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MANAGEMENT STRATEGIES FOR DENTINAL HYPERSENSITIVITY : A REVIEW

Dental Science

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ABSTRACT

Dentinal Hypersensitivity is a relatively common painful condition described as short sharp pain usually in response to stimulus in areas of dentinal exposure. Dentinal hypersensitivity is not life threatening, but it can be a particularly unpleasant sensation for patients dictating types of food and drinks ingested. Once sensitivity has become established the pulp may become irreversibly sensitive. Treatment is therefore aimed at not only restoring the original impermeability of the tubules by occluding them, but also controlling the neural elements within the pulp to dampen the external stimulatory effects. Mild and responsive dentinal hypersensitivity may be managed by less complex treatments such as Over the counter desensitizers and in office treatment be reserved for more severe recalcitrant cases. Clinicians should consider predisposing factors, removal of etiologic factors and dietary modifications to prevent recurrence. This article aims to inform practitioners on various management strategies for dentinal hypersensitivity.

KEYWORDS

INTRODUCTION

Dentinal hypersensitivity(DH) is a relatively common painful condition described as short sharp pain usually in response to stimulus in areas of dentinal exposure. Essentially, exposure of the dentine results from one of two processes, either removal of the enamel covering the crown of the tooth, or denudation of the root surface by loss of cementum and overlying periodontal tissues1 .Painful symptoms arising from exposed dentine are a common finding in the adult population affecting 1 in 7 of patients seeking dental treatment (Graf & Galasse 1977).

Dentinal hypersensitivity is not life threatening, but it can be a particularly unpleasant sensation for patients dictating types of food and drinks ingested. The condition most commonly affects the canines and premolars 2 peaking in incidence in the 2nd and 3rd decade of life and then again in the 50's[3].

Patients undergoing periodontal treatment are particularly susceptible to this condition due to gingival recession following periodontal surgery or loss of cementum following non-surgical periodontal therapy[4].

Prolonged exposure to the oral environment may cause the occlusion of dentinal tubules by the smear layer or pellicle. However this may not reduce DH. Once sensitivity has become established the pulp may become irreversibly sensitive. Treatment is therefore aimed at not only restoring the original impermeability of the tubules by occluding them, but also controlling the neural elements within the pulp to dampen the external stimulatory effects. Hence pain control is achieved by obliteration of the dentinal tubules or alteration of pulpal sensory activity, or both[5].

DIAGNOSIS

DH is characterized by short sharp pain arising from exposed dentin in response to stimuli typically thermal, evaporative, tactile, osmotic, or chemical and which can't be ascribed to any other form of dental defect or pathology6. Therefore the diagnosis of DH should be considered only after the practitioner has excluded other possible causes of pain , such as cracked tooth syndrome, fractured restoration, dental caries, post restorative sensitivity, pulpitis .3, 6. The most common stimuli used in clinical studies are thermal and tactile . However widely used stimulus methods have differences that affect their reproducibility and therefore long term monitoring of DH. Pain assessment presents another challenge in recording response as subjective feedback has to be translated to be objective data for recording. This involves the use of both unidimensional and multidimensional pain measurement systems.The most common unidimensional pain measurement system is the Visual analogue scale[7].

Visual analogue scale records a characteristic or attitude that is believed to range across a continuum of values. Pain is a continuous characteristic and doesn't take discrete steps, as a categorization of none, mild, moderate and severe would suggest. Operationally the VAS is usually a horizontal line,100mm in length, anchored by word descriptors at each end 8 .The ends are defined as the extreme limits of the parameter oriented from the left (no pain) to the right (Unbearable pain) 9 . The patient marks on the line the point that they feel best represents the intensity of pain at that time. The VAS score is determined by measuring in millimeters from the left hand end of the line to the point that the patient marks. However since such a method is subjective in nature, it is valuable in comparing changes in the same individual over a period of time but its use is limited when comparing a group of individuals[8].

