Review Article

ETIOLOGY AND MANAGEMENT OF HYPERSENSITIVITY OF TEETH - A REVIEW

ABSTRACT:

Hypersensitivity of teeth is a common clinical condition usually associated with exposed dentinal tubules. It is predominantly found in patients belonging to 20-60 years of age and most commonly affects the canines and premolars of both the arches. This article concisely reviews the etiology, patho-physiology, mechanism and management of dentin hypersensitivity. Treatment of dentin hypersensitivity should start with an accurate diagnosis and removal of the etiology. Differential diagnosis should be made and all other probable causes should be excluded. An often neglected phase of clinical management of dentin hypersensitivity is the identification and treatment of the causative factors. By removing the etiological factors, the condition can be even prevented from occurring or recurring. There are various treatment modalities available which can be used at home or may be professionally applied. The "at home" desensitizing agents include toothpastes, mouthwashes or chewing gums and they act by either occluding the dentinal tubules or blocking the neural transmission. The in-office treatments include application of potassium nitrate, fluorides, adhesive resins, copal varnish and oxalates. This article also discusses the recent treatment options like carbonated nano hydroxyapatite crystals bioglass, calcium silicate derivative of Portland cement, lasers and casein phosphopeptide.

KEYWORDS: Attrition; Abrasion; Abfraction; Dentin hypersensitivity; Desensitising agents; Tooth wear.

INTRODUCTION:

Definition of hypersensitivity of teeth

Various terms have been used to describe hypersensitivity of teeth. These terms are used based on the place of occurrence of hypersensitivity and include: cervical, root, dentin, cemental, and the terms sensitivity, and hypersensitivity. (Orchardson and Gillam, 2006; Porto, Andrade and Montes, 2009; Miglani, Aggarwal and Ahuja, 2010) All of these terms convey the same clinical conception and can be used interchangeably. According to the Canadian consensus document, hypersensitivity of teeth can be defined as "pain derived from exposed dentin in response to chemical, thermal tactile or osmotic stimuli which cannot be explained as arising from any other dental defect or disease". (Canadian Advisory Board on Dentin Hypersensitivity, 2003)

Dentin hypersensitivity is mainly due to the exposure of dentinal tubules by removal of enamel and cementum due to occlusal wear, psychological factors and anatomic variations. Treatment of dentin hypersensitivity should start with an accurate diagnosis and removal of the etiology. Differential diagnosis should be made and all other probable causes should be excluded. An often neglected phase of clinical management of dentin hypersensitivity is the identification and treatment of the causative factors. By removing the etiological factors, the condition can be even prevented from occurring or recurring.

Previously our department has published extensive research on various aspects of prosthetic dentistry ('Evaluation of Corrosive Behavior of Four Nickel–chromium Alloys in Artificial Saliva by Cyclic Polarization Test:An in vitro Study', 2017; Ganapathy, Kannan and Venugopalan, 2017; Jain, 2017a, 2017b; Ranganathan, Ganapathy and Jain, 2017; Ariga *et al.*, 2018; Gupta, Ariga and Deogade, 2018; Anbu *et al.*, 2019; Ashok and Ganapathy, 2019; Duraisamy *et al.*, 2019; Varghese, Ramesh and Veeraiyan, 2019), this vast research experience has inspired us to do a review on the etiology and management of hypersensitivity of teeth. This article aims to review concisely the etiology, pathophysiology, mechanism and management of hypersensitivity of teeth.

