Review Article

Indian Journal of Dental Education
Volume 6 Number 3, July - September 2013

Dentin Hypersensitivity: An Engima

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Abstract

The objective of this review is to inform practitioners about dentin hypersensitivity and its management. This review provides a general view of the aetiology, characteristics and treatment of dentinal hypersensitivity, so that professionals can use this information in the therapeutic management of this clinical condition. The presenting symptoms of sensitive teeth are multi-factorial, and from the perspective of restorative dentistry, make a differential diagnosis of true dentine hypersensitivity a challenge. Dentin hypersensitivity is diagnosed after elimination of other possible causes of the pain. Desensitizing treatment should be delivered systematically, beginning with prevention and at-home treatments. The latter may be supplemented with in-office modalities. The prevalence of Dentin hypersensitivity varies widely, depending on the mode of investigation. Potassium-containing toothpastes are the most widely used at-home treatments. Most in-office treatments employ some form of "barrier", either a topical solution or gel or an adhesive restorative material. The availability of a wide variety of treatment could be an indicator that there is still no effective desensitizing agent to completely resolve the patient's discomfort, or that it is difficult to treat, irrespective of the available treatment options.

Keywords: Dentin hypersensitivity; Desensitizing agents; Home-care; In-office treatment; Toothpaste.

Introduction

During routine dental examinations, our patients frequently inquire about dentinal hypersensitivity that was one episode or is chronic and recurring due to a given action, e.g., drinking cold beverages, eating hot foods, breathing in and out. This common complaint is defined as dentinal hypersensitivity, but it is also known as root sensitivity, or just sensitivity. Patients describe this phenomenon as sharp, short-lasting tooth pain, irrespective of the stimulus.

Definition

Dentine hypersensitivity is also referred as dentine sensitivity, dentinal hypersensitivity, cervical hypersensitivity/sensitivity, root hypersensitivity/sensitivity, cemental hypersensitivity/sensitivity. Holland *et al*[1] described dentinal hypersensitivity as "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". The first part of the definition provides a clinical description of dentine hypersensitivity whereas the second part aids in its differential diagnosis.

In dentine hypersensitivity, the definition highlights different stimuli inducing pain. Of these, cold or evaporative stimuli are usually identified as the most problematic for sufferers. Heat is not commonly reported perhaps because it is the exception to stimuli evoking pain, causing relatively slow inward movement of dentinal fluid.

Differential Diagnosis

Dentinal hypersensitivity has all the criteria to be considered a true pain syndrome. It is

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important to distinguish sensitivity pain, that of short duration, from pain of longer duration not treatable with desensitizing agents. A number of other dental conditions can give rise to pain symptoms similar to those of dentine hypersensitivity. Indeed, a definitive diagnosis of dentine hypersensitivity is reached through exclusion of the following conditions, which need a variety of treatment options for resolution.

