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REVIEW ARTICLE

Dentin Hypersensitivity: A Review of its Etiology, Mechanism, Prevention Strategies and Recent Advancements in its Management

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ABSTRACT

Dentinal hypersensitivity is one of the most common clinical problem for which patients seek treatment and visit dental clinics. Its incidence is on a rise probably because its etiology is not very well understood. Once initiated, it can either remain as sensitivity or it can trigger pain and creates severe discomfort for the patient. Number of theories have been put forward to explain the mechanism of hypersensitivity causing pain but hydrodynamic theory is most widely accepted nowadays. Treating dentinal hypersensitivity is a challenge for dental professionals. This article reviews the etiology, mechanism and prevention strategies of dentinal hypersensitivity and also discusses recent advancements in its management.

Keywords: Dentin hypersensitivity, Hydrodynamic theory, Dentinal tubules.

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INTRODUCTION

Dentin hypersensitivity (DH) is defined as diminutive and sharp pain which arises from exposed dentin surface in reaction to a stimulus of thermal, evaporative, tactile, osmotic or chemical origin.[1] DH is different from sensitivity which can be produced from other clinical conditions such as cracked tooth, fractured tooth or restoration, dental caries or microleakage from restorative material.[2] DH can affect people of any age group but the majority of affected people have been reported to be in the third and fourth decade of their life.[3] Some studies have suggested that DH affects more women than men.[4,5]

Prevalence

The prevalence of DH has been reported at 8 to 35% depending on the study of the population.[6] Its prevalence has been reported to be 60 to 98% in patients having history of periodontal disease.[7] Reason explaining this trend could be that in periodontal disease, bacteria can infiltrate the dentin to a significant distance.[8] It has been reported that around 69% of population in UK is experiencing some form of tooth sensitivity.[9] However, true or actual rates of prevalence may be distorted because most of the patients do not seek treatment from their dentists as they do not perceive DH as severe oral health problem.[10]

Etiology

Etiology of DH is not well understood. Various etiological and predisposing factors have been proposed to be associated with DH such as gingival recession, loss of enamel and patients habits.[11] Some of the etiological and predisposing factors associated with DH have been summarized in Table 1.

Gingival recession due to chronic periodontal disease is thought to be a very important etiological factor associated with DH.[13] Moreover, Dababneh et al (1999) suggested that hypersensitivity which occurs in association with periodontal disease might have an etiology which is different than the etiology of DH occurring alone because of possible penetration of dentinal tubules by bacteria.[14] Erosive potential of the food and drinks and certain habits of patients like bruxism, contribute in developing DH by affecting the tooth surfaces.

Mechanism of Dentin Hypersensitivity

Numbers of theories have been put forward in recent years to explain the mechanism of dentinal hypersensitivity causing pain. Most widely accepted theory till date is called hydrodynamic theory.[15] This theory was first put forward or postulated by Gysi in the year 1900 and was later developed by Brannstorm (1963).[16]

It was proposed in this theory that application of external stimuli such as cold or hot, tactile or osmotic pressure to

Table 1: Etiological and some predisposing factors associated with dentin hypersensitivity[12]

Etiological and predisposing factors
Loss of enamel
Denudation of cementum
Gingival recession
Attrition
Abrasion
Abfraction
Erosion (intrinsic and extrinsic)
Tooth malposition
Thinning, fenestration, absent buccal alveolar bone plate
Periodontal disease and its treatment
Periodontal surgery
Patient habits

exposed dentin will cause movement of dentinal fluid. The movement of the fluid in turn will stimulate mechano-receptors which are present near the base of the dentinal tubules and under the influence of certain physiological parameters; a pain response will be triggered. It is anticipated that mechanical activation of A-δ fibers which surround the odontoblasts, is responsible for this response.[17]

It has been suggested that almost 75% of DH patients will complain of pain when they receive cold stimuli.[18] A cold air syringe blast directed toward dentin is a clinical screening method for DH, as the air blast has both thermal and evaporative properties.[1] Intradental nerves are more effectively activated by the stimuli which makes the dentinal fluid to flow away from the pulp such as drying, cooling and hypertonic chemical stimuli as compared to the stimuli which causes the dentinal fluid to flow toward the pulp, such as probing or heating.[19]

