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Review

Diagnosis and treatment of dentinal hypersensitivity

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Abstract: This bibliographic review provides a general view of the etiology, characteristics and treatment of dentinal hypersensitivity, so that professionals can use this information in the therapeutic management of this clinical condition. For this purpose, the authors have analyzed whole texts of relevant articles on the subject. This study showed that the predisposing factors associated with the causes of dentinal hypersensitivity must be controlled or eliminated, by educating the patient regarding the excessive intake of acidic food, as well as providing guidance on the proper tooth brushing technique and analysis of occlusion. Effective treatment must be preceded by a proper diagnosis, established after the exclusion of any other possible causes of the pain. These cases must be managed efficiently, quickly and permanently. The availability of a wide variety of treatment could be an indicator that there is still no effective desensitizing agent to completely resolve the patient's discomfort, or that it is difficult to treat, irrespective of the available treatment options. Even with the large number of published studies, it has not been possible to reach a consensus about the product that represents the gold standard in the treatment of dentinal hypersensitivity. (J Oral Sci 51, 323-332, 2009)

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Introduction

Dentinal hypersensitivity (DH) is characterized by short sharp pain arising from exposed dentine 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 (1). A modification of this definition was suggested by the Canadian Advisory Board on Dentine Hypersensitivity (2) in 2003, which suggested that 'disease' should be substituted for 'pathology'. The definition provides a clinical descriptor of the condition and identifies DH as a distinct clinical entity.

Others terms to describe DH have been created by substituting the word dentinal, adding site descriptors, such as cervical or root, and combining this with either hypersensitivity or sensitivity. This practice resulted in a significant number of permutations to describe the apparently same condition (3) (Table 1).

Despite the existence of these various terms, several authors prefer the term DH, commonly used and accepted for many decades to describe a specific painful condition of teeth, which is distinct from others types of dentinal pain having different etiologies.

DH is a painful clinical condition that affects 8 to 57%

Table 1 Expressions in frequent usage for DH

Dentine Sensitivity
Dentine Hypersensitivity
Dentinal Hypersensitivity
Cervical Hypersensitivity/Sensitivity
Root Hypersensitivity/Sensitivity
Cemental Hypersensitivity/Sensitivity

of the adult population and is associated with the dentin exposure to the oral environment (4,5).

The difficulty found in treating DH is expressed by the enormous number of techniques and therapeutic alternatives to relieve it. Several methods and materials, such as varnishes, liners, restorative materials, dentinal adhesives (6), dentifrices and mouthwashes are used to reduce dental sensitivity (7).

Although there are a large number of techniques and therapeutic alternatives available in the literature with the purpose of relieving DH, generally speaking, professionals are confused about the etiology and diagnosis of DH resulting in the lack of confidence to approach this pathological process effectively (7,8).

The aim of this bibliographic review is to provide a general overview about the etiology, characteristics and treatment of DH, so that professionals can use this information for the therapeutic management of DH.

Literature Review

There are many DH studies. Nevertheless, most dental professionals are confused about the diagnosis, etiology and mechanisms of DH. Practitioners also report that they lack the confidence to manage the condition effectively (8) and this frequently leads to clinical failure.

Characteristics of DH

DH is a relatively common dental clinical condition in permanent teeth caused by dentin exposure to the oral environment as a consequence of loss of enamel and/or cementum. It is manifested in a manner that is physically and psychologically uncomfortable for the patient and it may be defined as acute pain of short duration caused by the presence of open dentinal tubules on an exposed dentinal surface (9).

The stimulus that triggers the onset of pain can be of thermal, chemical or mechanical origin. The most common complaint is caused by cold stimuli. Pain may also occur by chemical stimuli such as acidic foods (mainly fruit), sweets and rarely with salty foods. Mechanical stimulus frequently occurs when the patient rubs the sensitive area with a finger nail, or toothbrush bristles during brushing, setting off pain. The atmospheric air during mouth breathing, particularly in winter, which is associated with cold, or the air of a triple syringe by dehydration also causes pain (10-12).

