Review Article

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DENTINE HYPERSENSITIVITY: A REVIEW

Abdul Saheer[1*], Shanila Abdul Majeed[2], Pallavi SK[3]
1.Post Graduate student, Department of Public Health Dentistry,Vokkaligara Sangh Dental College And Hospital,Bangalore
2.Post graduate student,Department of Oral Medicine and Radiology, Dayanada Sagar College of Dental Science,KS layout,Bangalore
3.Reader,Department of Public Health Dentistry,Vokkaligara Sangh Dental College And Hospital,V.V.Puram,Bangalore

ABSTRACT:

Dentine hypersensitivity is one of the prevalent disease among aged population which results in compromising quality of life to a lesser extent. Literature explains various theories and explanations about the etiopathogenesis. Prevalence of dentine hypersensitivity varies among country to country and even within the same populations based on the study criteria used and age group of the study population.

Keywords: Dentine hypersensitivity,etiopathogenesis,Prevalence

INTRODUCTION:

Dentinal hypersensitivity is a common painful condition of the teeth, associated with the exposure of dentine. Tooth hypersensitivity, or more precisely known as dentine sensitivity or hypersensitivity. The suitability of the terms dentine sensitivity and dentine hypersensitivity have been questioned since both terms are often used to describe the same clinical condition. Although it has been suggested that true hypersensitivity can develop as a result of pulp inflammation, the symptoms are thought to be more severe and persistent than the typical short sharp pain of dentine hypersensitivity. [1]

Dentine hypersensitivity is a response from a non-noxious stimuli and a chronic condition with acute episodes whereas dentinal pain is a response from a noxious stimulus and usually an acute condition. Dentine hypersensitivity remains a worldwide under-reported and under-managed problem, despite making some dental treatments more stressful than necessary and having a negative impact on the patient's quality of life. Dentine hypersensitivity can be particularly uncomfortable and unpleasant for patients and can dictate types of foods and drinks ingested. Patients may describe the condition as dull or sharp, vague or specific and intermittent or constant. [2]

Dentine hypersensitivity is defined as “sharp short pain arising from exposed dentine in response to stimuli typically thermal, chemical, tactile or osmotic and which cannot be ascribed to any other form of dental defect or pathology. [3]

Terminologies

Dentine hypersensitivity/ Dentine sensitivity /Dentinal sensitivity-Pain or sensitivity felt at the exposed dentine surfaces

*Corresponding Author Address: Dr Abdul saheer Email: zaheer5475@gmail.com

Cervical hypersensitivity/sensitivity- Sensation of pain in the cervical region

Root hypersensitivity/sensitivity- Cemental hypersensitivity/sensitivity- Pain in the cemental regions due to the exposed root surfaces.[4]

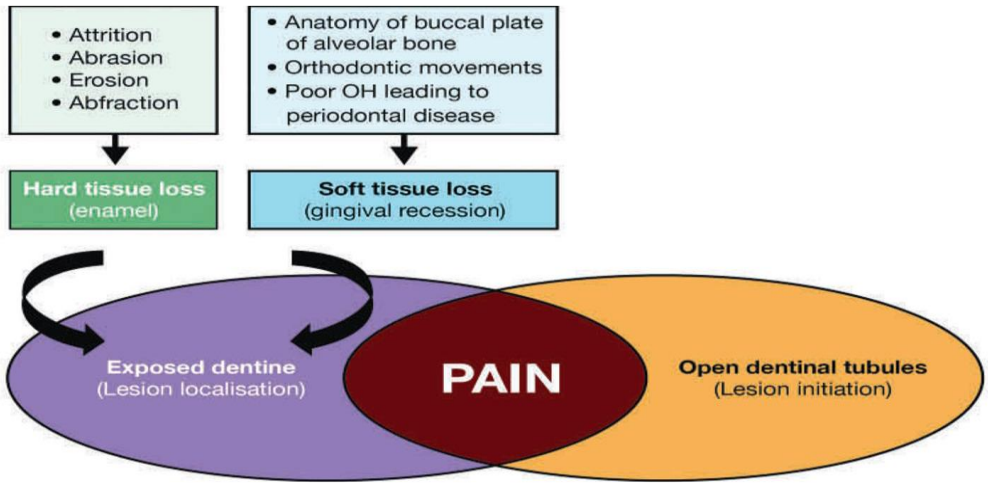
Phases in the development of dentine hypersentivity