PREVALENCE

The prevalence distribution and appearance of the disease have been reported differently in different studies. These differences are due to the differences in populations, habits, dietaries, and methods of investigation.(Miglani, Aggarwal and Ahuja, 2010) About 8 to 30% of the population are affected by hypersensitive tooth / teeth. (Graf and Galasse, 1977) The disease is prevalent in all the age groups. However, it is more prevalent in patients belonging to the age group 20-60 years and more prevalent in females. This would probably be attributed to dental hygiene and dietary habits. (Aranha, Pimenta and Marchi, 2009; Porto, Andrade and Montes, 2009; Cummins, 2010; Miglani, Aggarwal and Ahuja, 2010; Chu, Lam and Lo, 2011) The first premolars are the most affected teeth and the most common site involved is the cervical region on the buccal side. (Lilja, 1979) Several studies have reported dentin hypersensitivity to be predominantly associated with non-carious cervical lesions (NCCLs) in adult populations, with prevalence rates ranging from 5% to 85% (Lilja, 1979; Bartlett and Shah, 2006) and 2-8% to 74% respectively. (Que *et al.*, 2013) There are two common methods to determine the intensity of dentin hypersensitivity. One of them is through asking some questions from the patient and the other is through clinical examination. The prevalence distribution of dentin hypersensitivity in the first method is usually estimated higher than that of the second method. (Fischer, Fischer and Wennberg, 1992) In a study, it has been observed that the majority of the people with hypersensitive tooth / teeth do not pursue treatment of the disease. However, they may report it in a clinical visit to the dentist. This is perhaps due to the fact that they do not consider hypersensitivity of teeth as a specific disease. (Gillam *et al.*, 1999)

ANATOMY OF PULP DENTIN COMPLEX:

Dentin is considered as a vital tissue and has the capacity to respond to physiologic and pathologic stimuli. (Heymann, Swift and Ritter, 2013) Dentin is covered by enamel in the crown surface and by a thin layer of cementum in the root surface of the tooth. Dentin is sensitive to stimuli due to the extension of the odontoblastic process into the dentinal tubules and formation of dentin-pulp complex. (Orchardson and Gillam, 2006; Miglani, Aggarwal and Ahuja, 2010; Chu, Lam and Lo, 2011)

Dentin and pulp are histologically different. However, they are derived from the same embryonic origin - ectomesenchymal origin. The formation of dentin-pulp is associated and the development of dentin is affected by pulp and vice versa. Dentin has very minute tubules which are filled with an odontoblastic process. The processes are also surrounded by dentinal fluid which forms about 22% of the total volume of dentin. This dentinal fluid is derived from the blood vessels of the pulp. (Addy, 2000)

ETIOPATHOGENESIS

Dentin's sensitivity to stimuli does not lead to any problem while it is covered with protective tissues i.e., enamel and cementum. The dentin starts presenting hypersensitivity only when exposed to the oral environment which occurs due to the wear of these protective structures (enamel and cementum). (Addy, 2000; Orchardson and Gillam, 2006; Cummins, 2010; Miglani, Aggarwal and Ahuja, 2010)

Removal of enamel : The enamel layer can be removed by attrition due to occlusal wear, parafunctional habits (bruxism), improper tooth brushing (toothbrush abrasion), erosion from acids (Gastroesophageal reflux disorder - GERD), fracture of crown (Ellis class I and II), abfraction and defective restoration (improper marginal seal). (Wichgers and Emert, 1996)

Removal of cementum: Poor periodontal health due to incorrect tooth brushing and faulty restorations can lead to gingival recession and periodontal disease. This leads to the exposure of the root surface. Once exposed, the thin cementum layer (20 - 25 microns in thickness) can be easily removed by periodontal root scaling, abrasive pastes, acidic foods and toothbrushing, exposing the dentinal tubules. (Wichgers and Emert, 1996)

Anatomic variations: In about 10% of the individuals, the enamel and cementum do not meet exposing the dentinal tubules leading to hypersensitivity. (Wichgers and Emert, 1996)

Occlusal stress: Dentin hypersensitivity is closely related to occlusal stress, where the increased occlusal forces lead to an increase in the intrapulpal pressure, which in turn increases the movement and speed of dentinal fluid, leading to hypersensitivity of the tooth. (Berman, 1985)

Psychological factors: Hypersensitivity of teeth is considered as a chronic condition showing periods of exacerbation. Psychological component of chronic pain is to be considered. Many studies show that psychic tension reduces the tolerance threshold to external stimuli (eg: application of heat, cold, pressure). (Wichgers and Emert, 1996) Pain mechanism is complex and is proven that the neural activity initiated by peripheral stimuli may be modified by psychic and psychological factors. (McGrath, 1994)