Many theories have been used to explain the mechanisms of DH. An early hypothesis was the dentinal receptor mechanism theory, which suggests that DH is caused by the direct stimulation of sensory nerve endings in dentine. On the basis of microscopic and experimental data, it seems unlikely that neural cells exist in the sensory portion of the outer dentine (13). This theory is not well accepted.

The odontoblast transducer mechanism proposed by Rapp et al. (14) suggested that odontoblasts act as receptor cells, mediating changes in the membrane potential of the odontoblasts via synaptic junctions with nerves. This could result in the sensation of pain from the nerve endings located in the pulpodentinal border; however, evidence for the odontoblast transducer mechanism theory is generally lacking and inconclusive (15).

Pain, caused by the movement of fluid in the dentinal tubules (16), can be explained by the widely accepted "Hydrodynamic theory" proposed by Brännström and Astron in 1964 (17). According to this theory, the presence of lesions involving enamel and/or cementum loss in the cervical area and the consequent opening of dentinal tubules to the oral environment, under certain stimuli, allows the movement of dentinal fluid inside the tubules, indirectly stimulating the extremities of the pulp nerves, causing the pain sensation. Evidence of this hypothesis may be reviewed in the literature (18,19). Nevertheless, the mechanism by which the flow of fluid stimulates the nerve impulses is still unknown (12).

Physical stimulation is more difficult to explain through this theory although it is possible that mechanical abrasion of the exposed dentine surface may be sufficient to induce unwanted fluid flow within the dentinal tubules with resulting pain from the stimulated nerve fibers (20).

Pain has extremely variable characteristics, ranging from discrete discomfort to extreme severity. The level of pain varies among different teeth and different persons. It is related to individual tolerance of pain and to physical and emotional factors. It may be localized (one or two teeth) or generalized (several teeth) and in some cases, it may be felt in all four quadrants of the mouth (7).

Histologically, sensitive dentin presents widened dentinal tubules, two times larger when compared with tubules of normal dentin and in a greater number per area, when compared with the dentin without sensitivity (21).

At a macroscopic level, dentine exhibiting hyper-sensitivity appears no different from non-sensitive dentine. The status of the pulp in DH is not known, although symptoms would suggest minor inflammation as a result of the length of time that symptoms persist without developing into a true pulpitis (15).

Etiology and Prevalence

DH can manifest when dentin is exposed by enamel loss (lesions of abrasion, erosion or corrosion) followed by the constant action of acids, which keep the tubules open on the dentin surface, or because the root surface has been

denuded due to loss of structures such as cementum, which is easily removed by brushing or periodontal treatment (8), or more commonly, by the association of two or more of these factors (22,23). It may also be caused by gingival recession which occurs with aging, chronic periodontal disease and patient's deleterious habits (24).

Studies (7,25) indicate that dentin exposure may result in anatomic characteristics in the area of the enamel/cementum junction and/or enamel, or cementum loss due to one or more of the following processes:

1. Lack of or excessive tooth brushing. Traumatic brushing due to the poor position of vestibularized teeth, which makes them more subject to brushing trauma, or by excessive force or even lack of brushing, with consequent accumulation of dental plaque, causing gingival inflammation which may lead to periodontal complications and migration of the gingiva in the apical direction, exposing the cementum and then the root dentin (26). Excessive zeal in performing oral hygiene procedures is also pointed out as being responsible for the appearance of pain (22).

2. Low level of oral hygiene. Patients with a low level of oral hygiene have a high degree of periodontal tissue destruction, loss of supporting bone tissue and root exposure (27). Root exposure is related to DH and it can be aggravated by the action of acids secreted by bacteria capable of opening the dentinal tubules even further (28).

