Dentin Hypersensitivity: Etiology, Symptoms, Diagnosis and Recent Trends in Management

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Dentin hypersensitivity is dental pain which is sharp in nature and persist for short duration, a comes from exposed dentin surfaces in response to stimuli, mainly thermal, tactile, osmotic, chemical or electrical. The main cause of sensitivity is receding gums with exposure of root surfaces, loss of the cementum layer and smear layer, and tooth wear. Theoretically noticed, the reported incidence is usually higher than when clinical examination is used. Overall, it is estimated to affect about 20% of the general population to some degree. The diagnosis of dentin hypersensitivity may be challenging. It is a diagnosis of exclusion, reached once all other possible explanations for the pain have been ruled out. There is no universally accepted, gold-standard treatment which reliably relieves the pain of dental hypersensitivity in the long term, and consequently many treatments have been suggested which have varying degrees of efficacy when scientifically studied. Generally, they can be divided into in-office, or treatments which can be carried out at home, available over-the-counter or by prescription. Non-invasive, simple treatments which can be carried out at home should be attempted before in-office procedures are carried out.

Keywords: Dentin, Dentin tubule, Dentin hypersensitivity, Desensitizing agent

Introduction

Dentin hypersensitivity (DH [1], or DHS [2], or sensitive dentin [3], dentin sensitivity [4], cervical sensitivity [5], and cervical hypersensitivity [5]) is dental pain which is sharp in nature and persist for short duration, a comes from exposed dentin surfaces in response to stimuli, mainly thermal, tactile, osmotic, chemical or electrical; It has been recorded for over 2 millenium. The earliest treatment method was noticed back to 400 B.C. (Figure 1).

Causes

The main cause of sensitivity is receding gums with exposure of root surfaces, loss of the cementum layer and smear layer, and tooth wear [6]. Receding gums is because of long-term trauma from excessive or forceful toothbrushing, or abrasive toothpaste brushing (dental abrasion) [6,7], or a sign of chronic periodontitis (gum disease) [7]. The other less common causes are acid erosion (may be related to gastroesophageal reflux disease or excessive consumption of acidic foods and drinks), and periodontal root planing [7]. Other important cause is Dental bleaching of hypersensitivity [6].

According to some it includes smoking tobacco, which can wear down enamel and gum tissue, cracked teeth or grinding of teeth (bruxism) [8]. Dentine contains many thousands of microscopic tubular structures that radiate outwards from the pulp; these dentinal tubules are typically 0.5-2 μm in diameter.
Changes in the flow of the plasma-like biological fluid present in the dentinal tubules can trigger mechanoreceptors present on nerves located at the pulpal aspect, thereby eliciting a pain response. This hydrodynamic flow can be increased by cold, air pressure, drying, sugar, sour (dehydrating chemicals), or forces acting onto the tooth. Hot or cold food or drinks, and physical pressure are typical triggers in those individuals with teeth sensitivity (Figure 2).

**Epidemiology**

DH is undoubtedly a common condition [1,5]. Various studies and methods for detection of DH had been done because in differences in populations, the reported incidence ranges from 4%-74% [5]. Dentists may under-report DH due to difficulty in diagnosing and managing the condition [1]. Theoretically noticed, the reported incidence is usually higher than when clinical examination is used [5]. Overall, it is estimated to affect about 20% of the general population to some degree.

Sensitivity can affect people of any age, although those aged 20-50 years are more likely to be affected [5]. Females are more likely proneto develop DH when compared to males [5]. The condition is mostly noticed in the maxillary and mandibular canine (facial) and bicuspid teeth on the buccal aspect [5], especially in areas of periodontal attachment loss [7].

**Symptoms**

In respond to external stimulus that is cold pain is sharp and sudden [1,9]. About 75% of people with hypersensitivity reporting pain upon application of a cold stimulus [5]. some other stimuli may also trigger pain in DH, includes:
- Thermal – hot and cold drinks and foods [9], cold air, coolant water jet from a dental instrument.
- Electrical – electric pulp testers [6].
- Mechanical – tactile – dental probe during dental examination [6], periodontal scaling and root planing [6], tooth brushing [9].
- Osmotic – hypertonic solutions such as sugars [6].
- Evaporation – air blast from a dental instrument [6].
- Chemical – acids [6], e.g., dietary, gastric, acid etch during dental treatments.

The frequency and severity of pain with every stimulus occurs are variable [2].

**Diagnosis**

The diagnosis of DH may be challenging [2]. It is a diagnosis of exclusion, reached once all other possible explanations for the pain have been ruled out.