MANAGEMENT OF DENTINAL HYPERSENSITIVITY

Management of Dentinal Hypersensitivity aims at elimination of pain or discomfort. This can be achieved by two means

- Occlusion of dentinal tubules
- Interefering with transmission of nerve impulse

Occluding agents physically block exposed dentinal tubules, preventing any external stimuli from triggering the movement of

dentinal fluid and thereby blocking pain response 10 .These agents can be delivered by OTC products that patient can use at home or may be applied professionally in the dental office.

The second method to manage DH is to interrupt the neural response to pain stimuli. This may be achieved by direct ionic diffusion. 9,10. World Health Organization recommends that mild and responsive DH be managed by less complex treatments such as OTC desensitizers and in office treatment be reserved for more severe recalcitrant cases2 (See Table 1).

Sodium fluoride (NaF)

Home-use over-the-counter desensitizing agents that occlude the dentinal tubules are found in toothpastes, gels, and mouth rinses. One of the main active ingredients used in this manner is fluoride. The action of NaF may be attributed to the reaction that occurs between NaF and Calcium ions of dentinal fluid, and that leads to the formation of calcium fluoride crystals, which are deposited onto the openings of the dentinal tubules. However the crystal size of calcium fluoride (CaF2) being small, is diluted by saliva11, a single application of NaF would be ineffective in narrowing down the diameter of dentinal tubules and multiple applications would be necessary12.

Sodium mono fluorophosphates

It is effective in treating DH, however the mechanism of action is unclear13.Scanning Electron Microscopy failed to demonstrate any visual changes in the dentinal surface. Tubule occlusion , if any doesn't appear to be permanent14.

Iontophoresis is one such procedure whereby the desensitizing agents are penetrated deep into the open dentinal tubules with the help of electric current. It is most often used in conjuction with fluoride pastes or solutions and reportedly reduces DH 15.

Stannous fluoride

It is effective in controlling DH either in aqueous solution or in glycerine gelled with carboxy methyl cellulose. Acts by tubule occlusion which may be partial or complete. Alternatively it may precipitate on dentin surface leading to occlusion of exposed dentinal tubules 14. An aqueous Stannous fluoride gel product has been granted ADA seal of acceptance for the therapeutic prevention of caries16.

Potassium nitrate

Potassium nitrate is probably the most popular ingredient in OTC products for the treatment of DH. 5% Potassium nitrate is the concentration recognized by the FDA for this agent 17 .Potassium ions act by penetrating the dentinal tubules and block the repolarization of the myelinated A-fibers thus inactivating the action potential. 18, 19 20 .Hence desensitization is achieved. Importantly Potassium nitrate does not induce any pulpal changes and may be safely used in whitening trays to relieve hypersensitivity between whitening treatments[21].

Oxalates

Oxalates potentially offer an inexpensive and noninvasive chairside modality to treat DH. The potassium oxalate is a desensitizing agent which potentially obstruct the dentinal tubules and has been proven in a number of laboratory and clinical studies . This agent may act both by depolarization of the nerve endings (neural action) and by precipitation of calcium oxalate crystals in the dentinal tubules22.

Calcium oxalates crystals formed on dentin surface are easily removed by daily brushing. However acid etching with 35% phosphoric acid increasing the penetration depth of oxalate buffer leading to long lasting relief23.Oxalates have potential toxicity and are best avoided in generalized DH due to gingival and gastric irritation[24].

On the contrary Ben Balevi suggested that with the possible exception of 3 percent monohydrogen monopotassium oxalate alone, current evidence does not support the use of oxalates in the treatment of DH 25 .

Resin based formulations

Resin-based materials have been reported to successfully reduce dentin hypersensitivity26. An aqueous solution of glutaraldehyde and HEMA (hydroxyethylmethacrylate) e.g., Gluma Desensitizer (Hereaus-Kulzer) and Calm-It (Dentsply-Caulk), has been reported to be an effective desensitizing agent. The mechansim for tubule occlusion appears to be due to the effect of glutaraldehyde mainly through coagulation of proteins inside the dentinal tubules27.

