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DENTINAL HYPERSENSITIVITY

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Dentinal hypersensitivity

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ABSTRACT

Dentinal hypersensitivity (DHS) is a common and significant dental condition typically characterized by a brief sharp pain in response to exogenous, non-noxious stimuli. This condition negatively affects patients' quality of life and may disturb their eating, drinking, brushing, and sometimes even breathing. The exact nociceptive mechanisms of DHS have not been elucidated. In this paper, the authors carried out a narrative review and explored the etiopathogenesis, presumed mechanisms, risk factors, and epidemiology of DHS. They provided guidelines and suggestions for its diagnosis and management. The process of writing took place over one month, between August 30, and September 30, 2021. During this period, the authors sought relevant works online using various databases like PubMed, Core, Z-library, and Google scholar. Google and Edge search engines were used to obtain the required literature using keywords like "dentinal hypersensitivity", "tooth sensitivity", "cervical sensitivity", "dentin sensitivity", "cervical hypersensitivity", etc. The authors extracted and documented vital information from different original articles and textbooks based on the objectives of this work. Included in this paper were systematic reviews on DHS published in the English Language. Articles that are unrelated to the topic, whose full-text was not available, and articles that were not written in the English Language were excluded. The most important risk factors for DHS seem to be the frequency and methods of tooth brushing. Thus, dental healthcare personnel (especially dental therapists) should intensify their effort in educating patients on oral hygiene and proper tooth brushing techniques. A better understanding of the underlying nociceptive mechanisms of DHS will lead to the development of improved, simple, efficient, permanent, and low-cost management techniques for the treatment of patients experiencing DHS.

Keywords: *dentin, dentinal hypersensitivity, dentinal tubules, gingival recession, differential diagnosis*

INTRODUCTION

Dentinal hypersensitivity (DHS), colloquially known as teeth sensitivity is a widely researched topic in medical literature and one of the commonest complaints among patients who visit dental clinics (Liu et al., 2020). DHS has been described as an intense pain of short duration ensuing from exposed dentin which cannot be attributed to any other form of dental defects or pathology and is worsened by innocuous stimuli. These innocuous stimuli could be thermal, evaporative, tactile, osmotic, or chemical (Bandeca et al., 2017; Mendes et al., 2021; Tusharluthra et al., 2015). DHS involves acute tooth pain and often, it hinders the patient from engaging in normal daily habits (Bekes & Hirsch, 2013). More severe DHS can last more than 6 months and cause steady irritation, inducing psychological and emotional distress, which may trigger the development of chronic dental pain

conditions that would require management as neuropathic pain (Goh et al., 2016; Lima et al., 2017).

Prabhu et al. (2017) noted that dentinal hypersensitivity is one of the most familiar clinical diseases which causes significant pain to patients. DHS should be distinguished from other clinical conditions that present with similar symptoms at their different stages of progression; some of which are dental caries, microleakage, cracked tooth, fractured restorations, etc. (Arua et al., 2021). DHS is thought to develop when the underlying dentinal tubules and dental pulp nerves are exposed to external environmental stimuli (Gbadebo et al., 2016).

A variety of physical, chemical, pathological, biological, and/or developmental abnormalities that result in dental and/or periodontal damage or defects can cause the exposure of the dentinal tubules (West et al., 2013). Some of the clinical conditions that have been implicated in the development of DHS include enamel attrition, erosion, abrasion, abfraction, and gingival recession (Bartlett, 2011; Grippo et al., 2004; Kim, 2016). Some dental treatments such as crown preparation (Brännström 1996) and whitening procedures (Jorgensen & Carroll 2002; Hewlett 2007) can also expose the dentin and cause DHS. Abuzinadah and Alhaddad (2021) stated that about 47% of the general population experience DHS but Kanehira et al. (2015) reported an average of about 57%.

