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Dentine Hypersensitivity: Review of a Common Oral Health Problem

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Abstract

Dentine hypersensitivity remains an enigma. Although, it is known to usually present with sharp, short pain caused by one or more external stimuli, it is often inadequately understood. Controversies therefore abound concerning the appropriate terminology for the condition, variations in prevalence, and what is most important to the patients suffering from the condition - a treatment that provides immediate relief with long lasting effect. Available interventions using desensitizing agents provide only temporary relief. The interest of dental practitioners and researchers in dentine hypersensitivity remains very high as it is still a common complaint among patients globally. The myriad array of treatments available and claims of efficacy has not lessened the clinician's dilemma. As a guide and update for clinicians, the aim of this paper is to review the published literature and provide current opinion on the prevalence, aetiology, mechanism, clinical features, treatment as well as the concept of oral health related quality of life among patients with dentine hypersensitivity. A manual and PubMed literature search was conducted using the keywords: dentine hypersensitivity, tooth sensitivity, cervical dentine hypersensitivity, root dentine hypersensitivity in combination with the words prevalence, epidemiology, diagnosis, mechanism, aetiology, clinical features, treatment, oral health or quality of life. The recently developed treatment agents appear promising but will require more randomized clinical trials. Dentine hypersensitivity remains a common condition with significant effect on daily life. The incorporation of a measure of oral health related quality of life is recommended as one of the outcome measures for new treatment modalities.

Keywords: Dentine hypersensitivity; Prevalence; Aetiology; Treatment; Oral health related quality of life

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Introduction

Dentine hypersensitivity (DH) is a common dental complaint in adults and is one of the most painful and least successfully treated chronic problems of the teeth. It has been defined as being characterized by a short, sharp pain arising from exposed dentine in response to stimuli typically thermal, evaporative (air), tactile, osmotic or chemical, and which cannot be ascribed to any other form of dental defect or pathology [1]. It is a chronic condition that is dependent on dentine exposure as well as the patency of the dentinal tubules. The reported prevalence of DH presents a large variation, but by general consensus, it is said that between 10% and 30% of adults report the condition globally and thus has been referred to as the "common cold" of dentistry [2,3]. Since DH is related to the incidence of non-carious cervical lesions, the global prevalence is expected to increase as more people retain their teeth into older ages [4,5]. The aetiology of DH is multi-factorial with gingival recession, abrasion, erosion and attrition as the main predisposing factors [2]. Experience of discomfort in daily life and function is common among sufferers with significant impact on their quality of life (QoL) [3,6]. A successful treatment modality should therefore be able to alleviate the symptoms and reduce or eliminate the impact of the condition on the QoL of the patients. A myriad of agents and proprietary products have been proposed as treatment for DH. There is however lack of universal acceptance of any one

treatment, with no gold standard to compare new products. This review looks at the present knowledge of DH in the literature, new treatment regimens and the recent growing focus on the effect of the condition on QoL, viewing the patient as a whole rather than just the oral cavity.

Literature Review

Terminologies

It has been suggested that the term 'hypersensitive' is not appropriate to describe the condition as no evidence could be found to show that the dentine in this case differs from a normal dentine. And also that the pulp response of the "hypersensitive" tooth is not different from a normal one, thus the suggestion of the term "dentine sensitivity" (DS) [7]. However, it is known that not all exposed dentine is sensitive, thus a case could be made for both terms. But, the term "dentine hypersensitivity" (DH) has been in use for decades by clinicians. Furthermore, a definition for the term DH was proposed and adopted in the Canadian consensus document at an international workshop on DH in 2003 [8].

