Hypersensitivity- An Update

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Abstract

Dentinal hypersensitivity (DHS) can be defines as a short sharp pain that occurs as a response to various stimuli on exposure of dentinal tubules to the external environment and which cannot be described as any other disease. The most important part of the treatment is to identify the etiology behind the sensitivity and treating the condition accordingly. Differential diagnosis may include pulpitis, carious teeth, chipped tooth, cracked tooth syndrome etc. The treatment modalities depend upon whether the sensitivity is due to gingival recession, tooth wear lesion or periodontitis or related causes. This review highlights various strategies to counter this disease as well discusses various desensitizing agents recently in use like Novamin, Biomin, Calcium phosphate dentifrices, Nanoparticles and herbal desensitizing agents.

***Keywords**: Dentinal hypersensitivity, Desensitizing agent, Pain*

Introduction

Dentine hypersensitivity (DHS) is a very common but an exigent task to manage among the dentists. Although many agents and techniques have been studied and applied, it still remains a testing job to manage and treat this condition. It is a circumstance that affects the overall quality and standard of living of people.(1) As per Gillam et al, there is still a lack of buoyancy among the dentists in managing this condition.(2) The objective of this review is to provide an update on the modus operandi to treat dentinal hypersensitivity and to also provide an update on the latest desensitizing agents and procedures.

DHS can be clinically described as hyper responsiveness in the form of pain of the exposed or disclosed dentinal tubules to different types of stimulus. (3) Further, it has a characteristic short and sharp pain which occurs as a result of exposure to various types of stimuli like evaporative, osmotic, tactile, chemical or thermal and also it cannot be attributed to any other form of defect or pathology.(4) However, the Canadian Advisory Board proposed for modification of this description slightly by replacing the term pathology with disease.(5) Rationale for this is to challenge the clinicians to consider other possibilities for the discomfort or pain like cracked tooth syndrome, carious tooth, uncontoured restorations, fractured cusps etc.

Synonyms used for this condition are root sensitivity, cervical sensitivity, cementum sensitivity or hypersensitivity and dentine sensitivity.

Prevalence

The prevalence of hypersensitivity varies from 4 to 57% making it a very regular finding among the general population with a female predilection.(6) People with periodontitis are mostly affected. Around 60 to 90 % people with periodontitis have hypersensitivity. (7) Teeth affected include cuspids and bicuspids of both the arches, mostly the buccal site.(8) 90% of the dentinal hypersensitivity have been observed to be at the cervical region of the tooth.(9) However, clinically it has been observed that the occlusal sites have also been affected due to the excessive occlusal wear and various abusive habits. In a systematic review by Zeola et al in 2019, the prevalence ranges from 11 to 33 percent.(10) Dentinal hypersensitivity although being the most common pathology, has been treated with less success according to some studies.(11) Thus validating the purpose of this

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review. DHS has also been associated with the gingival recession among 23% individuals.[12] Hypersensitivity following or during periodontal therapy have also been observed.[13] Therefore, for the DHS to occur exposure of the dentinal tubules to the external environment is mandatory. However, the liaison between the wearing of tooth, gingival recession and abrasion has not been explored.

ETIOPATHOGENESIS

Three mechanisms have been proposed-

- Direct Innervation
- Odontoblast Receptor
- Hydrodynamic/ Fluid movement

Of the three theories that have been proposed, the fluid movement theory of Brannstrom and Astrom is the most widely acknowledged. [14] The reason being it stresses on the dynamics of fluid movement in the dentinal tubules that activates the nerve endings resulting in pain sensation. Although, the other two theories (Direct innervation and Odontoblastic receptor) provide evidence for the causation of sensitivity but are undermined by their respective drawbacks. Direct innervation theory fails to prove the synapse between the nerve fibres and the odontoblast and Odontoblast receptor theory fails to explain the extension of the odontoblastic processes to the DEJ. Hydrodynamic theory on the other hand states that when exposed dentinal tubules are subject to external stimuli, it results in alteration of the rate of fluid flow in the tubule causing a change in pressure that activates the nerve endings causing DHS.

