International Journal of Research in Health and Allied Sciences

Journal home page: www.ijrhas.com

Official Publication of "Society for Scientific Research and Studies" [Regd.]

ISSN: 2455-7803

REVIEW ARTICLE

Dentinal Hypersensitivity - A Review

[1]Neetu Bamel, [2]Sakshi Mehta, [3]Neena Vats, [4]Reena Kumari Gahlawat, [5]Pious Kumar Anthwal

[1]Senior Resident at Jan Nayak Chaudhary Devi Lal Dental College, Sirsa (Dept of Periodontics);
[2]Private practitioner at Janta Hospital Ellenabad,
[3]Private Practitioner at Smile Up Dentals Clinic, Gurugram,
[4]Senior Resident at D.A.V(C) Dental College, Yamunanagar (Dept. of Orthodontics),
[5]Senior Resident at D.A.V(C) Dental College, Yamunanagar (Dept. of Orthodontics)

***ABSTRACT*:**
Dentinal hypersensitivity is a common painful condition usually associated with exposure of dentinal tubules. The pain arising from dentinal hypersensitivity is extremely variable in character. A number of clinical conditions that may provide similar clinical features to that of dentinal hypersensitivity. Differential diagnosis should be made in order to provide correct treatment to the patients. This article concisely reviews etiology, mechanism and clinical management of the dentinal hypersensitivity. The management of DH requires a good understanding of the complexity of the problem as well as variety of treatment modalities.
Key words : DH-Dentinal Hypersensitivity.

Received: 12April, 2019 Revised: 24 April, 2019 Accepted: 15May, 2019

Corresponding author: Dr. Neetu Bamel, Senior Resident at Jan Nayak Chaudhary Devi Lal Dental College, Sirsa (Dept of Periodontics)

This article may be cited as: Bamel N, Mehta S, Vats N, Gahlawat RK, Anthwal PK. Dentinal Hypersensitivity - A Review. Int J Res Health Allied Sci 2019; 5(2):79-83.

Introduction:
Dentinal hypersensitivity (DH) is characterized by 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 form of dental defect or pathology[1].A modification of this definition was suggested by the Canadian Advisory Board on Dentine Hypersensitivity in 2003, which suggested that 'disease' should be substituted for 'pathology'. The definition provides a clinical descriptor of the condition and identifies DH as a distinct clinical entity[2].

Prevalence and Epidemiology :
DH is a painful clinical condition that affects 4 to 74% of the adult population and is associated with the dentin exposure to the oral environment.[(3-7)] The variation in the reports may be because of the difference in populations and different method of investigations.

A slightly higher incidence of DH is reported in females as compared to males. 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.[4-5] Canines and premolars of both the arches are the most affected teeth. Buccal aspect of cervical area is the commonly affected site.[8]

Etiology:
1. The most common cause of exposed dentinal tubules is the gingival recession.[9] The cause of the gingival recession may be inadequate width of the gingival, faulty brushing technique, pocket reduction periodontal procedure, osseous crown lengthening procedure.
2. Poor oral hygiene of the patients is related to DH poor oral hygiene of the patients. Poor oral hygiene leads to production of bacterial toxins which leads to dentinal tubules opening.

3. Another factor is the removal of dental cementum which covers the root or the root dentin itself during Scaling and Root planning or periodontal surgery. Von Troil et al found 50% of the patients undergoing SRP had dentine hypersensitivity after treatment.
4. Premature occlusal contact and occlusal contact with excessive force and premature occlusal contact leads to fracture of the enamel crystals in the cervical region.
5.Various Habit like excess intake of acidic fluid, horizontal toothbrushing, attrition,abrasion,abfraction lead to wide opening of dentinal tubules.