Causes of Exposure of Dentin Leading to Hypersensitivity

Dentin is covered by enamel in the crown region and by cementum in the radicular region. Once enamel or cementum is removed, the underlying dentin will be exposed along with the dentinal tubules, causing dentin hypersensitivity. Enamel is mostly removed by acid erosion and the removal of cementum is attributed to the overzealous tooth brushing or periodontal loss either due to disease or treatment. Most common factors leading to exposure of dentin are as follows:

Gingival Recession

Gingival recession is a multifactorial condition. It is seen in the population who usually have a high standard of oral hygiene or those who have poor standard of oral hygiene. In the population having good oral hygiene, overzealous tooth brushing and improper technique of tooth brushing causes gingival recession by damaging the gingiva and is recession is mostly seen on the buccal surfaces of the teeth.[20] In the population with poor oral hygiene, presence of periodontal disease and related periodontal conditions or periodontal tissue damage following a treatment, either surgical or nonsurgical, causes gingival recession.[21]

DH is attributed to any type of recession as stated earlier but it is most frequently associated with the loss of attachment of healthy gingival tissue.[22]

Acid Erosion

Acid erosion is the most aggressive type of wear as compared to abrasion and attrition. Enamel shows a high susceptibility to acid erosion. Acid can be derived from many sources such as gastric, intrinsic, and extrinsic sources which are dietary in origin.[23] Individual susceptibility results in the loss of enamel to acid erosion. When acid is in contact with the tooth, not only bulk loss of the hard tissue occurs but there is softening of the remaining surface as well.[24] Dentin being softer than enamel is also more susceptible to erosion and it shows an irreversible loss leading to exposure of dentinal tubules.[25]

Abrasivity

It is a well established fact that due to improper oral hygiene practices, abrasion of exposed dentin occurs but sufficient evidence to support this is still lacking.[26] Softening of enamel by the action of acids makes it much more susceptible to mechanical process like abrasion to such an extent that the action of tongue would be sufficient enough to remove eroded enamel.[27] Once enamel is removed, tooth brushing performed with abrasive toothpaste has the potential of abrading the dentin surface and can cause opening of the dentinal tubules if it is accompanied by erosive agents.

Prevention Strategies

Gingival recession is a consequence of periodontal disease which happens mostly because of improper oral hygiene. Advising the patients about proper technique of tooth brushing and providing them with oral hygiene maintenance instructions, can prevent DH.[14] It has been suggested that brushing should be avoided for at least 1 to 3 hours after the consumption of acidic food or drink in order to avoid abrasion which can lead to DH.[28] If the patient has a history of intrinsic erosion, patient should be referred and should be treated by a physician.[29]

Finally, communicating with the patients about their oral hygiene problems beforehand is very useful. Like advising the patients receiving periodontal treatment about the chances of occurrence of DH can help to prevent many predisposing factors.[30]

Treatment of Dentin Hypersensitivity

Tubule Occlusion

One of the logical conclusions to treat dentin hypersensitivity is to occlude the tubules. Although, we do not have full understanding of the pain transmission mechanism across dentin, but a reduction in dentin permeability as well as sensitivity occurs with occlusion of dentinal tubules.[31]

One of the treatment modalities for treating DH is tubule occlusion by constituents of toothpaste and it has been

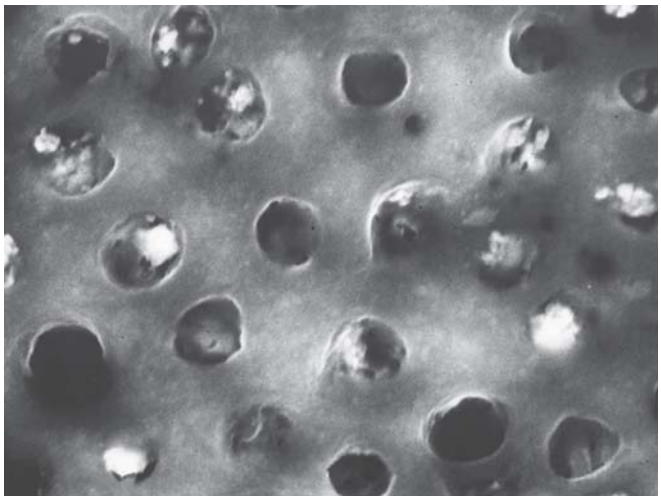

Fig. 1: SEM image, at 4500× magnification, showing dentinal tubules (*Courtesy*: Prof Robert Hill, QMUL, UK)

proposed that silica abrasives or other active agents can occlude the dentinal tubules.[5] But the effectiveness of agents occluding the tubules is dependent on their removal resistance. Some of the occluding agents could be resistant while others are acid liable and could be easily washed away.