Based on the studies, DH is developed in two phases.[1,5]

1. Lesion localization
2. Lesion initiation

1. **Lesion localization**- In the first phase, dentinal tubules, due to loss of enamels, are exposed by attrition, abrasion, erosion, and abfraction. However, dentinal exposure mostly occurs due to gingival recession along with the loss of cementum on the root surface of canines and premolars in the buccal surface. It is worth noticing that not all the exposed dentins are sensitive. Their calcified smear layer, as compared to non sensitive dentin, is thin and this leads to an increase in the fluid movement and consequently the pain response**.** [6,7]

2. **Lesion initiation-** In the second phase, for the exposed dentin to be sensitized, the tubular plugs and the smear layer are removed and consequently, dentinal tubular and pulp are exposed to the external environment.[6] Plug and smear layer on the surface of exposed dentine are composed of elements of protein and sediments which are derived from salivary calcium phosphates and seal the dentinal tubules inconsistently and transiently. The findings of laboratory research indicate that both mechanical and chemical factors are effective in removing the smear layer from the dentinal tubules. However, the results of clinical investigations, the mechanical factors are not the only key factors in removal of the smear layer and when they are accompanied with acidic foods or drinks they lead to the removal of smear layer**.** [15,20]Lesion initiation requires removal of cementum or smear layers. This is achieved by abrasive or erosive agents. The evidence available indicates erosion is the more dominant factor but can be potentiated by abrasion.



Structural differences between sensitive and non-sensitive dentine

There are the differences in the structure of sensitive *versus* non sensitive dentine. Sensitive dentine appears to have more dentinal tubules per unit area than non-sensitive dentine (eight times as many tubules at the root surface compared with non-sensitive teeth) and the channels are wider, with the average diameter of tubules in sensitive teeth being two times greater than that of tubules in non-sensitive teeth (0.83 _m *versus* 0.4 _m, respectively).[8] It has also been shown that smear layers in sensitive dentine are thinner and under calcified compared with those on non-sensitive dentine. [9] The greater number of open and wider tubules leads to increased fluid permeability through dentine and therefore increased stimulus transmission and, eventually, the pain response.

THEORIES OF DENTINE HYPERSENSITIVITY

1.Transduction /direct innervations theory:

According to this theory, the odontoblast has a special sensory function and the functional complex with the nerve ending in or near the odontoblastic layer acts as an excitatory synapse. The odontoblast and its process have been perceived as a transducer mechanism. This theory of dentinal sensation takes into consideration the "synaptic" like relationship between the odontoblastic processes. If the true synapse were present between these two elements to facilitate the transmission of dentinal sensation, then a neural transmitting substance such as acetylcholine would be expected in this area of the odontoblastic process and the predentine. There is no direct evidence for the presence of acetylcholine activity in the neural transmission in the pulp.[25] Direct mechanical stimulation of these nerves will initiate an action potential. There are many shortcomings of this theory. There is lack of evidence that outer dentin, which is usually the most sensitive part, is innervated. Developmental studies have shown that the plexus of Rashkow and intratubular nerves do not establish themselves until the tooth has erupted; yet, newly erupted tooth is sensitive. Moreover, pain inducers such as bradykinin fail to induce pain when applied to dentine, and bathing dentine with local anesthetic solutions does not prevent pain, which does so when applied to skin.[10]

2. Modulation theory:

According to Turkar, the nerve impulses in the pulp are modulated through the liberation of polypetides from the odontoblasts, when injured. These substances may selectively alter the permeability of the odontoblastic cell membrane through hyperpolarization, so that pulp neurons are more prone to discharge upon receipt of subsequent stimuli. When an

irritating stimulus comes to contact with the dentine, the odontoblast may become injured and subsequently release a variety of neuro transmitting agents as well as vasoactive and pain producing amines and proteins. These substances may modulate associated nerve fiber action potentials by increasing neuronal cyclic AMP levels through cell membrane adenylate cyclase receptors .[10]

