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Dentin Hypersentivity: A Literature Review

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***Abstract*---**Dentine hypersensitivity (DHS) is a widespread painful dental problem that is characterized by short sharp pain arising from exposed dentin in response to several different stimuli. This accepted definition, indicates the need to consider a differential diagnosis to rule out other causes of dental pain before the diagnosis of dentin hypersensitivity is made. The management strategy of this dental problem required a good understanding of the complexity of the condition and the variety of the treatment options. The aim of this review is to inform practitioners about dentin hypersensitivity, to provide a brief overview of the diagnosis, etiology and clinical management of dentin hypersensitivity.

***Keywords*---**hypersensitivity, dentine, desensitize, dental pain.

Introduction

Many dentists have some problems in determining the etiology, diagnosing and treating dental hypersensitivity and some limitations have been observed in this regard. Dentin hypersensitivity (DH) is a frequently chronic finding and a challenging condition to treat in dental clinical practice[1]. Dentin hypersensitivity can be defined as a short sharp pain that arises from the exposed dentin in response to thermal, tactile, osmotic, chemical, or evaporative stimuli that cannot be attributed to any other form of dental defect or pathology[2]. This condition impacts oral health-related quality of life[3,4], producing significant impairment on patients' daily life such as speaking, eating, drinking and tooth brushing[5,6]. The

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clinical management of DHS has been a challenge for clinician, various treatment modalities are available, but the success of any sound treatment plan is depended on taking a detailed clinical and dietary history, differentially diagnose the condition from other dental pain conditions and identify and manage etiological and predisposing factors[7-10]

All of these terms convey the same clinical conception and can be used interchangeably
Common terms which are used refer to dentin hypersensitivity
Dentin Hypersensitivity/ Sensitivity
Dentinal Hypersensitivity/ Sensitivity
Cervical Hypersensitivity/ Sensitivity
Root Hypersensitivity/ Sensitivity
Cemental Hypersensitivity/ Sensitivity[7-10]

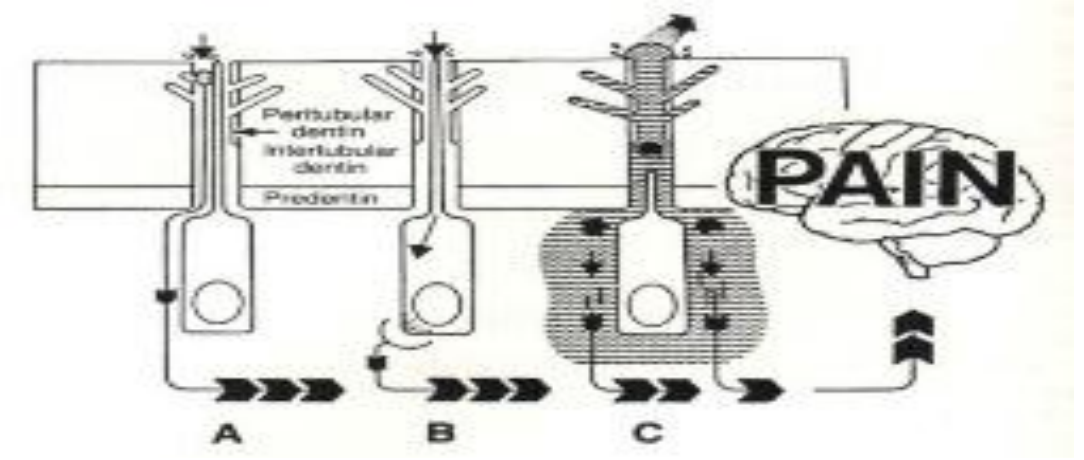
Prevalence and Epidemiology

Various studies showed that the incidence of DHS in most populations ranges between 10-30% of the general population and the age range varies from 20-50 years with the peak incidence occurring at the end of the third decade and decreases during the fourth and fifth decades of life[11-13].There are two common methods to determine the intensity of DH. One of them is through asking some questions from the patient and the other is through clinical examination. The prevalence distribution of DH in the first method is usually estimated higher than that of the second method[14].

