Dentine hypersensitivity

د.بناز جبار علي

Introduction

Dentine hypersensitivity (D.H.) is one of the most common complaints from patients in dental clinics. Characterized by a short, sharp pain, rapid onset & short duration usually resolves immediately after withdrawal of the stimulus. That arises from exposed dentin in response to non-noxious stimuli, typically thermal, evaporative, tactile, osmotic, or chemical, and that cannot be ascribed to any other form of dental defects or pathology, In more severe, long-standing cases, shorter or longer periods of standing dull or aching pain symptoms may be provoked.

Prevalence

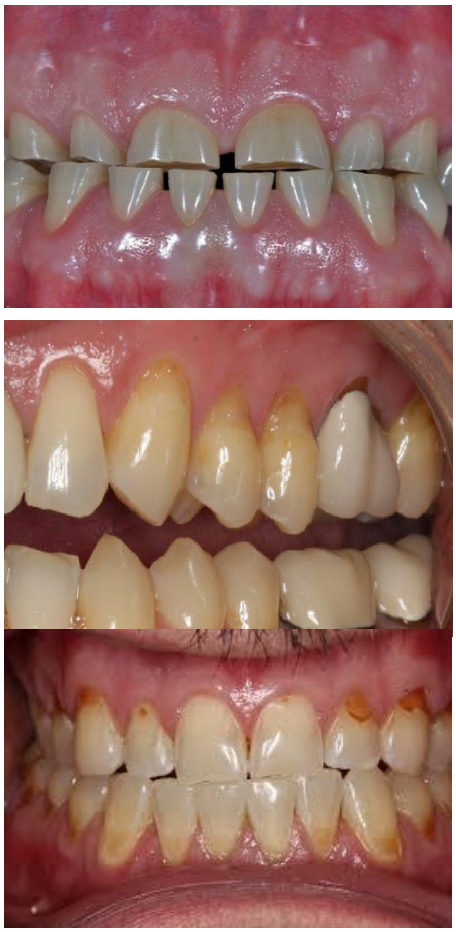
The prevalence, distribution, and appearance of the disease have been reported differently in different studies. Studies in the adult population have been reported that D.H. could affect as many as 1 in 7 of patients attending for dental treatment. Clinical studies and questionnaires on D.H. indicate a prevalence of 4% to 74% and the incidence ranging between 10-30%. D.H. could affect any age group; however, it is more prevalent in the patient with the age range of 30-40 (third decay) and more prevalent in female individuals. Regarding the type of teeth involved, canines and premolars of both arches are the most affected teeth. A buccal aspect of the cervical area is the commonly affected site.

D.H. can lead to both physical and psychological problems for the patient. Furthermore, it can have a negative effect on the quality of a person’s life, especially with regards to dietary selection, maintaining optimal dental hygiene, and beauty aspects.

Etiology

Dentin is covered and protected by hard tissues such as enamel or cementum. The dentin itself is a vital tissue consisting of dentinal tubules that maintain a tapered structure along the length from the pulpodentinal complex to the dentinoenamel junction. These tubules are occupied by odontoblastic processes. The odontoblastic processes may extend through the entire thickness of dentin from pulp to dentino-enamel junction. The odontoblastic processes are the extensions of odontoblasts,

which are the major cells of the pulp–dentin complex. The odontoblastic processes are surrounded by dentinal fluid inside the tubules. The dentinal fluid forms around 22% of the total volume of dentin. It is an ultrafiltrate of blood from the pulp via dentinal tubules and forms a communication medium between the pulp (via the odontoblastic layer) and the outer regions of the dentin. (is naturally sensitive because of extensions of odontoblasts and the formation of dentine–pulp complex). Exposure of dentin could be either to the removal of enamel covering the crown of the tooth or denudation of the root surface by loss of cementum & overlying periodontal tissues



1. Enamel loss may result from

a. Attrition relating to occlusal abnormalities.