Although research has established that DHS affects people of all ages, further studies have shown that it is strongly associated with age. People between the ages of 20-50 years are more often affected (Reshma et al., 2020), but it is most prevalent among those between 20-40 years (Kanehira et al., 2015; Tusharluthra, et al., 2015). DHS affects more women than men because the quest for teeth whitening is higher in women than in men (Ozen & Orhan, 2009; Spleith & Tachou, 2013). A study by Addy (2002) as cited in Gillam et al. (2013), showed that cuspids and first bicuspids are most frequently affected, followed by incisors and second bicuspids with molars being the least affected. DHS is mostly seen in patients with periodontal diseases and areas of gum recession especially at the buccal aspects of the cervical margins and may present on several teeth or one specific tooth (Arua et al., 2021; Reshma et al., 2020).

Many aspects of DHS are poorly understood by dental professionals, especially the etiology of DHS (Addy, 2005). Although the neurosensory mechanisms underlying DHS are not well understood, several theories have been proposed. These theories include the direct innervation theory, the transduction theory, the modulation theory, and the hydrodynamic theory (Vijay et al., 2011; Gilliam, 2021). Of these theories, the hydrodynamic theory which suggests that DHS is related to fluid movements within exposed dentinal tubules remains the most widely accepted explanation for DHS pain (Kim, 2016). Despite its wide acceptability, this theory has not accounted for all pain associated with DHS, nor has any effective treatment procedure that is consistent with this theory as the sole explanation for DHS been developed.

Diagnosis and treatment of DHS are further complicated by the fact that several dental conditions have symptoms that are similar to DHS at different stages of their progression. This presents diagnostic challenges for dental professionals especially new practitioners and can lead to delays in treatment further increasing patients' suffering (Davari et al., 2013; Liu et al., 2020). A survey of dentists and dental hygienists conducted by the Canadian Advisory Board on Dentin Hypersensitivity showed that nearly 50% of respondents reported a lack of confidence in managing patients with DHS-related pain (Canadian Advisory Board on Dentin Hypersensitivity [CABDH], 2003).

Despite the extensive studies that have been done on DHS, the condition remains one of the least satisfactorily treated dental conditions (Gbadebo et al., 2016). Several therapeutic approaches have been utilized in treating dentin hypersensitivity. Current approaches which include nerves desensitization and tubular occlusion (Gillam et al., 2013) involve topical rather than systemic treatment options because systemic treatments do not efficiently reach the pulp (Bandeca et al., 2017). However, for these treatments to achieve

immediate and enduring effects, they must be carried out in the dental clinic setting (Marto et al., 2019; Femiano et al., 2021). Even though DHS is among the commonest problems encountered by dental professionals, there is a lack of generally accepted strategies for differential diagnosis and dependable treatment methods (Liu et al., 2020). This paper aimed to review the presumed etiological factors that are responsible for the development and chronicity of DHS and to provide a summary of the current principles and strategies for differential diagnosis and management of DHS in dental practice.

PURPOSE OF THE STUDY

DHS is one of the commonest complaints among patients who visit dental clinics (Liu et al., 2020). Despite the extensive studies that have been done on DHS, many aspects of DHS are poorly understood by dental professionals and the condition remains one of the least satisfactorily treated dental conditions (Addy, 2005; Gbadebo et al., 2016). This paper aimed to review the etiopathogenesis, nociceptive mechanisms, risk factors, epidemiology, diagnosis, and management of DHS.

REVIEW METHODS

A narrative review was carried out to study the etiopathogenesis, diagnosis, and management of dentinal hypersensitivity. The process of writing took place over one month, between August 30, and September 30, 2021. During this period, relevant works were sought online using various databases like PubMed, Core, Z-library, and Google scholar. Published textbooks and articles were obtained. Google and Edge were the search engines used to find the required literature. Keywords such as "*dentinal hypersensitivity*", "*tooth sensitivity*", "*cervical sensitivity*", "*dentin sensitivity*", "*cervical hypersensitivity*", and so on were used. Vital information was extracted and documented from the different original articles, textbooks, and papers reviewed based on the objectives of this paper. Inclusion criteria in the study were systematic reviews on DHS published in the English Language. The authors excluded articles that are unrelated to the topic, whose full-text was not available, and articles that were not written in the English Language.

