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Review Article

Dentin Hypersensitivity: An Unsolved Enigma

Running Title: ***Dentin Hypersensitivity***

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Abstract

A specific condition which needs to be distinguished from other causes of dental pain and calls out for diagnosis is termed as Dentin Hypersensitivity. The literature published till date, states that lesions of dentinal hypersensitivity show presence of numerous, wider and open dentinal tubules at the dentinal surface and are patent to the pulp than non-sensitive dentin. Hence, the cause of dentin hypersensitivity is multifactorial and can manifest when dentin is exposed due to the loss of cementum or enamel, which may occur because of poor oral hygiene, excessive tooth brushing, exposure to chemical products, post-periodontal therapy, endogenous acids, as a side effect of bleaching, faulty occlusal contact or medication.

Key words: dentin hypersensitivity; root sensitivity; sensitive dentin; desensitizing agents; desensitizers; tooth sensitivity

Introduction:

Dentin hypersensitivity (DH) is a painful condition of teeth associated with short sharp pain arising from exposed dentin in response to various stimuli typically evaporative, osmotic, thermal, tactile or chemical. Despite extensive research, till date there is no gold standard developed for the treatment of dentinal hypersensitivity.1 Numerous terms were introduced in the literature for dentin hypersensitivity like, dentin sensitivity, dentin hypersensitivity, dentinal hypersensitivity, cervical hypersensitivity, cervical sensitivity, root hypersensitivity, root sensitivity, cemental sensitivity and cemental hypersensitivity.

The European Federation of Periodontology recommends the term 'Root Sensitivity'(RS) to describe: short, sharp pain from exposed dentin of periodontally involved teeth or following periodontal treatments (Sanz and Addy 2002). Certain points to be considered in support of this term: Periodontally involved teeth do not fit in the definition of DH, the prevalence of RS is very much higher compared to DH (Chabanski et al 1996), deep bacterial invasion of the dentin tubules has been reported with the periodontally affected teeth (Adriaens et al 1988).

To fathom the notion behind the extensive research carried out for dentinal hypersensitivity, it is important to condense dynamics of the entire pulp-dentin complex. The present literature review was being envisioned with a view to provide a succinct assessment of the DH's origin, pathophysiology, mechanism, and clinical therapy.

Definitions: 1-3

Harris 1878 defined 'Sensitive dentin' as "A painful condition of a tooth, attributed by some to the disturbance of the soft fibrils radiating from the pulp into the tubules of the tooth, or the conduction of shock of the instrument to the pulp".

Johnson et al 1982 defined DH as "An enigma, being frequently encountered but ill- understood".

Another definition suggested by Dowell and Addy in 1983 was adopted by the international workshop on the design and conduct of clinical trials for the treatment of the condition in 1997. The definition states, "Dentin hypersensitivity is 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 form of dental defect or pathology".

At an international meeting, Holland et al 1997, defined DH as short sharp pain arising from exposed dentin in response to stimuli, typically thermal, tactile, evaporative, osmotic or chemical and which cannot be ascribed to any other form of dental defect or 'pathology' ('pathology' as subsequently changed to 'disease') (Canadian Consensus Document 2003).

West in 2006 along with Addy and Smith defined DH as 'a specific condition which needs to be distinguished from other causes of dental pain and calls out for diagnoses.

ETIOLOGY: 4-7

A) Loss of enamel, or

B) Denudation of the root surface or loss of covering periodontal structures usually termed as 'gingival recession'.

Loss of enamel due to:

a) Atrrition associated with occlusal function

b) Abrasion from dietary components or habits such as tooth brushing

c)Erosion associated with environmental or dietary components

d)Iatrogenic

e) Abfraction

f) Tooth whitening procedures

g) Post-restorative sensitivity

h) Fractured restoration

Denudation of root surfaces occurs due to:

a) Advancing age

b) Cracked tooth

c)Chronic periodontal disease

d)Tooth abnormally positioned in the arch

e) Periodontal surgery

f) Incorrect tooth brushing habits

g) Root preparation

h) Repeated root planning for those 5-10% cases where cementum and enamel fail to meet each other could also expose dentin and result in dentinal hypersensitivity.

Tooth whitening procedures are often known to involve the use of bleaching agents like carbamide peroxide, which further breaks down into hydrogen peroxide, urea and bleaches the tooth. This causes dehydration within the tooth, and thus further initiates DH. However, the symptoms are known to be temporary and lasts for a shorter period of time.

In essence, it has also been suggested that the periodontal disease along with any non- surgical or surgical periodontal therapy may expose dentin, bacteria may further colonise the exposed sites and breakdown the smear layer for penetrate into the root dentin (Adriaens 1989) which inturn initiates further inflammation within the pulp. Chronic periodontal disease and its ongoing periodontal therapy, increases pulpal pressures, nerve spouting reduction in pain thresholds, scar tissue and reduced plasma proteins may worsen DH (Pashley 1984).

Developmental anomalies like amelogenesis imperfecta, dentinogenesis imperfecta as well as other conditions of enamel and/or dentin that predispose to hypomineralisation and hypoplasia.

Dietary factors (Erosive diets) and Gingival Recession are one of the most commonly associated factors. West et al 2013 carried out in-vivo study on 3,187 European adults of France, Finland, Spain, Italy, United Kingdom, Latvia and Estonia to investigate the presence of or otherwise absence of DH upon varied etiologies. The authors concluded the subject's experiencing DH upon clinical examination following an evaporative stimulus were 42%, 29% of these subjects' showed signs of tooth wear upon Basic Erosive Wear Examination (Bartlett et al 2013), 40% of the population having gingival recession experienced DH.

Tooth wear is an important factor associated with DH. It is irreversible, non-traumatic loss of dental hard tissues due to varied etiologies and further classified as: Attrition, Abrasion, Erosion, Abfraction and Ablation (Bartlett and Smith 2000; Ganss et al 2006). In dentistry, Smith and Knight 1984 were the first to distinguish tooth wear could be physiological and active or pathological in origin. The rate of tooth wear is comparatively fast in nature in young patients than adults and it could also be pathological in nature as opposed to physiological. As the pathological tooth wear progresses into dentin, the radius of dentinal tubules become larger and thus, the distance to the pulp decreases. As a result, the hydraulic conductance of dentinal fluid increases and further initiates symptoms associated with DH (Pashley 1990). Crucially, the physiological tooth wear takes time to show signs since the pulp initially lays down reparative or secondary dentin, which could prevent fluid flow within dentinal tubules and may also reduce DH (Krauser 1986). Erosion is considered to be the most common major factor responsible for tooth wear in Europe (Grippo et al 2004; Lussi 2006).45-48

Prevalence And Distribution: 8,9

In prevalence and distribution, literature evidence states that DH most commonly affects younger individuals aged between 20-40 years, specific teeth, tooth sides and sites. Females have been most commonly seen to be affected than males and at a younger mean age but not all literature evidence supports this statement. The available distribution data evidence states : Canines and first pre-molars, followed by incisors and second pre-molars and finally molars is the order of teeth most commonly affected; left sided teeth affected frequently more compared to right side along with their contralateral counterparts; the site of pre-dilection most commonly noted is buccal cervical region; also few authors comment that lesions could either show presence of plaque or may also be in absence of bacterial plaque (Addy et al 1987; Fischer et al 1992). A recent review on the burden of DH by Cunhan-Cruz suggested prevalence of DH is 10% with an average of 33% across the globe.