The higher incidence of DHS is reported in females than in males which may reflect hormonal influence and dietary practices[15,16] In contrary, one study showed no difference in prevalence of dentine hypersensitivity in either gender, suggesting overall that as many malesas females are susceptible[15]. Concerning the type of teeth involved, canines and premolars of both the arches are the most affected teeth. Also it was reported that buccal aspect of cervical area is the commonly affected site[16].

Theories And Mechanism Of Sensitivity

Several theories have been proposed over more than a century to explain the mechanism involved in dentine hypersensitivity[17].



Odontoblastic Transduction Theory

The odontoblast transducer theory proposed by Rapp et al. postulated that odontoblasts act as receptor cells, and transmit impulses via synaptic junctions to the nerve terminals causing the sensation of pain from the nerve endings located in the pulpodentine border[18]. However, evidence for the odontoblast transducer mechanism theory is deficient and unconvincing. This is because the majority of studies have shown that odontoblasts are matrix forming cells and they are not considered to be excitable cells, and no synapses have been revealed between odontoblasts and nerve terminals[19].

Neural Theory

This theory advocated that thermal, or mechanical stimuli, directly affect nerve endings within the dentine tubules through direct communication with the pulpal nerve endings[18]. Although this theory has been reinforced by the presence of unmediated nerve fibers in the outer layer of root dentine[20] and the presence of putative neurogenic polypeptides21, it is still considered theoretical with lack of solid evidences to support it.

Hydrodynamic Theory

The currently accepted mechanism of dentine hypersensitivity is the hydrodynamic theory which has been proposed by Brännström in 1964[22]. According to this theory, when the exposed dentin surface is subjected to thermal, chemical, tactile or evaporative stimuli, the fluid flow within the dentine tubules there will be increased[22].

This fluid movement within the dentine tubules causes an alteration in pressure and excites pressure-sensitive nerve receptors across the dentine. So the response of the excited pulpal nerves, mainly in intra dentine fibers, will be depended upon the intensity of stimuli in pain production[23]. Scanning electron microscopic (SEM) examination of hypersensitive dentin surface reviles the presence of widely open dentine tubules which is considered consistent with the hydrodynamic theory[24].

Accordingly, the number and the diameter of the dentine tubules are considered important factors in initiating pain from DHS[24,25]. Hence, the higher the number and greater the diameter of the open dentine tubules the more intense will be the pain from DHS. It has been noted that triggers such as cold stimuli stimulate fluid to flow away from the pulp creating more rapid and rigorous neural responses than heat stimuli, which cause somewhat sluggish fluid flow towards the pulp[24-26]. This is aligned with the observation that dentine hypersensitivity patients are more frequently complain of pain in response to cold stimuli than to heat[27].

Etiology Of Dentine Hypersensitivity

Dentin is covered by enamel in the crown region and by cementum in the radicular region. When the enamel or cementum is removed, the underlying dentin will be exposed along with the dentine tubules, producing dentin hypersensitivity. It has been postulated that DHS develops in two phases namely[28]

- lesion localization
- lesion initiation

Lesion localization occurs by loss of protective covering over the dentin, thus exposing it to external environment. Lesion initiation occurs after the protective covering of smear layer is removed, leading to exposure and opening of dentine tubules. Evidence already showed that the lesions of DHS have many more and wider open tubules than do non sensitive dentin[24,26].