Hypersensitivity of teeth is developed in two phases - Lesion localisation and lesion initiation. (R. Dababneh, Khouri and Addy, 1999; Barcellos *et al.*, 2012) In the first phase, dentinal tubules, due to loss of enamel / cementum, are exposed by attrition, abrasion, erosion, and abfraction. It was found that dentinal tubules are exposed mostly 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 dentinal tubules are sensitive. However, 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. (Addy, 2000; Lussi, 2006; Cummins, 2010) In the second phase, for the exposed dentin to be sensitized, the tubular plugs and the smear layer are removed and consequently, dentinal tubules and pulp are exposed to the external environment (Barcellos *et al.*, 2012). 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 the smear layer. (Zero and Lussi, 2005; Miglani, Aggarwal and Ahuja, 2010)

There is no evidence concerning the presence or absence of pulpal changes associated with hypersensitivity of teeth. (Dowell and Addy, 1983; Trowbridge, 1986) Some authors believe that there may be changes in the pulp dentin complex as a consequence of the exposed dentin and penetration of solutes and toxins from the oral environment, especially if there is a cavity or

caries, or if the plaque is on dentin. (Pashley, 1986; Olgart and Kerezoudis, 1994) According to Addy and Pearce, (Addy and Pearce, 1994) the sensitivity associated with plaque or caries (bacterial associated sensitivity) has a defined pathology and therefore is not dentinal hypersensitivity. It seems that Microbial plaque is not a significant factor in triggering DH. (Orchardson and Gillam, 2006) First, as mentioned previously, the canines and first premolars have the greatest recession and sensitivity. The same teeth also reveal the lowest buccal plaque scores. Secondly, teeth with DH are cleaned extremely by patients suffering from the condition. This would suggest that plaque does not produce dentin hypersensitivity itself nor does it act as a stimulus for pain. (Addy, Mostafa and Newcombe, 1987) However, the effect of plaque on DH is a controversial issue. (Barcellos *et al.*, 2012)

MECHANISM OF DENTIN HYPERSENSITIVITY

There are three mechanisms explaining hypersensitivity of teeth

Direct Innervation (DI) Theory

According to this theory the nerve endings enter dentin through pulp and extend upto the dentino enamel junction (DEJ) and the mechanical stimuli directly transmit the pain. However, there is little evidence to prove this theory. There is no enough evidence that can support the existence of nerves in the superficial dentin (where dentin is considered to be more sensitive). The plexus of Rashkov does not become mature until a complete tooth eruption. However, the newly developed teeth can be sensitive too. (*Dentin hypersensitivity: a review*, no date)

Odontoblast Receptor Theory

According to this theory, odontoblasts act as receptors of pain and transmit signals to the pulpal nerves. But this theory has also been rejected since the cellular matrix of odontoblasts is not capable of exciting and producing neural impulses. Furthermore, no synopsis has been found between odontoblasts and pulpal nerves. (Miglani, Aggarwal and Ahuja, 2010)

Fluid Movement / Hydrodynamic Theory

Hydrodynamic Theory for dentin sensitivity was first proposed by Brannstorm. (Brännström and Åström, 1964) This theory is the most widely accepted theory for dentin hypersensitivity. The theory has been proposed based on the movement of the fluid inside the dentinal tubules. The theory claims that tubules are open between the dentin surface which is exposed to the oral environment and pulp. (Brännström and Åström, 1964; Cunha-Cruz *et al.*, 2010) It is believed that dentin hypersensitivity is made as the result of fluid movement inside the dentinal tubules, which is further due to the thermal and physical changes, or as the result of formation of osmotic stimuli near the exposed dentine. This fluid movement stimulates a baroreceptor and leads to neural stimuli. The process is called the hydrodynamic theory of pain. (Gillam *et al.*, 1999; Miglani, Aggarwal and Ahuja, 2010) The movement of fluid can be towards the pulp or away from the pulp. Cooling, drying, evaporation, and hypertonic chemical stimuli cause the dentinal fluid to flow away from the dentin-pulp complex and lead to an increase in pain. (Gillam *et al.*, 1999) Heating causes the fluid to flow towards the pulp. About 75% of patients with dentin hypersensitivity feel pain in response to cold stimuli. (Lussi, 2006; Porto, Andrade and Montes, 2009; Miglani, Aggarwal and Ahuja, 2010)The number of tubules in sensitive dentin is eight times more than the number of tubules in non sensitive dentin. Also, the tubules of sensitive dentin are wider than those in non sensitive dentin. (Davari, Ataei and Assarzadeh, 2013)

