Review Article

DENTINE HYPERSENSITIVITY: REAL OR IMAGINED

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ABSTRACT

BACKGROUND:
Dentine hypersensitivity is a common presentation of cause of pain and or discomfort with mastication which has been shown to affect the quality of life of the affected individual. It is also a common cause of presentation at the dental clinics. However, the cause, diagnosis and possible management to give relief can be a dilemma for the clinician who at times may wonder if the sensation the individual is presenting with, is real or imagined.

AIM:
The purpose of this paper was to review dentine hypersensitivity in view of causes, diagnosis and management.

METHODOLOGY:
Articles used were found by searching the key words: dentine hypersensitivity, tooth wear lesions, desensitization, gingival recession, treatment of hypersensitivity.

RESULT:
There are a variety of causes of dentine hypersensitivity with a variety of ways to manage and provide adequate treatment, but the condition must be well understood.

CONCLUSION:
Many options of treatment are now available to eliminate the pain from dentine hypersensitivity and thus improving the quality of life of these patients because the sensitivity they experience is real.

KEYWORDS: dentine, hypersensitivity, tooth wear, desensitization, quality of life.

NigerJMed2016: 182-188
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INTRODUCTION

Dentine, the second layer of the tooth structure in an ideal anatomical position, is protected from the oral environment by enamel and cementum.[1,2] The dentinal tubules which occupy 1% (superficial dentine) to 30% (deep dentine) of the volume of intact dentine are filled with free dentinal fluid that occupies 1% of superficial dentin but about 22% of the total volume of deep dentin.[3] The outward flow of this fluid between the odontoblasts through the dentinal tubules is blocked peripherally by enamel on the crown and cementum on the root.[1] On exposure of the dentine however, this outward flow through the tubules is without hindrance and can trigger nerves along the pulpal canal of the dentine causing the pain termed "dentine sensitivity".[4]

Dentine hypersensitivity (DH) (also known as dentine sensitivity, cervical sensitivity/hypersensitivity etc) by general consensus and as concluded at an international workshop on DH has been defined as "condition characterized by short, sharp pain arising from exposed dentine in response to stimuli, typically thermal, evaporative, tactile, osmotic or chemical and which cannot be ascribed to any other dental defect or pathology".[5-8] The condition, etiology, and treatment of dentin hypersensitivity, or hyperalgesia, have been reported in the literature for over 100 years.

Many aspects of DH however is poorly understood by dental professionals, particularly the aetiology of DH[9] and also identifying it from other conditions such as reversible pulpitis that present with such dental pain.[10,11] Though studies[12-14] have been carried out on DH, the condition still remains one of the most painful and least satisfactorily treated chronic condition of the teeth. Much confusion has however been caused by conflicting views and opinions that make one wonder if dentine hypersensitivity is "Real or Imagined".

Epidemiology

The prevalence of Dentinal hypersensitivity varies greatly due to the variation in the approaches to the research which may be questionnaire or clinic based and it has been reported to be present in as low as 4%

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and as high as 74% of adult population, [8,13,15,16] with reported clinical prevalence of 15% [17]while the prevalence in periodontal patients has been found to be between 72 to 98%.[8] It affects ages 20-50years with the peak age between 20-40 years,[8,18] and higher incidence seen in females presumably due to their overall health care and better oral hygiene awareness.[8,17,19] The most affected teeth are the canines followed by the 1st premolars, 2nd premolar, the incisors and then the molars[7,20] with the most recorded affected site being the cervical area of the buccal surface of these teeth.[7,18,20]

Pathogenesis of DH

Much of the current opinion on dentin hypersensitivity is based on logical and sensible supposition rather than scientific evidence, and in attempt to explain the precise mechanism of pain transmission from the exposed dentin surface to the terminal nerve ending; various theories have been put forward.[2,21] These proposed theories, which are basically four, include the direct innervations theory, transducer theory, modulation theory by Rapp and the widely and currently accepted 'Brännström's' hydrodynamic theory,[2,8,21]

The direct innervations was one of the early proposed hypotheses which states that dentin is innervated and therefore there is direct stimulation of sensory cells that receive stimuli.[18] The modulation theory which is not so common proposes that nerve impulses are modulated by the release of certain polypeptides during pulp injury which may selectively alter the permeability of the odontoblastic cell membranes through hyperpolarization so that the pulp neurons are more prone to discharge on receipt of stimuli.[5]

Because odontoblasts are embryologically of neural crest derived mesenchymal cells, the odontoblast transducer theory states that odontoblasts act as receptor cells mediating changes in the odontoblasts via synaptic junctions with nerves, resulting in the sensation of pain from the nerve endings located in the pulpodentinal border.[5,21] Though odontoblasts have been found to be matrix forming cells they are not considered excitable and no synapses have been demonstrated between odontoblasts and nerve terminals.[3] However, recently a study[22] has shown that the odontoblasts are closely related to the nerve endings, and biological signals are probably transduced from them to the axons and vice versa thus promoting the odontoblastic transducer theory.