Gingival Recession

Gingival recession is usually recognized in patients with a high level of oral hygiene standards and in those who have poor level of oral hygiene. The causes of gingival recession in the population having good oral hygiene are due to overzealous tooth brushing, improper brushing technique or using of an excessive brushing forces and it is frequently seen on the buccal surfaces of the teeth [29]. On other hand, lack of tooth brushing, with consequent accumulation of dental plaque on root surfaces in patients with bad oral hygiene may lead to periodontal complications and migration of the gingiva in the apical direction, exposing the cementum and then to demineralization of tooth structures which could be associated with patency of dentine tubule orifices causing DHS[30,31]. Gingival recession is also a common side-effect of a periodontal treatment either surgical or nonsurgical one due to loss of attachment of healthy gingival tissue[32]

Erosion is the progressive loss of hard dental tissues by chemical processes or acids not produced by cariogenic bacteria as in case of acidic agents associated with regurgitation or extrinsic acids associated with dietary and medication sources. Repeated exposures to low Ph oral fluids lead to dissolution of the mineral content in the superficial layer of enamel, the result of which is loss of that tissue with consequent changes in tooth architecture. Inescapable dentine exposure results in dentin hypersensitivity. Abfraction lesions are wedge shaped defects developed at the cervical region of teeth and are not directly related to the

diet, periodontal disease or abrasion[33]. They occur as a result of mechanical overloading of cervical enamel regions initiated by cuspal flexure and occlusal overloading, resulting in fracture of the enamel crystals in this area with subsequent exposure of the underlying dentin[33,34].

Assessment of DHS

Traditionally DHS has been evaluated on the basis of the individual response to the presenting stimulus which could be in the form of verbal rating, visual analogue scales and questionnaires[35]. Commonly, DHS might either be assessed in terms of a stimulus intensity required to elicit pain called stimulus-based assessment or as a subjective evaluation of the pain caused by a distinct stimulus name response-based assessment[35-39].

The stimulus-based assessments depend on the measurement of an individual pain threshold on which the subject's response is held constant at the pain threshold, and the stimulus is varied with increasing and decreasing intensities[35-39]. Different devices have been used in these methods such as a calibrated probe where the tactile pressure applied to the tooth with a dental explorer tip can be varied and increased in steps of 10 g increments until the patient experienced discomfort[37-39].

Alternative thermal or electrical devices such as electrical pulp testers, dental pulp stethoscope and others have been used for applying graded thermal or electrical stimuli[38]. It was realized that, these stimulus-based methods have certain drawbacks such as repeated painful stimulation may cause a change in sensitivity and influence the outcome. Furthermore, it was reported that stimulusbased methods are often time consuming, which limit the number of teeth that can be tested with multiple stimuli in one appointment[40].

Verbal Rating Scales

For measuring of DHS, verbal rating scales (VRS) are used to grade the level of pain experience. Most pain scales utilize several pain descriptors, including 'no pain', 'weak', 'mild', 'moderate, 'strong', 'intense', and 'agonizing'. Numerical scores (0, 1, 2, 3, etc.) have been attached to these descriptors, and mean values are calculated[8]. However, the mathematical interpretation of the scoring system has been challenged, in that the scores are often arbitrarily assigned numerical values, and the assigned scores are analyzed as if these numbers reflected true quantitative differences in pain rather than simple qualitative differences[35]. Hence the main disadvantage of verbal descriptor scales is that they could be restrictive because they may not offer enough descriptions that can be placed in a continuous and ascending or descending order of severity of pain[38].

Visual Analogue Scale

The visual analogue scale (VAS) utilizes a line of 10 cm length anchored at the 2 extremes with descriptors representing the absolute minimum and the absolute maximum of pain a patient can experience from an external stimulus[41]. It was designed to overcome the shortcomings of VRS[42]. In VAS assessments, the patient

is asked to mark off the line such that it corresponds to the severity of the perceived pain and the pain intensity can be shown as an absolute value or as a percentage of the maximum[41].