A thorough patient history and clinical examination are required. The examination includes a pain provocation test by blasting air from a dental instrument onto the sensitive area, or gentle scratching with a dental probe [10]. If a negative result for the pain provocation test occurs, no treatment for dentinal hypersensitivity is indicated and another diagnosis should be sought, such as other causes of orofacial pain [10].

Inflammation of the dental pulp, termed pulpitis, produces true hypersensitivity of the nerves in the dental pulp [5]. Pulpitis is classified as irreversible when pulpal inflammation will irreversibly progress to pulpal necrosis due to compression of the venous microcirculation and tissue ischemia,
and reversible when the pulp is still capable of returning to a healthy, non-inflamed state, although usually dental treatment is required for this. Irreversible pulpitis is readily distinguishable from DH. There is poorly localized, severe pain which is aggravated by thermal stimuli, and which continues after the stimulus is removed. There also is typically spontaneous pain without any stimulus. Reversible pulpitis may not be so readily distinguishable from DH; however usually there will be some obvious sign such as a carious cavity, crack, etc. which indicates pulpitis. In contrast to pulpitis, the pain of DH is short and sharp.

**Table 1. Kind of intended mechanism of action**

<table>
<thead>
<tr>
<th>Intended mechanism of action</th>
<th>Examples</th>
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<tbody>
<tr>
<td>Nerve desensitization</td>
<td>Potassium nitrate</td>
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<td></td>
<td>Glutaraldehyde</td>
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<td>Silver nitrate</td>
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<td>Protein precipitation</td>
<td>Zinc chloride</td>
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<td>Strontium chloride hexahydrate</td>
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<td>Sodium fluoride</td>
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<td>Stannous fluoride</td>
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<td>Strontium chloride</td>
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<td>Plugging dentinal tubules</td>
<td>Potassium oxalate</td>
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<td>Calcium phosphate</td>
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<td></td>
<td>Calcium carbonate</td>
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<td>Bioactive glasses</td>
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<td>Dentin adhesive sealers</td>
<td>Fluoride varnishes</td>
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<td>Oxalic acid and resin</td>
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<td>Dentin bonding agents</td>
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<td>Lasers</td>
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**In-office treatment**

In-office treatments may be much more complex and they may include the application of dental sealants, having fillings put over the exposed root that is causing the sensitivity, or a recommendation to wear a specially made night guard or retainer if the problems are a result of teeth grinding. Other possible treatments include fluorides are also used because they decrease permeability of dentin in vitro. Also, potassium nitrate can be applied topically in an aqueous solution or an adhesive gel. Oxalate products are also used because they reduce dentin permeability and occlude tubules more consistently. However, while some studies have showed that oxalates reduced sensitivity, others reported that their effects did not differ significantly from those of a placebo. Nowadays, DH treatments use adhesives, which include varnishes, bonding agents and restorative materials because these materials offer improved desensitization [11].

Low-output lasers are also suggested for DH, including GaAlAs lasers and Nd:YAG laser [7]. They are thought to act by producing a transient reduction in action potential in C-fibers in the pulp, but Aδ-fibers are not affected [7] (Figure 2).
At-home treatments

At-home treatments include desensitizing toothpastes or dentifrices, potassium salts, mouthwashes and chewing gums.

A variety of toothpastes are marketed for DH, including compounds such as strontium chloride, strontium acetate, arginine, calcium carbonate, hydroxyapatite and calcium sodium phosphosilicate [1]. Desensitizing chewing gums and mouthwashes are also marketed [5].

Potassium-containing toothpastes are common; however, the mechanism by which they may reduce hypersensitivity is unclear. Animal research has demonstrated that potassium ions placed in deep dentin cavities cause nerve depolarization and prevent re-polarization. It is not known if this effect would occur with the twice-daily, transient and small increase in potassium ions in saliva that brushing with potassium-containing toothpaste creates. In individuals with DH associated with exposed root surfaces, brushing twice daily with toothpaste containing 5% potassium nitrate for six to eight weeks reduces reported sensitivity to tactile, thermal and air blast stimuli. However, meta analysis reported that these individuals’ subjective report of sensitivity did not significantly change after six to eight weeks of using the potassium nitrate toothpaste [9].

Since 2000, several trials have shown that potassium-containing toothpastes can be effective in reducing DH, although rinsing the mouth after brushing may reduce their efficacy [11].

Studies have found that mouthwashes containing potassium salts and fluorides can reduce dentine hypersensitivity, although rarely to any significant degree [11]. As of 2006, no controlled study of the effects of chewing gum containing potassium chloride has been made, although it has been reported as significantly reducing dentine hypersensitivity [11].

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

References