3. Periodontal therapy has been associated with DH due to the exposure of dentinal tubules after the removal of supra and/or subgingival calculi. Another factor is the removal of dental cementum which covers the root or the root dentin itself during periodontal scraping (29).

4. 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, called erosion, produces a more softened enamel zone (8,30). In the cervical area, the thinner enamel can be gradually dissolved and dentin becomes exposed to the oral environment (31). The acid environment can also open the dentinal tubules even further, leading to greater sensitivity. Moreover, this process can be associated with abrasion, particularly in the cases of an acidic diet or gastric reflux associated with brushing performed immediately after these processes (28).

5. Occlusal contact with excessive force and premature occlusal contact. Excessive occlusal forces have been related to tooth deformation and flexion, resulting in fracture of the enamel crystals in the cervical region, contributing to the exposure of coronal dentin, and in more severe cases, of coronal and root dentin (31,32). This lesion, classified as abfraction, is not directly related to the diet, periodontal disease or abrasion (32). However, it may be a predisposing factor to DH (27,33).

6. 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 tooth, results in root exposure, which may lead to DH (24).

Clinical studies and questionnaires on DH indicate a prevalence of 4% to 74%. It mostly affects individuals at the end of their third decade of life, causing patients great discomfort. In some cases, it may lead to emotional alterations and behavior changes. It is mostly found in permanent canines and premolars in both dental arches. The cervical region of the vestibular face of teeth is the most affected region (2,3,9,34-36).

Diagnosis and Clinical Management of DH

Clinical management of DH is based on proper diagnosis, considering its severity, localized or generalized condition, elimination of other possible causes of pain, elimination or prevention of the causes. This involves patient counseling about hygiene practices (type and hardness of toothbrush, brushing before or after meals), diet (frequency of food and acidic beverage intake) and other harmful habits (7).

A correct anamnesis associated with a careful clinical and radiographic examination allows DH to be differentiated from other pathologies that affect the teeth. Correct diagnosis is extremely important since the history may be clinically confounded with incipient caries, restorations in a poor state of conservation or performed recently, cracks or dental fractures and teeth with reversible or irreversible inflammatory processes of the pulp (3,5). Post-dental bleaching sensitivity is a major adverse effect of vital tooth bleaching mainly attributed to the penetration of the bleaching agent into the pulp chamber and it reflects reversible pulpitis (37). Taking these factors into consideration, it is necessary to exclude other forms of pain or dental sensitivity.

To obtain a conclusive diagnosis of DH, first carefully evaluate, investigate and compare among the other teeth, in order to eliminate other possible causes of pain, which could lead to confusion. A good clinical history is essential and questions asked by the professional may help to collect important information that will help in treatment.

Traditionally, dentists have used an exploratory probe or jets of air from a triple syringe on the exposed surface to provoke a response from the patient (7). Tactile stimulus with the use of a probe is the easiest, fastest and most precise method to identify the areas suspected of having DH (7). The method consists of touching the cervically exposed

dentin with a probe starting from the distal and working towards the mesial region, examining all the teeth in the area in which the patient reports pain.

The degree of severity of pain can be quantified by means of a descriptive scale: slight, moderate or intense pain (38), or a visual analogue scale – VAS; 0-10 (7).

The emotional component may be associated with painful sensitivity and regression of symptomatology may occur without any treatment or with the use of placebos. Spontaneous cure may occur by the natural remineralization process in the mouth, which promotes natural tubular occlusion of dentin (39), and pain may return because of the smear layer removal by food and acidic drinks (40) thus explaining the cyclic characteristic of DH (7).

After observing the severity and number of teeth involved, an active approach to DH can begin in the cases of generalized DH, by a home method followed by in-office treatment when the first option is not successful. However, when DH is restricted to a few teeth, one can opt for an in-office method as initial treatment (8).

Control of Dentinal Hypersensitivity

In 1935, Grossman (41) reported some requirements of ideal treatment for DH, which can still be applied nowadays. The treatment must act fast, be effective for long periods, be easy to apply, not irritate the pulp, not cause pain, not stain the teeth and be constantly effective.