DIAGNOSIS:

It is important to differentiate dentin hypersensitivity from other pathologies which present similar symptoms, allowing the establishment of an adequate treatment program A careful diagnosis includes a history and a thorough clinical and radiological examination. Before considering any treatment strategy for the management of dentin hypersensitivity, it is important to note from the published literature that there are a number of individuals who may be at risk for dentin hypersensitivity (Gillam and Orchardson, 2006) such as:

1. Overenthusiastic brushers
2. Periodontal treated patients
3. Bulimics
4. People with xerostomia

5. High-acid food/drink consumers
6. Older people exhibiting gingival recession
7. Chewing ‘smokeless’ or ‘snuff’ tobacco

The often forgotten or neglected phase in the treatment of dentin hypersensitivity is the diagnosis and eliminating or treating the main routes of dentin hypersensitivity. It is important to note the information about the tooth cleaning habits, including the frequency and duration, type of toothbrush, brushing technique, interval of toothbrush change and pressure applied during tooth brushing. It is also important to investigate the habits that may lead to gingival inflammation and recession. The contact with erosive agents must be questioned including those arising from living environment, working, medications, illness and diet. (Addy and West, 1994) History and nature of pain to be enquired. In general people with dentin hypersensitivity may have the following symptoms:

Pain provoked by:

1. Thermal stimuli (cold / hot food, air during the talking or even aspiration).
2. Chemicals (sweet and acid food)
3. Tactile (brushing and probing)

Nature of the pain:

1. Localised pain with greater definition of dental element
2. Acute pain with short duration
3. Pain vanishes with stimulus removal
4. Spontaneous remission periods
5. Coincidence with patient anxiety state (Berman, 1985)

Sometimes, pulpal inflammation complicates the diagnosis of dentin hypersensitivity. But dentin hypersensitivity differs from pain arising due to pulpal inflammation. When a stimulus is applied to a hypersensitive tooth, the patient will be readily able to locate the source of pain and discomfort, while pulpal pain can be lasting, intermittent and throbbing. The simplest method for diagnosis of dentin hypersensitivity is application of “air blast” on the tooth. Air stimulation usually reflects dentin hypersensitivity rather than a problem arising from the pulp. Careful

examination of other pathologies and dental defects must be considered including, cracked tooth syndrome, fractured restorations, chipped teeth, caries, post restorative sensitivity and teeth in hyperfunction, in order to exclude the alternative causes of pain. (Bissada, 1994)

TREATMENT FOR DENTIN HYPERSENSITIVITY

Removing the etiological factor and preventing dentin hypersensitivity:

The often forgotten or neglected phase in the treatment of dentin hypersensitivity is the diagnosis and eliminating or treating the main routes of dentin hypersensitivity. The etiologic factors can be improper tooth brushing, premature occlusal contacts, gingival recession, and the existence of a large amount of exogenous and endogenous acids in diets.

Improper tooth brushing: This includes using hard- or thick-bristle toothbrushes, brushing teeth with excessive pressure, excessive scrubbing at cervical areas or even missing to brush cervical areas. (Suge *et al.,* 2006) To avoid the DH due to improper tooth brushing: The patient should be taught the correct method of tooth brushing. (Miglani, Aggarwal and Ahuja, 2010) The patient should avoid the use of abrasive toothpastes. (Orchardson and Gillam, 2006) The patient should avoid brushing at least for one hour after consuming acid drinks or foods (due to the agonist effect of acidic erosion on tooth brush abrasion). (Orchardson and Gillam, 2006; Miglani, Aggarwal and Ahuja, 2010)

Premature occlusal contacts: Dentin hypersensitivity can be resolved through correction of occlusion or the use of an occlusal splint. (Gillam and Orchardson, 2006)

Gingival recession: Root coverage procedures such as graft or positioning flap might be adopted.