Treatment

Self-Applied And Office Supplied

Self-applied treatments to reduce sensitivity consist of materials that occlude dentinal tubules, coagulate or precipitate tubular fluids, encourage secondary dentin formation, or obstruct pulpal neural response. Desensitizing toothpastes that contain potassium salts, either nitrates or chlorides, are believed to act by depolarizing the nerve surrounding the odontoblastic process, resulting in interference of transmission. Usually it takes 2 weeks with twice-a-day usage to get a reduction in sensitivity. Instead of having patients brush with the toothpaste, the paste could be placed in a soft tray to increase contact time. Haywood and colleagues[43] recommended placing 5% potassium nitrate in bleaching trays to minimize sensitivity that may occur as a result of bleaching. Ten-minute to 30-minute applications seemed to assuage any sensitivity that may occur. Ideally, the desensitizing toothpaste should not have sodium lauryl sulfate because a large amount of this ingredient may cause tissue irritation.

Laser Treatment

Laser treatment has also been recommended for the treatment of dentin hypersensitivity. The treatment seems to be only transient, however, and the sensitivity returns in time. In order for a laser to actually alter the dentin surface, it has to melt and resolidify the surface. This effectively closes the dentinal tubules. This does not occur. It is felt that laser treatment reduces sensitivity by coagulation of protein and without altering the surface of the dentin.[44] Dicalcium phosphate-bioglass in combination with Nd:YAG laser treatment has sealed dentin tubules to a depth of 10 mm, and dicalcium phosphate-bioglass plus 30% phosphoric acid occluded exposed tubules up to 60 mm[45]

Fluoride Treatment

Patients can apply stannous fluoride in a 0.4% gel or sodium fluoride in a 0.5% mouth rinse or a 1.1% gel. Fluorides reduce the permeability of dentin probably by precipitation of insoluble calcium fluoride inside the dentinal tubules and reduce sensitivity[46] Gel-Kam Dentin Block (Colgate Oral Pharmaceuticals, New York, NY, USA) consists of 1.09% sodium fluoride, 0.4% stannous fluoride, and 0.14% hydrogen fluoride 8that can be applied in a tray.

Control Measure For Prevention Of Dhs And Removal Of The Etiological Factors

Various studies suggested that the dental practitioner should advice his/her patient to follow certain preventive measures to reduce both the frequency and intensity of DHS episodes. These measures are considered as self-care strategies.

- Avoid faulty tooth brushing to lower the risk of gingival recession and abrasion of exposed cementum and dentin[15,16]. It includes
  - Not to use a hard tooth brush use only a toothbrush with soft filaments.
  - Avoid using of an excessive pressure or force during brushing.
  - Brushing time should not be extended for prolonged period of time.
  - Excessive scrubbing at the cervical part of the tooth that damages to the supporting structures and causes gingival recession should be avoided.
  - Not to use large amounts of dentifrice or reapplying it during brushing.
  - Avoid using a highly abrasive tooth powder or paste.

- The use of an additional aids[15,47], such as:
  - Desensitizing dentifrices containing an active agent potassium salts such as potassium nitrate, potassium chloride or potassium citrate, where the potassium ions can decrease the excitability of A fibers, which surround the odontoblasts resulting in a significant reduction tooth sensitivity.
  - Remineralizing toothpastes containing sodium fluoride and calcium phosphates.
  - Mouth rinses and chewing gums that contain potassium or sodium salts.

- Avoid excessive flossing or improper use interproximal cleaning devices or toothpicks[8].
- Reduce the quantity and the frequency of taking foods containing acids[8].
- Avoid brushing for at least 30 minutes after taking acidic food or drinks[8].
- To take something alkaline or neutral after acidic drink[8]
- Maintain good oral hygiene[48].
- Wear occlusal splints to minimize tooth wear associated with para-functional habits like bruxism[49].

Conclusion

Clinically, DHS is a relatively common and significant dental problem for which patients look for treatment and visit dental clinics. There are many treatment modalities for DHS which the clinician may find successful in relieving the pain of DHS. The dental practitioner should first identify the causative or predisposing factor after taking a thorough history before the treatment plan is designed. The treatment strategy of the DHS should be begun with prevention, selfcare management and later may be supplemented with professional interventions depending on the severity of the case.

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