Dentin Hypersensitivity: Recent Concepts in Management

Vijay Mantri, Rahul Maria, Neeraj Alladwar, Savita Ghom

1Reader, Department of Conservative Dentistry and Endodontics, Modern Dental College and Research Center, Indore, Madhya Pradesh, India
2Professor, Department of Conservative Dentistry and Endodontics, Modern Dental College and Research Center, Indore, Madhya Pradesh, India
3Professor, Department of Orthodontics, Swargiya Dadasaheb Kalmegh Smruti Dental College and Hospital, Nagpur, Maharashtra, India
4Postgraduate Student, Department of Oral Medicine and Radiology, Chhattisgarh Dental College and Research Institute, Rajnandgaon, Chhattisgarh, India

Correspondence: Vijay Mantri, Reader, Department of Conservative Dentistry and Endodontics, Modern Dental College and Research Center, B-307, Staff Quarters, Airport Road, Gandhi Nagar, Indore, Madhya Pradesh, India
Email: vijayrmantir@yahoo.com

ABSTRACT

Tooth sensitivity is a very common clinical presentation which can cause considerable concern for patients. Dentin hypersensitivity (DH) is characterized by short sharp pain arising from exposed dentin in response to stimuli. The most widely accepted theory of how the pain occurs is Brannstrom’s hydrodynamic theory, fluid movement within the dentinal tubules. The condition generally involves the facial surfaces of teeth near the cervical aspect and is very common in premolars and canines. This condition is frequently encountered by dentists, periodontists, hygienists and dental therapists. Some dental professionals lack confidence in treating DH. 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 etiopathogenesis, incidence, diagnosis, prevention and management of dentinal hypersensitivity. DH is diagnosed after elimination of other possible causes of the pain. Any treatment plan for DH should include identifying and eliminating predisposing etiologic factors. Professionals should appreciate the role causative factors play in localizing and initiating hypersensitive lesions. It is important to identify these factors so that prevention can be included in the treatment plan. Treatments can be self-administered by the patient at home or be applied by a dental professional in the dental office. At-home methods tend to be simple and inexpensive and can treat simultaneously generalized DH affecting many teeth. Desensitizing treatment should be delivered systematically, beginning with prevention and at-home treatments. The latter may be supplemented with in-office modalities.

Keywords: Dentinal hypersensitivity, Prevention, In-office desensitizing agents, Oxalates, Dentin bonding agents.

INTRODUCTION

Dentin hypersensitivity (DH) is a relatively common, painful dental condition. Typically, the pain is short and sharp, and occurs in response to certain stimuli applied to exposed dentin.1

It is clinically described as an exaggerated response application of a stimulus to exposed dentin, regardless of its location.2,3

Dentinal hypersensitivity is characterized by short sharp pain arising from exposed dentin in response to stimuli typically thermal, evaporative, tactile, osmotic or chemical and which cannot be ascribed to any other form of dental defect or pathology.4

A modification of this definition was suggested by the Canadian Advisory Board on Dentin Hypersensitivity5 in 2003, which suggested that ‘disease’ should be substituted for ‘pathology’. The definition provides a clinical description of the condition and identifies DH as a distinct clinical entity. Others terms to describe DH have been created by substituting the word dentinal, adding site descriptors, such as cervical or root, and combining this with either hypersensitivity or sensitivity. This practice resulted in a significant number of permutations to describe the apparently same condition (Table 1).1

Despite the existence of these various terms, several authors prefer the term DH, commonly used and accepted for many decades, to describe a specific painful condition of teeth, which is distinct from others types of dentinal pain having different etiologies.

PREVALENCE AND EPIDEMIOLOGY

DH is a painful clinical condition that affects 8 to 57% of the adult population and is associated with the dentin exposure to the oral environment.2,6 The variations in the reports may be because of difference in populations and different methods of...
investigations. A slightly higher incidence of DH is reported in females than in males. While DH can affect the patient of any age, most affected patients are in the age group of 20 to 50 years, with a peak between 30 and 40 years of age.7 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.8

DH is a relatively common dental clinical condition in permanent teeth caused by dentin exposure to the oral environment as a consequence of loss of enamel and/or cementum. It is manifested in a manner that is physically and psychologically uncomfortable for the patient and it may be defined as acute pain of short duration caused by the presence of open dentinal tubules on an exposed dentinal surface.9

The stimulus that triggers the onset of pain can be of thermal, chemical or mechanical origin. The most common complaint is caused by cold 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 finger nail 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.10-12

PATHOGENESIS
It has been stated in the literature that DH develops in two phases: lesion localization and lesion initiation.13 Lesion localization occurs by loss of protective covering over the dentin, thereby exposing it to external environment. It includes loss of enamel via attrition, abrasion, erosion or abfraction. Another cause for lesion localization is gingival recession which can be due to toothbrush abrasion, pocket reduction surgery, tooth preparation for crown, excessive flossing or secondary to periodontal diseases.14 As stated earlier, not all exposed dentin is sensitive. For DH to occur, the lesion localization has to be initiated. It occurs after the protective covering of smear layer is removed, leading to exposure and opening of dentinal tubules.

