REVIEW ARTICLE

SENSE OF SENSITIVITY : A CLOSER LOOK AT DENTIN HYPERSENSITIVITY

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ABSTRACT: Dentin hypersensitivity (DH) is a common oral health problem affecting one or more teeth of many adult individuals on a global basis.
There is a growing awareness that dentin hypersensitivity is an increasingly important issue to be addressed, both from a diagnostic and a problem-management perspective, as caries prevention and periodontal disease management measures become increasingly successful, resulting in improved oral health status and functionality of the dentition throughout life.

DEFINITION

The current definition given by Holland and colleagues[1], states "Dentin hypersensitivity is 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 disease." There is some controversy regarding exactly what constitutes DH. The European Federation of Periodontology uses the term "root sensitivity" (RS) to describe the DH-like sensitivity seen as a result of periodontal disease or treatment.
It is conceivable that there may be a difference between long-standing DH in a patient with incipient recession and very good hygiene and that RS seen in a periodontal patient immediately following flap surgery[2].

PREVALENCE

The prevalence of DH in the adult population ranges between 8% and 35%[3].The peak prevalence of DH has been reported between the second and fifth decades of life. Orchardson and Collins reported a peak between 20 and 25 years, whereas Addy found peak prevalence to occur at the end of the third decade.
Fischer and colleagues found it to be between 40 and 49 years[2].

ETIOLOGY

There are many varieties of potential causes for dentin sensitivity. There is no principal cause.
The loss of enamel and removal of cementum from the root with exposure of dentin, however, is a major contributing factor.
In a normal tooth, dentin is covered in the crown by enamel and, in most areas of the root, by a thin layer of cementum. Each tooth contains many thousands of dentinal tubules, which are microscopic tubular structures that radiate outward from the pulp. These dentinal tubules are 0.52.0 µ in diameter and are connected to the pulp by the dentinal fluid For dentin hypersensitivity to occur, dentin must become exposed (a process termed "lesion localization") and dentin tubules must be opened and patent to the pulp (a process termed "lesion initiation").These two processes are multi-factorial[4].
DH is provoked when exposed dentin is subjected to stimuli such as osmotic changes, thermal changes, or mechanical stimuli such as toothbrushing.Loss of enamel or tooth structure occurs by attrition, abrasion, erosion ,abfraction, trauma whereas denudation of the root surface can occur as a result of gingival recession, periodontal therapy, or improper tooth brushing. Scaling and root planing may cause increased DH because they remove the thin outer layer of cementum in the cervical third of the root, exposing the dentinal tubules. Causes also include gingival recession due to root prominence and thin overlying mucosa, dehiscences and fenestrations, frenum pulls, and orthodontic movement, which causes a root to be moved outside its alveolar housing. Bleaching causes hypersensitivity by the change in osmolarity. These changes occur when bleaching agent penetrates through the enamel and the dentin and reaches the pulp.

MECHANISM

The classic hydrodynamic theory given by Brannstorm is the most accepted explanation of mechanism of dentin hypersensitivity. It suggests that dentin hypersensitivity is a result of movement of fluid within the dentin tubules. Structurally the dentin has over 300,000 dentinal tubules /mm sq. These are filled with the dentinal fluid. In a vital tooth there is a constant outward movement of this fluid. This theory states that whenever exposed dentin is stimulated by tactile chemical thermal or osmotic stimuli there is rapid movement of the fluid either towards or away from the pulp.

Most pain-producing stimuli, in particular the most problematic cold and evaporative stimuli, cause an outflow of dentin fluid. This results in a pressure change across the dentin which activates intra-dental nerve fibers, via a mechanoreceptor response, to cause pain.

In addition, the fluid movement in the tubules can cause an electrical discharge, known as "streaming potential," which may contribute by electrically stimulating a nerve response[4].

In contrast, heat causes a relatively slow retreat of dentin fluid, and the resultant pressure changes activate the nerve fibers in a less dramatic fashion, consistent with the fact that heat is generally a less problematic stimulus than cold.