Restorative treatment

Buccal cervical area is predisposed to DH since erosive and abrasive factors alone or in combination are most likely to impact at this site to expose dentine 28. In areas of significant tooth wear restorative materials like glass ionomer cements, composite resins can be used. However this treatment modality is best reserved for cases that do not respond to less invasive desensitizing protocols[5].

Lasers

The first use of laser for the treatment of dentine hypersensitivity was reported by Matsumoto et al. (1985)29. The lasers used for the treatment of dentine hypersensitivity are divided into two groups: low output power (low level) lasers [helium-neon (He-Ne) and Gallium/ Aluminum/Arsenide (GaAlAs) (diode) lasers], and middle output power lasers (Nd:YAG and CO2 lasers)[30].

Nd:Yag

The mechanism of Nd:YAG laser effects on dentine hypersensitivity is thought to be the laser-induced occlusion or narrowing of dentinal tubules (Lan & Liu 1995, 1996, Yonaga et al1999) as well as direct nerve analgesia (Whitters et al. 1995) [29].

GaAIA

These lasers mediate an analgesic effect related to depressed nerve transmission and blocking of depolarization of C afferent fibres 31.The combined use of the GaAlAs laser (830 nm wavelength) with fluoridation enhances treatment effectiveness by more than 20% over that of laser treatment only [32].

He Ne lasers

The first use of He-Ne laser for the treatment of dentine hypersensitivity was reported by Senda et al29. The mechanism involved is mostly unknown. According to physiological experiments, He-Ne laser irradiation does not affect peripheral A delta or C fiber nociceptors (Jarvis et al. 1990), but does affect action potential, which in the healthy nerve increased by 33% following a single transcutaneous irradiation[33].

Carbon dioxide (CO2) Laser

The first use of this laser for the treatment of dentine hypersensitivity was reported by Moritz et al. CO2 laser effects on dentine hypersensitivity are due to the occlusion or narrowing of dentinal tubules 34. There have been no reports on nerve analgesia by CO2 laser irradiation. CO2 laser with stannous fluoride gel effect tubule occlusion for upto 6 months[29].

Casein phosphopeptides

Amorphous calcium phosphate (ACP) was developed by Tung et al in 2003, mimics the natural process of dentinal sclerosis and provides effective biocompatible treatment for dentin hypersensitivity. ACP is precipitated under oral physiological conditions by the sequential application of calcium chloride (1.5mol/l) solution followed by potassium phosphate (1mol/l) maintained at a pH of 9.5[35].

G.C. tooth mousse is a commercial preparation containing ACP and casein phosphopeptide (CPP), a product developed by Prof Reynolds at the University of Melbourne 36. CPP stabilizes ACP and forms nano complexes with ACP at the tooth surface thereby providing a reservoir of calcium and phosphate ions which favors mineralization 37. Calcium phosphate compounds occlude the tubules by forming a calcium phosphate precipitate, while calcium hydroxide occludes the tubules and promotes peritubular dentin formation. 35,38. The efficacy of CPP ACP reported in the literature have varied from a rapid reduction in sensitivity, with a prolonged desensitizing effect37 to insufficient effectiveness and short-term therapeutic effect in treating hypersensitivity of dentine[39].

Bioglass

This bioactive glass contains calcium sodium phosphosilicate which rapidly release calcium, sodium, and phosphorous ions which form hydroxycarbonate apatite (HCA) which occludes dentinal tubules . The use of Bioglass in the management of DH has been shown by some products such as Novamin. It has been successfully incorporated into prophylaxis pastes thus providing relief from DH while ensuring effective stain removal[40].

CONCLUSION

In clinical practice, the approach to the management of DH has been

treatment based with little or no emphasis for the etiological and predisposing factors. Clinicians should evaluate the causes of DH and management strategies should include preventive measures.

Before embarking on any treatment protocol, it is necessary to consider operator knowledge and skill, patient preference, cost benefit ratio, severity of condition and number of teeth involved. As a general rule, treatment may initially involve non invasive and most cost effective modalities before considering complex and invasive procedures.

Removal or modification of etiological factors , dietary modification and oral hygiene will go a long way in ensuring a successful treatment . Regular reviews are recommended at appropriate intervals.

