

DENTIN HYPERSENSITIVITY

INTRODUCTION

Dentin sensitivity (DS) or dentinal hypersensitivity (DH) is one of the most commonly encountered clinical problems. It is clinically described as an exaggerated response to application of a stimulus to exposed dentin, regardless of its location. The terms DS or DH have been used interchangeably to describe the same clinical condition. True hypersensitivity can develop due to pulpal inflammation and can present the clinical features of irreversible pulpitis, i.e., severe and persistent pain, as compared with typical short sharp pain of DH. Majority of literature reviews dealing with this clinical condition have suggested the use of term DS and consider that the sharp pain is actually the normal pulpal response to the exposed dentin. But it is well known that all exposed dentin are not sensitive and the term DH has been used over the decades by the clinicians.[6,7] Therefore, both the terminologies can be used to describe the clinical condition. The condition has been defined by an international workshop on DH as follows “Dentin 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 dental defect or pathology”. Some authors have substituted the word “dentin” and added the site, such as cervical or root, resulting in various other terminologies (e.g., cervical sensitivity/hypersensitivity) to describe the same clinical condition.

PREVALENCE AND EPIDEMIOLOGY

DH is a painful clinical condition with an incidence ranging from 4 to 74%. The variations in the reports may be because of difference in populations and different methods of investigations. The methods employed are usually patient questionnaires or clinical examinations. Interestingly, the incidence of DH is much higher in patient questionnaires studies than in clinical studies which quote an incidence of mere 15%.

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 20–50 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. Buccal aspect of cervical area is the commonly affected site.

ETIOPATHOGENESIS

Anatomy of dentin pulp complex

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 dentin–pulp complex. Although dentin and pulp are histologically different, their origin is embryologically from the same precursor, i.e., the ectomesenchyme. Pulp is integrally connected to dentin, i.e., physiologic and/or pathologic reactions in one of the tissues will also affect the other. Dentin consists of small canal like spaces, dentinal tubules. These tubules occupied by odontoblastic processes. The odontoblastic processes may extend through the entire thickness of dentin from pulp to dentino-enamel junction. The odontoblastic processes are actually the extensions of odontoblasts, which are the major cells of pulp–dentin complex. The odontoblastic processes are surrounded by dentinal fluid inside the tubules. The dentinal fluid forms around 22% of total volume of dentin. It is an ultrafiltrate of blood from the pulp via dentinal tubules and forms a communication medium between the pulp (via the odontoblastic layer) and outer regions of the dentin.

Pathogenesis

It has been stated in the literature that DH develops in two phases: lesion localization and lesion initiation. 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. 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

Fluid movement/hydrodynamic theory

According to direct innervation theory, nerve endings penetrate dentin 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. Developmental studies have shown that the plexus of Rashkow and intratubular nerves do not establish themselves until the tooth has erupted; yet, newly erupted tooth is sensitive. Moreover, pain inducers such as bradykinin fail to induce pain when applied to dentin, and bathing dentin with local anesthetic solutions does not prevent pain, which does so when applied to skin.

Odontoblast receptor 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–dentin complex. This is similar to the activation of nerve fibers surrounding the hair by touching or applying pressure to the hair. The response of pulpal nerves, mainly A δ intradentinal afferent fibers, depends upon the pressure applied, i.e., intensity of stimuli. It has been noted that stimuli which tend to move the fluid away from the pulp–dentin 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. In spite of the fact that fluid movement inside the dentinal tubules produces pain, it should be noted that not all exposed dentin is sensitive. 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.

CLINICAL MANAGEMENT OF DH

Diagnosis

As 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. 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 using a visual analogue scale.

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 non-bacterial acids.

CLASSIFICATION OF DESENSITIZING AGENTS

Mode of administration

At home desensitizing agents

In-office treatment

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 ($\text{SiO}_2\text{-P}_2\text{O}_5\text{-CaO-Na}_2\text{O}$)

Dentin 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

At home desensitizing therapy

Grossman listed the requirements for an ideal dentin desensitizing agent as: rapidly acting with long-term effects, non-irritant to pulp, painless and easy to apply, and should not stain the tooth. Traditionally, the therapy for management of DH is primarily aimed at occluding the dentinal tubules or making coagulates inside the tubules. Patients are often prescribed over-the-counter desensitizing agents. These “at home” desensitizing agents include toothpastes, mouthwashes and chewing gums. 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. Various clinical studies have shown the efficacy of potassium salts in controlling the DH. It has been shown that toothpastes containing 5% potassium nitrate and 0.454% stannous significantly reduced the DH. Also, toothpastes containing potassium nitrate and fluorides have been shown to reduce post-bleaching sensitivity. The desensitizing toothpastes should be used with the help of a toothbrush with soft bristles. Patients should be advised to use minimal amount of water to prevent the dilution of the active agent. Along with the desensitizing toothpastes, mouthwashes and chewing gums containing potassium nitrate, sodium fluoride or potassium citrate are also recommended. The results of “at-home” desensitizing therapy should be reviewed after every 3–4 weeks. If there is no relief in DH, “in-office” therapy should be initiated.