Exogenous and endogenous acids (erosive agents): It has been proved that erosive agents have a role in the initiation and progression of dentin hypersensitivity. These agents can open dentinal tubules through removal of the smear layer, plugs and enamel. (Eisenburger and Addy, 2002)

Exogenous acids: carbonated drinks, citrus fruits, alcoholic drinks, yogurt, dairy products, and occupational hazard (such as workers in battery manufacturing plants). (Eisenburger and Addy, 2002; Miglani, Aggarwal and Ahuja, 2010) The patient's diet should be monitored for a while,

concerning the quality and the frequency of consumption of acidic foods so that the necessary recommendations can be offered to the patient. Recommendations such as using alkaline resources, like milk, or at least neutral materials, like water, after eating acidic foods, or having carbonated/acidic drinks with straw and avoiding to keep carbonated/acidic drinks in the mouth and tasting them. (Cummins, 2010)

Endogenous acids: Erosive agents with endogenous acids enter the mouth through reflux or gastro-esophageal regurgitation. (Mayhew, Jessee and Martin, 1998) These agents can be mostly found in patients with eating disorders. The patients are recommended to refer their doctors for the underlying. (Miglani, Aggarwal and Ahuja, 2010)

Poor oral hygiene: This initiates periodontal diseases leading to root exposure. It has been also reported that periodontal treatment that exposes more root surface could increase incidence of dentin hypersensitivity. (Troil *et al.*, 2002)

Desensitizing agents:

Grossman (Grossman, 1935) had given the characteristics of an ideal dentin desensitizing agent: rapidly acting, long-term effect, harmless to pulp, painless, easy to apply and should not stain the teeth.

Classification of desensitizing agents:

Classification based on the mechanism of action:

1. Disturb neural response to stimulus.
2. Block the flow of tubular liquid and therefore lead to occlusion of dentinal tubules.

Classification based on the mode of administration:

1. At home
2. In office

At home therapy:

Tooth dentifrice and toothpastes: Toothpastes are considered one among the most common over-the-counter materials in desensitizing. When the desensitizing toothpastes appeared on the market for the first time, they, those which contained strontium salt and fluoride, occluded dentinal tubules. However, those which contain formaldehyde, destroy vital elements inside the tubules. Nowadays, most of the desensitizing toothpastes contain potassium salts such as potassium chloride, potassium citrate, and potassium nitrate. (Cummins, 2010) The studies have revealed that potassium salts move along the dentinal tubules and through blocking the axonic action of the intra-dental nerve fibers decrease the excitability of the tooth. (Peacock and Orchardson, 1995; Miglani, Aggarwal and Ahuja, 2010) The patients are recommended to use the minimum amount of water so that the tooth pastes would have their maximum positive effects. (Miglani, Aggarwal and Ahuja, 2010) Several studies have also shown that remineralizing tooth pastes which contained sodium fluoride and calcium phosphates could reduce DH dramatically. (Sowinski *et al.*, 2000; Poulsen *et al.*, 2006; Markowitz, 2013) Recently, some tooth pastes and powders contain arginine and are proved to be effective through several clinical studies. They contain 8% arginine, calcium carbonate, and 1450 ppm fluoride and by establishing an alkaline environment, lead to the precipitation of more salivary calcium and phosphate on the surface and within the dentinal tubules. Furthermore, calcium carbonate, through attracting arginine, forms a molecule which is positively charged. (Cummins, 2009) Toothpastes with different ingredients and different concentrations of desensitizing agents and other agents such as anti-plaques and abrasives may have opposite effects on dentin hypersensitivity. However, in two of the studies which were done in 2005, these agents did not have a significant effect on the desensitizing property of the tooth pastes under study. (Orchardson and Gillam, 2006) The dentist should teach the patient the correct method of tooth brushing. Tooth powders should also be used with soft-bristled toothbrushes. There is no evidence to indicate a better result in using these powders through using fingers instead of toothbrushes. (Orchardson and Gillam, 2006)