DH can occur at any tooth, any site, where the dentin is exposed (lesion localisation) and where the dentinal tubules are patent (lesion initiation). Lesion localization could occur through dentin exposure caused by loss of enamel and/or gingival recession (with loss of cementum). Loss of enamel in absence of any trauma could be owing to a process involving attrition, abrasion and erosion alone or more usually in combination. On the other hand, Abfraction (cervical tensile stress) has been hypothesized to pre-dispose cervical enamel to abrasion and/or erosion (Grippo 1991). Erosion is another major factor associated with the loss of enamel, further leading to dissolution and demineralization of surface layers. The source of acid could be intrinsic as well as extrinsic. In-vitro studies till date indicate lesion initiation is most commonly investigated through scanning electron microscope (SEM) carried out on extracted teeth.

Absi et al 1987 concluded, sensitive dentin reveals large number of patent dentinal tubules. Furthermore, the dentinal tubules in non-sensitive dentin are covered by a 'smear layer' made up of collagen and hydroxyapatite (Pashley 1984), to initiate DH, this layer needs to be removed. Although toothbrushing alone is known to be very slow to remove a smear layer, nevertheless if used after an acid challenge, could readily make a way for patent dentinal tubules (Absi et al 1992). Similarly, dentifrices are also known to remove smear layers due to interaction with abrasive ingredients. Brannstrom 1966 and Pashley 1984 defined 'Smear

layer' as "A thin 'loose' layer consisting of organic collagen and glycosaminoglycans that form an adherent matrix over mineralized tissue arising from saliva and dentin particles that might occlude the dentin tubules." 10-13

Impact Of Dh On Oral Health-Related Quality Of Life: 14

The term 'Quality of Life' (QoL) was first introduced by the British economist Arthur Cecil Pigou (1920). It is defined as "As an individual's perception of his/her position in life, in the context of the culture and value systems in which he/she lives, and in relation to his/her expectations, goals and concerns (World Health Organization, 1993)." Further Oral health continues to have impact on health and disease on QoL is known as Health-related quality of life (HRQoL) (Naito et al 2006).

Oral health-related quality of life (OHRQoL) is one subset of HRQoL which focusses on oral health and oro-facial concerns. It is defined as "A multi-dimensional construct that reflects people's comfort when eating, sleeping and engaging in social interaction; their self-esteem; and their satisfaction with respect to their oral health (Department of Dental Health and Human Services (DHHS) 2000; US)".

Mechanism Of Dentin Hypersensitivity: 15

The principal cellular element extending at the pulp-dentin border are the Odontoblasts, they show presence of process extending approximately one-third the distance to the dentino-enamel junction (Byers and Sugaya 1995). Few physiological and gene expression studies demonstrated that these odontoblasts respond to pain provoking stimuli unlike sensory receptors (Magloire et al 2010, El Karim et al 2011, Chung et al 2013).

Stephan 1937 explained the persistence of pain associated post application of a stimulus is commonly associated with acute inflammation of pulp. Ishikawa et al 1969 studied that the areas associated with sensitive dentin show presence of patent dentinal tubules.

Johnson et al 1971 along with Brannstrom et al 1980 studied that in individuals showing complete absence of any symptoms associated with DH, was the result of formation of dead tracts with formation of irregular secondary dentin or laying down of sclerosed dentin. One of the most striking features associated with DH, is the Sensitivity. Pain is the most overwhelming sensation appreciated by the pulp-dentin complex. Although, numerous electron microscopic studies, histochemical and auto-radiographic studies have vividly described the relationship between the odontoblasts and the neural fibers of the pulp, the exact mechanism of transmission of the pain response from the dentin to the terminal nerve endings is only hypothesized. Lundy and Stanley 1969 explained that there might be more severe clinical responses commonly associated with acute histological changes in the pulp along with the inflammatory response scores recorded upon the response to cold stimuli. Newman 1978 and Addy 1982 stated, few adversities were encountered post periodontal therapy where the newly exposed surface dentin progressively becomes more sensitive during initial post-operative days and weeks.

Numerous theories put forwad to explain the mechanism of dh:

1)Direct neural stimulation

2)Transducer theory

3)Modulation theory

4)Gate control theory

5)Hydrodynamic theory

The most commonly accepted theory for the DH is the Hydrodynamic Theory.

Hydrodynamic theory:

Fish 1927 studied the movement of interstitial fluid inside the dentin and pulp. He postulated that the flow of this fluid could take place in either an outward or inward direction simulating

the pressure variations within the dentinal tubules is the basis for the transmission of sensations. Even when the intensity of pain response hardly refrains co-relating with the shift in the magnitude, the dentinal tubules act as conduits wherein the external stimuli evoke pain transduction mechanism (Chidchuangchai et al 2007).

In-vitro studies on mechanism of fluid movement inside the dentinal tubule in response to tactile, thermal, evaporative, chemical and osmotic stimuli were studied by Brannstrom and colleagues (Brannstrom 1962,1967). Later on, Brannstrom's theory was renamed as the "Hydrodynamic Theory". The hydrodynamic theory states, a novel adaptation of fluid flow sensing mechano-receptors to the unique anatomical environment of the pulp-dentin complex. Although, pain is produced due to rapid displacement of the dentinal tubule contents along the pulpo-dentinal border as opposed to the normal slow outward flow of the dentinal fluid (Brannstrom 1966). In-vitro application of physiological pulpal pressure of about 30mmHg (Beveridge and Brown 1965) for a period of 24 hours has resulted in a fluid flow of 0.6µl/sq of exposed fractured dentin (Johnson et al 1973). This gradient would vacant a patent dentinal tubule in a period of 24 hours. However, this pressure was insufficient to displace cells into the tubules when student in-vivo (Brannstrom and Astrom 1964); nevertheless, cell aspiration is common beneath 'leaky' restorations but it does not correlate with the occurrence of pain (Kramer 1955). However, pain is proved not only by the fluid shifts but also the intra-dental nerves respond directly to pain-provoking stimuli regardless of the dentin fluid shifts evoked by those stimuli (Chung et al 2013).

Nonetheless, the application of stimuli such as cold water (Brannstrom and Johnson 1970), hypertonic solutions, e.g; sugar, calcium chloride (Brannstrom 1962), absorbent material (Brannstrom and Astrom 1964, 1972), probing or air blasts (Brannstrom et al 1967) cause rapid outward movement of the tubular contents accompanied by the sensation of pain. Later on, it was estimated that the pain producing stimuli create an outward flow of fluid within the dentinal tubules every 2-4mm/sec (Berggren and Brannstrom 1965). Brannstrom and Johnson (1978) summarized that rapid flow within the pulpal region of the dentinal tubule could result in deformation, not only of the cellular processes but also of the nerve fibers which might be present in the dentinal tubules or adjacent to the pulp. The application of the heat caused an inward movement of the tubular contents along the pulpo-dentinal border which would refrain activation of the nerve endings and further deviate from severe pain. In the subsequent studies, Brannstrom and Astrom (1972) studied that an elevation in temperature by 30º C did not cause any pain, whereas an equivalent reduction in temperature invariably did so. Low temperature food and beverages, in part, stimulate intra-dental nerves directly by activating thermal receptors located at the nerve endings (Park et al 2006).