Other terms such as cervical dentine sensitivity (CDS) [9,10]. Cervical dentine hypersensitivity (CDH) [11], and root dentine sensitivity/hypersensitivity (RDS/RDH) [12] have been used. The latter has been used to describe sensitivity arising from periodontal disease and its treatment. The rationale is that sensitivity following periodontal therapy may be a distinct condition from that of DH. Addy [13], in a review, agreed that even though a distinction may be made between patients with relatively clean mouths complaining of DH from those who present with complains as a result of periodontal disease and/or its treatment, both DH and RDS/RDH are the same condition since they are provoked by hydrodynamic stimulus. Thus a clear distinction between spontaneously occurring DH and that following periodontal treatment is arbitrary [14]. Generally, all these terms have been used in the literature to describe the same condition affecting different parts of the tooth.

Prevalence

Prevalence rate for DH shows a wide variation with the reported prevalence values ranging from 1.34 to 98 percent [7-15]. The reasons suggested for the wide variation include differences in the target population, selection criteria, and the method of assessment or diagnosis [4]. It is however generally agreed that surveys that rely on patient questionnaires alone may greatly exaggerate the prevalence figures [2,4]. For example, in Brazil, Fischer et al. [16] reported a prevalence of 25% of self-reported tooth hypersensitivity, but only 17% of these were confirmed after clinical examination. However, hospital prevalence figures involving clinical diagnosis as opposed to questionnaire-based studies have also been found to vary widely. In Nigeria, Bamise et al. [15] reported a hospital prevalence of 1.34% in the southwest while Udoye [17] reported a higher prevalence of 16.3% in the southeast. Rees [18], Flynn et al. [19] and Chabanski et al. [9] reported varying prevalence rates between 3.8% and 73% in the UK, while Cunha-Cruz [20] and Albashaireh [21] reported 12.3% and 28.7% in the USA and Jordan respectively. The high prevalence in the study by Chabanski et al. [9] could be the use of subjects drawn from a specialist periodontology clinic. It has been reported that those individuals complaining of DH as a result of periodontal disease and/or its treatment have provided higher prevalence values in the region of 60–98% [9].

The general consensus is that DH tends to be commoner among females. A slightly lower prevalence has however been reported among females in a few studies [15,22]. Addy suggested that the higher incidence of DH reported in females may reflect better overall healthcare and oral hygiene [15]. Gillam et al. [23] however, suggested that in the absence of a significant difference, the higher female attendance at the dental clinics where these studies were done could be the reason. In relation to age and prevalence of DH, variations in the age group mostly affected have been reported among populations. Despite this wide variation, most studies of patients with DH are predominantly within the age range of 20 to 40 years with the peak occurrence at the end of the third decade [23]. There is generally a decrease in prevalence with increasing age recorded in most studies which has been attributed to natural desensitisation of sclerosis and secondary dentine formation that occurs with ageing [2].

Aetiology

There are numerous and varied aetiological and predisposing factors for DH but no prime cause have yet been identified. By definition, DH may arise as a result of loss of enamel and or root surface denudation with exposure of underlying dentine. Dentine is structurally and functionally related to the dental pulp and thus naturally sensitive. This inherent sensitivity is usually not a problem because other tissues cover the dentine. The loss of overlying enamel, as a part of tooth wear may result from attrition, abrasion, erosion or abfraction. However, enamel loss in reality is a combination of these processes but often with differing proportional effects. Though different authors have stated the tooth wear process believed to be prominent in the rate of enamel loss, the debate is far from over. Bamise et al. [24] and Özcan and Çanaci [25] reported attrition as the most common aetiological factor for DH in their studies. Bamise et al. [24] suggested that the attrition was due to consumption of coarse diets by the patients in their study.