3. **Gate control theory: Ronald Melzack and Patrick Wall, 1960**

When the dentine is irritated (for example-by cavity preparation) the pulpal nerves become activated from vibrations. The larger myelinated fibers may accommodate to the sensations. The smaller c fibers may tend to be maintained and not adjust to the stimulus. Thus as the low intensity "pain gates" from the larger fibers are closed, the high intensity "pain gates" from the smaller fibers are enhanced. "pain gates" may be opened by some stimuli, such as anxiety, and may be closed by distracting stimuli such as "audio analgesia" or 'gingival stimulation'. However the gate control theory does little to explain how pain responses from the dentine are transmitted and perceived by the nerve endings of the pulp. [10]

4. **Hydrodynamic theory: Proposed by Brännström and Astron in 1964 .**

According to Brännström's hydrodynamic theory, when an appropriate stimulus is applied to the outer dentin surface, there is a displacement of the contents of the dentinal tubules that gives rise to a mechanical stimulation of the pain at the pulpodentinal border. This theory is the most widely accepted until now and consider that the stimulation of the nerve endings next to the odontoblastic layer is provoked by the variation of the intrapulpal pressure toward the pulp or in the opposite direction, depending on the stimulus nature. The nerve fibers stimulation occurs because of the deformation of these fibers, caused by the fluid movement, leading to a widening of the nerve membrane ionic canals, allowing the entrance of Na++ in the cell, depolarizing the fibers and provoking pain. The presence of lesions involving enamel and/or cementum loss in the cervical area and the consequent opening of dentinal tubules to the oral environment, under certain stimuli, allows the movement of dentinal fluid inside the tubules, indirectly stimulating the extremities of the pulp nerves, causing the pain sensation.

The hydrodynamic theory is the most widely demonstrated and accepted physiopathological theory of DS. According to this theory, most pain-inducing stimuli increase centrifugal fluid flow within the dentinal tubules, giving rise to a pressure change throughout the entire dentine. This in turn activates the Aδ intradentinal nerves (of medium conduction velocity) at the pulp-dentinal interface, or within

the dentinal tubules - thereby generating pain. Two phases must coincide in order to produce DS. In effect, dentinal exposure (location of the lesion) must occur, and the dentinal tubular system must open or become permeable towards the pulp compartment (start of the lesion). Dentinal exposure may be secondary to loss of enamel or periodontal tissue (gingival recession). Enamel loss or dental wear is due to attrition, abrasion and erosion, and although dental erosion is the most important single factor to be taken into account, increased dentine wear and tubular aperture occurs when it acts in synergy with abrasion.

EPIDEMIOLOGY

Prevalence

Dentinal hypersensitivity appears to be a common problem with various reports globally indicating an incidence between 1 to 98 per cent of the population. One reason for this large discrepancy relates to the variations in the methods of data collection. The diversity of reports may be in part caused by different methods used to diagnose the condition and it is generally considered that surveys which rely on patient questionnaires alone greatly exaggerate the prevalence figures and thereby yield misleading data.

Several studies indicate that even though high percentages of a population may report to have sensitive teeth, a much smaller proportion are actually diagnosed as having cervical dentine hypersensitivity on the basis of defined clinical diagnostic criteria. In general, it appears that the incidence of hypersensitivity in most populations ranges between 10 to 30 per cent of the general population.[11]The incidence of DH can vary considerably depending on the cohort being studied with periodontal patients, patients with gingival recession and smokers with periodontitis showing the highest incidence of diagnosed dentinal hypersensitivity.

Most studies describe a preferential order of pain location according to the type of teeth involved. The **canines and first premolars** are the most affected teeth, followed by the **incisors and second premolars**. The molars are the teeth least affected by DS. As regards the anatomical location of DS, most cases are circumscribed to the **bucco-cervical region** of permanent teeth. [12]

