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Review Article

Treatment of Dental Hypersensitivity

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Abstract

Background: Dentin hypersensitivity (DH) is a short sharply painful reaction of the exposed and innervated pulp–dentin complex in response to stimuli being typically thermal, evaporative, tactile, osmotic, or chemical which reaction cannot be attributed to any dental defect or pathology. Exposed Dentin and tubules patent to both the oral cavity and the pulp. Clinical examination for DH would include a pain provocation test by a tactile stimulus, an evaporative air stimulus, or a cold stimulus. A number of dentalconditions can give rise to pain symptoms, which may mimic those of DH. Therefore, careful examination is necessary to exclude the conditions, which need different treatment options. When the patients do suffer from DH, there is a broad range of treatment options comprising home-use and professional approaches. It is advised to start with the less invasive home-use therapies and only expand to professional in office treatments when the home-use treatments are not effective. Most DH therapy work by either nerve desensitization or occlusion of exposed dental tubules.

Keywords: Sensitive teeth; Dentin hypersensitivity treatment; Dentin hypersensitivity diagnosis; Dentin tubule occlusion

Introduction

Teeth hypersensitivity is an exaggerated response to a sensory stimulus that usually causes no response in normal healthy teeth. As a source of chronic irritation, teeth hypersensitivity affects eating, drinking, and breathing. Hypersensitive teeth is characterized by transient pain in response to evaporative, tactile, thermal, electrical or chemo-osmotic stimulation of exposed dentin in teeth where no other defects or pathology exist [1].

Dentin hypersensitivity is one of the most common problems in dentistry, the management of dentin hypersensitivity always presented a challenge for clinicians [2]. The most common age of onset is between 30 and 40 but women experience a higher incidence of dentin hypersensitivity at a younger age than men [3].

It has been proposed that dentin hypersensitivity develops in two phases. First, lesion localization occurs by exposure of dentin, either by loss of enamel or by gingival recession. Gingival recession is the more important of these two factors. Normal tooth brushing will not remove enamel, but it has been cited in the aetiology of gingival recession [4].

Second, Lesion initiation. Not all exposed dentin is sensitive. The localized dentin hypersensitivity lesion. This occurs when the smear layer or tubular plugs are removed, which opens the outer ends of the dentinal tubules [5].

Etiology

Preoperative etiological factors

Bacterial: Dental caries produces different levels of teeth hypersensitivity that is mainly related to the depth of decay, dentin conductance and Pain threshold of the patient himself involved [6]. Greater degree of sensitivity happens when dental caries passes the DEJ. As caries penetrates further into the tooth, sensitivity lessens until pulp becomes involved [7].

Deeper in dentin and near the pulp, the number of dentinal tubules is higher, the bigger the diameter of the dentinal tubules, the shorter their length, the higher the permeability of the dentinal fluids and consequently the higher the degree of hypersensitivity [8].

Erosion: It is defined as the dissolution of teeth by acids which are not of bacterial origin. When an acid or an osmotic agent like sugar adhere to the margins of leaky restoration or exposed dentin that will affect the flow of dentinl fluid and result in hypersensitivity [9].

Extrinsic erosion results of exposure to extrinsic food, fluid or agents, such as citrus fruits, pickled food, fruit juice, carbonated drinks, wines, ciders, vitamin C, some mouth rinses with low PH and bleaching agents especially those delivered in a vacuum formed trays for home applications [10].

Intrinsic erosion may result from gastric reflux as in Hiatus hernia, alcoholism, eating disorders like bulimia nervosa [11].

Mechanical

I. Attrition

is defined as wear of teeth at sites of direct contact between teeth. Attrition is associated with occlusal function and can be aggravated by habits or parafunctional activities which is known as bruxism [12].

II. Bruxism

The aetiology of bruxism is unknown but it could be associated with:

A. Sleep disorders as obstructive sleep apnea and Snoring.

B. Malocclusion

C. High consumption of alcohol and heavy smooking

D. Stress, digestive problems.

E. Disorders as Huntington and parkinson's diseases

F. Drugs as: MDMA, cocaine

The bruxism results in hypersensitivity to heat and cold, fractured teeth and fillings, musculofacial pain and headache, stiffness and pain in the joints [13].

III. Abrasion

It is defined as the wear of teeth caused by objects other than other teeth such as tooth brush/toothpaste abrasion, scaling and root planning and pipe smoking [14].

IV. Abfraction

It is defined as the wear of teeth at the cervical portion as a result of occlusal loading that leads to cuspal flexture, this in turn results in compressive and tensile stresses at the cervical fulcrum area of the teeth with the resultant weakness and gradual loss of the cervical portion [15].

