DENTAL SCIENCE

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ABSTRACT

Dental hypersensitivity is a relatively common painful condition described as short sharp pain usually in response to stimulus in areas of dentinal exposure. Dental 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 therapy. Management of Dentinal Hypersensitivity aims at elimination of pain, post restorative sensitivity, pulpitis. 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.

INTRODUCTION

Dental 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 tissues. 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).

Dental 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.

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.

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.

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 pathology. 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. 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.

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 pain (unbearable pain) and none. 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.

MANAGEMENT OF DENTAL HYPERSENSITIVITY

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

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

Occluding agents physically block exposed dentinal tubules, preventing any external stimuli from triggering the movement of pain.
dentinal fluid and thereby blocking pain response. 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 iontophoresis. 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 cases.

**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 saliva, a single application of NaF would be ineffective in narrowing down the diameter of dentinal tubules and multiple applications would be necessary.

**Sodium mono fluorophosphates**

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

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 conjunction with fluoride pastes or solutions and reportedly reduces DH.

**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. An aqueous Stannous fluoride gel product has been granted ADA seal of acceptance for the therapeutic prevention of caries.

**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. 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.

**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 tubules.

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 relief. Oxalates have potential toxicity and are best avoided in generalized DH due to gingival and gastric irritation.

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

**Resin based formulations**

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

**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.

**Lasers**

The first use of laser for the treatment of dentine hypersensitivity was reported by Matsumoto et al. (1985). 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).

**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 al 1999) as well as direct nerve analgesia (Whitters et al. 1995).

**GaAlA**

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

**He Ne lasers**

The first use of this laser for the treatment of dentine hypersensitivity was reported by Senda et al. 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.

**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. There have been no reports on nerve analgesia by CO2 laser irradiation. CO2 laser with stannous fluoride gel effect tubule occlusion for up to 6 months.

**Casein phosphopeptides**

Amorphous calcium phosphate (ACP) was developed by Tung et al. 1993, mimics the natural process of dental sclerosis and provides an 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.

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 effect to insufficient effectiveness and short-term therapeutic effect in treating hypersensitivity of dentine.

**Bioglass**

This bioactive glass contains calcium sodium phosphosilicate which rapidly release calcium, sodium, and phosphorous ions which form hydroxyapatite (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.

**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”

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<thead>
<tr>
<th>Level</th>
<th>Strategy</th>
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<tbody>
<tr>
<td>1.</td>
<td>Non invasive intervention (At home/ OTC)</td>
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<tr>
<td>2.</td>
<td>Non invasive intervention (Professional/ In office)</td>
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<tr>
<td>3.</td>
<td>Invasive intervention</td>
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**General Guidelines**

1. Improve awareness regarding risk factors in etiology of Dental 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

**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 weeks after commencement of treatment to determine the effectiveness of the desensitizing treatment.**

**REFERENCES**