OVERVIEW

DHS has been defined as "a condition characterized by 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 dental defect or pathology" (Femiano et al., 2021). The dentin is an underlying structure of the tooth and occupies an ideal anatomical position. It is protected from the mechanical and chemical insult of the harsh oral environment by the enamel at the crown portion of the tooth and by the cementum at the root portion (Luukko et al., 2011; Borges et al., 2012).

The dentinal tubules occupy 1% (superficial dentin) to 30% (deep dentin) of the volume of intact dentin and are filled with free dentinal fluid that occupies 1% of superficial dentin but about 22% of the total volume of deep dentin (Pashley, 1996). The external drift of this fluid between the odontoblasts through the dentinal tubules is prevented perimetrically by enamel on the crown and cementum on the root. However, if the dentin is exposed, there will be nothing to stop this external drift through the tubules. This can trigger nerves along the pulpal canal of the dentin causing the pain termed "dentin sensitivity" (Matthews & Vongsavan, 1994; Soares et al., 2021).

DHS (also referred to as dentin sensitivity, dentinal hyperalgesia, cervical sensitivity, tooth sensitivity, etc.) has been reported in the literature for over a century (Gbadebo et al., 2016). The condition can mimic the clinical symptoms of other dental conditions such as reversible pulpitis making it difficult to diagnose. The severity of symptoms does not depend on the breadth and depth of lesions, but on the number of non-occluded tubules exposed to the oral cavity, so, the sealing of exposed tubules normally alleviates or eliminates the pain and discomfort of the patient (Femiano et al., 2021).

ETIOPATHOGENESIS

DHS has been documented as a clinically important dental problem for more than a century, the precise pathogenesis, particularly with the pain transduction mechanisms that play a role in DHS has not been clarified. Ideas about

its etiology are based mainly on data obtained from in vitro and in situ studies as well as from data obtained from epidemiological surveys (Liu et al., 2020). The chief etiological factor of DHS involves exposed dentin as a result of loss of enamel associated with tooth wear or trauma and/or as a result of gingival recession associated with exposure of root surfaces. Experts have determined that gingival recession, rather than cervical enamel loss, is the main predisposing factor for exposing the dentin surface. However, once the dentin is exposed erosion becomes the main factor in DHS initiation (Canadian Advisory Board on DHS, 2003). Tooth wear refers to the permanent loss of tooth structure and includes conditions such as abrasion, erosion, attrition, and abfraction (Kanehira et al., 2015).

There are two main stages in which DHS develops. First, the enamel layer protection of the tooth wears away by abrasion, erosion, attrition, and abfraction. Consequently, the dentinal tubules become exposed. Also, dentinal tubules may become exposed due to gingival recession along with the loss of cementum on the root surface of cuspids and bicuspids in the buccal surface (Arua et al., 2021). Gingival recession may be caused by toothbrush abrasion, pocket reduction surgery, tooth preparation for crowning, excessive flossing, or secondary to periodontal disease (Reshma, 2020). This stage is referred to as *lesion localization*.

The second stage termed *lesion initiation* occurs after the tubular plugs and the smear layer are removed and dentinal tubules and pulp are exposed to the external environment (Bubteina & Garoushi, 2015). The plug and smear layer on the surface of exposed dentin consists of protein and sediments derived from salivary calcium phosphates and seal the dentinal tubules inconsistently and transiently (Davari et al., 2013). Acidic soft drinks, citrus fruits, and fruit juices, alcoholic beverages, and many herbal teas remove the smear layer after a few minutes of exposure. Further, these acids can reduce the dentin surfaces' ability to resist abrasive forces due to enamel softening resulting in further dentin removal (Arua et al., 2021).

MECHANISM

A good measure of the present view on DHS is based on logical and sensible theories rather than on scientific evidence (Mantzourani & Sharma, 2013). Hence, attempts to expound on the exact mechanism of pain transmission from the exposed dentin surface to the terminal nerve ending have yielded several theories. These theories include the classic hydrodynamic theory, neural theory or direct innervation of dentinal tubules, and odontoblasts serving as sensory receptors (odontoblastic transduction theory) (Aminoshariae & Kulid, 2021).