It has been shown by the results of *in vitro* studies that the occlusion of tubules is achieved by a number of agents but their correlation to *in vivo* data is poor as there are oral challenges of daily activities as well.[22] It has been suggested by Gaffar (1999) that sodium fluoride occludes dentinal tubules by crystallization and fluid flow to the pulp is reduced which decreases DH.[32] But Lan et al concluded from the scanning electron microscopy (SEM) images of a study that occlusion of tubules by sodium fluoride is not that efficient (Fig. 1).[33]

It has also been proposed that the severity of DH is related to the increased width of dentinal tubules therefore an important variable which influences movement of fluid through the tubules is the functional radius of the tubules.[34] Poiseuilles law states that 'the movement of fluid is directly proportional to fourth power of radius'[35] and as a result any reduction in the permeability of dentin by the occlusion of tubules will be effective in treatment of DH.[36] To measure the dentinal fluid flow numerous techniques have been developed *in vitro*[37] and *in vivo* as well.[38] The hydraulic conductance model (Lp) was developed by Pashley (1986) which is based on determining the changes in dentin permeability to the simulated fluid flow of dentin after application of a putative active treatment.[39] The model is still considered as a standard to determine the ability of a desensitizing agent to reduce permeability of dentin.

Desensitization of Nerve by Potassium Salts

Currently potassium salts are the most common active ingredient used to desensitize dentin. 5% potassium nitrate in dentifrices has been in use since 1980's.[40] The active ingredient which is 2% potassium ion is released from dentifrices which contain potassium nitrate (5%), potassium chloride (3.75%) and potassium citrate (5.5%).[41] It is thought that the synapse between the nerve cells is blocked by potassium ions, which will reduce the nerve excitation and the pain associated with it.[42] Various reports about the clinical studies on dentifrice containing potassium have been published. Reports of six studies state that dentifrice which contains either 5% potassium nitrate or 3.75% potassium chloride, decreases DH significantly when it is compared with the baseline or negative controls.[43-48] Ajcharanukul et al (2007) did a study which demonstrated that DH in humans can be decreased by the use of potassium salts even through relatively thick dentin, by use of a controlled experimental design and visual analog pain scores (VAS), although the effect was transitory in nature.[49]

Potassium oxalate is another potassium salt which has demonstrated the formation of calcium oxalate crystals onto the tooth surface which are acid resistant and they occlude the dentinal tubules temporarily.[50]

The major challenge for researchers is that currently there is no mechanism to study and show potassium uptake clinically in the tooth to support the hypothesis of it acting as a desensitizing agent. Several investigators stated that desensitizing studies for the assessment and management are challenging due to the highly subjective nature of the response of the pain.[51] Although published literature shows clinical evidence supporting the role of potassium containing dentifrices in treating DH, but still the evidence is not convincing with regards to the proposed manner of nerve desensitization by dentifrices.[52]

Other Treatment Options

Calcium hydroxide can be used to block the tubules and decrease sensitivity[53] but it has been reported that its presence can cause irritation of the gingiva.[54] Sodium fluoride is also effective in tubule occlusion and nerve desensitization.[55] Other treatment possibilities include the use of dentin sealers like composites and glass ionomer cements but again their effect is temporary in nature as they can get washed away.[56]

CONCLUSION

Dentin hypersensitivity is a widespread oral problem which affects people of variety of age groups. A lot of research has been carried out to know the exact etiology of dentin hypersensitivity but concluding evidence is still missing. Various treatment options are now available for the treatment of dentin hypersensitivity. When a patient visits

a dental setting with the symptoms of hypersensitivity, causative or predisposing factor should be first identified by the practitioner after taking a thorough history and then a treatment plan should be designed accordingly to treat hypersensitivity.

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