Theories of dentinal hypersensitivity[10] : Three major mechanisms of dentinal sensitivity have been proposed in the literature:
1.Odontoblastic transduction theory
2. Neural theory
3. Hydrodynamic Theory

Odontoblastic transduction theory: According to this theory, a variety of mechanical and chemical stimuli leads to activation of the exposed odontoblastic processes present on the surface of the dentin. Basically on stimulus, there is the release of neurotransmitters and impulses are transmitted towards the nerve endings. But this theory is discarded because, no such neurotransmitters have been found to be produced or released by odontoblastic processes

Neural theory : According to this theory nerve ending with in the dentinal tubules is in the direct communication with the nerve fibers of the pulp. 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.[11] Moreover, pain inducers such as bradykinin fail to induce pain when applied to dentine, and bathing dentine with local anesthetic solutions does not prevent pain, which does so when applied to skin.[8]

Hydrodynamic Theory: Hydrodynamic theory proposed by Brannstrom and Astron in 1964.Hydrodynamic theory is the most widely accepted theory for dentinal hypersensitivity. This theory postulates that fluids within the dentinal tubules are disturbed either by temperature, physical or osmotic changes. The movement of the fluid stimulate a baroreceptor which leads to neural discharge. This theory is based on the movement of dentinal fluid within the dentinal tubules. This centrifugal fluid movement, in turn, activates the nerve endings at the end of dentinal tubules or at the pulp–dentine complex.[12] 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.[12] It has been noted that stimuli which tend to move the fluid away from the pulp–dentine 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.[12,14]

Diagnosis[14]
These are the important points that must be evaluated by the operato r during the diagnosis of dentine sensitivity:
1. History and nature of the pain
2. Number and location of the sensitive teeth
3. Area of origin of sensitivity from tooth
4. Intensity of pain, frequency and duration of each episode
5. Triggering factor or stimulus for dentinal hypersensitivity
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.
Various Methods used for diagnosis of dentinal hypersensitivity :

Mechanical Stimuli: In this, the dentinal surface of the teeth is rubbed with sharp tipped probe or Yeaple probe as mechanical pressure stimulators. In Yeaple probe, variation in forces is regulated by electromagnetic device. A tooth is said to be non- sensitive if the force is reached up to 70g without eliciting pain sensation.[14-15]

Chemical Stimuli: In this, hypertonic solutions of glucose and sucrose are used, which leads to change in osmotic pressure. It act as stimulator and causes movement of the intratubular fluid. Because the response given by the patients is difficult to control. That why this method is not preferred now a days.

Cold water testing: In this test we used a series of different temperatures syringes containing water. Between 0° to 200° C. During performing this test,it is suggested starting with the warmest water and gradually lowering the temperature with maintaining at least 3 minute gap. The temperature of the water is decreased by 50°C and the test is terminated when a painful response is recorded or when 0° C is reached. The temperature of the water is decreased by 50°C and the test is terminated when a painful response is recorded or when 0° C is reached[14-15].

Cold air blast from a dental air syringe: An air current is applied for 1
second at a pressure of 45psi and at a temperature of 19°-240°C through dental chair. Air current technique is generally used for screening[15].

Thermo-electric devices: A fine-tipped thermal probe is used in this method. It is placed on the surface of the tooth wherein heat or cold is continuously applied that allows quantification of the applied stimulus. The test is performed when temperature is 250°C; subsequently the temperature is reduced by every 50°C until the patient experiences the pain.[14-15]

Electrical Stimulation: This is more complex and usually consists of progressive elevation of the magnitude of the stimulus until a slight sense of pain is felt. However, due to current loss through the periodontium and the subsequent stimulation of the periodontium, false positive results can occur.[14-15]

Differential Diagnosis

Clinical features of the Dentinal hypersensitivity is similar to other dental conditions. Clinical and radiographic examination is necessary to elucidate the cause. Some of the clinical technique include pain response upon the pressure of tapping teeth(to indicate pulpitis/periodontal involvement),pain biting a stick (suggests crack tooth syndrome)and use of transilluminating light or dye(to diagnosis of fractures) and pain with recent restorations[16].