Desensitizing agents have been classified according to their mode of action (42); whether they are applied by the patient or professional, according to their chemical or physical properties (43); or by their reversible or irreversible characteristics (44). They may be found in the form of gels, dentifrices, mouthwashes, or agents to be applied topically, such as varnishes, resin composite, glass ionomer cement, dentinal adhesives, periodontal membranes and laser applications.

But it is difficult to classify them by their mode of action, because in the case of some substances, their desensitizing action has not yet been well explained. It is perhaps easier to classify them by their mode of administration: at home or professional (7).

Products sold in pharmacies and dispensing laboratories, such as dentifrices and solutions for mouthwashes have been widely tested and indicated for pain relief. Nevertheless, a variety of more complex and powerful products are available for use in dental clinics, such as potassium oxalate, fluorides, adhesives, resinous sealers, and others.

The advantage of using products available for home use is that they are immediately available for treatment, when compared with those applied by a professional. One disadvantage is that time is needed for remission of the symptoms (2-4 weeks), while theoretically, those applied in-office promote immediate relief. For generalized sensitivity involving several teeth, the use of a desensitizing dentifrice with strontium chloride and potassium nitrate produced relief in 2 weeks (45,46).

According to Pashley (47), products for in-office application are generally classified as those that do not polymerize, such as varnishes and precipitating agents, and those that undergo a setting reaction or polymerizing action, such as the conventional or resin-modified glass ionomer cements, and resinous adhesives. Other forms mentioned in the literature are the homeopathic medications (Plantago major) (48) and propolis (a mixture of resin, essential oils and wax, mixed with beeswax, amino acids, minerals, ethanol, vitamins A, B complex and E, pollen and bioflavonoids) (49). Nevertheless, information about the efficiency of these products is scarce.

Nowadays, two main methods are used in the treatment of DH: tubular occlusion and blockage of nerve activity by means of direct ionic diffusion, increasing the concentration of potassium ions acting on the pulpal nerve sensorial activity (50).

Occlusive therapies for the treatment of dentinal hypersensitivity are frequently proposed because it is believed that sealing the dentinal surface diminishes the movement of fluids inside the tubule and is capable of reducing DH (51). Among the substances indicated for this type of treatment are the oxalate, chloride and fluoride-based agents, either associated with iontophoresis, or not.

Topical application of fluoride by a professional has been recommended after periodontal treatment to relieve the patient's discomfort. There is also evidence that the home use of fluoridated products, as well as potassium nitrate and strontium acetate with fluoride, in the form of dentifrices and mouthwashes can benefit patients, by reducing sensitivity and dentin solubility, acting not only in reducing DH, but also in preventing caries (52). Contrary to the mentioned authors, Gillam and Orchardson (7) argue that, in spite of dentifrices with fluoride being widely used in Western countries, no significant reduction in DH has been perceived.

The use of desensitizing agents such as potassium nitrate and fluoride has also been proposed to reduce tooth sensitivity post-dental bleaching sensitivity (53,54).

Some researchers (55,56) have incorporated fluoride with iontophoresis, a technique that uses electricity to increase ion diffusion into the tissues. Iontophoresis with sodium fluoride is a method proposed for desensitizing dentin with the purpose of enabling deeper penetration of fluoride ion into the dentinal tubules, but it is not considered a simple

technique, because it involves the use of a specific appliance and it provides results similar to other simpler techniques. Dental iontophoresis is more frequently used in conjunction with dentifrices (57) and fluoridated solutions (58) and reduces DH (57,58).

Fluorides, such as sodium and stannous fluoride can reduce DH (59). The application of fluorides seems to create a barrier by precipitation of the calcium fluoride crystals which are formed especially in the inlet of the dentinal tubules. The precipitate is slowly soluble in saliva, which may explain the transitory action of this barrier (8).