The rate of flow of the fluid in the dentinal tubules is dependent on the diameter of the tubule according to Poiseuille's law. Thus, as the diameter of the dentinal tubule increases towards the pulp, the rate of flow thus increases and sensitizing the nerves near the junction of pulp and dentin to cause pain or sensitivity.[15] At a molecular level, the etiology of DHS has been based on 3 hypotheses, none of which can be discarded. The first hypothesis is based on the expression of various receptors (thermal, mechanical, chemical) by the afferent fibres. The second hypothesis is based on the passage of fluid within the tubules on stimulation. The third is based on the presence of ion channels on the odontoblastic processes that extends to the pulp-dentin complex.[16]

Clinically, it occurs due to the exposure of the dentin either due to removal of enamel (faulty tooth brushing, attrition, parafunctional habits, trauma from occlusion) or due to denudation of cementum due to loss of periodontium(recession, following periodontal surgery, periodontitis).[17] The pathogenesis can be described based on two phases namely Localization of lesion and Instigation of lesion. Localization is basically exposing the dentine to the external environment and initiation or instigation of lesion is through the erosive agents that eliminate cementum and the smear film.[18]

SCREENING OF DENTINAL HYPERSENSITIVITY

Screening of the patients on their first or subsequent dental visits is an age old but effective method to identify the sensitivity issue. Questions like whether the patient is having any discomfort in their daily activities like drinking, eating or while breathing through the mouth can help screen patients suffering from this condition. Frequently the initial symptoms is tend to be ignored by the patients leading to difficulty in managing DHS in later stages and more invasive procedures need to be used to manage the condition. Therefore, proper screening of the patients needs to be done to efficiently manage and treat in a cost effective process.

HISTORY

Proper history regarding the types of food or drinks that the patient consumes on a daily basis needs to be taken after proper screening of the condition. History regarding the type of pain also needs to be taken. Whether the pain lingers on consumption of cold foods and drinks and the localization of the pain also needs to be asked. The systemic health of the patient is an also an important factor that needs to be considered and hence proper history regarding medical and dental problems needs to be enquired. History regarding extreme intake of certain foods like citrus fruits, carbonated drinks and wines needs to be taken. A proper history would decide the road of management that the clinician needs to consider to manage the condition.

OBJECTIVE METHODS TO DETECT DENTINAL HYPERSENSITIVITY

The methods could be divided into two parts i.e. stimulus based assessments or response based assessments.

The stimulus based assessments include various types of stimuli like-

- Tactile
- Thermal
- Osmotic
- Electrical

ü Tactile stimuli – The simplest method is to use a sharp explorer and run the pointed end over the area of complaint.

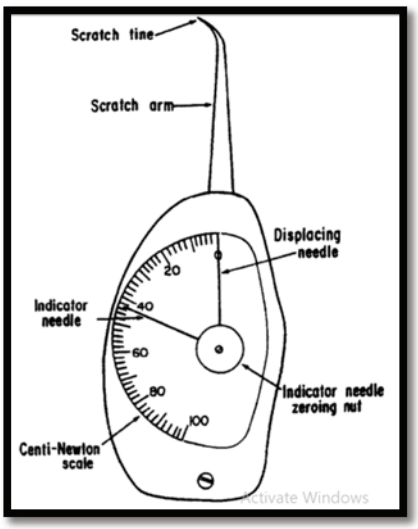

Figure 1 – Hand held scratch device

Kleinberg et al in 1990 devised an instrument to measure the sensitivity calculated by the force of displacement when pressure is applied as shown in Figure 1. A tooth is declared to have no sensitivity issues if it does not respond till 80 centi-newtons.[19]

Figure 2- Yeaple's probe

Another method was used by Schiff et al in 1994 using Yeaples probe (Figure 2) which is sensitive to pressure and uses electromagnetic force to control the amount of force applied.[20]

ü Thermal stimuli – Simplest method is to use a three way syringe at the sensitive site to record the findings. However, Smith and Ash et al in 1964 developed a device that uses a thermister probe to allow flow of current to that area and record the finding as shown in Figure 3.[21]