Attrition is defined as the wearing of the teeth surfaces due to normal or abnormal function.

b. Abrasion, which is wearing the teeth substance through an abnormal mechanical process as incorrect brushing leaves a deep V-shaped cervical lesion.

c. Erosion which is a chemical process (as acids) manifested as localized progressive destruction of enamel & dentine. The defects vary in shape from saucer-like depressions to deep wedge-like grooves.

Exposure to non-bacterial acids in the diet, chemical products, medication, drugs, or endogenous acids from reflux or regurgitation of stomach acid; that is, substances with low pH lead to the loss of dental structure by chemical dissolution without bacterial involvement. This process produces a more softened enamel zone. In the cervical area, the thinner enamel can be gradually dissolved, and the dentin becomes

exposed to the oral environment. The acid environment can also open the dentinal tubules even further, leading to greater sensitivity.

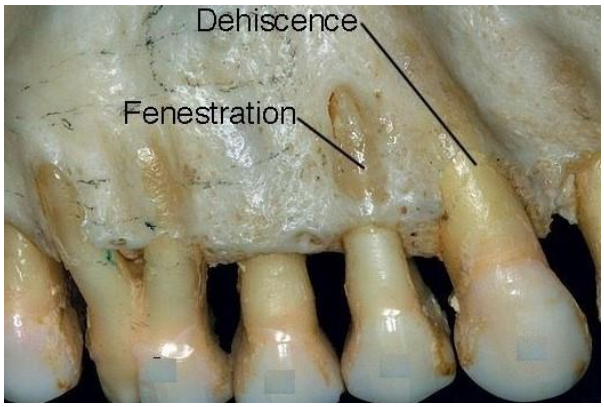
d. Habits as grasping things between teeth.

2. Cementum loss could be due to:

a. Gingival recession:- is the reduction of the height of the marginal gingiva to a location apical to the CEJ , which increase in severity with advancing age

. Gingival Recession may be caused by:

• Mechanical trauma: hard brush, vigorous technique



.Predisposing anatomic factors

1- Thin gingiva

2- Prominent roots

3- Dehiscences and Fenestrations

4- Frenum pulls

4 - Roots moved outside alveolar housing by orthodontic appliances.

• Faulty crown or restoration margins and Poorly designed partial dentures

• Periodontal disease

• Occlusal trauma and Trauma from teeth in opposing jaw

• Oral habits (tobacco smoking & chewing)

• Tooth position

• Healing response following periodontal surgery

b. Chronic periodontal disease as the root surface may become exposed as part of the disease process & the overlying cementum layer is thin & more easily removed.

c. Following periodontal therapy as scaling and root planing & periodontal surgery.

Scaling & root planing may lead to the removal of the thin cementum layer during periodontal scraping & expose the dentinal tubules, which induce hypersensitivity, which is transitory it reaches its peak in the first week after treatment & subside or disappear within few weeks.

Also, after periodontal surgery in which large root surface area is often exposed so leading to hypersensitivity. However, occasionally the condition may become a chronic pain problem and may persist for months or years. Patients appear to be especially at risk after periodontal surgery.

The increase in pain intensity after periodontal therapy may have one or both of the following two explanations.

Firstly, the smear layer formed on the root surface by the scaling procedure will be dissolved within a few days. This, in turn, will increase the hydraulic conductance of the involved dentinal tubules and thus decrease the peripheral resistance to fluid flow across dentin. Thereby pain sensations are more readily evoked.

Secondly, open dentinal tubules serve as pathways for diffusive transport of bacterial elements in the oral cavity to the pulp, which is likely to cause a localized inflammatory pulpal response.

The fact that root dentin hypersensitivity often disappears a few weeks after the scaling procedure is best explained by developing a natural occlusion of the exposed dentinal tubules by mineral deposits.

d. Physiological causes. The increase in the number of teeth with root exposure is evident as age advances. Dental extrusion, in the absence of an antagonist's tooth, results in root exposure, which may lead to D.H.

e. Anatomic variations: failure of meeting between enamel and cementum at CEJ.