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).

Fluorides

Traditionally, fluorides have been used as a caries preventive material which can help in remineralization of enamel/dentin. Also, various clinical trials have shown that application of

fluoride solution can decrease the DH. Fluorides decrease the dentinal permeability by precipitation of calcium fluoride crystals inside the dentinal tubules. These crystals are partially insoluble in saliva. SEM revealed granular precipitates in the peritubular dentin after application of fluorides. Various fluoride formulations are used to treat DH. These include sodium fluoride, stannous fluoride, sodium monofluorophosphate, fluorosilicates and fluoride combined with iontophoresis. Sodium fluoride has been used in dentifrices or may be professionally applied in a concentration of 2%. The precipitates formed by sodium fluoride can be mechanically removed by the action of saliva or mechanical action. Therefore, an addition of acid formulation is recommended. The acidulated sodium fluoride can form precipitates deep inside the tubules. Also, some authors have recommended the use of iontophoresis along with sodium fluoride. The electric current is supposed to increase the ion diffusion. A clinical study has shown that 0.4% stannous fluoride along with 0.717% of fluoride can provide an immediate effect after a 5 minute professional application. Stannous fluoride acts in a similar fashion as that of sodium fluoride, i.e., formation of calcium fluoride precipitates inside tubules. Also, SEM studies have shown that stannous fluoride itself can form insoluble precipitates over the exposed dentin. Fluorosilicates act by formation of precipitates of calcium phosphates from saliva. Ammonium hexafluorosilicate has been used as a desensitizing agent. It can present a continuous effect of dentinal tubule occlusion via precipitation of a mixture of calcium fluoride and fluoridated apatite. If the precipitate is predominantly composed of fluoridated apatite, it can form stable crystals deposited deep inside the dentinal tubules. These crystals are resistant to removal from the action of saliva, brushing or action of dietary substances.

Oxalates

Oxalates can reduce dentinal permeability and occlude dentinal tubules. Thirty percent 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. It has been shown that the effect of oxalates on DH diminishes over a period of time. This can be attributed to the removal of the calcium oxalate crystals by brushing or dietary acids. The condition can be improved by acid etching of the dentinal surface, thus increasing the penetration of calcium oxalate crystals deep into the dentinal tubules. Many vegetables like rhubarb, spinach and mint contain oxalates. It has been shown that phytocomplexes obtained from these natural products can reduce the dentinal permeability. This can also be followed by covering the exposed surface with a dental adhesive. Potassium oxalate can lead to gastric irritation. Therefore, it should not be used with a tray with prolonged placement.

Varnishes are commonly used useful in-office measures to treat DH. Copal varnish can be applied to cover the exposed dentinal surface. But its effect is for short term and is not recommended for long term management of DH. To improve its efficacy, removal of smear layer is advocated. Also, the varnishes can act as a vehicle for fluoride. The fluoride varnishes can be acidulated to increase the penetration of ions.

Bioglass

Bioglass was developed to stimulate the formation of new bone. It is used in orthopedics to cover the implants to promote union between implant and bone. It has been used in dentistry to fill up the osseous defects during periodontal surgery. It has been reported that a 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.[17] It helps to occlude the dentinal tubules by remineralization.

Laser

Laser is an acronym for light amplification by stimulated emission of radiations. It has been shown in various studies that lasers can be used in the effective management of DH. The mechanism of action of lasers in treating DH is not very clear. 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 phosphoserine sequences which get attached and stabilized with amorphous calcium phosphate (ACP).[64] The stabilized CPP-ACP prevents the dissolution of calcium and phosphate ions and maintains a supersaturated solution of

bioavailable calcium and phosphates.[64] Various studies have shown that CPP-ACP can effectively remineralize the enamel subsurface lesions.[65,66] By virtue of its remineralizing capacity, it has also been proposed by the manufacturers that it can also help in prevention and treatment of DH.