V. Cracked tooth syndrome

is defined as incomplete fracture of the vital teeth, can be involved in dentin only or extending to the pulp, Cracked tooth syndrome resulted in Teeth hypersensitivity with biting relieved with releasing the bite. Might involve severe spontaneous pain in case of pulp involvement. Can happen in sound teeth especially upper premolars, or most commonly in teeth that is restored with big restoration or direct gold [16].

VI. Thermal and idiopathic

which can result in reversible hypersensitivity that subside by treating the exposed dentin and preventing the cause, and irreversible pulpal damage [17].

VII. Postoperative (Iatrogenic)

a) Factors related to cavity preparation

b) Type of cutting instruments

Rotary instruments produce more heat generation than the hand instruments. Dull instruments might require higher pressure of cutting which will result in more heat generation. Heat can destruct pulpal tissue, coagulate protoplasm and even burn dentin. Proper cooling is mandatory with all rotary instrument [18].

C) Vibration

Cause a rebound response as a result of using eccentric burs, which can result in necrotizing effects on dentin.

D) Dentin Desiccation

Can result from heating of dentin during cutting, use of chemicals to sterile the cavity or use of air as a coolant for final cavity toilet.

Factors related to restorative phase and restorations

A. Polymerization shrinkage

The polymerization shrinkage results in stresses at the composite/tooth interface resulting in microleakage, microcracks or deformation of tooth structure [19]. Microleakage can result in secondary caries formation and the consequent teeth hypersensitivity. Stresses are greatest in cavities with high ratio of C factor (ratio of bonded surfaces to unbonded surfaces), decreasing C factor will result in decreasing stresses from polymerization shrinkage [20].

B. Undercured resin

Can result from a light source of inadequate intensity, or not close enough to the rein, or alight which is attenuated by passage through tooth structure or restoration. That results in a well cured surface covering incompletely cured layer, which will result in marginal fracture, open margin and chemical toxicity from the monomers or the bonding agent [21].

C. Microleakage

Any restoration though exhibits clinical satisfactory adaptation, shows some leakage. The ingress of fluids and microorganisms can be the cause of dentinal hypersensitivity in addition to the fluid movements within the dentinal tubules [22].

D. Cracked tooth

Pain on biting and eating citrus fruits, this sharp pain will disappear when pressure is released. Commonly happens in teeth

with large restoration, direct gold filling with excessive condensation forces and cast restoration without proper consideration for cusp protection [23].

E. Galvanism

When two dissimilar metallic restorations brought into contact the current will pass between them and a galvanic stimuli will be generated. Hypersensitivity is usually felt in the tooth containing the restorative with the lower potential in Amalgam [24].

Theories for Dentin Hypersensitivity

Odontoblastic transduction theory

According to this theory, odontoblastic processes are exposed on the dentin surface and can be excited by a variety of chemical and mechanical stimuli. As a result such stimulation neurotransmitters are released and impulses are transmitted towards the nerve endings.

Neural theory

It is an extension of the Odontoblastic transduction theory, this concept advocates that thermal or mechanical stimuli directly affect nerve endings within the dentinal tubules through direct communication with puipal nerve fibers. This theory is supported by the observation of presence of unmediated nerve fibers in the outer layer of the root dentin and the presence of putative neurogenic polypeptides. This theory is considered with no solid evidence to support it.

Hydrodynamic theory

It is the most accepted theory. This theory postulates that fluids within the dentinal tubules are disturbed by thermal, physical or osmotic changes. These fluid movements stimulate a baroreceptor which leads to neural discharge. The basis of this theory is the fact that the fluid filled dentinal tubules are open to the oral cavity at the dentin surface as well as within the pulp. The hydrodynamic theory postulates that perception of hypersensitivity occurs as a response to low intensity stimulation of afferent A delta nerve ends by sudden inward-outward movement of tubular fluid on application of non-noxious stimuli to exposed virgin dentin surface. These nerve fibers interweave with the odontoblasts and the dentinal tubules along a distance of 100 micrometer from the pulp surface. They are mylinated have relatively low stimulation threshold, and are poly modal. They serve as mechanoreceptors for low intensity non-noxious stimuli.

According to this theory, the hypersensitivity is a neural response of the mechano-receptor afferent nerve fibers to stimuli that are triggered at exposed dentin surface and are transduced through fluid-filled dentinal tubules. Consequently, there must be exposed, moist and vital dentin with patent dentinal tubules for such stimuli to be conducted to the mechanoreceptors and provoke the perception of hypersensitivity. The more the conductance of dentin the more the perception of the hypersensitivity in response to stimuli.

In the activation of the mechano-receptors the rate of change of the stimulus is more effective than its absolute value, so, suddenly applied or released pressure activates the A delta mechano-receptors causing perception of dentin hypersensitivity while steadily applied pressure does not cause that characteristic response, when it reaches high noxious values it causes tissue damage and evoke the C-fibers with perception of dull pain.