Three main mechanisms of dentin sensitivity are proposed

A. Direct Innervation (DI) theory

B. Odontoblast Receptor (OR) theory

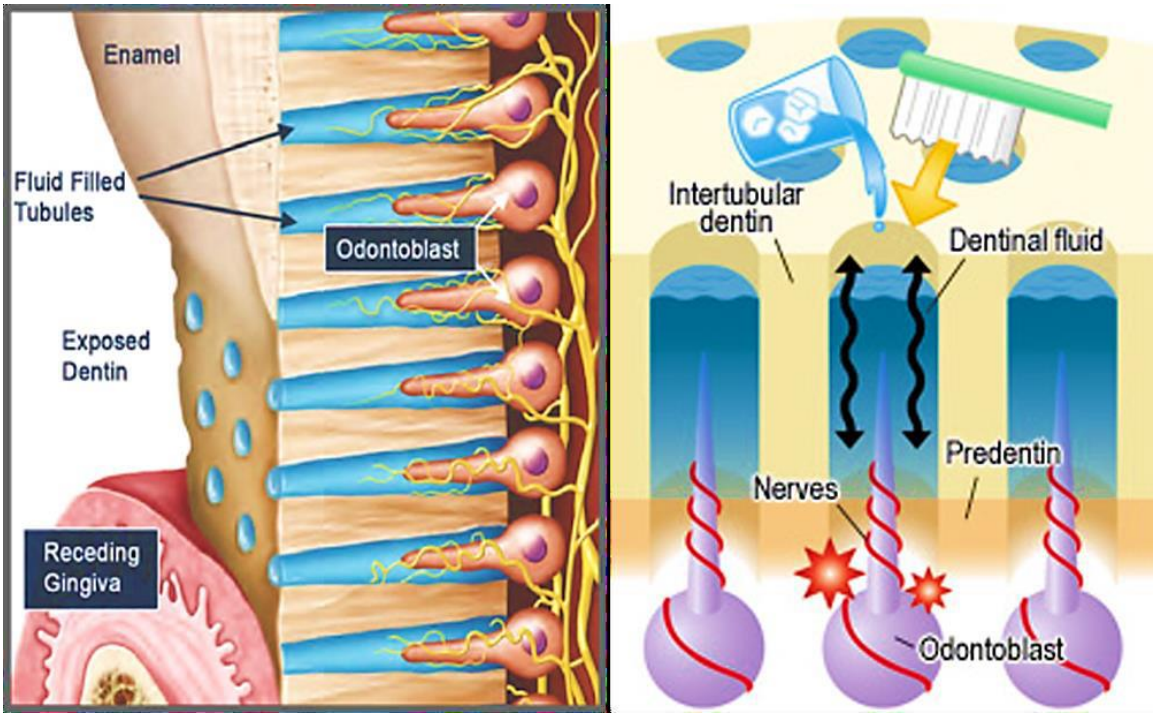
C. Fluid Movement/Hydrodynamic theory

According **to Direct Innervation (DI)theory**, nerve endings penetrate dentine and extend to the dentino-enamel junction. Direct mechanical stimulation of these nerves will initiate an action potential. There are many shortcomings of this theory. There is a lack of evidence that outer dentin, usually the most sensitive part, is innervated. Moreover, pain inducers such as bradykinin fail to induce pain when applied to dentine, and bathing dentine with local anesthetic solutions does not prevent pain, which does so when applied to the skin.

Odontoblast Receptor (OR) theory states that odontoblasts act as receptors by themselves and relay the signal to a nerve terminal. But a majority of studies have shown that odontoblasts are matrix forming cells and hence they are not considered to be excitable cells, and no synapses have been demonstrated between odontoblasts and nerve terminals

hydrodynamic theory. This theory is based on the presence and movement of fluid inside the dentinal tubules. This centrifugal fluid movement, in turn, activates the nerve endings at the end of dentinal tubules or the pulp–dentine complex. This is similar to activating nerve fibers surrounding the hair by touching or applying pressure to the hair. It has been noted that stimuli that tend to move the fluid away from the pulp–denitin complex produce more pain. These stimuli include cooling, drying, evaporation, and application of hypertonic chemical substances. Approximately 75% of patients with D.H. complain of pain with the application of cold stimuli. This theory is the most accepted one related to the explanation of D.H.