The perception of acute thermal stimulation is very well explained by the hydrodynamic theory. The coefficient of thermal expansion of the dentinal tubule fluid is about 10 times than that of the tubule wall. Therefore, application of heat causes expansion of the dentinal fluid and conversely, application of cold will cause in contraction of the fluid with both resulting in an excitation of the mechanoreceptors.

It is one of the common clinical findings that pain is experienced upon consumption of sweet and salty food. The pain subsides upon rinsing or brushing away. This again can be explained by movements of the tubular fluid. Fluids of a relatively low osmolarity (i.e dentinal tubule fluid) show tendency to flow towards solutions of higher osmolarity (i.e salt or sugar solutions), similarly no stimulus is felt upon consumption or application of iso-osmotic solutions.

The Anatomical Substrates of Dentinal Hypersensitivity: 16

In non-carious and periodontally healthy teeth, dentin is covered coronally by enamel and apically by cementum. These external layers seal the dentinal tubules so well that smallest of intra-dental fluid shifts occur smoothly in response to tactile, thermal, evaporative, chemical

and osmotic stimuli. Dentin reacts by hyperconductivity, upon the removal of surface layers of a tooth and exposure of the dentin. In this state, the hydrodynamic stimuli elicit intra-dental fluid shifts, further which evoke pain.

Dental pulp is richly innervated with a numerous nerve fiber. Only a few from those 1000- 2000 nerve endings found in each tooth reach the dentin. Of these, 75% are non-myelinated while 25% are myelinated. The myelinated nerves are further classified as A-α, A-β or A-δ nerve fibers depending upon the diameter of their axon and their conduction velocity.

Since, the dentin is highly permeable, it results in DH, in case it would not have permeability, it would be unlikely to show severe sensitivity. The procedures like root planning along with certain restorative procedures leave the dentin surface covered by a layer of burnished cutting debris commonly called as the smear layer (Pashley 1984). During these procedures, cutting debris is forced within the dentinal tubule orifices approximately 1µm, covered with further 1µm of smear layer. Additionally, this smear layer decreases the hydraulic conductance (capacity of dentin to react to pressure-driven, convective flow), about 10-30 times (Pashley et al 1981; Carrilho et al 2007). Furthermore, this smear layer along with smear plugs once dissolved by the organic acids present in the diet or that produced by micro-organisms present in bacterial plaque, expose the dentinal surface, making dentin more susceptible to sensitivity (Pashley 1986).

Physiological Factors Altering Dentin Permeability and Sensitivity: 17

Dentin experiences sensitivity, due to its inherent permeable nature. Under pathologic conditions, dentin becomes less permeable due to formation of reparative dentin. The reparative dentin generally has fewer dentinal tubules than the primary dentin, secondly it also has fewer nerves innervating the dentin. Since, diffusion and convection are two mechanisms responsible for the permeation of substances across dentin. Diffusion is the process by which substances from area of high concentration are transported to area of low concentration. The driving force in diffusion is the concentration gradient or chemical potential energy and not bulk movement of any fluid. Whereas in convection, there occurs bulk movement from an area of high hydrostatic pressure to an area of low hydrostatic pressure.

According to the hydrodynamic theory, the fluid movement is responsible for transducing numerous (physical, thermal, osmotic, evaporative and thermal) stimuli into electrical nerve activity. This type of dentinal fluid movement can be quantified by measuring the hydraulic conductance of the dentin, which is the reciprocal of resistance. This states that the dentin with inherent high conductance has a low resistance. Certain variables regulating hydraulic conductance of dentin are the length of the tubules (i.e thickness of dentin), the total number of tubules per unit surface area, the applied pressure, the viscosity of the fluid along with the radius of the tubules rose to the fourth power. These are expressed in the "Poiseuille-Hagen equation".

$$Q = \pi \Delta P N r^4$$

$$8\eta L$$

(Hagen-Poiseuille Law for capillary flow) Where Q = volumetric fluid flow (µL)

N = number of pores (i.e., tubules per unit area)

ΔP = hydrostatic pressure difference across dentin (cm H2O-1) r = radius of dentin tubules

η = viscosity of fluid

L = length of dentin tubules (cm)

$$Lp = Q / \Delta P A t$$

where Lp = hydraulic conductance (µL cm-2 min-1 cm H2O-1) Q= volumetric fluid flow (µL)

ΔP = hydrostatic pressure difference across dentin (cm H2O-1) A= area of dentin (cm2)

t = time in min

The mechanism of dentinal tubule fluid flow and diffusion follows certain physics laws. The measurement of fluid flow (Q) across dentin or its hydraulic conductance (Lp) is measured by calculating difference between hydrostatic pressure across dentin forcing through 1cm2 of dentinal surface area per min. the convective fluid flow along with diffusion are directly proportional to the number of patent dentinal tubules whereas, inversely proportional to their length. The rate of diffusion across the dentinal fluid varies with the square of the tubule radius, whereas the convective fluid flow throughout the dentinal tubule varies with the fourth power of radius. The relationship between the radius and convective fluid flow is steep owing to the laminar intratubular fluid flow (Merchant et al 1977). This statement states, smaller concentric cylinders of fluid slip beneath larger more stationary cylinders adjacent to the walls of the dentinal tubules. This causes frictional resistance to the fluid flow. Any tubule occluding agent, reduces radius of tubule anywhere throughout the complete length of the tubule by one-half, the resistance to fluid flow further increases not by one-half square as in diffusion, but by one- half to the fourth power. Hence, the reduction in radius of the dentinal tubule reduce diffusion to one-fourth of its original value but on the other hand, restrict the convective fluid flow to one-sixteenth of its original value. Thus, this relationship states that the dentinal tubules need not be completely sealed in order to help reduce the DH (Reeder et al 1978). Furthermore, the permeability of fluid flow depends upon the radius, density and length of the dentinal tubule. As we know the dentinal tubules provide pathway linking dentin stimulation upon nerve responses. Although the dentin refrains to remodel as a bone, the formation of smear layers, reparative dentin, peri-tubular and intra-tubular dentin reduces permeability of the dentinal tubule and thus DH (Mjor 2001).

Treatment Of Dentin Hypersensitivity: 18

According to Orchardson and Gillam (2006), the therapeutic strategies employed to relive this condition have been traditionally divided as: In-office or professionally applied products and over-the-counter (OTC/at-home) these may either block the tubules when applied to the exposed root surface or may also act by desensitizing nerves in the pulp. As stated by Orchardson and Gillam 2006; Gillam et al 2013) till date there is no gold standard treatment developed for treatment of DH. As per Grossman (1935) and Gillam (1997) certain ideal requirements of a desensitizing agent are: non-irritant to the pulp, relatively painless on application, ease of application, rapid in action, effective for a long time, without staining effects consistently effective.