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

• Direct innervation theory
• Odontoblast receptor theory
• Fluid movement/hydrodynamic theory.

According to direct innervation theory, nerve endings penetrate dentin and extend to the dentinoenamel junction.15 Direct mechanical stimulation of these nerves will initiate an action potential. There are many shortcomings of this theory.16

Odontoblast receptor theory states that odontoblasts act as receptors by themselves and relay the signal to a nerve terminal.17 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.18

Brannstrom (1964) has proposed that dentinal pain is due to hydrodynamic mechanism, i.e. fluid force.19 Scanning electron microscopic (SEM) analysis of hypersensitive dentin shows the presence of widely open dentinal tubules.20 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-dentin complex.18 As stated before, the hypersensitive dentin has more widely open tubules and thin/under calcified smear layer as compared with non-sensitive dentin. The wider tubules increase the fluid movement and thus the pain response.20,21

CLINICAL MANAGEMENT OF DH
Diagnosis
Like any other clinical condition, an accurate diagnosis is important before starting the management of DH. DH has features which are similar to other conditions like caries, fractured or chipped enamel/dentin, pain due to reversible pulpitis, and post dental bleaching sensitivity.8,22 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.4 A simple clinical method of diagnosing DH includes a jet of air or using an exploratory probe on the exposed dentin, in a mesiodistal direction, examining all the teeth in the area in which the patient complains of pain.23 The severity or degree of pain can be quantified either according to categorical scale (i.e. slight, moderate or severe pain) or using a visual analog scale.13

Prevention of DH/Removal of Etiological Factors
An often neglected phase of 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. The etiological factors include faulty tooth brushing, poor oral hygiene, premature contacts, gingival recession because of periodontal therapy or physiological reasons, and exogenous/endogenous nonbacterial acids.11

Faulty tooth brushing includes hard brushes, excessive forces, excessive scrubbing at the cervical areas or even lack of brushing which causes plaque accumulation and gingival recession.24 The patient should be taught the correct method of tooth brushing with the help of a model. Highly abrasive tooth powder or pastes should be avoided.13 Also, the patients should be instructed to avoid brushing for at least 2 hours after acidic drinks to prevent agonist effect of acidic erosion on toothbrush abrasion.
Erosive agents are also important agents in initiation and progression of DH. They tend to remove the enamel or open up the dentinal tubules.\textsuperscript{25,26} The erosive agents can be either exogenous dietary acids or endogenous acids. The exogenous dietary acids include carbonated drinks, citrus fruits, wines, yogurt and professional hazards (workers in battery manufacturing, wine tasters).\textsuperscript{25,26} A detailed dietary history should be taken. The quantity and frequency of the foods containing acids should be reduced. Patient should be advised to take something alkaline (milk) or at least neutral (water) after acidic drinks and to use a straw to sip the drink and avoid swishing it around the teeth. The endogenous acid comes from gastroesophageal reflux or regurgitation. It is also common in patients with eating disorders. The condition is characterized by generalized erosion of the palatal surfaces of maxillary anterior teeth.\textsuperscript{27} Such a patient should be referred to the medical practitioner for expert management of the underlying disease. An occlusal splint can be fabricated to cover the affected areas, to prevent their contact with the acids.

\section*{CLASSIFICATION OF DESENSITIZING AGENTS\textsuperscript{28}}

\begin{itemize}
  \item \textbf{I. Mode of administration}
    \begin{itemize}
      \item At home desensitizing agents
      \item In-office treatment
    \end{itemize}
  \item \textbf{II. On the basis of mechanism of action}
    \begin{itemize}
      \item Nerve desensitization
        \begin{itemize}
          \item Potassium nitrate
        \end{itemize}
      \item Cover or plugging dentinal tubules
        \begin{itemize}
          \item Ions/salts
            \begin{itemize}
              \item Aluminium
              \item Ammonium hexafluorosilicate
              \item Calcium hydroxide
              \item Calcium carbonate
              \item Calcium phosphate
              \item Calcium silicate
              \item Sodium citrate dibasic
              \item Fluorosilicate
              \item Potassium oxalate
              \item Silicate
              \item Sodium monofluorophosphate
              \item Sodium fluoride
              \item Sodium fluoride/Stannous fluoride combination
              \item Stannous fluoride
              \item Strontium acetate with fluoride
              \item Strontium chloride
            \end{itemize}
          \item Protein precipitants
            \begin{itemize}
              \item Formaldehyde
              \item Glutaraldehyde
              \item Silver nitrate
              \item Strontium chloride hexahydrate
              \item Zinc chloride
            \end{itemize}
          \end{itemize}
        \end{itemize}
    \end{itemize}
\end{itemize}