1. Cracked tooth syndrome, often in heavily restored teeth. Pain typically occurs on release of biting or tapping of a single cusp.
2. Abscessed or non- vital tooth. With periapical radiolucency or draining fistula; necrotic with sensitivity to occlusion; partially necrotic in one canal, with vital tissue elsewhere (in which case tooth tests vital to stimuli). Pain typically occurs spontaneously or upon occlusion or tapping.
3. Pulpal response to dental caries. Greatest degree of sensitivity experienced when dental decay passes the dentine-enamel junction. As caries penetrates further into the tooth, sensitivity lessens until pulp becomes involved.
4. Gingival recession. Often occurs post-periodontal surgery, when a large portion of the root is exposed, or due to ageing, mechanical trauma, frenum attachment pull or occlusal trauma.
5. Toothbrush abrasion. Caused by use of a hard toothbrush or a soft toothbrush with abrasive toothpaste or by aggressive brushing, and generally located on the side opposite the dominant hand. Abrasion may either instigate gingival recession or stem from greater accessibility to softer root surfaces from recession.
6. Abfraction lesions. Generally associated with occlusal trauma where the anatomic crown of the tooth has flexure. Although non-carious, these lesions can become very sensitive and even progress into the pulp. They may be multifactorial where abrasion and erosive forces combine to produce tooth surface loss.
7. Erosive lesions. Associated with acid reflux, hiatus hernia, purging, bulimia (intrinsic causes), and diet (extrinsic causes). Intrinsic acid lesions typically occur on the palatal surfaces, while extrinsic acid lesions tend to occur on the buccal surfaces. Consuming large quantities of carbonated cola drinks and fruit drinks, which have a very low p^H, causes tooth surface loss, as does toothbrushing following an acidic assault, which removes the acid-softened enamel or dentine.
8. Diet sensitivity. Generally associated with a low p^H material, such as fresh tomatoes, orange juice, cola drinks. Areas with exposed dentine are etched, causing sudden sensitivity. Diet choices may aggravate sensitivity from erosion.
9. Genetic sensitivity. Patients reporting history of sensitive teeth. It is not known whether sensitivity correlates to the 10% of teeth that do not have cementum covering all the dentine at the dentinoenamel junction, or is a factor of lower overall patient pain threshold values.
10. Restorative sensitivity. Triggered following placement of a restoration for several possible reasons: certain amalgams (such as Tytin) having a history of 24–48 hours sensitivity due to shrinkage, rather than the usual expansion, during setting; contamination of composites during placement or improper etching of the tooth on composites, which results in micro-leakage; improper tooth-drying technique; incorrect preparation of glass ionomer or zinc phosphate cements; general pulpal insult from cavity preparation technique; thermal or occlusal causes; galvanic reaction to dissimilar metals that creates a sudden shock or 'tin foil' taste in the mouth.
11. Medication sensitivity. Due to

medications that dry the mouth (e.g. antihistamines, high blood pressure medication), thereby compromising the protective effects of saliva and aggravating diet-related trauma or proliferating plaque. Even a reduction in salivary flow due to ageing or medications can lower the p^H of the saliva below the level at which caries occurs (6.0–6.8 for Dentine caries; < 5.5 for enamel caries) and increase erosive lesions to exposed dentine.

12. Bleaching sensitivity. Commonly associated with carbamide peroxide vital tooth bleaching and thought to be due to the by-products of 10 % carbamide peroxide (3% hydrogen peroxide and 7 % urea) readily passing through the enamel and dentine into the pulp in a matter of minutes. Sensitivity takes the form of a reversible pulpitis caused from the dentine fluid flow and pulpal contact of the material, which changes osmolarity, without apparent harm to the pulp. Sensitivity is caused by all other forms of bleaching (in-office, with or without light activation, and new, over-the-counter) and depends on peroxide concentration.
13. Fractured restorations and incorrectly placed dentine pins.
14. Lack of care while contouring restorations so the tooth is left in traumatic occlusion.
15. Palatogingival groove and other enamel invaginations.
16. Chipped teeth causing exposed dentine.
17. Vital bleaching.

Aetiology

Dentinal hypersensitivity can have multiple aetiologies. It is important that the patient's medical and social history, lifestyle, medications and supplements being taken, diet and food habits, and oral hygiene be thoroughly reviewed. Before making a diagnosis of dentinal hypersensitivity, other oral conditions must be ruled out, including occlusal trauma, caries, defective restorations, fractured or cracked teeth, potential reversible or irreversible pulpal pathology, or gingival conditions.

The primary cause of dentin hypersensitivity is loss of enamel on the tooth crown and gum recession exposing the tooth root. Tooth wear due to the irreversible loss of tooth structure and includes conditions such as abrasion, erosion, attrition, and abfraction. Enamel can also be lost as a result of aggressive or incorrect toothbrushing, overconsumption of acidic food, and tooth grinding caused by stress and parafunctional behaviours and iatrogenic during restorative procedures and tooth preparation. The frequent intake of food and beverages such as fruits, lemon tea, fruit juice, and soft drinks can cause tooth erosion and dentine hypersensitivity. Gum recession may occur as a result of aggressive and incorrect toothbrushing, as well as periodontal diseases. Some dental operative and surgical procedures can also cause the gum to move away from the normal position at the crown-root junction. Dentin hypersensitivity has also been reported after external tooth bleaching.

