Dentine hypersensitivity

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Introduction

Dentine hypersensitivity (DH) is defined as a 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 dental defect or pathology.

The main symptoms of dentine hypersensitivity (D.H) is a sharp pain of rapid onset & short duration provoked by different stimuli & usually resolves immediately after withdrawal of the stimulus. In more severe, long-standing cases, shorter or longer periods of lingering, dull or aching pain symptoms may be provoked.

The prevalence, distribution, and appearance of the disease have been reported differently in different studies. Studies in the adult population have been reported that DH could affect as many as 1 in 7 of patients attending for dental treatment. DH could affect any age group; however, it is more prevalent in the patient with the age range of 30-40 (third decay) and more prevalent in female individuals.
Regarding the type of teeth involved, canines and premolars of both the arches are the most affected teeth. Buccal aspect of cervical area is the commonly affected site.

DH can be triggered by various stimuli:

• Cold in the most common stimuli such as atmospheric air during mouth breathing, particularly in winter.
• Chemical stimuli such as acidic foods (mainly fruit), sweets and rarely with salty foods
• Mechanical stimulus; example, when the patient rubs the sensitive area with a fingernail, tooth brushing, or the air of a triple syringe by dehydration.

Etiology

Dentin is covered and protected by hard tissues such as enamel or cementum. Dentin itself is a vital tissue, consisting of dentinal tubules, and is naturally sensitive because of extensions of odontoblasts and formation of dentine–pulp complex.

Exposure of dentin could be either to removal of enamel covering the crown of the tooth or denudation of the root surface by loss of cementum & overlying periodontal tissues.

1. Enamel loss may result from (Fig 1)

a. Attrition relating to occlusal abnormalities.
b. Abrasion.
c. Erosion ..

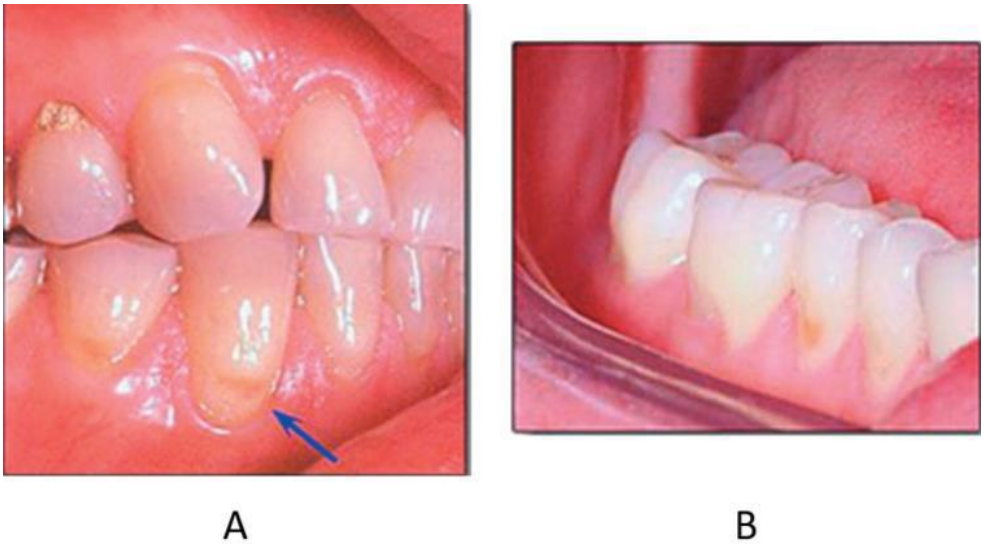

Figure 1: (A) Tooth abrasion, (B) tooth erosion

d. Habits as grasping things between teeth.

2. Cementum loss could be due to:

a. Gingival recession :

Gingival Recession may be caused by:

• Mechanical trauma: hard brush, vigorous technique .Predisposing anatomic factors is thin gingiva , Prominent roots ,Dehiscences ,Fenestrations
• Faulty crown or restoration margins
• Periodontal disease

• Occlusal trauma
• Trauma from teeth in opposing jaw
• Oral habits (tobacco smoking & chewing)
• Poorly designed partial dentures
• Tooth position
• Healing response following periodontal surgery

b. Chronic periodontal disease

As the root surface may become exposed as part of the disease process & the overlying cementum layer is thin & more easily removed.

c. Physiological causes. The increase in the number of teeth with root exposure is evident, as age advances.

d. Anatomic variations: failure of meeting between enamel and cementum at CEJ.

e. Following periodontal therapy

As scaling and root planing & periodontal surgery.

Scaling & root planing may lead to removal of the thin cementum layer during periodontal scraping & expose the dentinal tubules which induce the hypersensitivity which is transitory it reaches the peak in the first week after treatment & subside or disappear within few weeks.