There is also the unpopular modulation theory which suggests that nerve impulses are modulated by the release of certain polypeptides during pulp injury which may selectively change the permeability of the odontoblastic cell membranes through hyperpolarization so that the pulp neurons are more prone to discharge on receipt of stimuli (Gbadebo et al., 2016).

Following are the theories frequently discussed in the literature:

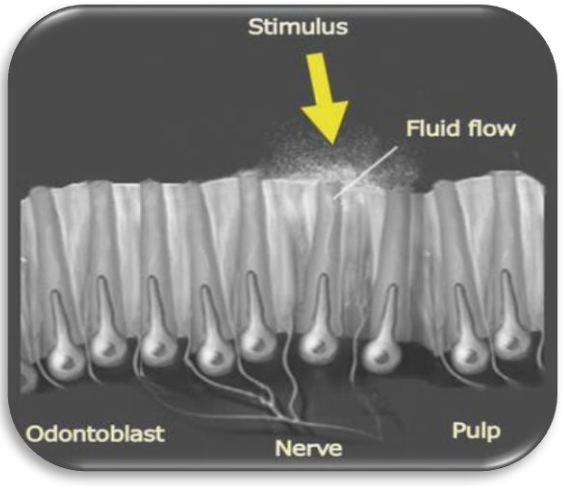
i. **The Direct Innervation Theory**: this theory supposed that dentin is innervated and so there is direct stimulation of sensory cells that receive stimuli (West, 2006). The nerve's endings enter dentin through pulp and extend to dentinoenamel junction (DEJ) and the mechanical stimuli directly transmit the pain (Cummins, 2010). But there is little evidence to prove this theory. First, because there is little proof that can support the existence of nerve in the superficial dentin where dentin has the most sensitivity; and also, because the plexus of Rashkov do not become mature until complete tooth eruption. However, newly developed teeth can likewise be sensitive (Borges et al, 2012, Chu & Lo, 2010).

ii. **The Odontoblastic Transduction Theory**: this theory was proposed by Rapp et al., (1967) and states that odontoblastic processes on the exposed dentinal surface could get excited by chemical and mechanical stimuli. These odontoblasts are

of neural crest origin and can act as receptor cells. Neurotransmitters are released on nerve excitation and impulses are transmitted to nerve endings. Nevertheless, till now, neurotransmitters have not been detected with odontoblastic processes. Most of the odontoblastic processes extend only 0.5 to 1 mm from the pulpal end. Dentin remains sensitive even after aspiration of dentinal tubules or nerve injury (Peeran & Ramalingam, 2021).

iii. **The Hydrodynamic/Fluid Movement Theory**: this was first proposed by Alfred Gysi, a Swiss dentist in 1900 but was scientifically proven by Brannstorm and Astrom (1972). It is the most popular of the theories of DHS and proposes that fluids within the dentinal tubules are disturbed by thermal, physical, or osmotic changes. These fluid changes or movements stimulate a baroreceptor which leads to the neural discharge of A-β and A-δ fibers. The basis of this theory (as illustrated in the figure below) is that the fluid-filled dentinal tubules are open to the oral cavity at the dentin surface and also within the pulp. This theory suggests that changes in the flow of the fluid present in the dentinal tubules can trigger receptors present on nerves located at the pulpal side thereby deriving a pain response (Peeran & Ramalingam, 2021).

Figure 1:
Brännström's hydrodynamic theory



Adapted from Kanehira et al. (2015)

It is important to note that these theories are not mutually exclusive. Thus, several of them may contribute to dentinal sensitivity. Knowledge of these mechanisms may prompt the development of therapeutic drugs that aim to disrupt these mechanisms, leading to more effective treatments for pulpal pain (Aminoshariae & Kulid, 2021).

RISK FACTORS

Savage et al. (2019) opined that the most important risk factors for DHS seem to be the frequency and characteristics of tooth brushing. The result of a study by Mafla and Lopez-Moncayo (2016) showed that individuals who used toothpaste with a relative dentin abrasivity (RDA) higher than 70, had gingival recession (GR), and received periodontal therapy in the last month increased the risk for DHS. The study also showed some clinical but not statistical associations between DHS and type of toothbrush bristles, pH of artificial fruit juices, a quantity of carbonated drinks per week, or pH of alcohol. There were no significant associations between DHS and psychological factors. However, subjects with higher perceived psychological stress and obsessive-compulsive symptoms had clinical greater odds of DHS (Mafla & Lopez-Moncayo, 2016).