In general, the “hypersensitive” dentin has more widely open tubules and thin/under calcified smear layer as compared with “non-sensitive” dentine. The wider tubules increase the fluid movement and thus the pain response.



Diagnosis

The diagnosis of D.H. requires exclusion of other dental and periodontal conditions that might cause pain; Differential diagnosis is indispensable to exclude other conditions with similar symptoms where dentin is exposed and sensitive.

Differential diagnosis:

Dental-related reasons

1. Cracked teeth.

2. Fractured restoration and Marginal leakage.

3. Traumatized or Chipped teeth.

4. Dental caries.

5. Periodontal disease.

6. Post-restorative sensitivity and bleaching sensitivity.

7. Palatogingival groove.

8. Pulpitis.

Non-Odontogenic origin

1. Musculoskeletal
2. Neuropathic
3. Neurovascular
4. Inflammatory (sinusitis)
5. Systemic (cardiac, herpes zoster, sickle cell anemia, neoplasm)
6. Psychogenic
7. Referred pain

Useful diagnostic tools are:

1. Air/water syringe (thermal).

2. Very sharp dental explorer (touch).

3. Percussion testing.

4. Thermal tests such as an ice cube.

5. Tactile testing by applying different degrees of pressure (Yeaple probe).

Methods of measuring D.H.

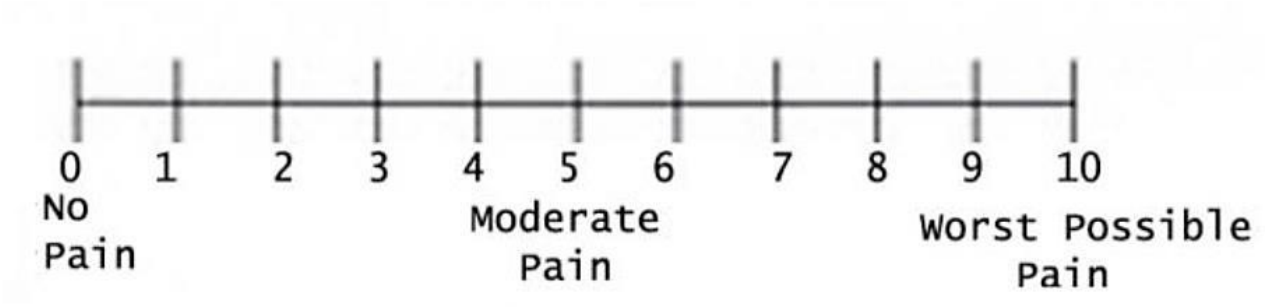
Although D.H. is a subjective sensation that varies greatly from one individual to another and in the same individual at different times.

The severity or degree of pain can be quantified either according to

categorical scale :-(i.e., slight, moderate or severe pain)

A Visual Analogue Scale (VAS): is a measurement instrument that tries to measure a characteristic or attitude that is believed to range across a continuum of values and cannot easily be directly measured**.**

Operationally a VAS is usually a horizontal line, 100 mm in length, anchored by word descriptors at each end. The patient marks on the line the point that they feel represents their perception of their current state. The VAS score is determined by measuring in millimeters from the left-hand end of the line to the point that the patient marks



Treatment of D.H.

Patients should be aware that several factors must be considered during the treatment of tooth hypersensitivity, including the history and severity of the problem, as well as the physical findings of the tooth or teeth involved.

A proper diagnosis is required before any treatment can be initiated so that pathologic causes of pain should be ruled out before attempting to treat the hypersensitivity

D.H. can be triggered by various stimuli:

• Cold in the most common stimuli such as atmospheric air during mouth breathing, particularly in winter.

• Chemical stimuli such as acidic foods (mainly fruit), sweets and rarely with salty foods

• Mechanical stimulus; for example, when the patient rubs the sensitive area with a fingernail, toothbrushing, or the air of a triple syringe by dehydration.

The following information on how to cope with the problem should also be given to the patient:

1. Hypersensitivity appears as a result of the exposure of dentin, which is inevitable if calculus, biofilm, and their products, which are buried in the root, are to be removed.