Also, after periodontal surgery in which large root surface area often exposed so leading to hyper sensitivity. However, occasionally the condition may become a chronic pain problem and may persist for months or years. Patients appear to be especially at risk after periodontal surgery.

The increase in pain intensity after periodontal therapy may have one or both of the following two explanations:

Firstly, the smear layer formed on the root surface by the scaling procedure will be dissolved within a few days. This in turn will increase the hydraulic conductance of the involved dentinal tubules and thus decrease the peripheral resistance to fluid flow across dentin. Thereby pain sensations are more readily evoked.

Secondly, open dentinal tubules serve as pathways for diffusive transport of bacterial elements in the oral cavity to the pulp, which is likely to cause a localized inflammatory pulpal response.

The fact that root dentin hypersensitivity often disappears a few weeks after the scaling procedure is best explained by the development of a natural occlusion of the exposed dentinal tubules by mineral deposits.

Theories of DH

Three main mechanisms of dentin sensitivity are proposed (Fig 2):

A. Direct Innervation (DI) theory

B. Odontoblast Receptor (OR) theory

C. Fluid Movement/Hydrodynamic theory

According to DI theory, nerve endings penetrate dentine and extend to the dentino-enamel junction. 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. 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.

OR theory states that odontoblasts act as receptors by themselves and relay the signal to a nerve terminal. But majority of studies have shown that odontoblasts are matrix forming cells and hence they are not considered to be excitable cells, and no synapses have been demonstrated between odontoblasts and nerve terminals

Brannstrom (1964) has proposed that dentinal pain is due to hydrodynamic mechanism, i.e., fluid force. Scanning electron microscopic (SEM) analysis of “hypersensitive” dentin shows the presence of widely open dentinal tubules. The presence of wide tubules in hypersensitive dentin is consistent with the hydrodynamic theory. This theory is based on the presence and movement of fluid inside 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. This is similar to the activation of nerve fibers surrounding the hair by touching or applying pressure to the hair. It has been noted that stimuli which tend to move the fluid away from the pulp–denitin 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. This theory is the most accepted one related to the explanation of DH.

In general, the “hypersensitive” dentin has more widely open tubules and thin/under calcified smear layer as compared with “non-sensitive” dentine. The wider tubules increase the fluid movement and thus the pain response.

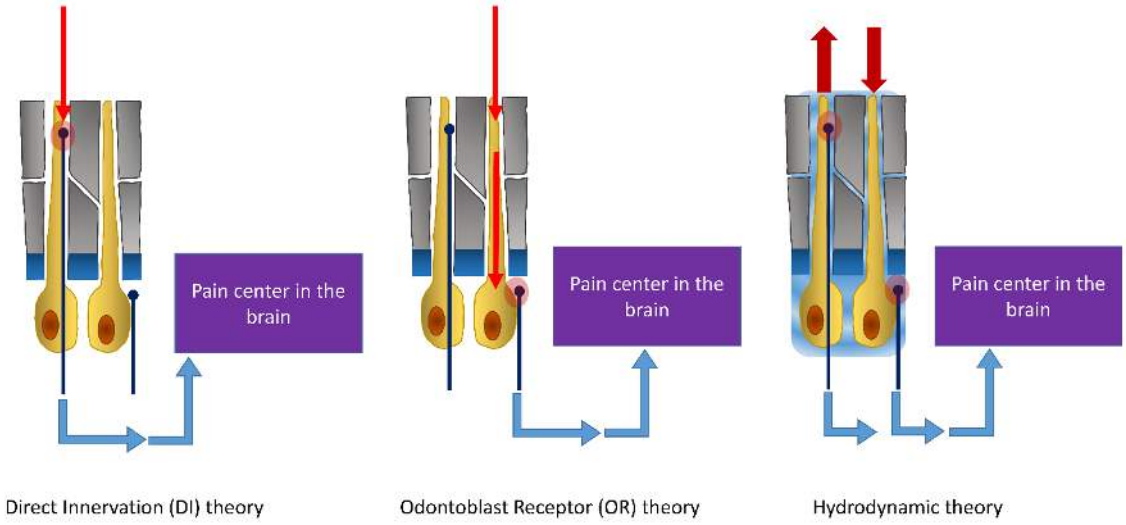

Figure 2: Theories of DH

Diagnosis of DH

Diagnosis of DH starts with a thorough clinical history and examination. The other causes of dental pain should be excluded before a definite diagnosis of DH is made. Some of these techniques include:

• Pain response upon the pressure of tapping teeth (to indicate pulpitis/periodontal involvement)

• Pain on biting a stick (suggests fracture), use of transilluminating light or dyes (to diagnose fractures),

• Pain associated with recent restorations.

A simple clinical method of diagnosing DH includes a jet of air or using an exploratory probe on the exposed dentin, in a mesio-distal direction, examining all the teeth in the area in which the patient complains of pain.