In contrast, available literature indicates that majority of studies favour erosion and abrasion as the main types of tooth wear lesions causing DH. It has been documented that the abrasive effect of toothbrushing and toothpaste especially silica-based toothpastes causes gingival recession, root exposure and dentine abrasion [25,26]. It has however been suggested that the abrasivity of tooth brushing alone on enamel and dentine is too minimal to cause DH and that the brushing techniques and the relative dentine abrasivity (RDA) of the toothpaste considerably contributes to the development of DH [13,27]. Özcan and Çanaci however argued that the abrasive effect of toothbrush on the gingiva could result in gingival recession with subsequent exposure of the softer root surface to the abrasive effect of tooth brushing [25]. The smear layer that occludes the abraded dentine undergoes dissolution by erosion from acidic

drinks and food [28,29], leaving the tubules patent. Abrasion and erosion may act either in an additive or a synergistic manner in this tooth wear process [27]. The effect of erosion in the initiation of DH is supported by Bamise et al. [24] who reported a strong association between DH and consumption of citrus drinks and carbonated drinks in patients with DH. McCraken et al. [30] also found an association between the frequency of toothbrushing, gingival recession and DH. This is supported by studies that show that DH is commoner on the buccal cervical areas of the teeth on the left side of the mouth [17-24]. Sehmi and Olley [31] attributed the association between gingival recession and DH to higher brushing forces received by this area predisposing it to gingival recession, abrasion and DH. Abfraction, which are microfractures due to stresses at the cervical junction of the teeth have also been proposed and accepted to be a cause of DH [24-29]. It is however the least aetiological factor associated with DH [24,25] and the least studied among the aetiology factors. There is thus an indication for further studies to characterize the abfraction phenomenon and its contribution to the development of DH [30,31].

Mechanism of dentine hypersensitivity

Subsequent to dentine exposure and loss of the "protective" smear layer over the dentine, pain results from thermal, evaporative (air), tactile, osmotic or chemical stimuli introduced into the mouth. Several theories such as the odontoblastic transduction theory, neural theory and hydrodynamic theory have been proposed to explain how the triggering stimuli elicit the pain of DH. With no conclusive evidence to support the odontoblastic transduction and the neural theory, the hydrodynamic hypothesis first proposed by Gysi [32] with confirmatory evidence produced by Brännström and colleagues [33,34] remains the most widely accepted theory of DH. This theory postulates that rapid shifts, in either direction, of the fluids within the dentinal tubules, following stimulus application, result in activation of sensory nerves in the pulp/inner dentine region of the tooth. The basis of this theory is that the fluid-filled dentinal tubules are open to the oral cavity at the dentine surface as well as within the pulp. Microscopic examination reveals that patent dentinal tubules are more numerous and wider in hypersensitive dentine than in non-sensitive dentine [2]. This thus enhances fluid permeability through dentine and as such increase the possibility for stimulus transmission and subsequent pain response [33]. These observations are consistent with the hypothesis that dentinal pain is mediated by a hydrodynamic mechanism.

Clinical features

Patients with DH usually complain of and present with discomfort, inability to brush their teeth and pain on application of stimuli including cold, air, acidic drinks and tooth brushing. DH can be a particularly uncomfortable and unpleasant sensation for patients and can dictate types of foods and drinks ingested. The pain arising from DH is extremely variable in character, ranging in intensity from mild discomfort to extreme severity and may emanate from a single tooth or several teeth. The degree of pain varies in different teeth and in different persons. A spontaneous change in the degree of DH over time has been described, and has been ascribed to possible neurogenic inflammation resulting from chronic low-grade stimulation [14].

There are conflicting reports on the teeth commonly affected by DH. Most studies however agree that it is most common in first premolars and canines [16,18]. The reason is most likely due to the location of both types of teeth at the corner of the dental arch, making them most susceptible to toothbrush abrasion. For this reason, they are prone to gingival recession and tooth defects. Another point to support this assertion is the minimal response to stimuli on the lingual surface when compared to the buccal surfaces of the teeth [9,35]. In addition, the general intra oral distribution of DH is noted to resemble that of gingival recession in right-handed individuals where teeth on the left and buccal surfaces are commonly affected compared to other sites in the mouth [2,36].