Mouthwashes and chewing gums: Findings indicate that mouthwashes which contain potassium nitrate and fluoride reduce dentin hypersensitivity. Mouthwashes used for dentin

hypersensitivity are broadly classified into fluoride containing and non - fluoride containing. (Barcellos *et al.*, 2012)

Non - fluoride desensitising mouthwash: They include strontium salts (i.e., chloride, bromide, iodine, acetate, edetate, nitrate and lactate. They also include sodium chloride and sodium bicarbonate and flavouring agents. Sodium chloride and sodium bicarbonate produce a solution that is isotonic thereby non irritating to the oral mucosa, act to maintain and restore the integrity of the tissues of mouth and throat. (Barcellos *et al.*, 2012)

Fluoride containing desensitising mouthwash: Fluoride desensitizing mouthwash formulations include sodium fluoride, at the concentration 0.2%, is considered effective as a desensitising agent. A combination of potassium nitrate at the concentration of 3% and sodium fluoride at the concentration of 0.2% have therapeutic effect to alleviate dentin hypersensitivity. (Barcellos *et al.*, 2012)

A few studies have also been done on the chewing gums containing potassium chloride. However, the results of such studies are not very reliable. (Gillam *et al.*, 1999; Orchardson and Gillam, 2006) About 2-4 weeks after at- home therapies, the degree of dentin hypersensitivity would be reinvestigated. If the pain still existed, the patient should start the next phase of the therapy; in-office therapy. (Orchardson and Gillam, 2006; Porto, Andrade and Montes, 2009; Miglani, Aggarwal and Ahuja, 2010)

In-office therapy

Theoretically, in-office therapy of DH should lead to immediate relief of the pain. However, practically, this might not be the case. (Miglani, Aggarwal and Ahuja, 2010) Classification of different types of clinical desensitizing agents is based on their mechanism of action and includes occluding dentinal tubules and disturbing the transmission of nerve impulses.

Disturbing the transmission of nerve impulses agent

Only potassium salts (potassium nitrate) and perhaps laser can be placed into the group of disturbers of nerve impulse transmission. Before the discovery of local anesthesia, the dentists used a series of chemical materials such as silver nitrate, zinc chloride, and arsenic compounds to

alleviate dentine discomfort. Nowadays, less poisonous materials are used for desensitizing. (Orchardson and Gillam, 2006; Barcellos *et al.*, 2012)

Potassium nitrate: It is available in two forms of aqueous solution and adhesive gel. As it was stated above, the number of potassium ions decrease when they enter dentinal tubules and decrease the excitability of nerves that transmit pain. (Miglani, Aggarwal and Ahuja, 2010)

Occluding dentinal tubules agents

Fluorides: There are many articles on the effectiveness of fluorides in decreasing dentin hypersensitivity. Fluorides precipitate calcium fluoride crystals inside dentinal tubules, and thus decrease dentinal permeability. (R. H. Dababneh, Khouri and Addy, 1999)These crystals are almost insoluble. Sodium fluoride with a 2 % concentration is used in the office. The precipitate which is formed by sodium fluoride can be removed by the saliva or mechanical scrubbing. Therefore, acid has been added to the formula so that the resultant acidulated sodium fluoride can form precipitates deep in the tubules. (Miglani, Aggarwal and Ahuja, 2010) Fluorides and fluoro-silicates can be used in combination with iontophoresis, which through electrical current can increase ionic diffusion. (Porto, Andrade and Montes, 2009) Stannous fluoride has the same effect as sodium fluoride. If the precipitate of apatite fluoride forms, it can resist salivary action, tooth brushing and dietary substances' action. (Morris, Davis and Richardson, 1999; Walters, 2005)

Oxalates: Oxalates can occlude dentinal tubules and reduce permeability of dentine, up to 98% . (Pillon, Romani and Schmidt, 2004; Vieira *et al.*, 2009) The application of 28% potassium oxalate can lead to the formation of calcium oxalate in the depth of dentinal tubules. However, findings have indicated that the reduction of dentin hypersensitivity induced by oxalate, remains for a short time. To increase the effectiveness of oxalate, the surface of the tooth can be etched. (Sauro *et al.*, 2006) Potassium oxalate can lead to some digestive disorders so it should not be used for a long term. (Miglani, Aggarwal and Ahuja, 2010)