TABLE 1
Management strategy for dentinal hypersensitivity a "hierarchial model"

General Guidelines
1. Improve awareness regarding risk factors in etiology of Dentinal Hypersensitivity
2. Suggest dietary modifications (Avoidance of dietary acids)
3. Recommend appropriate tooth brushing technique.

Level 1: Non invasive intervention (At home/ OTC)
1. Fluoride and Potassium nitrate dentifrices
2. Mouth washes/chewing gum
3. CPP ACP

Level 2: Non invasive intervention (Professional/ In office)
1. Fluorides(sodium fluoride , stannous fluoride)
2. Oxalates (Potassium Oxalate, Aluminium Oxalate)
3. Varnish

Level 3: Invasive Intervention
1. Dentin Bonding Agents HEMA
2. Lasers
3. Restorative material
4. Periodontal surgery.

DH severity should be reassessed two to four weeks after commencement of treatment to determine the effectiveness of the desensitizing treatment.

REFERENCES

1. Dowell.P, Addy.M. Dentin hypersensitivity A review. J clin peiodontol1983;10:341-350.
2. Ochardson R ,David .G. Managing dentin hypersensitivity. JADA;2006;137;990-998
3. Walter .P.A. Dentinal hypersensitivity : A review. J Contemporary Dent Practice 2005;6;110.
4. Curro F. Tooth hypersensitivity in the spectrum of pain. Dent Clin No Amer 1990;34; 429-37.
5. Bartold. P.M. Dentinal hypersensitivity : a review. ADJ 2006;51;212-218
6. Addy. M .Dentine hypersensitivity: new perspectives on an old problem : IDJ 2002;52 ; 5: 367-375.
7. Ide .M, Wilson.RF, Ashley.FP. The reproducibility of methods of assessment for cervical dentine hypersensitivity:J Clin Periodontol 2001; 28: 16–22.
8. Agnes Paul-Dauphin, Francis Guillemin, Jean-Marc Virion. Am J Epidemiol1999; 150:1117-27
9. Gould. D. Visual analogue scale: Journal of clinical nursing 2001;10:697-706
10. Cummins D. Dentin hypersensitivity: From to a breakthrough therapy for everyday sensitivity relief. J Clin Dent 2009; 20 (Sp Is):1-9.
11. Everett FG, Hall WB, Phatak NM. Treatment of hypersensitive dentin. JOral Ther Pharmacol.1966;2:300-10
12 Corona S AM, Do Nascimento TN, Catirse ABE et al. Clinical evaluation of low-level laser therapy and fluoride varnish for treating cervical dentinal hypersensitivity. J Oral Rehab 2003; 30: 1183-1189
13. Addy M, Dowell P. Dentine hypersensitivity – A review. Clinical and in vitro evaluation of treatments. J Clin Periodontol1983;10:351-363.
14. Tarbet WJ, Silverman G, Stolman JM, Fratarcangelo PA. Clinical evaluation of a new treatment for dentinal hypersensitivity. J Periodontol 1980;51:535-540.
15. Wilson JM, Fry BW, Walton RE, Gangarosa LP. Fluoride levels in dentin after iontophoresis of 2 % sodium fluoride. J Dent Res 1984;63(6):897-900
16. Jacobsen PL, Bruce G. Clinical dentin hypersensitivity: understanding the causes and prescribing a treatment. J. Contemporary Dent Practice 2001;2(1):1-8.
17. Federal register ,vol 57 no 91 ,may 11,1992; 20114-20115.
18 . Peacock J M, Orchardson R. Effects of potassium ions on action potential conduction in A andC fibers of rat spinal nerves: J Dent Res 1995; 74:634- 41.
19. Kim S Hypersensitive teeth. Desensitization of pulpal sensory nerves : Jendod 12, 482 - 85
20. Kim JL, Karastathis D. Dentinal hypersensitivity management. Dental Hygiene Theory and Practice .:3rd ed. St. Louis, MO: Saunders-Elsevier. 2010:726- 35.