Treatment of DH

Spontaneous cure may occur by the natural remineralization process in the mouth, which promotes natural tubular occlusion of dentin, and pain may return because of the smear layer removal by food and acidic drinks thus explaining the cyclic characteristic of DH.

After observing the severity and number of teeth involved, an active approach to DH can begin in the cases of generalized DH, by a home method followed by in-office treatment when the first option is not successful.

This principle of treatment is based on using different types of desensitizing agents that either occlude or reduce the diameter of dentinal tubules, which minimize the fluid movement in these tubules, and hence reducing the pain. In severe cases, where no remedy is achieved with any advice or treatment approach, pulpectomy and root filling or even extraction may be the last resort.

Hypersensitivity may prevent proper plaque control, therefore treatment of hypersensitivity with plaque control measures may be created. Plaque control is an important integral part of the prevention and treatment of root dentin hypersensitivity. It has been observed clinically that, with time, teeth in patients with excellent oral hygiene habits develop hard, smooth and insensitive root surfaces. However, when severe symptoms of root hypersensitivity have emerged it is difficult to motivate the patient to maintain the degree of plaque control that is necessary to allow for a natural occlusion of the dentinal tubules. In such situations desensitizing agent may be beneficial which has a reasonable capacity to block the tubular openings, at least temporarily, so that proper oral hygiene measures can be reinforced.

Classification of desensitizing agents

1- Mode of administration :

A- At home desensitizing therapy

These “at home” desensitizing agents include toothpastes, mouthwashes and chewing gums. Toothpastes are widely indicated, particularly because of their low cost, ease of use and home application. They are effective but it often takes four to eight weeks to achieve pain relief in addition to the need of patient’s compliance.

B- In-office desensitizing agents

Dental professionals can deliver a wider range of more complex and more potent desensitizing treatment with immediate relief from pain of DH. A variety of office applied agents are currently available, which include cavity varnishes, calcium compounds, oxalates, resins and adhesives, restorative materials, laser treatment and an aqueous solution of glutaraldehyde and hydroxyethyl methacrylate.

2- On the basis of mechanism of action :

1. Nerve desensitization, e.g. Potassium nitrate
2. Anti-inflammatory agents, e.g. Corticosteroids
3. Cover or plugging dentinal tubules

a. Plugging (sclerosing) dentinal tubules

i- Ions/salts, e.g. Sodium monofluorophosphate, Stannous fluoride
ii- Protein precipitants, e.g. Silver nitrate
b. Dentine sealers, e.g. Glass ionomer cements, Varnishes
c. Periodontal soft tissue grafting
d. Crown placement/restorative material
e. Lasers

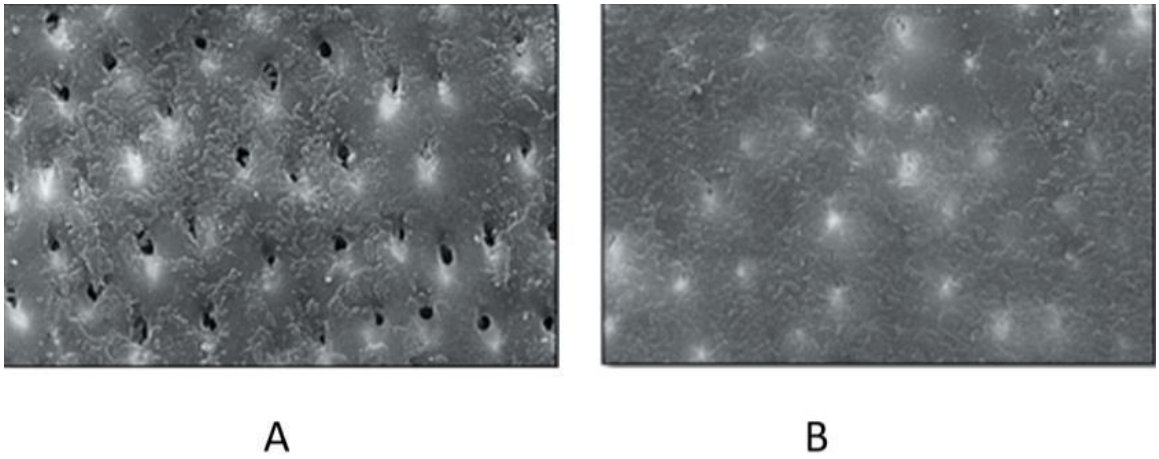

Figure 3: Open tubules (A), (B) Closed tubules following treatment with SnF2 dentifrice.

Dentinal hypersensitivity is a relatively common and significant dental problem which can be successfully managed by a very wide variety of procedures, agents and formulations applied
locally, either “in office” or “at home”. It is clear that some products appear to be more effective than others.