2. Hypersensitivity slowly disappears in a few weeks.

3. An important factor in reducing hypersensitivity is biofilm control.

4. Desensitizing agents do not produce immediate relief and must be used for several days or even weeks to produce results

After observing the severity and number of teeth involved, an active approach to D.H. can begin in the cases of generalized D.H. by a home method followed by in-office treatment when the first option is not successful. This principle of treatment is based on using different types of desensitizing agents that either occlude or reduce the diameter of dentinal tubules, which minimize the fluid movement in these tubules and hence reducing the pain. In severe cases, where no remedy is achieved with any advice or treatment approach, pulpectomy and root filling or even extraction may be the last resort.

Hypersensitivity may prevent proper plaque control; therefore treatment of hypersensitivity with plaque control measures may be created. Plaque control is an important integral part of the prevention and treatment of root dentin hypersensitivity.

Classification of desensitizing agents

Desensitizing agents need to have certain requirements that include:

1. Non-irritant to the pulp.

2. Relatively painless on application.

3. Easily applied.

4. Rapid in action.

5. Effective for a long time without staining effects

1- Mode of administration

A- At home desensitizing therapy

These “at home” desensitizing agents include toothpaste, mouthwashes, and chewing gums. Toothpaste is widely indicated, mainly because of its low cost, ease of use, and home application. They are effective, but it often takes four to eight weeks to achieve pain relief in addition to the need for patient’s compliance.

B- In-office desensitizing agents

Dental professionals can deliver a wider range of more complex and more potent desensitizing treatment with immediate relief from the pain of D.H. A variety of office applied agents are currently available, including cavity varnishes, calcium compounds, oxalates, resins and adhesives, restorative materials, laser treatment, and an aqueous solution of glutaraldehyde and hydroxyethyl methacrylate.

2- On the basis of the mechanism of action

1. Nerve desensitization

- Potassium nitrate

2. Anti-inflammatory agents

- Corticosteroids

3. Cover or plugging dentinal tubules

a. Plugging (sclerosing) dentinal tubules

i- Ions/salts

- Calcium hydroxide

Ferrous oxide

- Potassium oxalate

- Sodium monofluorophosphate

- Sodium fluoride

- Sodium fluoride/stannous fluoride combination

- Stannous fluoride

- Strontium chloride

- Bioactive glasses ($SiO_2–P_2O_5–CaO–Na_2O$)

ii- Protein precipitants

- Formaldehyde/Glutaraldehyde

- Silver nitrate

- Strontium chloride hexahydrate

- Casein phosphopeptides

- Burnishing

- Fluoride iontophoresis

b. Dentine sealers

- Glass ionomer cement

- Composites

- Resins

- Varnishes

- Sealants

- Methyl methacrylate

c. Periodontal soft tissue grafting

d. Crown placement/restorative material

e. Lasers

1. Nerve desensitization

- Potassium nitrate:- Reduces the excitability of the nerve transmitting pain

Potassium salts act by diffusion along the dentinal tubules and decreasing the excitability of the interdental nerve fibers, Potassium ions are thought to act by blocking the action potential generated in intradental nerves, The toothpaste (5% KNO3), and a professionally applied sealant product (6% ferric oxalate) both produced some obliteration of the tubuli.

Also, toothpastes containing potassium nitrate and fluorides have been shown to reduce post-bleaching sensitivity. The desensitizing toothpastes should be used with the help of a toothbrush with soft bristles. Patients should be advised to use a minimal amount of water to prevent the dilution of the active agent; in addition with the desensitizing mouthwashes and chewing gums containing potassium nitrate, sodium fluoride, or potassium citrate are also recommended. The results of “at-home” desensitizing therapy should be reviewed after every 3–4 weeks. If there is no relief in D.H., “in-office” therapy should be initiated.