Arua et al. (2021) added that people who suffer from bruxism, have xerostomia, consume high-acid food/drink, are obsessive brushers, have received periodontal treatment, as well as bulimics, and older people with gingival recession are more prone to developing DHS.

EPIDEMIOLOGY

DHS has a reported prevalence range of 4% to 57%. This wide range is believed to be due to differences in the population, the setting and the clinical procedure used to assess DHS, and also differences in patient perception (Cummins 2009; Zeola et al., 2019). The cuspids and first bicuspids are most frequently affected, followed by incisors and second bicuspids with molars being least affected. The buccal cervical regions are the most commonly affected (Addy, 2002; Bartlett, 2011). In a study by Orchardson and Collins (1987) 90% of cases of the hypersensitive area were reported to be at the cervical margin. But occlusal/buccal regions have become more often affected in

young adults. This is probably due to the consumption of a high acidic diet that erodes tooth surfaces and wrong methods of tooth brushing as well as the use of highly abrasive dentifrice leading to tooth wear (Jaeggi & Lussi, 2006).

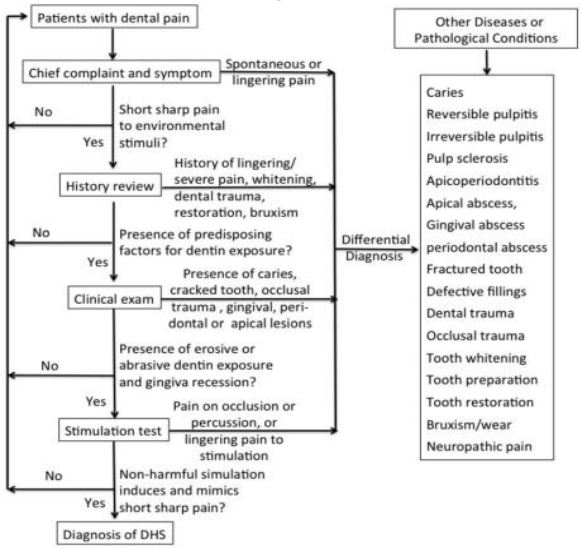
DHS can present at any age, but the majority of individuals range from 20-50 years with a peak in prevalence in the age range 30-39 years (Soares et al., 2021). DHS is more prevalent in women than men. This could be attributed to hormonal influence and dietary practices as well as the higher quest for tooth whitening. Also, because of the high record of periodontal disease and tooth whitening treatments, patients in developed countries are more prone to DHS (West, Sanz, et al., 2013). A study by Savage et al. (2019) revealed that the prevalence of DHS in young Nigerian adults (18-35years) is low compared to their European counterparts. The study suggests that about one in every three young Nigerian adults (32.8%) may have DHS. This is low in comparison with a similar European study by West et al. (2013) which reported a prevalence of 41.9%. However, Savage et al. (2019) noted that the incidence of DHS among Nigerian adults may be rising.

DIAGNOSIS

An accurate diagnosis for DHS is crucial to formulating a suitable and effective treatment (Liu et al., 2020). The diagnosis is often based on the subject's self-report of pain and requires exclusion of other dental and periodontal conditions that might elicit pain. The distinctive response in DHS to specific stimuli is pain that is sharp, localized, and ephemeral, and usually lessens once the stimulus is eradicated (Gbadebo et al., 2016). Differential diagnosis is essential to exclude other conditions with similar symptoms where dentin is exposed and sensitive, such as chipped teeth, fractured cusps, cracked teeth, caries, and restorations with poor marginal adaptation (Kanehira et al., 2015).