The desensitizing products for in-office use based on their mechanism of action are classified as

i)the products which do not polymerise (varnishes/ precipitants/ primers containing HEMA),

ii)the products that undergo setting or polymerization reactions (conventional glass ionomer cements / compomers, adhesive resin primers, adhesive resin bonding systems); or whether these agents are used in conjunction with

iii)the mouthguards,

iv)iontophoresis combined with fluoride pastes or solutions or

v)lasers (Pashley 2000).

The management of DH should be based on a step-wise approach based on the extent and severity of the condition (Orchardson and Gillam. 2006). An inherent way associated with this approach is removal of the underlying pre-disposing factors, diet counselling, improper toothbrushing techniques along with appropriate monitoring by the concerned clinician. According to the recent UK guidelines published on the management by Gillam et al, has stressed significance of combined treatment approach and not employing only a single approach for treating DH. It recommends treatment strategies based upon etiology and pre-disposing factors of specific conditions, namely,

a) patients with good oral hygiene associated with the DH

b) patients with loss of tooth surface layer

c)periodontally affected teeth.

In-Office (Professionally Applied Agents: Depending upon the severity and extent of DH, the clinician may opt for any in-office treatment.

FLOURIDE APPLICATION: The fluoride application in the form of gels, varnishes and resins has been recommended by Cummins (2010). The solutions, gels and varnishes having high concentration of fluoride have reported to give instant and long-term relief from DH (Pradeep 2012, Schmidlin and Sahrman 2013), a single topical application reported to relieve DH for 3-6 months (Al-Sabbagh et al 2009; Glockner 2013; Ritter et al 2006), combined fluoride application along with iontophoresis also reported to give relief from DH (Gangarosa and Park 1978, Da Rosa et al 2013). The fluorides mainly act by forming fluoride rich hydroxyapatite layer i.e fluoroapatite layer which is more resistant to acid breakdown.

Although, more studies need to be conducted to evaluate effectiveness of fluoride application to relieve DH (Sharif et al 2013).19

5% Sodium fluoride solutions… (Lokumsky et al 1941)

Sodium fluoride, kaolin and glycerine paste (Hoyt & Bibby 1943)

0.6% Sodium silicofluoride application… (Bhatia et al 1953)

Silicic acid + Calcium of tooth → gel → insulating barrier (Everett et al 1966)

10% Stannous fluoride (Blank and Charbeneau 1986)

Potassium Salts: The mechanism by which potassium salts act is that they react with the exposed dentin followed by precipitation of calcium compound crystals onto the dentin surface and within the dentinal tubules which further occlude the tubules thus relieving DH (Pereira et al 2005; Calabria et al 2012; Cummins 2010; Markowitz and Pashley 2008). Potassium has also been associated with oxalate formation to reduce DH, the 3 types of oxalate formulations mainly 3% mono-hydrogen monopotassium, 6% ferric 30% dipotassium have been associated with occlusion of dentinal tubules and further reduce DH (Al-Sabbagh et al 2009, Pashley et al 1987, Schmidlin and Sahrman 2013). The oxalate crystals formed have been reported to be more resistant to acid breakdown compared to precipitate fluoride crystals (Pereira et al 2005; Calabria et al 2012). The oxalate-containing phyto-complexes may however, show effective alternative to traditional oxalate preparations in treating DH (Al-Sabbagh et al 2009). An in-vitro study conducted by Sauro et al (2006) stated that these oxalates containing phyto-complexes have shown to reduce dentin permeability following a single application and thus reducing DH. 20

Calcium Phosphate Compounds: Calcium hydroxide has been reported to reduce DH immediately in 90% of treated teeth in clinical studies conducted by Al-Sabbagh et al 2009; Levin et al 1973. In-vitro studies conducted on calcium phosphate compounds in comparison with potassium oxalate have shown better effectiveness in occlusion of dentinal tubules (Al-Sabbagh et al 2009; Suge et al 2005). Casein-phosphopeptide-amorphous calcium phosphate (CPP-ACP) is a derivative of milk, known to remineralise enamel as well as dentin (Reynolds 1997). ACP in the presence of saliva is known to be converted to hydroxyapatite. At the tooth surface interface, both calcium and phosphate and are able to precipitate as stable hydroxyapatite (Nongonierma and Fitzgerald 2012). Reynolds et al 2008 carried out study on dentifrices incorporated with CPP- ACP for the treatment of DH, the authors concluded enhanced tubule occlusion with CPP- ACP group owing to remineralised dentin technology. Few other calcium-containing desensitisers formulations containing arginine and calcium carbonate have known to occlude dentinal tubules and provide long lasting relief from DH (Pei et al 2013; Schiff et al 200). Arginine is a naturally occurring amino-acid present in the saliva which presents positive charge. On the other hand, since dentin has a negative charge, arginine favours deposition of calcium on the dentin surface. The arginine, calcium carbonate and phosphate complex physically occlude the dentinal tubules and resists the intra-oral acid challenges (Cummins et al 2010; Pei et al 2013). A Con-focal Laser Scanning Microscope (CLSM) study conducted by Calabria et al 2012 to evaluate effectiveness of Pro-Argin technology, the authors concluded this technology was effective in depositing crystals over the dentin surface and thus occluding the subsurface of the dentin.21

Calcium Sodium Phosphosilicate (Bioactive Glass): The bioactive glasses have inherent ability to release silicon (Si), Calcium (Ca) and Phosphate (PO4) ions and thus initiate the process named as osteo-stimulation i.e upregulation and activation of a family of genes in osteoprogenitor cells (Price et al 1997; Sollazzo et al 2010). The proposed mechanism of action through dentifrice and polishing pastes is through precipitation of hydroxycarbonate apatite (HCA) on the dentin surface and subsequently within the dentinal tubules, as proposed by Lynch et al 2012. In-office prophylaxis pastes application of arginine, calcium carbonate along with bioactive glasses have reported to provide relief from DH (Cummins 2010; Pradeep and Sharma 2010). 21

Dentin Bonding Agents / Adhesives/ Adhesive Restorations:

The literature published till date states that the application of dentin bonding agents (DBA) in various forms has been reported to provide relief from DH. The application of phosphoric acid promotes the interlocking of the resin material within the surface of the dentin. These agents are less aggressive to the dentin-pulp complex compared to other etch- rinse systems. Further, they promote a micro-mechanical interlocking through a hybridization process, and presence of functional monomers stabilizes chemical interaction with the calcium ions from the residual hydroxyapatite from the submicron hybrid layer (Pei et al 2013; Inoue et al 2005). Additionally, the functional monomers too interact with the calcium ions from the smear layer, resulting in better chemical bonding. However, formation of such superficial layer on the outer surface of the dentin, resists outward flow of fluid movement, thus decreasing DH. Fransisconi et al 2009 studied stress distribution at the cervical region of the tooth, and concluded that the cervical stresses are present in larger amounts during mastication and toothbrushing, further these may present a challenge to the DBA or other adhesive restorations to resist forces, since resin tags are known to penetrate only superficially within the dentinal tubules. Aranha et al 2009 and Lopes Aranha 2013 advised repeated applications of the DBA for immediate pain relief from DH. Glutaraldehyde containing products have been reported to reduce DH by their mechanism of protein precipitation (Schupbach et al 1997; Glockner 2013). Qin et al 2006 reported glutaraldehyde containing adhesives promoted long-lasting pain relief from DH.22

Laser Technology: 23 The word ‘LASER’ is an acronym for ‘Light Amplification by

Stimulated Emission of Radiation’.

