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An Approach On Dentin Hypersensitivity And Its Management

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ABSTRACT
Dentin hypersensitivity (DH) is characterized by short andsharp pain arising from exposed dentin in response to stimuli. It is the most widely accepted theory of how the pain occurs is Brannstrom′s hydrodynamic theory, fluid movement within the dentinal tubules. The condition usually affects the facial surfaces of teeth near the cervical aspect and is common in premolars and canines. This condition is frequently encountered by dentists, periodontists, hygienists as well as dental therapists. Some dentists lack confidence in treating DH. The management of this condition generally requires a good understanding of the complexity of the problem, as well as the variety of treatments available. This review briefly explains the etiopathogenesis, incidence, diagnosis, prevention and management of dentinal hypersensitivity.

***Keywords :Dentinal tubule, dentine hypersensitivity, pain, sensitivity, dentin bonding agents, prevention*.**

1.Introduction

Dentin hypersensitivity (DH) is characterized by sharp and short pain arising from exposed dentin in response to stimuli. The foremost widely accepted theory of how the pain occurs is Brannstrom′s hydrodynamic theory, fluid movement within dentinal tubules. Typically, the pain is usually brief , sharp, and occurs in response to certain stimuli applied to exposed dentin. Its clinically described as exaggerated response application of a stimulus to exposed dentin, no matter its location. [1]

II. Definition

Dentin hypersensitivity (DH) is characterized by short sharp pain arising from exposed dentin in response to stimuli typicallyevaporative, thermal, tactile, osmotic, or chemical and which cannot be ascribed to any other defect or pathology. [2]

III. Prevalence and epidemiology

DH is a painful clinical condition that affects 8-57% of the adult population and is associated with dentin exposure to the oral environment. A rather higher incidence of DH is reported in females than in males. Age group of 20-50 years is most affected with a peak between 30 and 40 years of age.In both arches,canines and premolars are most affected. The most commonly affected site is the buccal aspect of cervical area .[1]

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IV. Pathogenesis

DH occurs in two phases: lesion localization and lesion initiation.[3] Lesion localization occurs by loss of protective covering over the dentin thereby exposing it to the external environment. It includes loss ofthe enamel via attrition, erosion, abrasion or abfraction. Gingival recession can also cause lesion localization by toothbrush, pocket reduction surgery, abrasion, tooth preparation for crown or excessive flossing.[4] In the initiation of the lesion, protective covering of smear layer is removed, leading to exposure and opening of tubules.

V.Mechanism of Hypersensitivity

Odontoblastic transduction theory: (Rapp et al, 1968)[5]

According to this theory, odontoblastic processes which are exposed on the dentin surface can be excited by a variety of chemical and mechanical stimuli. As a result of such stimulation, neurotransmitters are released and impulses are transmitted towards the nerve endings.To date there is no neurotransmitters found to be produced or released by odontoblastic processes.

Hydrodynamic theory: (Brannstrom et al, 1960)[6]

By far,the most widely accepted theory for dental hypersensitivity, proposed by Brannstorm and co-workers in the late 1960's. This theory postulates that fluid within the dentinal tubules are disturbed either by temperature, physical or osmotic changes and that these fluid changes or movements stimulate baroreceptor which leads to neural discharge.The basis of this theory is that fluid filled dentinal tubules are open to the oral cavity at the dentin surface as well as within the pulp.An increased outward fluid flow causes a pressure change across the dentin ,distorting the A-d fibers by a mechanoreceptor action,causing shootingsharp pain.

Neural theory: (Frank RM et al, 1988)[7]

As an extension of odontoblastic theory,this concept advocates that thermal or mechanical stimuli,directly affect nerve endings within the dentinal tubules through direct communication with the nerve fibers of pulp.

VI. Clinical assessment of dentine hypersensitivity[3]

Subjective evaluation

Verbal rate scales:(Karcioglu et al., 2018)Used to assess pain experiences. It is alsoknown as verbal pain scores and verbal descriptor scales. It is self-report which consists of a number of statements designed to describe pain intensity and duration.

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Visual analogue scale: It is a measurement instrument that tries to measure a characteristic or attitude that is believed to range across the continuum of values and it cannot be easily and directly measured.

Mc Gill word description: It isused to evaluate a person experiencing significant, strong pain. It can be used to monitor the pain overtime and to determine effectiveness of any intervention. It can be translated to several languages.