Management Of Dentinal Hypersensitivity

Review of literature on Dentinal hypersensitivity provides with a general guideline to be followed in the management of dentinal hypersensitivity. The various steps to be followed includes[17]:

1.History and examination to establish proper diagnosis
2. Remove all the etiology factor .
3.Treatment based on severity of problem.
a)Mild generalized sensitivity-use of OTC desensitizing products (toothpastes, gels, etc)
b)Localized moderate to severe sensitivity-use of In-office products (primers, varnishes, sealants, etc)
c) In severe cases, pulpal extirpation and extraction may be the treatment of choice
d) Review on an appropriate basis and reassess if pain persists.

There are two main approaches for the treatment of the dentinal hypersensitivity either blocking the dentinal tubules or blocking the nerve activity.

Dentrifice:

Dentifrices are the most common vehicles for desensitizing agents. Majority of the toothpastes contains potassium nitrate, sodium fluoride, formaldehyde, monofluorphosphate and stannous fluoride, fluoride iontophoresis, calcium hydroxide. They are widely indicated, particularly because of their low cost, ease of use and home application.[18]

Potassium nitrate

Potassium nitrate in bioadhesive gels at 5% and 10% has also been shown to be effective in reducing dentinal hypersensitivity[19].Importantly ,it has shown that potassium nitrate does not induce any pulpal changes[20].Despite these encouraging findings it is interesting to note that a recent Cochrane Database Systematic Review failed to find strong evidence supporting the efficacy of potassium nitrate toothpaste for dentine hypersensitivity[21].The mechanism of action of potassium nitrate is largely unknown, although an oxidizing effect or blocking of tubules by crystallization has been proposed but not proven[10,22].

Calcium hydroxide

Green et al[23], Mcfall et al[24] have reported on the effectiveness of calcium hydroxide in managing dentinal hypersensitivity. Its mode of action has been proposed to be via increasing mineralization and blockage of the exposed dentinal tubules. But disadvantage of the calcium hydroxide, it causes irritation of the gingival tissues and action of calcium hydroxide is diminishes rapidly[10].

Sodium fluoride

Minkov et al[25] and Kerns et al[26] have shown that treatment of exposed root surfaces with fluoride toothpaste and concentrated fluoride solutions is very efficient in managing dentinal hypersensitivity. Tal et al reported that it causes precipitation of the fluoride compounds mechanically blocking exposed dentinal tubules or fluoride within the tubules blocking transmission of stimuli[27].

Strontium Chloride:

It act as protein precipitants and their mechanism is through organic precipitants and their mechanism odontoblast denaturation forming sealing film[18]. It prevents the movement of dentinal fluid. Minkoff and Axelrod[28] concluded that regular home use of the 10% strontium chloride is effective in reducing dentinal hypersensitivity.

Stannous fluoride

The mode of action of the Stannous fluoride appears to be through the induction of a high mineral content calcific barrier leading to occlusion of the exposed dentinal tubules.[29] Stannous fluoride are used either an aqueous solution or in glycerine with carboxymethyl cellulose in the treatment of DH.

Fluoride iontophoresis

Iontophoresis is the process of influencing ionic motion with the help of electric current[10]. It act as a desensitizing procedure in conjunction with sodium fluoride[24]. Studies report that there is an immediate reduction in sensitivity after treatment with iontophoresis, but the symptoms gradually return over the ensuing six months[30].

Oxalates

The mechanism of action of the oxalates by reducing the dentinal permeability and occlude dentinal permeability. Thirty percent potassium oxalate had shown a 98% reduction in dentinal permeability. Also, topical application of 3% potassium oxalate reduced DH after periodontal therapy.[31]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. This results in a better sealing as compared with an intact smear layer[11].Potassium oxalate can lead to gastric irritation. Therefore, it should not be used with a tray with prolonged placement[8].