Application Of Lasers in The Treatment of Dentinal Hypersensitivity: 24

Maiman in 1960 developed ruby laser and from then the clinician’s used lasers in dental practice. Laser is a device that transforms polychromatic light into a monochromatic wave into the visible, infrared and ultraviolet regions with all the waves in phase capable of mobilizing immense heat and power once focusses at close range. Matsumoto et al 1985 was the first to use Nd: YAG laser for the treatment of DH.

Kimura et al 2000 stated clinical effectiveness of various types of lasers (Er:YAG, Er Cr: YSGG, CO2, GaAlAs, Diode laser and low-level diode laser) in treatment of DH. According to Lin et al 2013, Nd: YAG laser promotes analgesic effect whereas GaAlAs laser acts possibly by interrupting neural transmission (Tengrungsun and Sangkla 2008) compared to other high-power lasers and thus gives pain relief instantly form DH. The lasers mainly cause ‘melting effect of the dentin’ and this primarily occludes the patent dentinal tubules. Few studies have stated

low potency lasers are known to diminish the neural response to hypersensitive dentin (Bader et al 2014).

The tissues act by the following mechanisms once irradiated:

a) Melting and re-crystallization of the dentinal tubules, resulting in complete occlusion of the patent dentinal tubules.

b) Increase in the action potential of the nerve, by acting on the peripheral nociceptors.

c) Analgesic effect is caused due to decreased nerve transmission by blocking the depolarization of slowly conducting C-fiber afferents and also rapidly conducting A- fiber afferents.

d) The laser energy interferes with the sodium pump mechanism, changes the cell membrane permeability and/or temporarily alters the endings of the sensory axons.

e) Evaporation of the dentinal fluid through the thermal effect of the laser, thus causing a reduction in sensitivity.

f) antimicrobial action of the laser, results in reduction of bacterial noxious products.

g) Placebo effect is seen in the clinical trials conducted on DH (Hawthorne effect).

Kimura et al (2000)

Low output lasers (LLLT)	**Middle output lasers**
He-Ne (5.2%-100%)	Nd-YAG (51%-100%)
Diode lasers (Ga-Al-As) (30%-100%) 780nm; 830nm; 940nm	CO_2 Lasers (60%-100%)
Tubule narrowing and occlusion	**Nerve analgesia**
Nd-YAG CO_2 Lasers	Diode lasers He-Ne Nd-YAG

Carbondioxide (Co2) Laser: Carbondioxide laser (CO2) is a laser with wavelength of 10,600nm and can be used in both pulsed as well as continuous wave modes. It was first used at outpowers of 0.5 and 1 W, and the continuous wave mode by Moritz et al (1996) for the management of DH. The samples were irradiated for 0.5 to 5 sec, and irradiation was repeated 5-10 times. The treatment effectiveness ranged from 59.8 to 100%. It is the most frequently discussed laser and is active in a gas medium that incorporates a sealed tube containing a gaseous mixture of CO2 molecules pumped via electrical discharge current. CO2 laser is known to have powerful ablative properties owing to its absorption coefficient which is low in water (103) whereas high in hydroxyapatite (104). Occlusion or narrowing of dentinal tubules is the most common effect seen on irradiation with CO2 laser (Moritz et al 1995). Irradiation with CO2 laser at moderate density, causes sealing of the dentinal tubules, as well as reduction in permeability (Bonin et al 1991). Fayad et al reported the CO2 laser could also cause dentinal dessication further resulting in reduction of DH. CO2 laser irradiation at 0.3W for 0.1sec on dentinal tubules usually results in 2-8mm of sealing depth (Kimura et al 1998). The CO2 laser works upon the waveguide theory, this theory states, once the laser energy is redistributed along the interface of two dissimilar layers, the bond between these layers is weakened. This mainly occurs at the cemento-dentinal junction, further causing removal of excessive cementum resulting in DH and cervical de-mineralization.

Studies	Irradiation parameters	Effectiveness in %
Moritz et al (1980)	At 0.5W for 5 sec and for 6 times	59.8- 98.6%
Zhang et al (1998)	At 1W, Continous wave for 0.5sec for 5-10 times	50-100%
Moritz et al (1998)	At 0.5W, continuous wave for 5 sec for 6 times	94.5-96.5%

There are two methods by which CO2 laser acts:

a) Direct Method: Irradiation is carried out on the exposed dentin with CO2 laser only.

b) Indirect Method: Irradiation is carried out as first described by Moritz et al (1995,1996, 1998) in combination with fluoride gel.

In the indirect method, stannous fluoride gel is first applied to the patent dentinal tubules followed by laser irradiation through the gel. Output power of 0.5sec and 1W with the continuous wave (CW) mode were used for 0.5sec to 5 sec for 5-10 times. This procedure had dual advantage of laser as well as fluoride therapy which aids in long term management of DH.

Other Restorative Based Procedures:

Periodontal Plastic Surgery (Root Coverage Procedures): 25

One of the major signs of periodontal disease is gingival recession, which is known to be associated with DH as a result of the exposure of the root surface. Numerous root coverage procedures have been used to cover the exposed root surface till date, which not only cover the exposed root but along with that also decrease the DH, since the patent tubules are covered by healthy periodontium (Orchardson and Gillam 2006; Lin and Gillam 2012). According to Douglas de Oliveira et al 2013, root coverage procedures (partial or complete root coverage) have shown to reduce pain and associated discomfort thus improving quality of life of patient. The clinician is advised to select appropriate root coverage procedure depending upon patient's gingival biotype. According to Seibert and Lindhe 1989; Zweers et al 2014, three different gingival biotypes have been published in the literature till date: thin scalloped, thick flat and lastly thick scalloped biotype. The thin and scalloped gingival biotype presents more pronounced contour of the gingival margin, with a greater height of the interdental papilla along with underlying thin ridge of buccal bone which at times could present fenestrations. The second type i.e thick and flat gingival biotype presents thicker bone surrounding the teeth covered by thicker and smoother gingival contour, with reduced height of the interdental papilla. The third biotype i.e thick and scalloped presents characteristics of the above both of them. The thin biotypes can easily tear

during probing and surgical intervention and root coverage may not show optimum results, whereas, the thick gingival biotype has shown optimum results of root coverage (Seibert and Lindhe 1989; Muller et al 2000).