Objective assessment

Mechanical / tactile stimuli,Explorer Probe,Mechanical Pressure Stimulation,Yeaple Probe,Chemical /osmotic stimuli, Electrical stimuli, Electrical pulp tester and Dental pulp stethoscope are the objective assessments done.

Evaporative stimuli

Cold air blast, Air thermal system,Air jet stimulator and Temptonic device are evaporative stimuli that can be used.

Thermal stimulation

Cold water testing, Thermo electric device,Ice Stick, Heat or Cold Airand Ethyl Chlorideare thermal stimulation methods.

VII.Differential diagnosis [1]

Chipped teeth, Cracked tooth syndrome, Fractured restoration, Traumatic occlusion, Post operative pain and Dental caries are various differential diagnosis.

VIII.Natural desensitization [8]

Sclerosis of dentin,Secondary dentin,Tertiary dentin,Smear layer and Calculus formation.

IX. Management

The most common form of management is the placement of topically applied agent applier either by a dental professional or by the patient at home.

X. Mechanisms of desensitization:

Mechanisms of desensitization are nerve depolarization , occlusion of dentinal tubule orifices and newer occluding agents.

XI. Classification of desensitizing agents [9]

Mode of Administration

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The desensitizing agents are often applied by the patient reception or by the dentist or hygienist in the dental office.

On the basis of mechanism of action

Nerve desensitization ,Potassium nitrate, Protein precipitation ,Glutaraldehyde,Silvernitrate ,Zinc chloride and Strontium chloride hexahydrate are used.

Plugging dentinal tubules

Sodium fluoride,Stannous fluoride,Strontium chloride,Potassium oxalate and Calciumphosphate ,Bio active glasses (SiO2 – P2O5- CaO-Na2O).

Dentin adhesive sealers

Fluoride varnishes ,Oxalic acid and resin,Glassionomer cements, Composites and Dentin Bonding agents are various sealers used.

Fluoride iontophoresis

Lasers

Neodymium: yttrium aluminum garnet (Nd- YAG) laser,Gallium-aluminum-arsenide laser (GaA1As) and Erbium-YAG laser can be used.

Self applied / at home desensitizing therapy [10]

Strontium Chloride, Strontium acetate ,Potassium Nitrate,Oxalates,Fluorides like Sodium Fluoride,SodiumSilicofluoride,Stannous fluoride, Sodium monofluorophosphate and Sodium Citrate Pluronic F-127 gel

In office treatment [11]

Cavity Varnishes,Resins and Adhesives ,Seal & Protect and AdmiraProtect,CalciumCompounds,Corticosteroids,FlourideIontophoresis-Pivati 1947,Galvani and VoltaHypothesis,Induction of secondary dentin formation and Induction of parasthesia on odontoblastic process Tubule occlusion are various in office treatment.

Lasers for treatment of dental hypersensitivity [12]

Low Output Power Laser used are He Ne Laser and GaAlAs Laserand Middle Output Power Laser are Nd:YAG Laser and CO_2 Laser.

XII. Recent advances in treatment materials[13]

BIO Glass, Portland Cement,Prehybridized Dentin,Carbonate Hydroxyapatite Nanocrystals,Casein Phosphopeptide and Proarginare the recent advances in treating dentinal hypersensitivity.

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XIII. Guidelines for patients:(Drisko 2002)[14]

Avoid using large amounts of dentifrice during brushing, hard-medium tooth brushes, brushing with excessive pressure , brushing immediately after taking acidic foods,excessive flossing or improper use of other interproximal cleaning devices and rinsing or eating for 1hour after desensitizing treatment.

XIV. Guidelines for dental professionals: (Drisko 2002) [14]

Avoid over-instrumenting root surfaces during SRP particularly in cervical areas ofthe tooth, over polishing exposed dentin during stain removal, violating biologic width during restoration placement and burning gingival tissues during in office bleaching.

XV.Conclusion

The increase in longevity of dentition through periodontal therapy and plaque control procedures may increase the incidence of dentine hypersensitivity. The ultimate goal in the treatment of dentine hypersensitivity is the immediate and permanent relief of pains.Once the definitive diagnosis of dentine hypersensitivity has been made after considering a differential diagnosis, a careful assessment of the etiologic factors must be considered, and if identified correctly , it should be managed appropriately. This may enhance the outcome of the currently used desensitizing agents and ensure more successful management.

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