Skills and tact should be applied to gain the necessary information relating to a patient's history screening, identification of etiologic and predisposing factors, particularly dietary and oral hygiene habits associated with erosion and abrasion. This will help to exclude other dental conditions that present with dental pain similar to that of DHS and to make a definite diagnosis of DHS and ultimately lead to a successful treatment strategy (Bubteina & Garoushi, 2015). A simple clinical method of diagnosing dentinal hypersensitivity includes the jet of air or using an exploratory probe on the exposed dentin, in a mesiodistal direction, by examining all the teeth. The severity or degree of pain can be quantified either according to a categorical scale or using a visual analog scale (Tusharluthra et al., 2015). A step-by-step diagnostic approach for DHS is shown in the flow chart below.

Figure 2:
Flow chart for the differential diagnostic approach to DHS



Adapted from Liu et al. (2020)

MANAGEMENT

The first step to successfully managing the condition is to address any underlying causes of DHS (Arua et al., 2021). Based on the mechanism of DHS, Liu et al. (2020) identified the following management strategies:

i. Oral hygiene education and brushing technique instruction for prevention of DHS

ii. Behavioral control and elimination of predisposing factors for DHS

iii. Non-invasive treatments for pain relief through occluding dentin tubules and blocking nociceptive transduction/transmission

iv. Restoration or surgical treatments for dental hard and soft tissue defects.

Desensitization of the nerve tissue to block nociceptive transduction can be achieved by modifying the neural response within the dentin 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 restabilize and bring relief to the patient (Gbadebo et al., 2016).

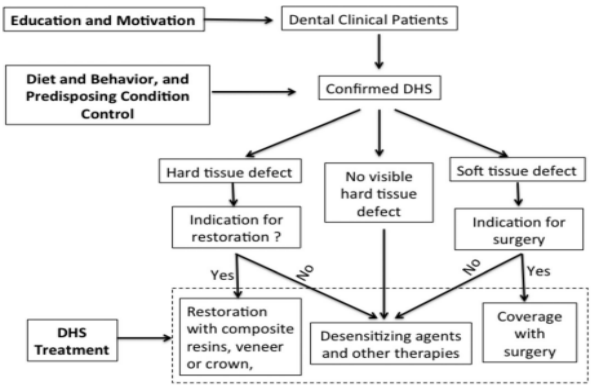
According to Gbadebo et al., (2016), the distal terminal ends of the exposed dentinal tubules can be occluded through secondary dentin formation or mineralization or by using compounds that can precipitate an accumulation of denatured protein or a calcified plugging layer. These substances include strontium salts, sodium fluoride, stannous fluoride, monofluorophosphate, oxalates or fluoridated agents, casein phosphopeptide (CPP), 8% arginine, and calcium carbonate combination.

Gbadebo et al., (2016) further posited that dentifrices are the most common vehicles for these desensitizing agents and they are widely indicated, mainly because of their cost-effectiveness, ease of use, and home application. However, these agents too can be available as varnishes to be painted on the tooth surface in-office and as mouthwashes.

Another strategy to cover the exposed surface of the dentinal tubules can be achieved by utilizing connective tissue graft procedures and/or dental restorations. 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 (Gbadebo et al., 2016).

Below is a diagrammatic guide for the management of DHS (strategies for managing DHS), as put forward by Liu et al. (2020):

Figure 3:
Strategies for managing DHS



Adapted from Liu et al. (2020)

CONCLUSION

DHS is a common and important dental problem. It is an exaggerated response to non-injurious stimuli, often characterized by short sharp pain arising from the stimulation of exposed dentin. For DHS to develop, exposure of the dentinal tubules is usually a prerequisite. Accurate diagnosis is vital to formulating an appropriate management strategy for DHS. This can be achieved through a differential diagnosis using exclusion criteria. Although the exact nociceptive mechanisms of DHS have not been elucidated, current theories have yielded some effective management strategies. Future treatment modalities for DHS that might combine the benefits of being both non-invasive and permanent yet cost-effective are being developed.

Healthy habits such as proper tooth brushing techniques, dietary control, and routine dental visits reduce the susceptibility to DHS. Nerve desensitization, dentinal tubules occlusion, restoration, and surgical treatment are the main management strategies for DHS. Specially formulated toothpaste are the most common desensitizing agents and they are extensively indicated because they are cost-effective and easy to use at home.

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