The Hydrodynamic theory first proposed by Gysi in1900[23] but first proved scientifically in 1964 by Brannstorm and Astrom,[24] has been the most widely accepted of the theories of DH which states that the sensitive dentin is based on the stimulus-induced fluid flow in the dentinal tubules and consequent nociceptor activation in the pulp/dentin border area. The intradental myelinated A-β and some A-δfibres are thought to respond to stimuli that displace the fluid in the dentinal tubules resulting in the characteristic short, sharp pain of the dentin hypersensitivity.[1,25]

It has also been shown that patency of the dentinal tubules is an important feature of sensitive dentine[26] with more sensitive teeth having many more (eight times) and wider (two times) tubules at the buccal cervical area compared to non-sensitive teeth.[12]

The fluid movement in the dentine is quantified by measuring the hydraulic conductance of dentin and the more and wider the tubules the higher the conductance of the fluid and the more the pain responses.[1]

Aetiology of DH

Although DH is as a result of dentinal exposure, not all exposed dentine is sensitive. Literatures have stated that DH develops in two phases which are lesion localization and lesion initiation.[26] Lesion localization occurs by loss of protective covering over the dentin, thereby exposing it to external environment. It includes 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.[7] For DH to occur, the lesion localization has to be initiated i.e a number of dentin tubules in close proximity to each other must be patent from the pulp to the oral environment and this occurs after the protective covering of smear layer is removed, leading to exposure and opening of dentinal tubules.[7]

Dentine exposure may be due to loss of enamel or exposure of the cementum through periodontal tissue loss. Various causes of dentine exposure have been documented with gingival recession being one of the major causes.[19,27] Gingival recession is more common as patient age and causes of gingival recession include the loss of the underlying alveolar bone which provides local blood supply to the buccal gingivae leading to loss of buccal gingivae.[28] Thus absent or thin fenestrated buccal bone predisposes to gingival recession. The alveolar thickness can also be affected by tooth anatomy, position and orthodontic movement.[29] Gingival recession is also caused indirectly by poor oral hygiene which leads to periodontal diseases and subsequent tissue loss.[30] The use of toothbrush is also a documented cause of gingival recession,[9,31,32] with the number of times it is used being a predisposing factor to recession of gingival tissue. Also reported is gingival

recession with good oral hygiene or improved oral hygiene which may be associated with excessive and traumatic tooth brushing thereby leading to apical migration of gingivae and exposure of the dentine.[9] The most brushed teeth with the lowest plaque scores exhibited the most gingival recession which led to the description of gingival recession/dentin hypersensitivity as "toothbrush disease." Gingival recession is also a common feature in young adults with intra- and perioral piercing as they may cause gingival tissue trauma.[33], Periodontal treatment and surgeries are also factors that have been implicated in the recession of gingival leading to remodeling and apical shift of gingival tissue.[34]

Hard tissue tooth loss with subsequent exposure of dentine secondary to tooth wear such as cervical abrasion, abfraction, erosion, and attrition are well documented.[2,9,21]. Abrasion which is the physical wear as a result of mechanical processes involving foreign substances or objects, is a major factor in the aetiology of non carious cervical lesion (NCCL)[35] resulting in angular wedge-shaped cervical lesions, generally on the buccal surfaces of maxillary canines and premolars, although such lesions can be found on the lingual surfaces of molars. Though tooth brushing with paste is the most common cause, nutrition behavior is another contributory factor.[36] The stiffness and configuration of the toothbrush bristles with abrasiveness of toothpaste in combination with force,[37] the tooth brushing method, frequency of brushing, and the duration of brushing[38] all contribute to loss of tooth structure.

Erosion is defined as the loss of tooth structure by chemical dissolution resulting from extrinsic or intrinsic acids acting on plaque free surfaces.[35] The extrinsic acid is from dietary sources of acids such as citrus fruit and drinks, acidic wines, carbonated drinks etc and intrinsic acids are largely gastric acid from inadvertent gastro esophageal reflux disease, from psychogenic vomiting syndromes (bulimia) or from the side effects of drugs that irritate the gastric mucosa or cause nausea and vomiting.