Over-the-counter (otc) products:

The ideal requirements of any OTC product according to Mantzourani and Sharma (2013):

i.Should be able to provide instant relief

ii.Easy of availability

iii.Should be low in cost

The desensitizing products and formulations readily available in pharmacies or any other commercial outlets and that could constitute variety of toothpastes, gels or mouthwash formulations. Advantages of these OTC products are that they are available even without a medical prescription, bit their indications for use are limited for a small period of time. These OTC formulations could also be recommended by the clinician along with on-going in-office treatments.

Flouride Containing Toothpaste, Gels or Mouthwash Formulations: 19

Fluoride was first proposed as a desensitizing agent by Lukomsky in 1941. The literature published till date states that the fluoride ions are known to mechanically block the dentinal tubules, or even the labile fluoride present in the organic matrix of the dentin could also block the transmission of the stimuli. This supports the Brannstrom's hydrodynamic theory, favoured by Schiff et al (1994). Duckworth et al (1994) carried out a study to assess deposition of fluoride using sodium fluoride and found it superior to sodium mono-fluorophosphate.

Cury and Tenuta (2014) stated fluoride is the most important therapeutic substance used in toothpaste formulations that is essential for reduction in dental caries. Along with fluoride, few other therapeutic substances like zinc, triclosan, pyrophosphates, etc could be added for reducing the biofilm formation and calculus formation, halitosis. The effectiveness of OTC products is dependent on the individual's oral hygiene, frequency of application or usage of mouthwashes on a twice daily basis (Petersson 2013). The mode of action and duration of action differs in each OTC product as per its active ingredient. Any OTC may require atleast 2-4 weeks of continuous usage or application to show its action (Talioti et al 2014). Toothpastes containing various formulations of fluoride like sodium fluoride (NaF), stannous fluoride

(SnF2), amine fluoride (AmF) is commonly used to treat DH (Morris et al 1999; Plagmann et al 1997; Pradeep et al 2012; Rosing et al 2009). The mode of action of fluoride is based on its ability to react with hydroxyapatite and further form fluoroapatite, fluorapatite is more resistant to acid breakdown and thus fluoride is an essential anticariogenic element added to the toothpaste formulations (Featherstone 2000). In addition to fluoride, other metal ions like zinc, tin, strontium, potassium, calcium carbonate, strontium along with few abrasive compounds like alumina, silica, calcium carbonate, zinc, etc are added which may also have synergistic effect on the surface of the dentin and further aid in blocking of dentinal tubules (Gillam 1992; Petersson 2013). A study conducted by Pradeep et al (2012) however, reported that amine fluoride added in a toothpaste formulation gave similar results when compared with calcium sodium phosphosilicate and potassium nitrate were equally effective in reducing DH.

Commercial Toothpaste andMouthwashes	Mechanism of Action	Compounds
Colgate Sensitive Pro-Relief (Pro-Argin Technology)	Dentinal tubule occlusion	Calcium carbonate and arginine (0.8%) – an amino acid naturally found in saliva, PVM/MA copolymer, pyrophosphates and 0.05% sodium fluoride in an alcohol-free mouthwash
Sensodyne Repair andProtect (Novamin technology)	Dentinal tubule occlusion	Flouride, calcium, phosphateions
Sensodyne Pronamel	Sealing of dentinal tubules	Fluoride and Potassium nitrate
Sensodyne Rapid Relief		8% strontium acetate in asilica base, 1,040 ppm sodium fluoride
SootheRx (Novamin technology)		Calcium sodium-Phosphosilicate
AMFLOR		3.85% Amine fluoride
Fluoridex (Phillips Oral Healthcare)	Clinician prescribed toothpaste formulations	5,000 ppm Fluoride
Colgate gel and sensitive		Stannous fluoride 0.4% w/v, 1,000 ppm F
Colgate Durphat		2,800 ppm Fluoride
Potassium containing products	Neural desensitisation	Potassium chloride, citrate, nitrate and oxalate and fluoride compounds
Sensitive toothpaste (Himalaya herbals)	Dentin tubule occlusion and neural desensitization (Other products)	Spinach- natural oxalate compound

Sodium Fluoride:

Hoyt and Bibby (1943) studied various preparations of sodium fluoride and came to a conclusion that a 33% sodium fluoride paste (incorporating equal parts of kaolin and glycerin) was extremely effective in relieving DH. Single application of 2% acidulated sodium fluoride to human teeth which were extracted later at intervals, produced fluoride levels which

maintained for 14 days (Ehrlich et al 1975). However, scanning electron microscopy (SEM) of the extracted teeth showed granular precipitation in peri-tubular dentin upto 6 months with clinical improvement, generally of considerable duration, was noted to mechanical stimuli. These findings reflect natural desensitizing process (Karlsson and Penney 1975). The pretreatment with 10% strontium chloride followed by 2% sodium fluoride showed additive effect in reducing DH (Gedalia et al 1978), although sodium fluoride alone was also equally effective.

Few comparative studies evaluating effectiveness of sodium fluoride with and without iontophoresis have shown better results. Murthy et al (1973) found that iontophoresis with sodium fluoride application showed decline in DH when compared to 33% sodium fluoride application. Minkow et al

(1975) carried out a study using 2% sodium fluoride solution with and without iontophoresis, stated that both groups showed effective results. Reduction in DH followed by iontophoresis was seen due to electrical stimulation of secondary dentin formation (Schaeffer et al 1971). However, positive effect was applied after single application for a short period of time due to induction of secondary dentin formation. Selvig et al 1968 stated fluoride application after iontophoresis would aid in resistance of the dentin to acid decalcification, and to precipitations to the exposed dentinal tubules (Selvig et al 1968, Furseth 1970). Tal et al (1976) proposed that precipitated fluoride compounds blocking mechanically, or labile fluoride in the organic matrix of dentin blocking the transmission of stimuli biochemically, are the probable desensitizing factors.

Stannous Fluoride:

Aqueous solutions of stannous fluoride in low concentrations effectively decrease DH (Miller et al 1969). Nevertheless, stannous fluoride in glycerin gelled with carboxy methyl cellulose showed better results than placebo gel in a double-blind clinical trial on exposure to thermal, chemical and tactile stimuli. Two mechanisms have been put forward for stannous fluoride: Firstly, as stated by Kutscher (1967), stannous fluoride acts as an enzyme that inactivates the odontoblasts process and secondly it is known to induce high mineral content that further creates a calcific barrier upon the dentin surface (Furseth 1970). The stannous fluoride with stannous chloride and sodium fluoride, is known to precipitate on the surface of the dentin and thus cause occlusion of the dentinal tubules as demonstrated by in-vitro studies (Blunden et al 1981). Penney and Karlsson (1976) states that the tin diffuses readily compared to fluoride within the dentin. The authors stated that the reason behind this could be the double availability of the fluoride ion and/or that the stannous ion forms more insoluble salts with phosphate than fluoride does with calcium. Although, it is noteworthy that fluoride is of molecular size to the hydroxyl group, whereas the size of tin is much smaller comparatively. As a result, ionic exchange for tin occurs less likely.

