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Dentine Hypersensitivity - A Review of Mechanisms and Treatment Alternatives

Dr. Rajul Vivek[1], Dr. Romesh Soni[2], Dr. T.P. Chaturvedi[3], Dr. Atul Bhatnagar[4],

Ph.D Research Scholar, Department of Prosthodontics, Faculty of Dental Sciences, IMS, BHU
Assistant Professor, Department of Prosthodontics, Faculty of Dental Sciences, IMS, BHU
Professor & Head, Department of Orthodontics, Faculty Of Dental Sciences, IMS, BHU
Associate Professor, Department of Prosthodontics, Faculty of Dental Sciences, IMS, BHU

I. Introduction

Dentin hypersensitivity following tooth preparation is a frequently encountered oral health problem. Dentin hypersensitivity is a "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."[1]

The terms DS or DH have been used interchangeably to describe the same clinical condition. True hypersensitivity can develop due to pulpal inflammation and can present the clinical features of irreversible pulpitis, i.e., severe and persistent pain, as compared with typical short sharp pain of DH.[2] Women are more frequently affected, and at a younger mean age. [3] Dentin hypersensitivity is most commonly observed in the buccal-cervical regions of the canine and pre-molar teeth, sites, which are also most susceptible to gingival recession.[4] This review will address the etiology of the condition commonly referred to as "dentinal hypersensitivity" or "tooth sensitivity."

Prevalence

Dentinal hypersensitivity is generally reported by the patient after experiencing a sharp pain caused by one of several different stimuli. Dentinal hypersensitivity has been shown to peak in 20 to 30 year olds and then rise again when in their 50's.[5, 6] The condition generally involves the facial surfaces of teeth near the cervical aspect and is very common in premolars and canines.[7] Patients undergoing periodontal treatment are particularly susceptible to this condition because of the recession following periodontal surgery or loss of cementum following non-surgical periodontal therapy.[8]

In addition periodontal disease and improper brushing habits can also result in gingival recession accompanied by sensitive teeth.

Theories of dentin hypersensivity

Several theories have been cited to explain the mechanism involved in dentinal hypersensitivity.[9] The transducer theory, the modulation theory, the "gate" control and vibration theory, and the hydrodynamic theory have all been presented and discussed throughout the years. The latter, "hydrodynamic theory", developed in the 1960's and based upon two decades of research, is widely accepted as the cause of tooth sensitivity.[10] Assumptions of the hydrodynamic theory conclude that when the fluids within the dentinal tubules are subjected to temperature changes or physical osmotic changes, the movement stimulates a nerve receptor sensitive to pressure, which leads to the transmission of the stimuli.

Based on the hydrodynamic theory, dentinal hypersensitivity is a transient tooth pain. The disease is characterized by a short, sharp pain arising from exposed dentin in response to a stimulus that cannot be ascribed to any other form of dental defect or pathology.[11,12] Therefore, in order to exhibit a response to the stimuli, the tubules would have to be open at the dentin surface as well as the pulpal surface of the tooth.

The most important variable affecting the fluid flow in dentin is the radius of the tubuli. If the radius is reduced by one-half, the fluid flow within the tubuli falls to one-sixteenth of its original rate. Consequently, the creation of a smear layer or obliteration of the tubule can greatly increase the effectiveness of the treatment of this malady.[13, 14]

Diagnosis

As like any other clinical condition, an accurate diagnosis is important before starting the management of DH. DH has features which are similar to other conditions like caries, fractured or chipped enamel/dentine, pain due to reversible pulpitis, and post dental bleaching sensitivity.[15, 16] 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), and pain associated with recent restorations.[17] 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.[18] The severity or degree of pain can be quantified either according to categorical scale (i.e., slight, moderate or severe pain) or using a visual analogue scale.[19]

Treatment

Treating dentinal hypersensitivity can be challenging for the dental professional because of the difficulty related to measuring the pain response since the response varies from patient to patient. In addition if the dentin exposure is due to personal habits, it may be difficult for patients to change their behavior(s). If the diagnosis confirms dentinal hypersensitivity in the absence of underlying diseases or structural problems, then the following steps can be initiated: (1) remove the risk factors by educating the patient about dietary acids and other oral care habits; (2) recommend different toothbrushing methods, if appropriate; (3) initiate treatment by recommending a desensitizing agent for home use; or (4) applying topical desensitizing agents professionally.

Treatment can be invasive in nature or noninvasive. Invasive procedures may include gingival surgery, application of resins, or a pulpectomy Non-invasive treatment options are topical agents and dentifrices that contain a desensitizing active ingredient. These are considered to be the simplest, cost-effective, and efficacious first line of treatment for most patients.[20] According to the literature, the most widely available desensitizing toothpaste ingredient is potassium nitrate.[21] The potassium ions are thought to block the synapse between nerve cells, reducing nerve excitation and the associated pain. Another active ingredient that exhibits a similar mechanism is potassium chloride.

Other active agents that have been proven to be effective as a desensitizing agent are dentin sealers (resins), sodium citrate, and sodium monofluorophosphate.

Method of Action

In situ research shows root dentin treated with stannous fluoride exhibits tubule occlusion.[22]Several other studies using analysis by scanning electron microscopy showed that partial or complete occlusion of dentin tubules occurred after treatment with SNF2. In reported a tin-rich surface deposit forms in vitro and in situ with two weeks use of an anhydrous 0.4% stannous fluoride gel, providing nearly complete surface coverage and occlusion of the tubules. When the tubules are blocked, the stimulation of the mechanoreceptors does not occur, thus, preventing the pain response.

II. Conclusions

Dentinal hypersensitivity is a problem that plagues many dental patients. When a patient presents with dentinal hypersensitivity symptoms, they should be examined and informed of the multiple treatment options that may be necessary to eliminate the problem. The patient should be responsible for the decision making process since some of their daily habits may be contributing to the problem and if not changed the condition will persist.

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