Sodium Fluoride or mixture of fluoride can reduce hypersensitivity[35].

In Office Management

- **Cavity Varnish[36]**

Dentin often becomes insensitive when open tubules are covered with a thin film of varnish. Effective means of providing temporary relief. Duraphatfluorstat fluoride containing varnish, duraflor, can be used.

- **Anti Inflammatory Agents[37]**

Marteller reported that when a liner consisting of 1% prednisolone in combination with 25% gum camphor was applied to the walls of cavities, it was completely effective in preventing post operative thermal sensitivity as it caused obliteration of tubules.

- **Treatments That Partially Obturate Dentinal Tubules**

  - **Calcium Compounds:** $Ca(OH)_2$ may block dentinal tubules by providing calcium ions that may tie up the loop protein radicals or promote peritubular dentin formation[38]. Large quantities of $Ca(OH)_2$ will irritate the tissue[39].

  - **Dibasic Calcium Phosphate[40]**: 93% cent of patients reported significant relief. This was

because mineral was deposited near the surface of tubules.

- **Fluoride Compounds: Sodium Fluoride** leads to precipitation of CaF_2 crystals, thus reducing the functional radius of dentinal tubules. Size of the crystal is very small approx 0.05 microns and so a single application has little effect.[41] **Acidulated sodium fluoride** concentration of fluoride in dentin treated with acidulated NaF was significantly higher than dentin treated with NaF. **Sodium Silicofluoride**: A saturated solution (0.6%) of sodium silicofluoride when applied for 5 minutes caused the silica acid to form a gel with the calcium of the tooth, thus producing an insulating barrier[42]. **Stannous Fluoride** Topical application of 0.717% aqueous SnF_2 provided relief from sensitivity[43]. **Fluoride iontophoresis** transfers fluoride ions more deeply into the dentinal tubules that cannot be achieved with topical application of fluoride[44]. It is reported to provide long-term relief from hypersensitive dentin[45].

- **Strontium Chloride:** strontium deposits are produced by an exchange with calcium in the dentin resulting in recrystallisation in the form of a strontium apatite complex[46].

Fig 3: Sensi stop strips.

- **Oxalates:** The oxalate reacts with the calcium ions of dentine and forms calcium oxalate crystals inside the dentinal tubules as well as on the dentinal surface. Thirty percent potassium oxalate had shown a 98% reduction in dentinal permeability[47]. Introduction of an over-the-counter thin, flexible strip coated with oxalate-gel (Crest Sensi-Stop Strips, P&G) is worn at each sensitive site for 10 minutes, applied half on the gingival tissue and half on the tooth surface of each sensitive site reduces sensitivity effectively[48].

- **Dentin resin and adhesives:** the objective in employing resins and adhesives is to seal the dentinal tubules to prevent pain producing stimuli from reaching the pulp. Several investigators have demonstrated immediate and enduring relief of pain for periods of upto 18 months following treatment. GLUMA is a dentin bonding system that includes glutaraldehde primer & 35% HEMA (hydroxyethylmethacrylate). It provides an attachment to dentin that is immediate & strong[49]. GLUMA is very effective (Fig. 4).

Fig 4: GLUMA

- **Bioglass:** A formulation of bioglass can promote infiltration and remineralization of dentinal tubules. It has been shown by some products such as NovaMin[50] (NovaMin Technology Inc., FL, USA).

- **Portland cement:** Calcium silicate cement derived from Portland cement help in the management of DH. It helps to occlude the dentinal tubles by remineralization[10].

- **Lasers:**

The Neodymium-dopedytttrium aluminum garnet (Nd:YAG) has been used successfully to treat dentin hypersensitivity, the power levels used only 1/10th to 1/20th of that required for surgical procedures[51]. It appears that the mechanism of action is by tubule occlusion. CO2 laser irradiation and stannous fluoride gel has also been shown to be effective for inducing tubule occlusion for up to six months after treatment[52].

- **Surgical Correction:**

Soft tissue grafts are performed on teeth that have lost their attached gingiva. The exposed dentin is covered and the sensitivity is eliminated[1].

CONCLUSION

Tooth sensitivity is a very common clinical presentation which can cause considerable concern for the patients. "An enigma" frequently encountered but not well understood. Management of such a condition requires a good understanding of the complexity of the problem as well as the variety of treatment available. The ultimate goal in the treatment of dentine hypersensitivity is the immediate and permanent relief of pain. Professionals should identify the causative factors so that prevention can also be included in the treatment plan.

CONFLICTS OF INTEREST

The authors declare they have no potential conflict of interests regarding this article.

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