Most affected patients are in the **20-50 years** of age interval, with a peak between **30-40 years** of age. As regards to the sex distribution, DS affects women more often than men. [13] It has been suggested that with the lifespan of the general population increasing, and more people keeping their teeth longer, hypersensitivity will increase in prevalence. This seems to make sense on the basis that gingival recession and loss of enamel and cementum is more prevalent in older individuals. The

above assumptions are somewhat confounded by reports in the literature which indicate that most sufferers of dentine hypersensitivity range in age from **20 to 40 years** with the peak incidence occurring at the **end of the third decade** and decreases during the fourth and fifth decades of life**.** [14] This may be explained by the decrease in permeability of dentine and neural sensitivity with ageing. Such responses may arise from the natural desensitization of sclerosis and secondary dentine formation. In addition, long-term use of fluoride dentifrices can add to the occlusion of open dentine tubules resulting in a decrease in sensitivity**.**

Reasons for the variability of prevalence values recorded in the literature, including: the different study designs used to assess the condition, variation in patients' oral hygiene habits,consumption of erosive foods and drinks, and the type of setting where the study was carried out. It was claimed that questionnaire studies tend to overestimate the prevalence as mentioned previously. Virtually 50% or more of adults have complained of dentine hypersensitivity in studies based on patients' perception of this problem**.** [14] Furthermore, studies carried out at hospitals or specialty practices tend to report higher prevalence values. The prevalence was observed to be greater in patients referred to specialist periodontology clinics and hospital clinics than in general practice patient populations, presumably because of the greater risk of root exposure as a result of periodontal attachment loss and gingival recession following periodontal treatment.

Even though numerous studies have been conducted on this topic, a bulk of them has focused on a specifically targeted group of general practices, which in turn, might have biased the findings. Accordingly, there is a clear need to explore the nature of dentine hypersensitivity in a randomly selected sample of general practices. Such an investigation would no doubt enhance the understanding of this problem amongst the dental practitioners and consequently benefit patients to maintain better oral health

Summary of Various prevalence studies

Study	Country	No	Study type	Setting	Prevalence (%)	Peak of age	M: F ratio	Commonly affected teeth	% with GRa [1]
Bamise et al. [16](1991)	Nigeria	2165	Q[2]+ CE[3]	University	1.34%	4th decade	1.4:1	Molars	12.8%
Rees and Addy [17] (2003)	UK	5477	Q + CE	GDP[6]	2.8%	4th decade	1:1.5	Max[7]1st molars	93%
Rees et al.[18](2004)	Hong Kong	226	Q + CE	PSC[8]	67.7%	5th decade	1:1.5	Mand[9]incisors	76.8%

Clayton et al.[19](2008)	UK	250	Q	GDP	50%	3rd decade	1:1	Mand right sextant	NA [10]
Tanni and Awartani [20] (1987)	Saudi Arabia	259	Q + CE	GDP + PSC	GDP 42.4% PSC 60.3%	4th decade	GDP 1:4 PSC 1:2	Max molars and mand anteriors	5%
Gillam et al. [13](1966)	UK and Korea	557	Q	GDP	52- 55.4%	3rd and 4th decades	NA	NA	NA
Fischer et al. [11](1991)	Brazil	635	Q + CE	Marine dental clinic	17%	M: 6th, F: 3rd decade	1:1	Incisors and premolars	NA
Flynn et al. [21](1987)	UK	369	Q + CE	University	8.7%	4th decade	1:1	Premolars	NA
Gillam et al.[22] (1999)	UK	277	Q	GDP [6]	52%	3rd decade	1:1.4	NA	NA
Chabanski et al.[23] (2008)	UK	51	Q + CE	PSC	72.5-98%	5th decade	1:1	Molars	NA
Liu et al. [24](2006)	Taiwan	780	Q + CE	University	32%	NA	1:1	Premolars & molars	23%
Vijaya et al[25] (2013)	India	655	Q+CE	University	55%	3rd decade	1:0.8	Molars	NA
Rane P et [26]al(2013)	India	960	Q	Hospital	42.5%	4th decade	1:0.9	Lower anteriors	NA
Dhaliwal JS et al[27](2012)	India	650	Q+CE	Rural population	25%	6th decade	1:1.3	Lower anteriors	NA

1gingival recession,
2questionnaire,
3clinical examination,
4sensitivity to air blast,
5periodontal disease assessment,
6general dental practice,
7maxillary,
8periodontal specialty clinic,
9mandibular,
10not applicable,
11cold water mouth rinse

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