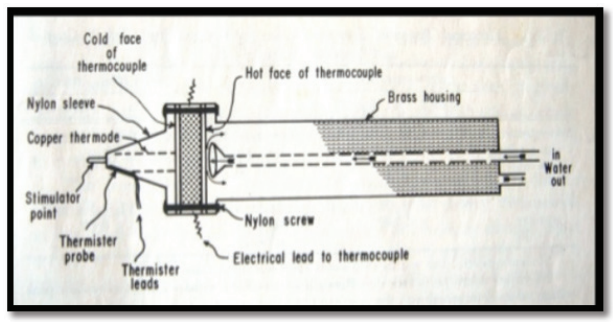

Figure 3- Smith and Ash device

ü Osmotic stimuli – Mcfall and Hamrick et al in 1987 devised an osmotic method to identify hypersensitive teeth by the use of sweet stimuli using the principle of osmosis.[22]

ü Evaporative stimuli – Brannstrom and Astrom et al first demonstrated that air blast can cause movement of fluid due to the evaporation of the fluid on that area.[14]

These assessments have some limitations that needs to be considered which is repeated stimulus can result in wrong outcomes as the threshold will increase and also the number of teeth that would be tested in a particular appointment would be less.[23]

However the response based assessments like Visual analog scale(VAS), Verbal response scale(VRS) would provide a more consistent outcome since a steady stimulus would be applied.[24]

DIFFERENTIAL DIAGNOSIS OF DHS

It is very important to eliminate the other possibilities that could cause similar kind of pain as that of sensitivity. Some of the defects can be eliminated by taking proper history like whether the pain lingers following removal of stimuli or how long the pain does last or is the pain occuring at night time only. Some defects that could be in the differential include broken tooth (bite test), cracked tooth syndrome, carious tooth with periapical pathology(tooth tender on percussion), gingival infection or inflammation(dull pain of pain deep into bone).[25] Symptoms like dull pain, lingering pain, referred pain from other sites removes the possibility of DHS. On exclusion of these defects it can be concluded that the pain is due to hypersensitivity.

MANAGEMENT OF DENTINAL HYPERSENSITIVITY

Management strategies should be based on the etiology of the DHS. The main objective should be to remove the etiological factor to prevent relapse of the condition and therefore the management should be strategized accordingly.

A. If the condition is mechanical trauma causing gingival recession, then it can be managed by various ways such as History of good plaque control, Diagnosis of mechanical trauma being the underlying cause, Patient should be made aware of the involved sites, Patient should be educated about brushing technique (Modified Stillman's technique), Patient should be advised to refrain from consumption of carbonated drinks and sweet foods, At office desensitizing treatment for pain relief, At home desensitizing agent and mouthwash should be advised with appropriate brushing technique, Follow up should be done for the patient regarding severity of DHS and brushing technique should be assessed, In case the DHS persists, surgical intervention to be done for root coverage.

B. If the condition is tooth wear lesions, then it can be managed by various ways such as History of excessive enamel loss and etiology to be identified (traumatic brushing, parafunctional habits, endogenous acids or exogenous dietary habits), Severity of tooth wear to be recorded with an apt index, Patient advocated an oral hygiene agenda to prevent tooth wear and diet dairy needs to be maintained, High fluoride remineralization is advised, Professional desensitizing treatment to be done, Systemic condition to be checked by medical practitioner for GERD, At home desensitizing toothpaste to be advised with appropriate brushing technique to avoid trauma by extreme brushing, Regular follow up to be done for tooth wear and potential etiology to be discussed, along with assessment of diet diary, Endodontic followed by prosthodontic intervention to be done if DHS persists.

C. If the condition is periodontal disease, then it can be managed by the following ways such as Previous history of periodontal problems and poor oral hygiene, Patient shown involved site and probable cause for exposure to be explained to the patient, Risk factors of periodontal disease to be discussed with patient with proper medical and personal history regarding deleterious food habits, Patient advised appropriate at home oral hygiene maintenance protocol, Management to be done non surgically followed by treatment for DHS, Periodontal parameter to be recorded for specific sites, Follow up to be done, If it persists, surgical treatment to be done alone with at home desensitizing procedures and maintenance of oral hygiene, Follow up with proper recording of periodontal parameters with desensitizing treatment to be done.