"Treating the Twinge": TREATMENT OPTIONS

1. Self applied treatment
2. Office applied treatment

Self applied treatment Self applied desensitizing agents The ideal patient-applied desensitizing agent should be nonirritating to the pulp, leave no deposits on tooth surfaces or restorations, and should not be irritating to the soft tissues. To ensure compliance the product should be easily applied, have a rapid onset,produce long-lasting relief, and provide effective relief from DH.

STRONTIUM CHLORIDE

Strontium has been suggested to have several effects on teeth, including a cariostatic effect which is alleged to be most effective in the pre-eruptive phase of tooth formation. Strontium can substitute for calcium in activating secretory mechanisms and can also possibly affect or modulate the pulpal cholinergic and adrenergic mechanisms involved in DH[5].

The manner in which strontium affects DH has not been elucidated, but it has been proposed that the ions occlude dentinal tubules by binding to the tooth substance and stimulating reparative dentin formation. It has also been suggested that strontium ions have the capacity to reduce sensory nerve activity, but less effectively than potassium ions[6].

However, Kishore and colleagues[7] reported that strontium chloride is more effective than potassium nitrate. These workers demonstrated that 10% and 2% strontium chloride significantly reduced DH, while a 5% solution of potassium nitrate did not show a significant reduction in DH.

POTASSIUM

Potassium salts block neural transmission at the pulp and depolarize the nerve around the odontoblasts. Potassium nitrate has been incorporated into both toothpastes and mouthrinses for use as a treatment for DH. Kim reported for the first time that the potassium ion is the active portion of potassium nitrate. Greenhill and Pashley found potassium nitrate ineffective in decreasing dentinal fluid flow in vitro coated dentin, even at 30% concentration. This suggests a lack of effect on fluid flow. Products containing potassium have been studied to evaluate the efficacy of potassium nitrate as a desensitizing agent. Tarbet and colleagues found that a 5% potassium nitrate paste reduced DH effectively at 1 week and up to 4 weeks.

Tarbet and colleagues also compared the abilities of strontium chloride, dibasic sodium citrate, formaldehyde and potassium nitrate to desensitize hypersensitive teeth and reported that 5% potassium nitrate was the most effective in reducing DH[5].

DIBASIC SODIUM CITRATE

In an in vitro study by Greenhill and Pashley,a 19% reduction in dentinal fluid flow was thought to be attributable to dentinal tubule obturation. In a number of doubleblind, placebo-controlled studies, dibasic sodium citrate was significantly superior to a placebo in reducing DH[5].

FLUORIDE-BASED INTERVENTIONS

Clinical trials have shown that some fluoride dentifrices or concentrated fluoride solutions are effective in producing favorable results in the treatment of DH. Tal and colleagues proposed that precipitated fluoride compounds may reduce DH by occlusion of the dentinal tubules. In a 3

month study, Kanouse and Ash reported that subjects using a monofluorophosphate (MFP) dentifrice had a statistically significant increased tolerance to cold and hot in comparison to subjects using a placebo dentifrice. Stannous fluoride has also been proposed as a treatment for DH. Thrash and colleagues noted that topical application of 0.717% aqueous stannous fluoride (SnF2) gave patients immediate relief from DH. Blong and colleagues found that application of 0.4% SnF2 gel was also an effective treatment for DH[5].

In office treatment

CAVITY VARNISH

Brannstrom suggested the application of cavity lining and varnishes under restorations, so that the smear layer plugging open tubules is retained. In a narrative review of DH interventions, Wycoff recommended the use of a copal varnish, since covering exposed dentin with a thin film of varnish often renders it nonhypersensitive. For more sustained relief, a 2% sodium fluoride-containing varnish can be applied. The duration of effect of this varnish is usually 3 months.

Corona and colleagues compared the galium-aluminium-arsenide laser and sodium fluoride varnish (Duraphat) in the treatment of DH. They found no statistically significant difference between fluoride varnish and laser.