Varnishes: Varnishes can act as a means to help other materials increase their therapeutic effect. Fluoride varnishes combine with acid to increase its effectiveness. (*Management of dentin hypersensitivity*, no date; Hack and Thompson, 1994) Copal varnish is used to cover the exposed

dentine. However, its effect remains for a short period of time and it needs to be applied several times. (Hack and Thompson, 1994)

Adhesive resins: Adhesive systems, unlike the other local desensitizing agents which have a short-term effect, exhibit a long-term or permanent effect. These adhesives include varnishes, bonding agents, and repairing resin composites. The composites can effectively seal dentinal tubules through forming a hybrid layer. (Orchardson and Gillam, 2006) The old adhesives formed the hybrid layer through removing the smear layer and etching the dentinal surface so that deep resin tags could be formed. (Holland *et al.*, 1997; Duran and Sengun, 2004) The new adhesives, however, act in a way that the smear layer will be modified and incorporated into the hybrid layer. (*Clinical evaluation of the role of glutardialdehyde in a one-bottle adhesive*, no date a) It is claimed that the recent dentin bonding agents (DBA) can manage or prevent dentin hypersensitivity. For example, Gluma desensitizing agent (Heraeus Kulzer; Ca, USA) includes hydroxyethyl methacrylate (HEMA), benzalkonium chloride, glutaraldehyde and fluoride. Glutaraldehyde can lead to protein coagulation within dentinal tubules. HEMA can cause resin tags to be formed and dentinal tubules to be occluded. (*Clinical evaluation of the role of glutardialdehyde in a one-bottle adhesive*, no date b; Miglani, Aggarwal and Ahuja, 2010) Gluma has shown good results in management of dentin hypersensitivity in clinical trials. (*Clinical evaluation of the role of glutardialdehyde in a one-bottle adhesive*, no date a)

NEWER METHODS IN THE MANAGEMENT OF DENTIN HYPERSENSITIVITY:

At home:

Carbonated hydroxyapatite nanocrystals: More recently, the toothpastes and mouthrinses containing carbonated hydroxyapatite nanocrystals are being studied. These have high reactivity by which they bind to enamel and dentine apatite producing a biomimetic coating. (Khetawat, 2015) An in vitro study in 2011 concluded that toothpastes containing nano-hydroxyapatite revealed higher remineralizing effects when compared to amine fluoride toothpastes. (Low, Allen and Kontogiorgos, 2015)

In office:

Bioglass: Bioglass has been produced to stimulate bone formation. It is employed to fill the osseous defects during periodontal surgery. (Hench *et al.,* 1971) There are some reports which indicate the effectiveness of bioglass in mineralization and infiltration of dentinal tubules. Its main component is silicate which acts as a nucleus for precipitation of calcium and phosphate. Scanning electron microscopic (SEM) analysis has shown that the application of bioglass causes the formation of an apatite layer which further leads to the occlusion of dentinal tubules. (Forsback, Areva and Salonen, 2004; Vollenweider *et al.*, 2007)

Calcium silicate coating derived from Portland cement: Some researchers have shown that silicate cement which is derived from Portland cement by adding calcium chloride can be effective in management of dentin hypersensitivity and help the occlusion of tubules through remineralization. (Porto, Andrade and Montes, 2009) The dimensions of the powder particles size are micrometrical. Cumulative percentage of particles between 1.5–3 micron size may be approximately 70–80%. The small size makes the particles able to enter into open dentinal tubules. Portland cements mixed with water form a plastic and soft paste that may be applied and spread on the dentin surface. The hypothesis that the application of such paste on the dentinal surface should occlude dentinal tubules with water resistant plugs appears reasonable. (Gandolfi *et al.*, 2008)