21. Haywood VB, Caughman WF, Frazier KB, Myers ML. Tray delivery of potassium nitrate-fluoride to reduce bleaching sensitivity. Quintessence Int. 2001;32(2):105-9.
22. Orchardson, R.; Gillam, D.G. The efficacy of potassium salts agents for treating dentin hypersensitivity. J.Orofac. Pain,2000 ; 14 (1): 9-19.
23. Isabel C.C.M. Porto. Diagnosis and treatment of Dentinal hypersensitivity ; Journal of oral science ,2009 : 51; 3,323-332 .
24. Guo C , Mc Martin KE . The cytotoxicity of oxalates ,metabolite of ethylene glycol ,is due to calcium oxalate monohydrate formation . Toxicology 30:347 -355.
25. Oxalates may not be effective In treating dentin hypersensitivityn 2012 JADA :(6) ;143
26. Duran I, SengunA. The long term effectiveness of five current desensitizing products on cervical dentine hypersensitivity . J Oral Rehabil 2004;31: 351- 56.
27. Schupback P, lutz F . Closing of dentinal tubules by Gluma desensitizer . Eur J Oral Sci 1997 ;105;414 -421.
28. Addy M. Dentine hypersensitivity: new perspectives on an old problem; International Dental Journal 2002 :52; 5 (Supplement 1)
29. Kimura Y, Wilder-Smith P, Yonaga K, Matsumoto K: Treatment of dentine hypersensitivity by lasers: a review.J Clin Periodontol 2000; 27: 715–721.
30. Shriprasad Sarapur, Shilpashree H.S. Dentin Hypersensitivity: A Review international journal of dental clinics 2012 :4 ; 1
31. Wakabayashi, H., Hamba, M., Matsumoto, K. & Nakayama, T. Electrophysiological study of irradiation of semiconductor laser on the activity of the trigeminal subnucleues caudal neurons. 1992:Journal of Japanese Society for Laser Dentistry 3, 65–74.
32. Liu, H.-C. & Lan, W.H.The combined effectiveness of the semiconductor laser with Duraphat in the treatment of dentin hypersensitivity. Journal of Clinical Laser Medicine & Surgery 1994 :12;315–319.
33. Rochkind, S., Nissan, M., Barr-Nea, L.Response of peripheral nerve to He-Ne laser: experimental studies. Lasers in Surgery and Medicine 1987: 7, 441–443
34. Moritz, A., Gutknecht, N., Schoop, U., Wernisch, J., Lampert, F. & Sperr, W.Effects of CO2 laser irradiation on treatment of hypersensitive dental necks: results of an in vitro study. Journal of Clinical Laser Medicine & Surgery 1995:13; 397– 400.
35. Geiger S, Matalon S, Blasbalg J, Tung M, Eichmiller F. The clinical effect of amorphous calcium phosphate (ACP) on root surface hypersensitivity. Operative Dentistry 2003; 28 (5): 496-500
36. Reynolds E, Cain C, Webber E. Anticariogenicity of calcium phosphate complexes of tryptic casein phosphopeptides in the rat. Journal of Dental Research1995;74(6):1272-9.
37. Rosaiah.K, Aruna.K. Clinical Efficacy of Amorphous Calcium Phosphate, G.C. Tooth Mousse and Gluma Desensitizer in Treating Dentin Hypersensitivity. International journal of dental clinics 2011: 3 (1)
38. Tung MS, Bowen HJ, Derkson GD. Effects of calcium phosphate solution on dentin permeability J Endodont.1993;19:283
39. Kowalczyk A, Botuliński B, Jaworska M, Kierklo A, Pawińska M, Dąbrowska E. Evaluation of the product based on RecaldentTM technology in the treatment of dentin hypersensitivity; Advances in MedicalSciences:2006; 51 :1 ·
40. Jennings DT, McKenzie KM, Greenspan DC. Quantitative analysis of tubule occlusion using Novamin(sodium calcium phosphosilicate). J Dent Res. 2004; 83(Spec Iss A): 2416.