Varnish : It has been recommended for the treatment of DH, but its action is transitory and usually lasts only a few hours. The varnishes can act as a vehicle for fluoride. The fluoride varnishes can be acidulated to increase the penetration of ions. But its effect is for short term and is not recommended for long term management of DH[32].

Resins: Traditionally, resin composites or dentin bonding agents are used as desensitizing agents. It effectively seals the dentinal tubules and prevents DH.It can provide a more durable and long lasting dentine desensitizing effect. Various clinical studies have demonstrated the effectiveness of adhesives in management of DH[33-35]. The conventional dentin bonding agents (DBA) removes the smear layer, etches the dentinal surface and forms deep dentinal resin tags inside the dentinal tubules. The combined dentin–resin layer (consisting of penetrating resinous tags) has been termed as hybrid layer. It effectively seals the dentinal tubules and prevents DH[33-35]. Newer bonding agents modify the smear layer and incorporate it in into the hybrid layer[36]. Recently, some dentin bonding agents have been introduced in the market with the sole purpose of treating DH[8].

Gluteraldehyde:

Gluteraldehyde causes coagulation of the salivary proteins in the dentinal tubules[36]. HEMA causes deep resinous tags and blocks the dentinal tubules[36]. Gluma has shown promising results in the clinical trials[36-37].

Laser

Light amplification by stimulated emission of radiations (LASER) has been shown in various studies that lasers can be used in the effective management of DH[38-41]. It has also been proposed that lasers coagulate the proteins inside the dentinal tubules and block the movement of fluid. Both the Nd:YAG and CO_2 lasers have been studied for their use in managing dentinal hypersensitivity. Some authors have shown that Nd–YAG laser application occluded the dentinal tubules.[40-41] The Nd:YAG laser has been used in conjunction with sodium fluoride varnish with encouraging results showing up to 90 per cent of the dentinal tubules being occluded through use of this combined therapy. Laser is thought to act by affecting the neural transmission in the dentinal tubules[41]. It has also been proposed that lasers coagulate the proteins inside the dentinal tubules and block the movement of fluid. CO_2 laser irradiation and stannous fluoride gel has also been shown to be effective for inducing tubule occlusion for up to six months after treatment[42-43.] While still largely experimental, this technique requires further scientific investigation before it becomes a clinically acceptable means of treatment[8].

Bioglass

Bioglass was developed to stimulate the formation of new bone[44]. It has been reported that a formulation of bioglass can promote infiltration and remineralization of dentinal tubules. The basic component of bioglass is silica, which acts as a nucleation site for precipitation of calcium and phosphate and forms a apatite layer[45].

Conclusion: From review of literature**,** It is noticed that an effective treatment must be preceded by proper diagnosis. The dentist must explore all possibilities, form a definitive diagnosis, then, implement management strategies that will help reduce or eliminate the sensitivity. There is wide variety of tropical and professional desensitizing agents[14]. In future, gene therapy may be used to block the increased production of nerve growth factor (NGF) by pulpal fibroblasts near the lesion which are thought to contribute to tooth hypersensitivity after restorative procedures[46].

References:

1. Dowell P, Addy M (1983) Dentine hypersensitivity – a review. Aetiology, symptoms and theories of pain production. J Clin Periodontol 10, 341-350.
2. Canadian Advisory Board on Dentin Hypersensitivity (2003) Consensus-based recommendations for the diagnosis and management of dentin hypersensitivity J Can Dent Assoc 69, 221-226.
3. Rees JS, Jin U, Lam S, Kudanowska I, Vowles R. The prevalence of dentine hypersensitivity in a hospital clinic population in Hong Kong. J Dent 2003;31:453-61.
4. Flynn J, Galloway R, Orchardson R. The incidence of hypersensitive teeth in the west of Scotland. J Dent 1985;13:230-6.
5. Fischer C, Fischer RG, Wennberg A. Prevalence and distribution of cervical dentine hypersensitivity in a population in Rio de Janeiro, Brazil.J Dent 1992;20:272-6.
6. Irwin CR, McCusker P. Prevalence of dentine hypersensitivity in general dental population. J Irish Dent Assoc 1997;43:7-9.
7. Taani DQ, Awartani F. Prevalence and distribution of dentin hypersensitivity and plaque in a dental hospital population. Quintessence Int 2001;32:372-6.
8. Miglani S, Aggarwal V ,Ahuja B. Dentinal hypersensitivity :Recent trends in management. J Conserv Dent 2019;13:218-24.
9. Peter L. Jacobsen, Gretchen Bruce. Clinical Dentin Hypersensitivity:Understanding the Causes and Prescribing a Treatment. The journal of contemporary dental practice 2001;2:1

