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DENTINAL HYPERSENSITIVITY: A REVIEW ON EFFECTIVE TREATMENT WITH POTASSIUM NITRATE CONTAINING DENTIFRICE

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ABSTRACT

Dentinal hypersensitivity is a very common dental disease worldwide, which can cause various problems to patients. The management of this condition requires a good understanding of the complexity of the problem, as well as the variety of treatments available. Various agents are commonly employed like calcium salts, fluorides, ferrous oxide, and strontium chloride etc. This review considers the treatment of dentinal hypersensitivity using the dentifrices containing the nerve desensitizing agent Potassium Nitrate.

KEY WORDS: **Dentin, Hypersensitivity, Potassium Nitrate, Dentifrices, Dentinal Hypersensitivity (DH)**

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INTRODUCTION

Dentin hypersensitivity is best defined as 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] This is one of the most commonly encountered dental problems, and estimates of its prevalence in the adult dentate population range from 8% to 57%.[2, 3]It is very common between 20-30 years people and again show in people over 50 year in age. When thermal, tactile, osmotic and mechanical stimuli, such as tooth brushing, sweet and sour foods, and hot or cold beverages/water, are applied to the exposed dentin, patients feel a short sharp pain which is termed dentin hypersensitivity.[4]Dentine may become exposed via several means. For example, the enamel or cementum which normally covers the dentine surface may be removed or denuded as a result of attrition, abrasion or erosion. Alternatively, in some individuals the cementum and enamel which normally cover the dentine do not meet and result in dentine exposure as a result of a developmental anomaly. In general, it appears that dentinal hypersensitivity is rarely a result of just one of the above factors, but rather a combination of more than one factor. Regardless of the aetiology of dentine exposure, one feature appears to be in common and that is open dentinal tubules which provide a direct link between the external environment and the internal pulp of the tooth. If the tubules are not exposed it seems unlikely that hypersensitivity will be found. In areas of sensitive dentine the apertures of the dentine tubules are patent and these results in more stimuli having closer contact with the dental pulp.[5] 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.[6]

THEORIES BEHIND HYPERSENSITIVITY

Odontoblastic transduction theory

According to this theory, odontoblastic processes are exposed on the dentine surface and can be excited by a variety of chemical and mechanical stimuli[7, 8]. As a result of such stimulation neurotransmitters are released and impulses are transmitted towards the nerve endings. To date no neurotransmitters have been found to be produced or released by odontoblastic processes.

Neural theory

As an extension of the odontoblastic theory, this concept advocates that thermal, or mechanical stimuli, directly affect nerve endings within the dentinal tubules through direct communication with pulpal nerve fibers.While this theory has been supported by the observation of the presence of unmyelinated nerve fibers in the outer layer of root dentine[9] and the presence of putative neurogenic polypeptides[10], this theory is still considered theoretical with little solid evidence to support it.

Hydrodynamic theory

By far the most widely accepted theory for dentinal hypersensitivity is the hydrodynamic theory proposed by Brannstrom and co-workers[11,12].This theory postulates that fluids within the dentinal tubules are disturbed either by temperature, physical or osmotic changes and that these fluid changes or movements stimulate a baroreceptor which leads to neural discharge. The basis of this theory is that the fluid filled dentinal tubules are open to the oral cavity at the dentine surface as well as within the pulp.

ANATOMY OF DENTINE PULP COMPLEX

Dentine 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.[13] Although dentin and

pulp are histologically different, their origin is embryologically from the same precursor, i.e., the ectomesenchyme.[13] Pulp is integrally connected to dentine, i.e., physiologic and/or pathologic reactions in one of the tissues will also affect the other. Dentin consists of small canal like spaces, dentinal tubules. These tubules occupied by odontoblastic processes.[13] The odontoblastic processes may extend through the entire thickness of dentin from pulp to dentino-enamel junction. The odontoblastic processes are actually the extensions of odontoblasts, which are the major cells of pulp-dentin complex.[13] Theodontoblastic processes are surrounded by dentinal fluid inside the tubules. The dentinal fluid forms around 22% of total volume of dentin.[13] It is an ultrafiltrate of blood from the pulp via dentinal tubules and forms a communication medium between the pulp (via the odontoblastic layer) and outer regions of the dentin.

PATHOGENESIS

DH develops in two phases: lesion localization and lesion initiation.[14] Lesion localization occurs by loss of protective covering over the dentin, thereby exposing it to the external environment. It includes the loss of enamel via attrition, abrasion, erosion or abfraction. Another cause for lesion localization is gingival recession which can be due to toothbrush abrasion, pocket reduction surgery, tooth preparation for crown, excessive flossing or secondary to periodontal diseases.[15] Not all exposed dentine is sensitive. For DH to occur, the lesion localization has to be initiated. It occurs after the protective covering of smear layer is removed, leading to exposure and opening of dentinal tubules.