Mechanism of sensitivity

The most widely accepted mechanism of dentinal sensitivity is the hydrodynamic theory, first described by Brännström. In this model, the aspiration of odontoblasts into the dentinal tubules, as an immediate effect of physical stimuli applied to exposed dentin, results in the outward flow of the tubular contents (dentinal fluids) through capillary action. The changes to the dentinal surface lead to stimulation of the A-type nerve fibers surrounding the odontoblasts. For there to be a stimulus response, the tubules must be open at both the dentinal interface and within the pulp. Sensitive teeth have up to eight times the number of open dentinal tubules per surface area compared to nonresponsive teeth. Another theory is an alteration in pulpal sensory nerve activity. The treatment of exposed, open dentinal tubules is based upon

the physiology of the stimulus response.

Episodic nature of dentine hypersensitivity

For dentine hypersensitivity to occur, not only does the dentine need to become exposed (lesion localization) and but the tubules need to be patent to the pulp (lesion initiation). Many people have dentine exposed to the oral environment owing to loss of cementum and D or enamel, but clinical experience indicates that only a proportion of those people suffer from dentine hypersensitivity. In vitro studies indicate that erosion from acidic soft drinks causes rapid loss of the smear layer resulting in the wide opening of tubules[2], and similarly most toothpastes readily remove the smear layer to expose tubules.[2] However, toothbrushing can also replace the smear layer, creating a dynamic environment.[2]

The width of the tubule is very important, as the rate of fluid flow is dependent on the fourth power of the radius. If the tubule diameter doubles, a 16-fold increase in fluid flow results. Sensitive teeth have many more (8 times) and wider (2 times) tubules at the buccal cervical area compared with nonsensitive teeth.[3] A higher velocity of fluid flow also occurs in tubules of smaller diameter, possibly provoking pain sensations. Dentine will only be sensitive if the tubules are patent from the pulp to the oral environment, and this patency will change with production and removal of the smear, hence resulting in an episodic condition.[3]

Behavioral changes

Patients with unresolved hypersensitivity over many years provide the dental professional with varied behavioral and postural clues, some of which are easily recognized. These include avoidance of routine dental exams, necessary treatment and follow-up care, reluctance to schedule planned treatment or follow-up care, insistence on the use of local anesthesia for even the most minor of dental treatments, tense facial muscles, tooth clenching, a rigid torso, holding hands tightly on the arm rest, crossed arms, an awkward head position and an inability to follow routine instructions for head and body positioning.[4]

Epidemiology

The prevalence of dentine hypersensitivity ranges from 4% to 57%. Although the age range for dentin hypersensitivity varies from 15-70 years, the peak incidence is between 20 to 40 years.[5]

The highest incidence of dentine hypersensitivity has been reported on the buccal cervical area of teeth. The teeth most commonly affected are canines> premolars> incisors> molars.[5]

Women are more frequently affected and at a younger mean age.[6]

A significantly higher proportion of left vs right contralateral teeth was reported in right-handed patients with dentine hypersensitivity. [5]

Addy and his colleagues reported that all sensitive teeth have very low plaque scores, suggesting that toothbrushing with dentifrice may facilitate the development of dentin hypersensitivity.[7]

Management

The development of a sound treatment plan for any oral health condition should consider causative factors. Similarly, any treatment plan for dentine hypersensitivity should include identifying and eliminating predisposing etiologic factors such as endogenous or exogenous acids and toothbrush trauma.

After observing the severity and number of teeth involved, an active approach to dentine hypersensitivity can begin in the cases of generalized dentine hypersensitivity , by a home method followed by in-office treatment when the first option is not successful. However, when dentine hypersensitivity is restricted to a few teeth, one can opt for an in-office method as initial treatment.

Preventive management recommendations[8]

Suggestions for patients:

1. Avoid using large amounts of dentifrice or reapplying it during brushing.
2. Avoid medium- or hard-bristle toothbrushes.
3. Avoid brushing teeth immediately after ingesting acidic foods.
4. Avoid brushing teeth with excessive pressure or for an extended period of time.
5. Avoid excessive flossing or improper use of other interproximal cleaning devices.
6. Avoid "picking" or scratching at the gumline or using toothpicks inappropriately.