Erosive tooth wear, or acid wear, occurs in a two-stage process where the acids soften the tooth surface through demineralization within seconds and may get re-hardened through the action of saliva and fluoride within 1 to 2 hours.[21] During the softened period, if the enamel is subject to frictional or abrasive forces the surface will be permanently removed resulting cumulatively over time as an erosive lesion. Thus patient should avoid brushing 2 hours after taking this acidic diet to prevent the agonistic effect of acidic erosion on toothbrush abrasion and thereby prevent DH.[21]

Abfraction is caused by occlusal contact with excessive force and premature occlusal contact, this leads to tooth deformation and flexion, resulting in microfracture of the enamel crystals in the cervical region and contributing to the exposure of coronal dentin, and in more severe cases, of coronal and root dentin.[39] This has been documented to be one of the causes of DH,[36] however it may be co-destructive rather than a direct causal, whereby abrasion and/or erosive process are potentiated.[40]

Occlusal trauma, frenal attachments, cracked tooth and post bleaching sensitivity from hypertonic bleaching agent that stimulates flow of dentinal fluid are other causative factors that may contribute to recession and hypersensitivity however sensitivity from a tooth undergoing vital bleaching may be more of pulpal pain.[41] Also physiologically as age advances, there is exposure of root dentine which may predispose to DH.[10]

For all the above mentioned causes to lead to DH, the tubules need to be patent to the oral environment (lesion initiation) for there to be continuous outflow of fluid from the open tubules[42] and when stimuli are then applied to the dentin, the rate of flow is increased, in turn exciting the nervous system and resulting in a pain sensation.

The exposed dentine surface may have either patent dentin tubules or is covered by a smear layer of oral debris such as calcium or toothpaste ingredients.[43] The smear layer covering which is protective is acid labile and can be removed by most acidic soft drinks, citrus fruits and fruit juices, some alcoholic beverages and many herbal teas. Oral acidity which involves pH and buffer capacity of the saliva can also affect the protective effect of the smear layer.[43] Also the dentine tubules can be made patent by erosion, abrasion, attrition.[7] Toothbrushing and brushing with non occluding toothpaste due to their abrasiveness,[44] can open the tubules and thereby leading to pain.

Lesion initiation in dentin hypersensitivity can be induced by synergistic action of abrasive and erosion forces resulting in tubule opening and dentin wear.

Diagnosis

Due to the multifactorial aetiology of DH, a good history and evaluation of the patient is paramount to effective management. In diagnosing DH, it must be differentiated from other conditions that may cause teeth sensitivity and appropriate diagnosis must be made before any treatment is initiated. The characteristic response in DH to specific stimuli is pain that is sharp, localized, and brief, and usually

diminishes following removal of the stimulus.[13] Clinical conditions such as: post restorative sensitivity, postoperative sensitivity from bleaching, cracked tooth, fractured teeth or restorations, dental caries, and irreversible pulpitis should however be ruled out.[2,10] The history should include information regarding faulty tooth brushing which includes excessive brushing and scrubbing of the cervical region, or lack of tooth brushing which may lead to accumulation of plaque and subsequent gingival recession, harmful habits, frequency of intake of carbonated and acidic drinks.

Recent recommendations by Holland et al.[6] suggest that DH may be evaluated either in terms of the stimulus intensity required to evoke pain (stimulus-based assessment), or as the subjective evaluation of the pain produced by a stimulus (response-based assessment). Stimulus-based methods usually involve the measurement of a pain threshold which can be completed using pain measuring scale visual analogue scale (VAS), while response-based methods involve the estimation of pain severity with use of exploratory probe (tactile stimulus) or jets of air from a triple syringe on the exposed surface to provoke a response from the patient[45] which may identify the areas suspected of having DH and also confirm if the sensation the patient is experiencing is "Real or Imagined".

Treatment of DH

Based on the mechanism of dentinal hypersensitivity, three fundamental treatment strategies have been discussed.[46]

First strategy is to desensitize the nerve tissue by modifying the neural response within the dentine tubule. Potassium nitrate has been found to work by this mechanism, by increasing the extracellular potassium ion concentration and thus depolarizing the nerve. This disrupts the ionic tubular membrane transmission and prevents sending pain signals to the brain until ionic concentrations restabilizes and causes relief to the patient.[47]

Occluding the distal terminal ends of the exposed dentinal tubules is another strategy. This can be achieved through secondary dentine formation or mineralization or by using compounds that can precipitate an accumulation of denatured protein or a calcified plugging layer. These substances include strontium salts, sodium fluoride, stannous fluoride, monofluorophosphate, oxalates or fluoridated agents, casein phosphopeptide (CPP), 8% arginine and calcium carbonate combination.48 Various studies [45,46,49,50] have been done to show the effectiveness of these various desensitizing agents in treatment of DH and these are the commonest dentine hypersensitivity management strategy.