Diagnosis of dentine hypersensitivity

The first part of the definition of DH describes a clinical entity, but other conditions may present with similar symptoms. The second part of the definition therefore considers differential diagnosis that requires different management strategies [4]. For appropriate diagnosis, Gernhardt [37] has recommended a verbal screening, during which the patient is asked if there is pain when eating or drinking hot, iced, cold, or acidic food or beverages. Confirmation of at least one of these questions would necessitate obtaining a complete history from the patient. The pain arising from DH is usually described as being rapid in onset, sharp in character, and of short duration [2,38]. Occasionally, the pain may persist as a dull or vague sensation in the affected tooth after removal of the stimulus. Patients also report a wide variety of pain-producing conditions and a large combination of stimuli has been recorded in the literature [2,37]. Hypersensitive teeth may be sensitive to one form of stimulus but not to others. The reason is unknown and requires further investigation. It is agreed however that the most common stimuli eliciting pain in patients suffering from DH is cold stimulus [2,12].

The history will assist in identifying the risk factors the patient may have for hypersensitivity and should include: the dietary history; oral hygiene practices including tooth brushing technique, frequency, duration and timing of brushing; previous dental therapies like professional tooth cleaning, vital tooth bleaching and restorative procedures. The effect of these factors as well as the presence of other factors, and their contribution to the establishment of the condition will be ascertained during intraoral examination. A clinical examination would be necessary to confirm clinical signs associated with the DH (e.g., dental erosion, gingival recession, and exposed cervical dentine) in patients with a positive history suggestive of DH. Since DH is a diagnosis of exclusion, a thorough differential diagnosis is very important to eliminate all other forms of dental pain that present with similar symptoms. These includes but is not limited to cracked tooth syndrome, fractured restorations, restorations left in traumatic occlusion, dental caries, root caries, postoperative

sensitivity, marginal leakage of restorations, and pulpitis [33,38]. Furthermore, clinical examination specific for DH is necessary in cases with positive results in the first examination steps. The most commonly used diagnostic tools are air jet from an air-water syringe and scratching the tooth surface with a sharp dental explorer. These tactile and evaporative stimuli should reliably provoke the DH-associated pain.

Management of dentine hypersensitivity

Management strategies for DH which include prevention are usually more successful than treatment alone. Identifying and controlling the aetiological factors can do this most effectively. Preventive measures are primarily aimed at reducing the risk of exposing dentine, either as a result of the removal of enamel, usually caused by erosion, or the removal of cementum, most often attributed to either overzealous tooth brushing in a healthy mouth or periodontal disease and or treatment [28-39]. Ciaramicoli [40] while assessing the effectiveness of lasers demonstrated the importance of prevention in a study that showed that the efficacy of laser treatment of DH increased when aetiological factors were removed.

Treatment

Numerous agents have been investigated with claims of various degrees of effectiveness, including corticosteroids, silver nitrate, zinc and strontium chloride, formaldehyde, glutaraldehyde, calcium hydroxide, sodium citrate, potassium oxalate, resin adhesives and fluorides. Treatment approaches attempt to reduce pain by: either targeting the pulpal nerves to block neural transmission; or occluding the dentine tubules, thereby blocking the hydrodynamic mechanism [2,38].

Generally, these agents may be in the form of dentifrices or gels and mouth rinses or in the form of topically applied agents such as resins, varnishes, primers and dentine bonding agents. Procedures such as periodontal grafting procedures and laser therapy have also been used. Grossman [41] listed the requirements for an ideal dentine desensitising agent as: rapidly acting with long-term effects, non-irritant to pulp, painless and easy to apply, and should not stain the tooth. In spite of the different array of surface treatments for DH, no single treatment meets all of the ideal criteria. There is therefore no gold standard with which to compare new product proposed for treatment of DH. At present, treatment choice seems to depend mostly on the practitioner's experiences and personal preferences [38]. Many topical desensitising agents do not adhere to the dentine surface and are lost eventually, thus limiting their efficiency over time [35]. Furthermore, some toothpaste, specially designed for treating DH have a low abrasion coefficient and cannot withstand the abrasive effects of toothpastes, with loss of treatment efficiency over time [42].