Suggestions for dental professionals:

1. Avoid over-instrumenting the root surfaces during scaling and root planing, particularly in the cervical area of the tooth.
2. Avoid over-polishing exposed dentine during stain removal.
3. Avoid violating the biological width during restoration placement, as this may cause recession.
4. Avoid burning the gingival tissues during in-office bleaching, and advise patients to be careful when using home bleaching products.

At-home treatment procedures

Desensitizing agents intended for at-home use by patients generally are simple to administer.

Desensitizing toothpastes/dentifrices: Toothpastes are the most widely used dentifrices for delivering over-the-counter desensitizing agents. The first desensitizing toothpastes to appear on the market claimed either to occlude dentinal tubules (those that contained strontium salts and fluorides) or destroy vital elements within the tubules (those that contained formaldehyde). Now, most desensitizing toothpastes contain a potassium salt such as potassium nitrate, potassium chloride or potassium citrate, though it has been reported that a remineralizing toothpaste containing sodium fluoride and calcium phosphates reduced dentine hypersensitivity. [9]

Toothpaste application: Practitioners should educate patients on how to use dentifrices and monitor their toothbrushing techniques. Dentifrices should be applied by tooth brushing. There is no evidence to suggest that finger application of the paste increases effectiveness. Many patients habitually rinse their mouths with water after toothbrushing. Rinsing with water may cause the active agent to be diluted and cleared from the mouth and, thus, reduce the efficacy of the caries reducing effect of fluoride toothpastes.[10]

Mouthwashes and chewing gums: Studies have found that mouthwashes containing potassium nitrate and sodium fluoride[11], potassium citrate or sodium fluoride or a mixture of fluorides[12] can reduce dentine hypersensitivity. Another study[13] concluded that a chewing gum containing potassium chloride significantly reduced dentine hypersensitivity, but the study did not include a control group.

Dentine hypersensitivity severity should be reassessed two to four weeks after commencement of treatment to determine the effectiveness of the first level of desensitizing treatment. If at-home care fails to reduce dentine hypersensitivity compared with baseline levels, the next level of treatment, an in-office method, should be started.

In-office treatment procedures[14]

Dental professionals can deliver a wider range of more complex and more potent desensitizing treatment.

1. *Nerve desensitization:* Potassium nitrate
2. *Anti-inflammatory agents:* Corticosteroids
3. Cover or plugging dentinal tubules;

a. Plugging (sclerosing) dentinal tubules
   - Ions/salts

i. Calcium hydroxide
ii. Ferrous oxide
iii. Potassium oxalate
iv. Sodium monofluorophosphate
v. Sodium fluoride
vi. Sodium fluoride/stannous fluoride combination
vii. Stannous fluoride
viii. Strontium chloride

- Protein precipitants
  i. Formaldehyde
  ii. Glutaraldehyde
  iii. Silver nitrate
  iv. Strontium chloride hexahydrate
- Casein phosphopeptides
- Burnishing
- Fluoride iontophoresis

b. Dentine sealers
   i. Glass ionomer cements
   ii. Composites
   iii. Resins
   iv. Varnishes
   v. Sealants
   vi. Methyl methacrylate
c. Periodontal soft tissue grafting
d. Crown placement/restorative material
e. Lasers

If the symptoms still persists, then the offending tooth is either root canal treated or extracted.

Several criteria are recognized as constituting an ideal desensitizing agent. These include not irritating the pulp, being relatively painless to apply, easily applied, rapid action, permanently effective and should not discolour the teeth. Overall, patient responses are very subjective and thus treatment results are largely dependent upon the individual's pain threshold.

Conclusion

As part of the routine dental examination and during every recall appointment, dental professionals should include in their patient questions queries about whether there are any sensitive teeth. There are many causes of and treatments for dentine hypersensitivity. Patients with dentinal hypersensitivity should be evaluated based upon risk factors and a proper diagnosis made, after which a treatment plan can be outlined for the patient. In most circumstances, the least invasive, most cost-effective treatment is the use of an effective desensitizing toothpaste. Based on the identified cause, a combination of individualized instructions on proper oral health behaviors, use of self-care products, and professional treatment, including recent and novel technologies that have been introduced may be required to manage the problem.

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