DESENSITIZING AGENTS

Scherman and Jacobsen et al in 1992 classified based on the basis of –[26]

- Method of application
- Treatment at home
- Treatment at dental office

v Mechanism of action

v DESENSITIZING NERVE- KNO_3

v PRECIPITATION OF PROTEIN COMPOUNDS- $ZnCl_2$, $AgNO_3$, Gluteraldehyde

v DENTINAL TUBULE OCCLUSION- NaF, SnF, KHC_2O_4, $Ca_3(PO_4)_2$, $CaCO_3$, Arginine

v ADHESIVE SEALERS- Flouride varnish, GIC, Composite, Resins

v LASERS

v HERBAL DESENSITIZING AGENT- Propolis, Triphala, Lavanga, Palakya, Cinnamon

There are various desensitizing agents available in market such as

A. Potassium nitrate(SENSODYNE PRONAMEL): It's mechanism of action is due to its oxidizing nature and/or due its crystallization process. Zawaideh et al in 2017 stated that ProNamel reduced the change in microhardness occuring due to acids.[27]

B. Oxalates (Oxagel): It's mechanism action is as a precipitation of oxalates and occlusion of tubules thus preventing dentinal fluid flow.[28] Pereira et al in 2005 stated that the oxalates tend to be resistant to dissolution by acids.[29] However Cunha Cruz et al in 2011 stated that there is no added benefit from the use of oxalates.[30]

C. Calcium Phosphate dentrifices(G.C TOOTH MOUSSE): It's mechanism action is as combining ACP and Casein Phosphophopeptide to localize at the plaque area and results in remineralization of enamel at a very fast rate.[31] Tung et al in 2003 stated that they imitate the natural process of sclerosing dentin and is very biologically compatible.[31]

D. Amorphous calcium sodium phosphosilicate (NOVAMIN): It's mechanism action is as effectively delivering silica, calcium, phosphorus and forms an occlusive layer over dentin. Kakodkar G et al in 2013 stated that it has the dual advantage of bleaching and densensitization.[32] Millemann J et al in 2012 stated that it has the potential clear white spot lesions.[33]

E. Fluoride containing Bioglass (BIOMIN, NUPRO): It's mechanism action is as high flouride release and the substantivity for 8-12 hours and its microparticles contents that help penetrate the tubules deeper providing better effect. Cruz et al in 2018 stated that incorporation of bioactive glass helps accentuate the process of treating DHS.[34] Reddy et al in 2019 concluded that Biomin proved to be better than other dentrifices like strontium chloride and potassium nitrate at the end of 4 weeks.[35]

F. Arginine (PRO ARGIN TECHNOLOGY): It's mechanism action is as using the physiological pH of saliva for deposition of a calcium rich layer in tubules and its arginine, calcium carbonate and bicarbonate contents as a buffer. Hamlin et al in 2009 concluded that dentrifice containing 8% Arginine and calcium carbonate significantly reduced hypersensitivity when as a pre procedural toothpaste. Bekes et al in 2017 conducted a study to evaluate the efficacy of *% Arginine and calcium carbonate and concluded that it reduced DHS over a period of 8 weeks.[36]

G. Nanoparticles Hydroxyapatite (DESENSIBILIZE NANO-P PASTE): It's mechanism action is as a Nano hydroxyapatitie particles have the same structure, morphology as the HA in enamel and dentin, thus replaces it better. Wang et al 2016 concluded that Desensibilize Nano-P paste effectively treated DHS significantly comparable with Pro argin toothpaste and potassium nitrate.[37]

H. TRIPHALA: It's mechanism action is as obtunding action on the dentinal nerves for its eugenol content. Kumari et al in 2013 stated that a herbal desensitizing dentrifice containing Triphala, spinach, suryakshara helps in reducing DHS over a period of 12 weeks.[38]

I. PROPOLIS: It's mechanism action is as occlusion of the dentinal tubules for its resins, wax calcium and aluminium contents and thus preventing fluid movement. Kripal et al 2019 in an invitro study showed that Propolis varnish significantly reduced the open tubules, thus has a desensitizing effect.[39]

Conclusion

Dentinal hypersensitivity is a pathology that needs to be managed carefully and in meticulous manner. In this review several strategies and desensitizing agents

have been discussed which if used as and when required could possibly treat hypersensitivity issues. However, there are many desensitizing agents that are undergoing research which could provide faster relief and thus give an effective treatment to the patients.

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Ethical Issues: Approved

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