Thrash (60) compared the effect of 0.4% stannous fluoride gel and an aqueous solution with 0.717% of fluoride and concluded that this aqueous solution provides an immediate effect when applied for 3 to 5 minutes in-office. The stannous fluoride gel has a gradual effect and it can be used by the patient at home to obtain a long-term effect.

In the study conducted by Suge et al. (61), ammonium hexafluorosilicate [$(NH_4)_2SiF_6$] was considered useful in the treatment of DH, because it induces precipitation of calcium phosphate from saliva, presenting a continuous effect of dentin tubular occlusion in an environment that simulated the oral environment. Treatment with fluoro-silicate (SiF) could play an important role in obtaining durable occlusion because some silica composites induce the formation of apatite. The open tubules were completely obliterated with the precipitation of calcium silicate-phosphate (62).

The precipitate seemed to be a mixture of calcium fluoride and fluoridated apatite. The former is subsaturated in comparison with saliva and thus remains in the oral environment for a short period, however, if the precipitate is predominantly fluoridated apatite, a stable occlusion can be expected, because this composite is supersaturated in comparison with saliva (61) and it is found deeply deposited in the dentinal tubules (63). Another positive aspect is that this composite does not present inconvenience of dentin pigmentation by the precipitation of silver ions from silver diamine fluoride (26).

The precipitate formed by substances used in the treatment of DH can disappear by the action of saliva, mechanical factors, such as brushing or chemical factors such as food, acidic beverages and the acid from dental biofilm (61). However, the crystals deposited inside the dentinal tubules at a depth of 60-70 μm, such as the ones formed after treatment with fluorosilicates, are difficult to remove. Moreover, demineralization caused by these acids, which exacerbate dentinal sensitivity, can be prevented by the acid resistance of the tooth after treatment with fluorosilicates, which is higher than when acidulated phosphate fluoride and sodium fluoride are used (39).

Topical use of 3% potassium oxalate on exposed dentin after periodontal procedures results in a reduction of DH (64). The desensitizing action of potassium oxalate occurs by the deposition of calcium oxalate crystals on the dentin surface. Oxalate reacts with the dentin calcium and promotes deposition of calcium oxalate crystals on the dentin surface and/or inside its tubules, significantly reducing hydraulic conductivity inherent to this structure, sealing the tubules more effectively than the intact smear layer. If the hydrodynamic mechanism is responsible for pain, this effect observed after the application of potassium oxalate leads to the reduction of DH (12).

The calcium oxalate crystals formed on the dentin surface are easily removed by daily brushing. However, when dentin is previously etched with 35% phosphoric acid, the penetration depth of oxalate buffer into the dentinal tubules is about 6-7 μm (65) and thus, pain relief can be expected for a longer period. The application of potassium oxalate on the etched dentin can also be associated with a covering of dentinal adhesives (66).

In vitro studies have shown that phytocomplexes containing oxalate derived from rhubarb stalks (*Rhubarb rhaponicum*) and spinach leaves (*Spinacia oleracia*) promote occlusion of dentinal tubules by the formation of acid resistant calcium oxalate crystals on the dentin surface and inside its tubules, and may be effective for topical treatment of DH. An adequate commercial product formula for clinical application is being tested (16) and it seems to be a promising alternative.

However, there are limitations to the clinical use of potassium oxalate due to its potential toxicity. Professionals must avoid its application with a mold for the treatment of generalized DH, because it may result in gastric irritation (67).

Copal varnish has also been recommended for the treatment of DH, but its action is transitory and usually lasts only a few hours. Nevertheless, it may serve as a vehicle for fluoride and success has been obtained due to this factor. According to Hack (68), it is desirable to remove the smear layer before the cavity varnish is applied, otherwise it will remain on the smear layer surface and it will not promote obliteration of the tubules. The use of fluoridated varnish is indicated for the treatment of DH because it has shown to be very effective by having an immediate effect, and being easy to apply and handle.