2. Anti-inflammatory agents

- Corticosteroids:-

Anti-inflammatory agents such as corticosteroids have been proposed for use to manage dentine hypersensitivity. However, trials have not found them to be

particularly useful. While it is presumed that these agents may induce mineralization leading to tubule occlusion, this view has yet to be validated, and the

validity of using such agents has been questioned

3. Cover or plugging dentinal tubules

a. Plugging (sclerosing) dentinal tubules

- Calcium hydroxide:- Several studies have reported on the effectiveness of calcium hydroxide in managing dentinal hypersensitivity. Its mode of action has been proposed via occlusion of dentinal tubules through the binding of loose protein radicals by calcium ions and increasing mineralization of the exposed dentine. Although immediately effective, the action of calcium hydroxide diminishes rapidly, requiring multiple applications to maintain its effect. (in –office)

Fluorides:-

Many clinical studies have shown that treatment of exposed root surfaces with fluoride toothpaste and concentrated fluoride solutions is very efficient in managing dentinal hypersensitivity. The improvement appears to be due to an increase in the resistance of dentine to acid decalcification and precipitations in the exposed dentinal tubules. Many studies believe that the probable desensitizing effects of fluoride are related to precipitated fluoride compounds mechanically blocking exposed dentinal tubules or fluoride within the tubules blocking the transmission of stimuli. **Fluorides decrease the dentinal permeability by precipitation of calcium fluoride crystals inside the dentinal tubules. These crystals are partially insoluble in saliva**, Various fluoride formulations are used to treat D.H. These include **sodium fluoride, stannous fluoride, sodium monofluorophosphate.**

The precipitates formed by sodium fluoride can be mechanically removed by the action of saliva or mechanical action. Therefore, an addition of an acid formulation is recommended. The acidulated sodium fluoride can form precipitates deep inside the tubules. Also, some authors have recommended the use of ionotophoresis along with sodium fluoride**.**

Stannous fluoride acts similarly as that of sodium fluoride, i.e., formation of calcium fluoride precipitates inside tubules

Oxalates:- Reacts with the calcium ions of dentine and forms calcium oxalate crystals inside the dentinal tubules as well as on the dentinal surface. Topical application of 3% potassium oxalate. . It has been shown that the effect of oxalates on D.H. diminishes over a period of time. This can be attributed to the removal of the calcium oxalate crystals by brushing or dietary acids

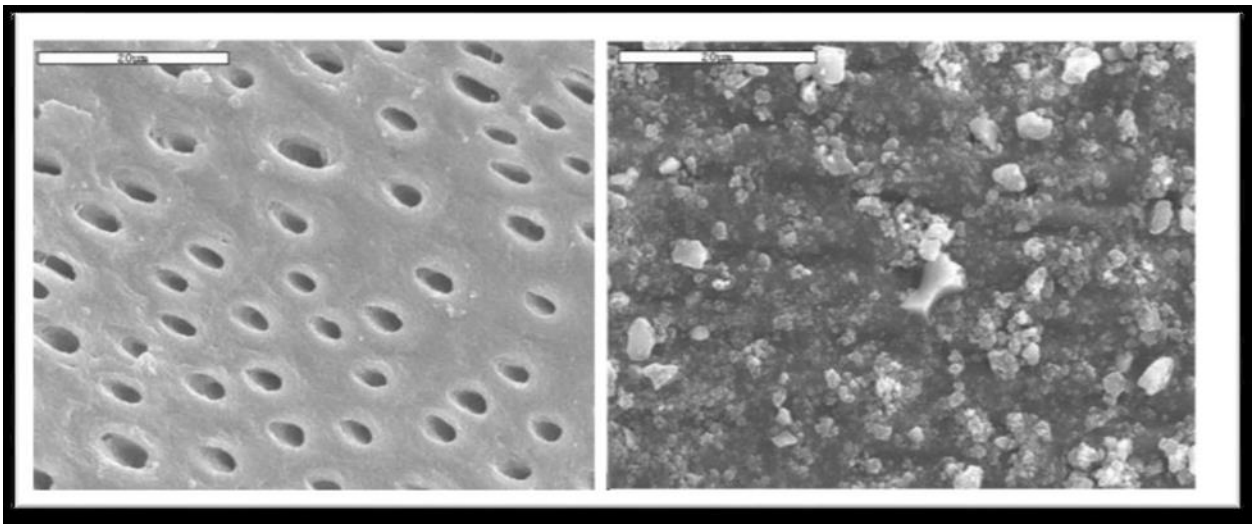
Action can be improved by acid etching of the dentinal surface, thus increasing the penetration of calcium oxalate crystals deep into the dentinal tubules