Objective assessment of the reduction in dentinal pain by the treatment agents is difficult. Generally, observation on pain reduction or abolition is subjective and subject to individual interpretation, since pain perception depends on several factors. The lack of standardized methods used in clinical trials of treatment modalities for DH was cited by the authors of the consensus report on DH in 1994 as the major problem [14]. Thus; the authors emphasized the need for well-designed protocols for carrying out controlled clinical trials for investigating the effectiveness of treatments for DH. In response to this, Holland et al. [1] in 1997 proposed guidelines for the design and conduct of clinical trials on DH. Their recommendation included the method of assessment of dentinal pain, and the need for both negative control/placebo and positive control agent group when testing the effectiveness of a new agent for treatment of DH.

"At home" treatment

The "at home" treatment agents includes dentifrices, mouthwashes and chewing gums. Toothpastes are the most widely used dentifrices for delivering over-the-counter desensitising agents with as high as 67.9% of individuals reporting DH using a desensitising toothpaste on a regular basis in a study done by Lavigne [43]. Toothpastes are widely indicated, particularly because of their low cost, ease of use and home application. They are effective but it often takes four to eight weeks to achieve pain relief [43]. Other challenges associated with the use of toothpastes as desensitising agents include compliance, difficulty of delivery to specific sites and requirement for continuous use which also encourages noncompliance. Presently, most desensitising toothpastes contain a potassium salt such as potassium nitrate, potassium chloride or potassium citrate. However, conclusions drawn from a systematic review by Karim and Gillam [44] did not find available evidence that strongly supports the efficacy of potassium salts in reducing DH.

In-office treatment

Dental professionals can deliver a wider range of more complex and more potent desensitising treatment with immediate relief from pain of DH. A variety of office applied agents are currently available, which include cavity varnishes, calcium compounds, oxalates, resins and adhesives, restorative materials, laser treatment and an aqueous solution of glutaraldehyde and hydroxyethyl methacrylate. These products generally occlude and seal the exposed dentinal tubules and in most cases a macroscopic coating is apparent [38,44]. Besides the application of various agents to the exposed sensitive dentine, the use of periodontal grafts and guided tissue regenerative procedures have been advocated for treatment of root DH in the presence of gingival recession [38]. Laser treatment has also been recommended for treatment of DH. Studies have reported that the neodymium: yttrium-aluminum-garnet (YAG) laser, the erbium:YAG laser and galium-aluminium-arsenide low level laser all reduce DH [45-47]. A more recent literature review on the use of lasers in treatment of DH has suggested that though it has a slight clinical advantage over topical medicaments, further studies with larger sample-sizes, long-term, high-quality randomised controlled clinical trials are needed before definitive conclusions can be made [48].

Recent developments in the treatment of dentine hypersensitivity

Over the last decade, advances have been made in various areas of management of DH. This include advances in toothbrush technology, abrasivity of toothpastes, new agents for treatment of DH, improved material science as a result of nanotechnology, improvements in periodontal flap procedures, and QoL of patients suffering from DH. Toothbrush technology has brought improvements to the standards of safety, design, texture, type of filament, etc. which, together with a dentifrice formulation, has both oral health and cosmetic benefits. The RDA scale has been introduced as a measure of the abrasive potential of commercial toothpastes [39]. This allows comparison between different pastes as a higher RDA indicates a greater tendency to cause abrasion, a factor that may also hamper the efficacy of desensitising toothpastes [26-49]. Toothpaste with low or moderate RDA should therefore be recommended for safety, and as a preventive advice on tooth wear.