Sodium Mono-Fluorophosphate:

The anti-caries effectiveness of commercially available dentifrices consists of 0.76% sodium mono-fluorophosphate has been well documented in numerous clinical trials (Accepted Dental Therapeutics 1971/1972). Furthermore, few studies conducted by Bolden et al 1968, Hazen et al 1968, Kanouse and Ash 1969, Hernandez et al 1972 have stated dentifrices containing 0.76% sodium mono-fluorophosphate is effective in treating DH.

Hernandez et al (1972) carried out a comparative study for 6 weeks on sodium mono- fluorophosphate (61.3%) and strontium chloride (37.8%) and the results showed statistically significant reduction in the incidence of hypersensitive surfaces when compared with the control (19.5%). The authors states, 6 weeks post cessation of dentifrices, the sodium mono-fluorophosphate group showed statistically significant improvement of 41.4% compared to baseline. The results suggested a carry-over effect and furthermore the greatest number of subjects experiencing a total desensitizing effect belong to the sodium mono-fluorophosphate group.

In contrast, Shapiro et al (1970) carried out a comparative study on strontium chloride and sodium mono-fluorphosphate for 8 weeks period, but this study did not gain significant results in reducing DH when compared to control groups. Forward (1980) stated that ionic fluorides, sodium and tin show incompatibility to form covalent bonds in fluoride from sodium mono- fluorophosphate. The covalent bond between the fluorine atom and the phosphorous atom is not readily separated and as a result, the formation of soluble fluorides such as calcium fluoride does not occur to greater extent.

Strontium Salts Containing Products:

Earlier strontium salts were used as Strontium chloride and more recently they are also available as strontium acetate. In-vitro studies conducted by Ling and Gillam 1996; West 2008 stated that the mechanism of action of strontium salts (chloride/acetate) is mainly by causing the dentinal tubule occlusion. Addy et al (1987) and Banfield et al (2004) carried out in-vitro studies on strontium acetate toothpaste and the authors concluded that the results were promising. Recently, the strontium acetate-based toothpaste reformulated as Sensodyne Rapid Relief comprising of 8% strontium acetate in silica base with 1,040 ppm sodium fluoride. The abrasive action of silica showed better tubule occlusion on the etched dentinal surface. Banfield et al (2004) found an interesting finding that silica layer was intact upon regular mouth-rinsing in patients suffering from DH which proved strontium salts showed better binding to the dentinal surface, but this still has to be proved through in-vivo studies. Schiff et al (2009) carried out a 16-week crossover design study comparing Colgate Sensitive Pro-relief and Sensodyne Rapid Relief, subjects were advised 8 weeks toothbrushing with these toothpastes. The authors concluded subjects in the Colgate Sensitive Pro-relief group experienced better results and significant reduction of mean DH values when compared with the subjects in Sensodyne Rapid Relief group.

Potassium- Containing Products: 20

Mouthwashes and Toothpastes comprising Potassium formulations (nitrate, chlorine and citrate) have been reported to be effective in reducing DH compared to placebo control groups (Orchardson and Gillam 2000; Frechoso et al 2003; Markowitz 2009). Amongst all the available potassium containing formulations, 5% potassium nitrate toothpaste formulation has been extensively studied and reported better results till date (Rosing et al 2009). They act by diffusion of potassium ions along the dentinal tubules thereby decreasing the excitability of the intra-dental A fibers by blocking their axonic action subsequently declining the DH (Schmidlin and Sahrman 2013; Boneta et al 2013). Similar study conducted by Gillam et al (1996) with 3% potassium nitrate concentration formulation with sodium fluoride mouthrinse was known to significantly reduce DH compared to a sodium fluoride mouthwash after 2-6 weeks. Pereira and Chavas (2001) carried out a comparative study, all subjects in both the groups were advised to use the toothpaste formulations for a period of 2 weeks. The authors concluded that at the end of 2 weeks, there were no significant differences observed between both the groups using tactile and thermal stimuli. However, at 6 weeks, both the 0.2% sodium fluoride mouthwash group and 3% potassium nitrate group demonstrated a significant reduction in DH upon exposure to cold blast of air compared to the control 0.2% sodium fluoride mouthwash. Another study conducted by Jackson et al (2000) and Panagakos et al (2009), on usage of potassium containing toothpaste, was found not much effective in reducing DH when compared to regular fluoride containing toothpaste.

Recently, 1.4% potassium oxalate (Listerine Advanced Defence Sensitive (LADS)) containing mouthwash has been developed by Johnson and Johnson Consumer and Personal Products Worldwide, Skillman and Morris Plains, NJ, USA. Sharma et al 2013 conducted two randomized clinical trials (RCTs) of 5 days and 4 weeks to check effectiveness of LADS. The authors concluded that in both the studies, the potassium oxalate mouthwash significantly declined the intensity of DH compared to the negative control groups. The authors added that the combination of potassium and oxalate together would cause dual mode of action in reducing DH (namely, nerve desensitization caused by potassium and tubule occlusion caused by oxalates). Furthermore, the authors stated, the tubule occlusion associated with LADS was found more stable to resists acid challenge compared to other test products.

Calcium Containing Compounds

Casein Phosphopeptide – Amorphous Calcium Phosphate (Cpp-Acp): 21

Casein Phosphopeptide (CPP) developed a water-based sugar free toothpaste formulation named as (GC Tooth Moose, Recaldent technology) it has dual action, anticaries as well as remineralization of enamel and dentin.

As stated by Reynolds et al (1998), the CPP component provides calcium (Ca) and potassium ions (PO4) at the tooth surface, this stabilizes the ACP component of the CPP and ensures delivery of Ca and PO4 ions onto the tooth surface for remineralization to CPP, and formulated as CPP-ACP, this subsequently prevented dissolution of both the Ca as well as PO4 ions that supersaturate saliva (Reynolds 1997). Few studies conducted by Lata et al 2010; Cai et al 2003 have reported that CPP-ACP has ability to effectively remineralise the enamel subsurface lesions. The manufacturer's suggested CPP-ACP is the agent that not only effectively remineralizes enamel and dentin but is also an ideal agent to treat DH, a number of in-vivo and in-vitro studies have proven the same. The calcium phosphate formulation deposits a mineral rich precipitate onto the dentin surface, which further blocks the dentinal tubules thereby reducing DH (Ebisu 2002; Gandolfi et al 2010; Walsh 2010). Wegehaupt et al (2011), however, concluded the results of their study were not statistically significant and claimed availability of insufficient data on CPP-ACP to reduce DH.

Functionalised Tri-Calcium Phosphate (F-Tcp): 21

The f-TCP is commercially available as a 950-ppm fluoride toothpaste (Clinpro Tooth Crème; 3M ESPE, Saint Paul, MN, USA). According to Vanichvatana and Auychai (2013), f-TCP is prepared by milling of solid-state ball of beta-tricalcium phosphate and sodium lauryl sulphate. Its further acts by preventing Ca ions from prematurely interacting with iconic fluoride and forming calcium fluoride, thus delivering more fluoride along with Ca ions to the enamel surface (Karlinsey et al 2010, 2012). Studies conducted by Karlinsky et al 2009; Mensinkai et al 2012 reported f-TCP has proved remineralising effects in both in-vitro and in-vivo studies. Asaizumi et al (2013) assessed the effect of 0.21% and 1.1 % NaF toothpaste containing f- TCP on remineralization of white spots in-vitro. While, Mensinkai et al (2012) carried out a comparative in-situ remineralization study of white-spot enamel lesions with non-fluoride toothpaste of 500 and 1,100 ppm. The authors concluded f-TCP group yielded superior results in remineralization compared to the non-fluoride group.