Strontium chloride:- Reduces fluid movement in the dentin tubules by occluding or sclerosing the tubules

-Bioactive glasses (SiO2–P2O5–CaO–Na2O):- NovaMin is a known component made of bio-active glass particulates with a median size of less than 20 microns.,when NovaMin comes in contact with saliva or any aqueous media, its active ingredient, inorganic chemical calcium sodium phosphor silicate, binds to the tooth surface in order to initiate the remineralization process on the tooth enamel.

This is performed by providing silica, calcium, phosphorous and sodium ions to the tooth structure [7, 8]. A localized transient increase in pH occurs during the initial exposure of the mineral due to the release of sodium. This rise in pH helps the calcium and phosphate form the NovaMin particles, followed by calcium and phosphorous found in saliva to form a calcium phosphate (ca-p) layer. As the particles' reaction continues and deposition of calcium phosphate complex takes place, this layer crystalizes into a calcium hydroxyl apatite, also known as hydroxyl carbonate apatite Antibacterial effect of NovaMin tooth paste has beendocumented against several periodontal pathogens. Sodium Ion is released for several days providing a long term re-mineralization potential

It's available as toothpaste containing NovaMin or In-office chair side desensitization using NovaMin powder



A) Open dentin tubules (B) Closed tubules following NovaMin application

- Formaldehyde/Glutaraldehyde :- formaldehyde and glutaraldehyde, through their ability to precipitate salivary proteins in dentinal tubules, can be used to

manage dentinal hypersensitivity, they should be used with extreme caution to ensure they do not come in contact with the vital gingival tissues. (Protein precipitants)

b. Dentine adhesive sealers

Varnishes :- are commonly used useful in-office measures to treat D.H. Copal varnish can be applied to cover the exposed dentinal surface. But its effect is for the short term and is not recommended for long-term management of D.H. To improve its efficacy, the removal of the smear layer is advocated. Also, the varnishes can act as a vehicle for fluoride. The fluoride varnishes can be acidulated to increase the penetration of ions.

Resin-based dental adhesive systems;- can provide a more durable and long lasting dentine desensitizing effect. The adhesive resins can seal the dentinal tubules effectively by forming a hybrid layer. Various clinical studies have demonstrated the effectiveness of adhesives in the management of D.H. Traditionally, resin composites or dentin bonding agents are used as desensitizing agents. The conventional dentin bonding agents (DBA) removes the smear layer, etches the dentinal surface, and forms deep dentinal resin tags inside the dentinal tubules. The combined dentin–resin layer

(consisting of penetrating resinous tags) has been termed as a hybrid layer. It effectively seals the dentinal tubules and prevents D.H.

- results have been good, but problems arise when the adhesive breaks away, resulting in exposure of the tubules. ---This technique is generally reserved for cases of specific and localized dentinal hypersensitivity rather than generalized dentinal pain.

Restorative materials :-(Glass ionomer cement, Composites)

The use of restorative materials is generally an invasive solution to the problem of hypersensitivity. Commonly used materials include composite resins and glass ionomer restorations. Occlude dentinal tubules and restore tooth contours

Indicated in case of significant loss of cervical tooth structure or case of failure of other less invasive desensitizing protocols

c. Periodontal soft tissue grafting:- There are numerous soft tissue grafting procedures that can be carried out to cover exposed root surfaces, including lateral sliding grafts, free gingival grafts, connective tissue grafts, and coronally repositioned

flaps. Soft tissue grafting for localized recession defects requires careful

planning and an understanding of the anatomical defect to be treated. In general, soft tissue grafting for the management of sensitivity is not regarded as a very predictable treatment strategy.

d. Crown placement/restorative material

e. Lasers:- Coagulate protein inside dentinal tubules, thus preventing fluid movement

- Enhance the action of other desensitising agents such as sodium fluoride and stannous fluoride.