Management of dentin Hypersensitivity

Management of the etiological factors

It is important to identify these factors so that prevention can be included in the treatment plan before starting the Active management of Dentin hypersensitivity which usually involve a combination of home care and in-office therapies [25].

Home care

Desensitizing toothpastes/dentifrices: Tooth-pastes 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). Use of standardized fluoridated dentifrice can abrades the dentin and leads to hypersensitivity. Some reports indicate that some individuals experience hypersensitivity as a result of using tartar control dentifrices, possibly due to pyrophosphate content. Now, most desensitizing toothpastes contain a potassium salt such as potassium nitrate, potassium chloride or potassium citrate, though one study [26] reported that a remineralizing toothpaste containing sodium fluoride and calcium phosphates reduced Dentine hypersensitivity.

Potassium salts: pastes containing potassium chloride or potassium citrate have been made available. Potassium ions are thought to diffuse along dentinal tubules and decrease the excitability of intradental nerves by altering their membrane potential [27]. The efficacy of potassium nitrate to reduce Dentine hypersensitivity. Several trial results of potassium-containing toothpastes have been published. Some of these studies compared different tooth-paste formulations. For instance, six studies [28].

Toothbrush and toothpaste application: Use of a soft or ultra-soft manual toothbrush with soft end rounded bristles lowers the risk of gingival recession and abrasion of exposed cementum and dentin. With powered toothbrush less pressure is required on the teeth, they require a light grasp to remove plaque [29]. Dentifrices should be applied by toothbrushing. 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 [30].

Mouthwashes and chewing gums: Studies have found that mouthwashes containing potassium nitrate and sodium fluoride, [31] can reduce Dentine hypersensitivity. 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 [32].

Dietary Modifications: Controlling the consumption of acidic food and drinks such as citrus fruits, wine, pickled foods and carbonated beverages. Avoiding brushing immediately after ingestion of acidic food, as it may accelerate the combined effect of abrasion and erosion, rinsing with water is recommended before brushing [32].

In-office professional treatment

Fluoride: Fluorides such as sodium fluoride and stannous fluoride can reduce dentin sensitivity. Fluorides decrease the permeability of dentin in vitro, possibly by precipitation of insoluble calcium fluoride within the tubules [33].

Ionto-phoresis: This procedure uses electricity to enhance diffusion of ions into the tissues. Dental iontophoresis is used most often in conjunction with fluoride pastes or solutions and reportedly reduces Dentin hypersensitivity [34].

Potassium nitrate: the use of a custom-made soft tray filled with a dentifrice containing potassium nitrate. The tray delivery system increases the medicament-tooth contact time, thus increasing the effectiveness of the potassium nitrate [35].

Oxalates: 30 percent potassium oxalate caused a 98 percent reduction in dentin permeability in vitro. Since then, numerous oxalate-based desensitizing products have become available [36].

Calcium phosphates: Calcium phosphates may reduce dentin sensitivity effectively. Calcium phosphates occlude dentinal tubules in vitro and decrease in vitro dentin permeability [37].

NovaMin: is the brand name of a particulate bioactive glass that is used in dental care products. It consists of 45% SiO_2, 24.5% Na_2O, 24.5% CaO and 6% P_2O_5. it delivers an ionic form of calcium, phosphorus, silica, and sodium which are necessary for bone and tooth mineralization. When microscopic particles of NovaMin are exposed to water, they release mineral ions that become available for the natural remineralization process [38].

Propolis: a natural resinous substance collected by bees, it has antibacterial agent (tt-farnesol) and inhibitor of glycosyl transferase (apigen), it has a positive effect in the control of dentinal hypersensitivity. It was found that Propolis occluded the dentinal tubules in periodontally involved and recession teeth [39].

Casein Phosphopeptides: It is a water based topical cream, sugar free with bioavailable calcium and phosphate, in the form of CPP-ACP (casein phosphopeptides- amorphous calcium phosphates. Recent studies reported that it provides extra teeth protection and neutralize acids from acidogenic bacteria and from other external and internal acid sources [40].

Lasers: The effectiveness of lasers for treating dentine hypersensitivity varies from 5 to 100% yttrium-aluminum-garnet (YAG) laser, the erbium: YAG laser and galium-aluminium-arsenide low level laser all reduce Dentin hypersensitivity [41].

Conclusion

Dentinal hypersensitivity is a chronic and challenging condition. Therefore, a comprehensive assessment is essential as is appropriate treatment to decrease the dentinal flow and relieve discomfort. Professional treatment options along with the use of a desensitizing dentifrice should be considered before performing operative and whitening procedures and periodontal therapy that may result in discomfort due to hypersensitivity. A desensitizing product should be used during the treatment and continued during the post-treatment period to decrease patient discomfort with the sensitivity. This will increase patient compliance during treatment, resulting in enhanced outcomes without discomfort to the patient.

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