Pro-Argin Based Toothpaste and Mouthwash Formulations: 26

Pro-Argin is a combination of calcium carbonate and arginine complex i.e prepared on Kleinberg's original formulation Sensistat (Kleinberg 2002). Colgate Sensitive Pro-relief is a commercially available toothpaste, Colgate Palmolive Co, New York a mouthrinse based on Pro-Argin technology. Arginine is a naturally occuring amino acid in saliva which on combination with calcium carbonate forms a calcium-arginine complex onto the tooth surface and within the dentinal tubules (Kleinberg 2002; Cummins 2010). Arginine bicarbonate is the most frequent form used in the toothpaste formulation is naturally found in saliva and has inherent adhesive properties that help in occluding dentinal tubules. Arginine bicarbonate is prepared from arginine by titrating the arginine with gaseous carbondioxide (CO2) until a pH of approximately 7 is achieved. At this pH, arginine is completely converted to arginine bicarbonate and then further the pH is adjusted between 8 and 9, which facilitates complex formation and binding of the arginine to the tooth surface. The presence of calcium phosphate ions during this process, facilitates dentinal tubule occlusion (Kleinberg et al 2003). The Colgate Sensitive Plus dentifrice used in the present study consists of calcium carbonate, sorbitol, arginine bicarbonate, sodium lauryl sulphate, flavor, sodium mono fluorophosphate, sodium carboxymethyl cellulose, sodium bicarbonate, tetrasodium pyrophosphate, titanium dioxide, sodium saccharin, benzyl alcohol, xanthan, gum, limonene in aqueous base.

RCTs conducted on Pro-Argin containing toothpaste have reported to reduce DH when compared to either placebo or other products (Petrou et al 2009; Boneta 2013). Mouthwashes containing 0.8% of arginine, PVM/MA copolymer, pyrophosphates and 0.05% of sodium fluoride in an alcohol-free base was found effective compared to control mouthwashes in reducing DH (Hu et al 2013).

Calcium Sodium Phosphosilicate (Bioactive Glass): 26

Bioactive glasses are commercially available as NovaMin developed by NovaMin Technology Inc (Alachua, FL, USA) based on the original 45S5 Bioglass formulation by Larry Hench (US Biomaterials Corp, Jacksonville, FL, USA; now GSK) (Hench 2006).

The proposed mode of action is by precipitating of hydroxycarbonate apatite (HCA) onto the dentin surface and subsequently occluding the dentinal tubules (Litkowski et al 1998; Gillam et al 2002; Vollenweider et al 2007). Advantage of the precipitated HCA layer was chemically and structurally similar to both natural enamel and dentin (Burwell 2006; Gendreau et al 2011). The layer of fluoroapatite (FAp) rather than HCA is more resistant to the intra-oral acid attack. In the presence of saliva, the bioactive glass particles dissolve releasing calcium and phosphate ions. The apatite like material preferentially blocks the highly mineralized peri-tubular dentin within the dentinal tubule. It is postulated that the HCA within the peri-tubular dentin preferentially nucleates apatite crystals. Bioactive glasses have also known to remineralise the dentin (Mneimne et al 2014). It is known that the bioactive glasses dissolve more rapidly and form apatite at acidic pHs of 7 or higher associated with dental caries. The fall of pH in caries or acid erosion challenge simultaneously releases Ca and PO4 ions. Hence, bioactive glasses are considered smart materials for treating DH. Bioactive glasses comprise particles of about 5 μm in size, and thus aid in occlusion of dentinal tubules, whilst the larger particles in the distribution take much longer to dissolve. NovaMin toothpaste formulations are similar to HA toothpaste containing a carboxyl functional polymer that provides required viscosity to the toothpaste. However, the polymer also plays an important role in chelating Ca ions in apatite and Ca ions in NovaMin glass and acts to promote the adhesion of the glass particles to the tooth and prevents the particles from being washed away by salivary flow. The NovaMin toothpaste formulations are prepared in glycerol rather than water based, and this is to prevent the glass reacting with water during the storage of the toothpaste.

Nano-Hydroxyapatite (Nhap) Containing Dentifrices: 26

Recently, Nanohydroxyapatite (nHAP) containing dentifrices have been marketed to be effective both as a remineralising agent and desensitising agent (Calabria et al 2012, Hanning 2010, Tschoppe et al 2011). These dentifrices contain nano-sized particles, which act in a similar way to hydroxyapatite in regard to their morphology, structure and crystalline arrangement (Vandiver et al 2005). This technology is based upon the bioactivation of natural compounds with a view to improve stability of the surface precipitate on the tooth surface following an acid challenge. The nHAP containing dentifrices are known to maintain a topical state of supersaturation at the tooth surface. On the other hand, it favours crystal growth and deposition at the tooth surface aiding in surrogation of symptoms associated with DH. The nHAP containing dentifrice named as Aclaim consisted of sorbitol, glycerin, silica, purified water, hydroxyapatite, cocamidopropyl betaine, hydroxyethyl cellulose, titanium dioxide, flavour, sodium saccharin.

Advances in in-vitro testing technologies help to quantitatively determine the effectiveness of specific treatment.

Certain Advanced Laboratory Methodologies: 27

1)Dentin permeability evaluation – fluid flow rate (hydraulic conductance model)

2)Environmental scanning electron microscopy (ESEM)

3)Energy dispersive x-ray spectroscopy (EDS, EDX OR XEDS) also known as Energy dispersive X-ray analysis (EDXA) or Energy dispersive X-ray micro-analysis (EDXMA).

4)Atomic Force Microscopy (AFM)

5)Raman and Micro-raman spectroscopy

6)Fourier transform infrared spectroscopy (FTIR)

7)Confocal laser scanning microscopy (CLSM)

8)Focussed ion beam scanning electron microscopy (FIB SEM)

9)Solid state nuclear magnetic resonance (NMR) spectroscopy

10)Al, Si, F, P magic angle spinning nuclear magnetic resonance (MAS-NMR)

Conclusion:

Consider a differential diagnosis and deal with any dental pathology which could cause similar 'dentinal' symptoms. Identify aetiological and predisposing factors, these will mainly relate to abrasion and erosion. Remove, reduce or modify aetiological and predisposing factors. This will usually involve dietary advice to minimise erosion and oral hygiene advice to minimise abrasion. Treatment strategies will vary, being dependent on each individual case, and in particular on the number of teeth involved and the severity of symptoms. However, some improvement for a majority of cases will be obtained by the recommendation of a desensitising toothpaste.

Conflict of Interest: None

Author Contributions:

Dr Sandhya Shobhange - Writing of the manuscript.

Dr Nasheer Shaikh - Compiling data for the manuscript.

Dr Sharayu Dhande - Assessing of manuscript.

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