10. P Bartold. Dentinal Hypersensitivity: a review. Australian Dental Journal 2006;51:(3):212-218
11. Orchardson R, Cadden SW. An update on the physiology of the dentinepulp complex. Dent Update 2001;28:200-9.
12. Pashley DH. Dynamics of the pulpo-dentinal complex. Crit Rev Oral Biol Med 1996;7:104-33.
13. Chidchuangchai W, Vongsavan N, Matthews B. Sensory transduction mechanisms responsible for pain caused by cold stimulation of dentine in man. Arch Oral Biol 2007;52:154-60.
14. Suchetha A, B.S. Keshava Prasad , Apoorva SM , Lakshmi P. Dentinal Hypersensitivity - A Review.Indian Journal of dental sciences2013;5:112-16.
15. Ricarte JM, Matoses VF, Llacer VJF. Dentinal Sensitivity: Concept and methodology for its objective evaluation. Med Oral Patol Oral Cir Buccal. 2008; 13(3): E 201-E 206
16. Dowell P, Addy M. Dentine Hypersensitivity: A review: Aetiology, symptoms and theories of pain production. J Clin Periodontol 1983;10:341-50.
17. Gillam DG, Orchardson R. Advances in the treatment of root dentin sensitivity: Mechanisms and treatment principles. Endodontic Topics 2001;13:13-33.
18. Porto IC.C.M.,Andrade A.K.M.,Montes M.A.J.R. Diagnosis and treatment of dentinal hypersensitivity.Journal of Oral Science 2009;51(3):323-32.
19. Frechoso SC, Menendez M, Guisasola C, Arregui I, Tejerina JM,Sicilia A. Evaluation of the efficacy of two potassium nitrate bioadhesive gels (5% and 10%) in the treatment of dentine hypersensitivity. A randomized clinical trial. J Clin Periodontol 2003;30:315-20.
20. Tarbet WJ, Buckner A, Stark MM, Fratarcangelo PA, Augsburger R. The pulpal effects of brushing with a 5 percent potassium nitrate paste used for desensitization. Oral Surg Oral Med Oral Pathol 1981;51:600-02.
21. Poulsen S, Errboe M, Hovgaard O, Worthington HW. Potassium nitrate toothpaste for dentine hypersensitivity. Cochrane Database Syst Rev 2001;2:CD001476.
22. Greenhill JD, Pashley DH. The effects of desensitizing agents on the hydraulic conductance of human dentin in vitro. J Dent Res1981;60:686-98.
23. Green BL, Green M, McFall WT Jr. Calcium hydroxide and potassium nitrate as desensitizing agents for hypersensitive root surfaces. J Periodontol 1977;48:667-72.
24. McFall WT. A review of the active agents available fortreatment of dentinal hypersensitivity. Endod Dent Traumatol 1986;2:141-49
25. Minkov B, Marmari I, Gedalia I, Garfunkel A. The effectiveness of sodium fluoride treatment with and without iontophoresis on the reduction of hypersensitive dentin. J Periodontol 1975;46:246-249.
26. Kerns DG, Scheidt MJ, Pashley DH, Horner JA, Strong SL, Van Dyke TE. Dentinal tubule occlusion and root hypersensitivity. J Periodontol 1991;62:421-428.
27. Tal M, Orion M, Gedalia I, Ehrlich J. X-ray diffraction and scanning electron microscope investigations of fluoride-treated dentine in man. Arch Oral Biol 1976;21:285-90.
28. Minkoff S, Axelrod S. Efficacy of strontium chloride in dental hypersensitivity. J Periondontol .1987;58: 470-74.