CORTICOSTEROIDS

It has been suggested that the application of anti-inflammatory drugs such as glucocorticoids to cavity preparations and exposed dentin may reduce DH by way of their effect on pain mediators. However, there is little experimental evidence to support or refute the use of such agents. In a double-blind study, Lawson and Huff found that paramethasone had a significant desensitizing action. Furseth and Mjor have reported complete obturation of dentinal tubules; hence, reduction of dentin permeability after an application of a corticosteroids preparation to exposed dentin. Mjor reported less pulpal inflammation following restoration with a corticosteroid-containing cement than with amalgam in a nonhuman primate model.

CALCIUM COMPOUNDS

Calcium hydroxide [Ca (OH)2] has been used many years for the treatment of DH, particularly after root planing. Ca (OH)2 has little or no direct effect on dentin sensory nerve activity. However, it is thought that it induces peritubular dentin mineralization and, subsequently, less hypersensitive dentin[5]. Suge and colleagues examined occlusion of dentinal tubules by scanning electron microscopy (SEM) and reported that calcium phosphate precipitation was more effective than potassium oxalate in occluding dentinal tubules. Levin and colleagues found that the application of Ca(OH)2 paste to hypersensitive exposed dentin resulted in an immediate decrease of DH in over 90% of treated teeth. Jorkjend and Tronstad applied a paste of Ca(OH)2 to the exposed root surface following periodontal surgery[5].

OXALATES

Oxalate-containing products are a popular agent for in-office treatment of DH. Oxalate desensitizing agents are easy to apply, safe, relatively inexpensive, and well-tolerated by patients. It has also been shown that potassium oxalate has both dentinal tubule obturation properties and inhibitory effects caused by the potassium ions actions on nerve activity. Oxalate ions react with calcium to form insoluble calcium oxalate crystals that bind tightly to dentin and obturate the dentinal tubules. It has been shown that a 3% monohydrogen monopotassium oxalate releases a high concentration of calcium ions and accelerates crystal formation[5].

RESINS AND ADHESIVES

The rationale for the use of resins and adhesives is to seal the dentinal tubules and hence to preclude the transmission of pain causing stimuli to the pulpal nerve fibers. Brannstrom and colleagues found that impregnating the dentinal tubules with a restorative resin material resulted in significant relief from DH. Wycoff suggested that adhesives be used for severe cases of DH that were unresponsive to other interventions. A combination product consisting of an aqueous solution of 5% glutaraldehyde and 35% hydroxyethyl methacrylate (Gluma Desensitizer) has been reported to be an effective desensitizing agent for up to 9 months. The glutaraldehyde intrinsically blocks dentinal tubules counteracting the hydrodynamic mechanism that leads to DH[5].

LASERS

A recent review of the literature by Kimura and colleagues reported that effectiveness of laser treatment of DH ranged from 5% to 100%. However, these authors also reported that the laser was less effective in cases of severe DH. Moritz and colleagues examined the long-term effects of combined carbon dioxide (CO2) laser treatment and

fluoridation on DH. He found that 97% of laser group showed complete relief from DH compared with conventional fluoridation. Furthermore, SEM examination at 18 months post treatment showed complete closure of dentinal tubules[5].

Zhang and colleagues studied the effect of irradiated teeth by CO2 laser on the pulp and in the treatment of DH. He found that all patients were free of DH immediately following laser treatment. Over 3 months, the CO_2 laser treatment reduced DH to air stimulus by 50% without obvious loss of pulpal vitality[8]. Lan and Liu reported that the neodymium: yttrium-aluminum-garnet (Nd:YAG) laser reduced DH to air by 65% and to tactile stimulation by 77%. A combination of dental laser with dentin bonding agents has been reported to be an efficient method for improving dentin bonding.

GINGIVAL AUGMENTATION

Gingival reconstructive surgical periodontal procedures, such as root coverage grafts, have been used to treat DH due to gingival recession. Although many investigators have reported on the efficacy of various methods of soft tissue augmentation for root coverage, it was not possible to locate any studies which were designed to specifically test such grafting as a treatment for DH[5].

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