\section*{At Home Desensitizing Therapy}

Traditionally, the therapy for management of DH is primarily aimed at occluding the dentinal tubules or making coagulates inside the tubules.\textsuperscript{13} Patients are often prescribed over-the-counter desensitizing agents. These “at home” desensitizing agents include toothpastes, mouthwashes and chewing gums.\textsuperscript{13} Majority of the toothpastes contain potassium salts (potassium nitrate, potassium chloride or potassium citrate), sodium fluoride, strontium chloride, dibasic sodium citrate, formaldehyde, sodium monofluorophosphate and stannous fluoride. Potassium salts act by diffusion along the dentinal tubules and decreasing the excitability of the intradental nerve fibers by blocking the axonic action.\textsuperscript{29,30} Along with the desensitizing toothpastes, mouthwashes and chewing gums containing potassium nitrate, sodium fluoride or potassium citrate are also recommended.\textsuperscript{13} The results of “at-home” desensitizing therapy should be reviewed after every 3 to 4 weeks. If there is no relief in DH, “in-office” therapy should be initiated.

\section*{In-office Desensitizing Agents}

Theoretically, the in-office desensitizing therapy should provide an immediate relief from the symptoms of DH. The in-office desensitizing agents can be classified as the materials which undergo a setting reaction (glass ionomer cement, composites) and which do not undergo a setting reaction (varnishes, oxalates).

\section*{Fluorides}

Various clinical trials have shown that application of fluoride solution can decrease the DH.\textsuperscript{31,32} Fluorides decrease the dentinal permeability by precipitation of calcium fluoride crystals inside the dentinal tubules.\textsuperscript{13} These crystals are partially insoluble in saliva. SEM revealed granular precipitates in the peritubular dentin after application of fluorides.\textsuperscript{33} Various fluoride formulations are used to treat DH. These include sodium...
fluoride, stannous fluoride, sodium monofluorophosphate, fluoroapatite, and fluoride combined with iontophoresis. Sodium fluoride has been used in dentifrices or may be professionally applied in a concentration of 2%.

**Oxalates**

Oxalates can reduce dentinal permeability and occlude dentinal tubules. About 30% potassium oxalate had shown a 98% reduction in dentinal permeability. Also, topical application of 3% potassium oxalate reduced DH after periodontal therapy. The oxalate reacts with the calcium ions of dentin and forms calcium oxalate crystals inside the dentinal tubules as well as on the dentinal surface. This results in a better sealing as compared with an intact smear layer.

**Adhesive Materials**

The adhesive resins can seal the dentinal tubules effectively by forming a hybrid layer. Various clinical studies have demonstrated the effectiveness of adhesives in management of DH. Newer bonding agents modify the smear layer and incorporate it into the hybrid layer. Recently, some dentin bonding agents have been introduced in the market with the sole purpose of treating DH. Gluma desensitizer (Heraeus Kulzer, Hanau, Germany) contains hydroxyethyl methacrylate (HEMA), benzalkonium chloride, gluteraldehyde and fluoride. Gluteraldehyde causes coagulation of the proteins inside the dentinal tubules. It reacts with the serum albumin in the dentinal fluid, causing its precipitation. HEMA forms deep resinous tags and occludes the dentinal tubules. Gluma has shown promising results in the clinical trials.

**Bioglass**

It has been reported that the formulation of bioglass can promote infiltration and remineralization of dentinal tubules. The basic component is silica, which acts as a nucleation site for precipitation of calcium and phosphate. SEM analysis has shown that bioglass application forms an apatite layer, which occludes the dentinal tubules. The use of bioglass in management of DH has been shown by some products, such as NovaMin (NovaMin Technology Inc., FL, USA).

**Portland Cement**

Some authors have shown that calcium silicate cement derived from Portland cement can help in the management of DH. It helps to occlude the dentinal tubules by remineralization.

**Laser**

It has been shown in various studies that lasers can be used in the effective management of DH. Some authors have shown that Nd-YAG laser application occluded the dentinal tubules. GaAlA laser is thought to act by affecting the neural transmission in the dentinal tubules. It has also been proposed that lasers coagulate the proteins inside the dentinal tubules and block the movement of fluid.

**Casein Phosphopeptide—Amorphous Calcium Phosphate**

Recently, milk protein casein has been used to develop a remineralizing agent (GC Tooth Mousse). The casein phosphopeptide (CPP) contains phosphoseryl sequences which get attached and stabilized with amorphous calcium phosphate (ACP). The stabilized CPP-ACP prevents the dissolution of calcium and phosphate ions and maintains a supersaturated solution of bioavailable calcium and phosphates.

**Management Strategy**

- Take a detailed clinical and dietary history
- Differentially diagnose the condition from other dental pain conditions
- Identify and manage etiological and predisposing factors
- In case of mild-to-moderate sensitivity, advice at-home desensitizing therapy
- If there is no relief or in case of severe sensitivity, initiate in-office treatment
- In extreme cases, if patient does not respond to the therapy and there are individual teeth exhibiting the symptoms, then endodontic therapy can be initiated
- A regular review should be made with an emphasis on prevention of the condition.

**REFERENCES**