Casein-phosphopeptide-amorphous calcium phosphate (CPP)-(ACP): Recently, a remineralizing agent has been produced out of milk casein proteins and has appeared on the market under the name GC Tooth Mousse (GC Asia Pty. Ltd.; Japan). CPP containing phosphoseryl sequences can be helpful in attaching and stabilizing ACP. CPP-ACP remineralizes the early lesions of enamel subsurface. (Cai *et al.,* 2003) The manufacturing factory has claimed that the product can be effective in prevention and treatment of dentin hypersensitivity. Tung et al., have postulated that the materials CPP-ACP and propolis precipitate and obstruct the dentinal tubules and decrease dentinal permeability by 85% or more. (Geiger *et al.,* 2003)

Laser: According to previous researches, the effect of laser on the treatment of dentin hypersensitivity is different and might be between 5-100% based on the type of laser and therapeutic parameters such as the laser's length of beam; the amount of time spent on the use of

laser; and the intensity of laser. (Gerschman, Ruben and Gebart-Eaglemont, 1994; He *et al.*, 2011; Sgolastra *et al.*, 2011) Different mechanisms of action have been proposed for lasers, its effect on the dentine and its effect on reducing dentin hypersensitivity. They include:

1. Occlusion through coagulation of the proteins of the fluid inside the dentinal tubules
2. Occlusion of tubules through partial sub-melting
3. Discharging of internal tubular nerve (Sgolastra *et al.*, 2011)

The lasers used for the treatment of dentine hypersensitivity are divided into two groups:

1. Low output power (low-level) lasers [(He- Ne) helium-neon and (GaAlAs) gallium-aluminum arsenide (diode) lasers]

   Mechanism: In low output power lasers, a small fraction of the laser's energy is transmitted through enamel or dentin to reach the pulp tissue. Low-power laser therapy is an appropriate treatment strategy to promote bio modulatory effects, minimize pain and reduce inflammatory processes. Its use has been widely accepted and approved due to satisfactory results reported in the literature.

2. Middle output power (Carbon Dioxide Laser (CO2), neodymium- or erbium-doped yttrium aluminum garnet (Nd:YAG, Er:YAG lasers) and erbium, chromium doped: yttrium, scandium, gallium and garnet (Er,Cr:YSGG) lasers)

   Mechanism: The effects of high-power lasers, such as the carbon dioxide, Nd:YAG, Er:YAG and Er,Cr:YSGG lasers, are related to an increase in surface temperature which can result in the complete closure of dentinal tubules after recrystallization of the dentinal surface. (Asnaashari and Moeini, 2013)

In a systematic review of the articles, published through the years 2000-2010, on the effects of laser therapy on treating Dentin hypersensitivity, it has generally been claimed that laser therapy for the treatment of dentin hypersensitivity is preferred to other relevant local therapies. (Sgolastra *et al.*, 2011) However, further clinical long-term studies in many different samples and better qualities need to be done to prove this claim. (He *et al.*, 2011) In addition, this type of therapy is highly acceptable to patients because its proper usage has no negative impacts. So far,

there has been no report of adverse reactions or pulp damage in the studies. Thus the use of lasers in treatment of dentin hypersensitivity is both logical and acceptable. (He *et al.*, 2011; Sgolastra *et al.*, 2011)

The patient must be informed in different phases of the therapy:

1. Taking a detailed clinical and dietary history
2. Correct diagnosis of the condition concerning the differential diagnosis of other painful conditions. Diagnosing and treating of the probable etiologic factors causing dentin hypersensitivity
3. Initiating the at-home treatment in cases of mild-to-moderate sensitivity
4. Initiating in-office treatment in cases of severe sensitivity or when one or two teeth were involved
5. EConsidering RCT in cases were at-home and in-office treatments were not effective or when a number of teeth

CONCLUSION:

Dentin hypersensitivity is mainly due to the exposure of dentinal tubules by removal of enamel and cementum due to occlusal wear, psychological factors and anatomic variations. Reasons for the development of dentin hypersensitivity should be diagnosed early and appropriate treatment planning to be done by eliminating the causative agent and use of desensitising agents either at home or in dental office to prevent further tooth wear and sequelae.

COMPETING INTERESTS DISCLAIMER:

Authors have declared that no competing interests exist. The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

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