CLINICAL SIGNIFICANCE OF HYPERSENSITIVITY

Dentinal hypersensitivity, while neither life threatening nor a serious dental problem, can be a particularly uncomfortable and unpleasant sensation for patients and can dictate types of foods and drinks ingested. Patients may describe the condition as dull or sharp, vague or specific and intermittent or constant. Teeth causing such symptoms are rarely considered to be as seriously affected as those affected by caries, endodontic problems or periodontal disease, however, the condition is nonetheless of sufficient concern to warrant appropriate and proper management. In most instances the condition can be managed by patients through appropriate home care using properly prescribed over-the-counter products. These features of the condition negate, in the majority of cases, the need for expensive and lengthy professional care. Notwithstanding the above, the condition still requires an appropriate differential diagnosis since carious exposureofdentine surfaces, inflamed pulps or cracked cusps can produce symptoms similar to cervical dentinal hypersensitivity.[16]

CLINICAL MANAGEMENT OF DH

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.[6, 17] 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 trans-illuminating light or dyes (to diagnose fractures), and pain associated with recent restorations.[18] 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.[19] 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.[15]

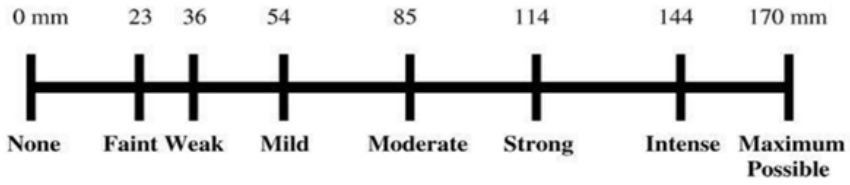

Figure 1
Heft Parker visual analogue scale[20]

TREATMENT STRATEGIES FOR DENTINAL HYPERSENSITIVITY[5]

1. Nerve desensitization
   Potassium Nitrate
2. Anti-inflammatory agents
   Corticosteroids
3. Cover or plugging dentinal tubules
   a. Plugging (sclerosing) dentinal tubules
   - Ions/salts
     - ♦ Calcium hydroxide
     - ♦ Ferrous oxide
     - ♦ Potassium oxalate
     - ♦ Sodium monofluorophosphate
     - ♦ Sodium fluoride
     - ♦ Sodium fluoride stannous fluoride combination
     - ♦ Stannous fluoride
     - ♦ Strontium chloride
   - Protein precipitants
     - ♦ Formaldehyde
     - ♦ Glutaraldehyde
     - ♦ Silver nitrate
     - ♦ Strontium chloride hexahydrate
   - Casein phosphopeptides
   - Burnishing
   - Fluoride iontophoresis

   b. Dentine sealers
   - Glass ionomer cements
   - Composites
   - Resins
   - Varnishes
   - Sealants
   - Methyl methacrylate

   c. Periodontal soft tissue grafting
   d. Crown placement/restorative material
   e. Lasers

NERVE DESENSITIZATION USING POTASSIUM NITRATE

A number of studies have reported the efficacy of potassium nitrate for managing dentinal hyper-sensitivity.[21,22,23,24] While the Hodosh study was the first to report that potassium nitrate was a “superior desensitizer” this study was not well controlled and it was not until the studies of Tarbet.[22,23] Patients are often prescribed over the counter desensitizing agents. These "at home" desensitizing agents include toothpastes, mouthwashes and chewing gums containing potassium salts (potassium nitrate, potassium chloride or potassium citrate), sodium fluoride, strontium chloride, dibasic sodium citrate, formaldehyde, sodium monofluorphosphate and stannous fluoride.[15] Potassium salts act by diffusion along the dentinal tubules and decreasing the excitability of the intradental nerve fibers by blocking the axonic action.[14,15] According to the FDA, for a potassium nitrate toothpaste to claim to be desensitizing it must contain 5% of the ingredient. Potassium nitrate penetrates the enamel and dentin to travel to the pulp and creates a calming effect on the nerve. This effect can be thought of as “anesthetic-like”.[25] Previously it was believed that NO_3^- is the active agent is potassium nitrate, however [26, 27] showed that NO_3^- anion is not effective as desensitizing agent and K^+ is an effective desensitizing agent regardless of the anion with which it is combined and further K^+ had a reversible effect, that is, they did not appear to damage the dentinal sensory apparatus.The results of "at-home" desensitizing therapy should be reviewed after every 3-4 weeks. If there is no relief in DH, "in-office" therapy should be initiated.[28] Theoretically the in-office desensitizing therapy should provide an immediate relief from the symptoms of DH. The in-office desensitizing agents can be classified as the materials which undergo a setting reaction (glass ionomer cement, composites) and which do not undergo a setting reaction (varnishes, oxalates).[28]

CONCLUSION

Dentinal hypersensitivity is a very common problem and now it is successfully managed by a large variety of products. Treatment may be started at home methods such as desensitizing dentifrices containing 5% potassium nitrate. This alone may solve the condition, but if not, an in-office treatment may be used. The products developed for at home application, Potassium nitrate containing dentifrices has been extensively studied and proved to be not only safe but can also give benefits to patient suffering from dentinal hypersensitivity up to a satisfactory extent.

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