Recent research suggests that Arginine, an amino acid, which occurs naturally in saliva and calcium carbonate significantly, increased DH relief [50,51]. The formulation derived from this combination, Pro-Argin, has been described as a novel occlusion technology based on the fact that 8% arginine and calcium carbonate are believed to seal exposed dentinal tubules with a plug that contains arginine, calcium carbonate and phosphate thereby providing relief for DH [52]. This claim has been supported by the results of studies which demonstrated relief of DH for variable periods following the use of Pro-Argin mouthwash or paste [52,53]. Although previous systematic reviews have recommended arginine-containing toothpastes for treatment of DH [50,51]. West et al. [49] and Yang et al. [54] in more recent reviews of clinical trials and Idon et al. [55] in a recent clinical trial did not find enough evidence to recommend the professionally applied formulation for treatment of DH over the other available agents.

Recently, NovaMin®, a product based on bioglass technology containing 15% of calcium sodium phosphosilicate (CSPS) has been introduced into the market. In an aqueous environment, such as saliva, the CSPS releases sodium ions, which leads to an increase in the local pH. This process results in the rapid formation and precipitation of a calcium hydroxyapatite mineral layer on the dentin surface [56]. Although Zhu et al. [57] concluded that CSPS was more effective than negative control based on a review of several studies, Talioti et al. [58] in their review concluded that its effectiveness is only supported by low-level quality of evidence. So, though effective as an in-office treatment agent, it is not recommended over the other professionally applied agents.

Improved material science with the aid of nanotechnology has found a role to play in the treatment of DH. This includes the use of 3-layered membrane for guided tissue regeneration in regenerative procedures for patients with DH associated with localised gingival recession [59]. The incorporation of nanoparticles in composite resins, and fluoroaluminosilicate glass has given rise to a new class of materials with improved resistance to abrasion [60]. The use of nanocomposites and nanoionomers to treat cervical DH with structural tooth loss as a result of abrasion may better be able to withstand the abrasive effects of toothpastes, and thus prolong treatment efficiency over time.

DH and oral health related quality of life (OHRQoL)

Until recently, the management of DH did not consider the effect of the condition on the oral health related quality of life (OHRQoL) of sufferers. More recent concepts of health shift the focus to the effect of the condition on the lives of those affected. A substantial number of patients with DH experience symptoms severe enough to interfere with their eating, drinking, oral hygiene habits and sometimes even breathing [3,15]. These symptoms are highly relevant from the patients' point of view and often have a considerable adverse impact on their daily QoL [3,6]. The concept of OHRQoL among patients with DH is therefore gaining popularity with growing awareness of the importance of patients' perspective in pre and post treatment evaluation. Very few researchers have however assessed the impact of DH on the QoL of the patients or effect of intervention on the QoL. In two separate studies Bekes et al. [61,62] used the Oral Health Impact Profile (OHIP), a widely used generic multi-item questionnaire to assess the impairment of OHRQoL caused by DH. They were able to show a significantly better QoL in the general population in comparison to patients suffering from DH, as well as improvement in the QoL of patients after treatment intervention. Idon et al. [63] in their study also used the OHIP and reported significantly improved prevalence of impact and overall QoL following in-office treatment of patients that presented with DH. Using a different OHRQoL measure, specific for DH, the dentine hypersensitivity experience questionnaire (DHEQ). Machuca et al. [64] assessed the impact of DH on QoL in a group of patients, arriving at the same result as the earlier studies; that DH significantly impacts on QoL.

Conclusion

DH largely still remains an enigma, poorly understood and is essentially a diagnosis made by exclusion. Though an old problem, the prevalence of DH remains high, and in spite of the vast literature on the subject and the new advances in treatment, there is still lack of a consensus among researchers on the ideal treatment modality. It may however be comforting to know that a lot of research is still ongoing in achieving the ultimate goal in the treatment of DH which is immediate and permanent relief of pain. In line with the growing acceptance of the QoL model of health, assessment of new treatment interventions should include patients' perception of treatment outcome.

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