29. Furseth R. A study of experimentally exposed and fluoride treated dental cementum in pigs. Acta Odont Scand 1970;28:833-50.
30. Kern DA, McQuade MJ, Scheidt MJ, Hanson B, Van Dyke TE. Effectiveness of sodium fluoride on tooth hypersensitivity with and without iontophoresis. J Periodontol 1989;60:386-89.
31. Pillon FL, Romani IG, Schmidt ER. Effect of a 3% potassium oxalate topical application on dentinal hypersensitivity after subgingival scaling and root planing. J Periodontol 2004;75:1461-4.
32. Hack GD, Thompson VP. Occlusion of dentinal tubules with cavity varnishes. Archs Oral Biol 1994;39:S149.
33. Duran I, Sengun A. The long-term effectiveness of five current desensitizing products on cervical dentine sensitivity. J Oral Rehab 2004;31:351-6.
34. Prati C, Cervellati F, Sanasi V, Montebugnoli L. Treatment of cervical dentin hypersensitivity with resin adhesives: 4-week evaluation. Am J Dent 2001;14:378-82.
35. Baysan A, Lynch E. Treatment of cervical sensitivity with a root sealant. Am J Dent 2003;16:135-8.
36. Dondi dall'Orologio G, Lone A, Finger WJ. Clinical evaluation of the role of glutardialdehyde in a one-bottle adhesive. Am J Dent 2002;15:330-4.
37. Dondi dall'Orologio G, Lorenzi R, Anselmi M, Grisso V. Dentin desensitizing effects of Gluma Alternate, Health-Dent Desensitizer and Scotchbond Multi-Purpose. Am J Dent 1999;12:103-6.
38. Kimura Y, Wilder-Smith P, Yonaga K, Matsumoto K. Treatment of dentine hypersensitivity by lasers: A review. J Clin Periodontol 2000;27:715-21.
39. McCarthy D, Gillam DG, Parson DJ. *In vitro* effects of laser radiation on dentine surfaces. J Dent Res 1997;76:233.
40. Schwarz F, Arweiler N, Georg T, Reich E. Desensitizing effects of an Er:YAG laser on hypersensitive dentine. J Clin Periodontol 2002;29:211-5.
41. Corona SA, Nascimento TN, Catirse AB, Lizarelli RF, Dinelli W, Palma-Dibb RG. Clinical evaluation of low-level laser therapy and fluoride varnish for treating cervical dentinal hypersensitivity. J Oral Rehabil 2003;30:1183-9.
42. Moritz A, Schoop U, Goharkhay K, et al. Long-term effects of CO2 laser irradiation on treatment of hypersensitive dental necks: results of an in vivo study. J Clin Laser Med Surg 1998;16:211- 15.
43. Ciaramicoli MT, Carvalho RC, Eduardo CP. Treatment of cervical dentin hypersensitivity using neodymium: Yttrium aluminum- garnet laser. Clinical evaluation. Lasers Surg Med 2003;33:358-62.
44. Hench LL, Splinter RJ, Allen WC, Greenlee TK. Bonding mechanisms at interface of ceramic prosthetic materials. J Biomed Mater Res Symp1971;2:117-41.
45. Forsback AP, Areva S, Salonen JI. Mineralization of dentin induced by treatment with bioactive glass S53P4 *In vitro*. Acta Odontol Scand 2004;62:14-20.
46. Connie Hastings Drisko. Dentine hypersensitivity - dental hygiene and periodontal Considerations. International Dental Journal.2002;52,385-03.