Dentifrices are the most common vehicles for these desensitizing agents[14,47,48] and they are widely indicated, particularly because of their low cost, ease of use and home application.[10,14] However these agents too can be available as varnishes to be painted on the tooth surface in-office [2] and as mouthwashes.[51]

Iontophoresis is another clinical technique that utilizes a charged electrical current to accelerate and precipitate insoluble calcium to occlude open dentinal tubules using topical fluoride gels which has been found to be more effective than topical fluoride application.[52,53]

The use of calcium hydroxide in office to occlude dentinal tubules has also been advocated. The high alkaline pH of the calcium hydroxide provokes odontoblastic protein coagulation precipitating the proteins and thus occluding the tubules, reducing the hydraulic conductance and thereby reducing pain.[54]

Adhesive resin impregnation is another clinical technique that has increased in popularity in recent years [2] and is currently considered one of the most definitive and rapidly acting methods of desensitization employed when there is no loss of tooth structure.[10] These adhesives in the form of varnishes and resin bonding agents reduce sensitivity with the application of a dentin adhesive to form a hybrid layer, with the barrier preventing continued diffusion of toxins and bacterial invasion towards the pulp while producing minimal adverse pulpal inflammation. The new trend in the use of adhesives for DH is use of self etch adhesive system, which prevent the collapse of collagen network and makes use of non rinse technique and simple to apply.[55] Though they produce immediate relief, they are easily removed.[56]

Bioglass which contains silica as its basic component has also been reported that it can promote infiltration and remineralization of dentinal tubules,[57] by acting as a nucleation site for precipitation of calcium and phosphate. It forms an apatite layer, which occludes the dentinal tubules,[57] and its use in bringing relief in management of DH has been documented. [38,58]

Also use of calcium silicate derived from Portland cement as been shown to help in the management of DH. It helps to occlude the dentinal tubules by remineralization.[26] The calcium silicate crystals which are 1.5-3 microns in size[2] have ability to penetrate dentinal tubules and occlude them protecting them from saliva immersion and acid penetration.[59]

Use of laser as a means of occluding the tubules has also been advocated and has been found to be more effective than other treatments,[60,61] giving about 5% to 100% effectiveness although this diminishes in severe DH.[61] While the Nd:YAG laser causes melting of dentin and closure of exposed dentinal tubules without dentine surface cracking,[62] leading to reduction of permeability and hydraulic conductance,[63] the low level laser therapy induces changes to neural transmission networks within the dentinal pulp.[64] Though reported to be very effective, when compared with conventional approaches, its high cost, complexity of use, decreasing effectiveness over time, the controversy about its mechanism of action and efficacy, limit its clinical utility.[26]

The final treatment strategy is to cover the exposed surface of the dentinal tubules. This can be achieved by utilizing connective tissue graft procedures and/or dental restorations.[65] The periodontal procedures include free, autogenous-mucosal grafts, subepithelial connective tissue grafts, a coronally advanced flap technique, guided periodontal tissue regeneration, and acellular dermal matrix grafts.[36,65]

Restorative coverage may be needed when the structural integrity of the tooth is compromised with aesthetics and functional reestablishment of deeper lesions necessary.[2,10,65] Depending on the severity of tooth loss, restorative coverage include use of conventional glass-ionomer cements, Resin-modified glass-ionomer, compomers, flowable or hybrid composites, composite or porcelain veneers, composite or all-ceramic class V inlays and crowns and porcelain-fused-to-metal crowns and bridges.[65] There may also be need for root canal treatment if attempts to achieve pain remission with a more conservative procedure fail.[10]

Quality of life with DH:

DH is a relatively common oral complaint frequently reported in clinical dental practice which manifests in manner that is physically and psychologically uncomfortable for the patient.[66] According to Addy,[8] 52% patients with DH fail to seek treatment to desensitize their teeth because they do not perceive dentine hypersensitivity to be a severe oral health problem. Thus many people live with sensitivity which interferes with their eating, drinking, oral hygiene habit, sometimes even their breathing and possibly their psychology.[66,67] A German study [67] showed that patients with sensitive teeth reported substantial Oral hygiene Related Quality of life (OHRQoL) impairment, which may have an influence on how patients should be treated. Annoyance has been shown to be one of the emotional reactions to the inconvenience and discomfort caused by the sensitivity, while functional impact included restrictions in performing everyday tasks such as eating, drinking, looking after their tooth being outside on a cold and windy day and avoidance of sporting activities.[68] The majority however try to cope with the situation by avoiding the tooth or things that can trigger the pain.

Conclusion

To obtain a conclusive diagnosis of DH, there is need to first carefully evaluate, investigate and compare among the other teeth, in order to eliminate other possible causes of pain, which could lead to confusion. Good clinical history is very essential which may reveal important information that will help in treatment.

There is also need for frequent review of the cases to be sure that you have positively affected the patients' life and improved on the reduction/ablation of the sensitivity. Many options however are now available to treat dentine hypersensitivity and eliminate the problem, in order to improve the quality of life of the patients because the sensitivity they experience is real.

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