Dentifrices

Dentifrices are the most common vehicles for de-sensitizing agents. They are widely indicated, particularly because of their low cost, ease of use and home application.

They present complex formulae with several ingredients, among them desensitizing agents such as strontium chloride, potassium nitrate, dibasic sodium citrate, formaldehyde, sodium fluoride, sodium monofluorphosphate and stannous fluoride (51,69-71).

The mechanism of action is based on the obliteration of dentinal tubules, by the precipitation of calcium phosphate on the dentin surface and calcium is the most frequent component present in the dentifrices (70). Many dentifrices contain abrasives (calcium carbonate, aluminum, calcium phosphate, silicate, etc) which may also cause obliteration of the tubules by the abrasive or indirectly by the formation of a smear layer during brushing (71,72).

Dentifrices must be applied with a toothbrush. There is no evidence that application by friction with the finger increases their efficacy (2). It must be recommended to the patient to use a reduced quantity of water, and after brushing, avoid rinsing with water, because this can dilute the active agent, which will be expelled, reducing the desired effect (73).

Silver nitrate reduces DH by fast coagulation of the Tomes processes forming silver albuminate, which acquires a dark color when exposed to light, blackening the tooth surface. The subsequent use of sodium chloride reduces the pigmentation. Thus, due to tooth darkening, this technique is not well accepted among patients. Studies using dentin disks obtained from extracted teeth showed that the presence of proteins in the dentinal tubules has little to do with the reduction of dentin hydraulic conductivity caused by the silver nitrate (62).

Unlike other products, potassium nitrate does not diminish dentin hydraulic conductivity, or promote obstruction of dentinal tubules by the deposition of crystals. According to Wilchgers and Ermert (28) and Kim (74), potassium nitrate has an effective desensitizing action. It is believed that the increase in the concentration of extracellular potassium around the nerve fibers causes their depolarization, avoids repolarization and blocks the axonic action and passage of nerve stimulus, thus inactivating the action potential.

Strontium chloride and zinc chloride are protein precipitants and their mechanism is through organic precipitation and odontoblast denaturation forming a sealing film that prevents fluid movement and has an occlusive action. After conducting studies, Minkoff and Axelrod (75) concluded that regular home use of dentifrices with 10% strontium chloride is an efficient means of reducing DH.

Strontium can react with fluoride if the two components are present in the same formula, thus, an alternative to avoid this interaction is to use fluoride and strontium acetate (76,77).

Adhesive Materials

Adhesive restorative materials and dentinal adhesives are considered dentinal tubule sealers. Some studies have investigated the role of these materials on the exposed dentin of cervical lesions and the results showed an acceptable durability, except when there are fractures in the material (50).

When there is no loss of dental structure, dentinal adhesives in the form of bonding agents and varnishes can be indicated. They produce an immediate effect, but they are easily removed (78).

Resinous dentinal desensitizers, such as Gluma Desensitizer® (Heraeus Kulzer) are products which unite dentin and they can effectively seal the dentinal tubule openings. They were designed to produce an immediate long-term effect, and clinically they have been shown to fulfill these requirements. These materials are relatively new on the market and they are promising for the treatment of dentinal hypersensitivity. Basically, in their composition they have: hydroxyethyl methacrylate (HEMA), benzalkonium chloride, glutaraldehyde and fluoride.

HEMA physically blocks the dentinal tubules and glutaraldehyde causes coagulation of plasma proteins of the tubule fluid, resulting in the reduction of dentinal permeability. HEMA can be absorbed by dentin and collagen and glutaraldehyde can form cross-links with bovine serum collagen and albumin. These results, found by Qin et al. (79), suggest that Gluma acts as a desensitizer by means of two reactions. First, the glutaraldehyde reacts with part of the serum albumin in the dentinal fluid which induces albumin precipitation, and then a second reaction of glutaraldehyde with albumin induces HEMA polymerization.

Resin composites and glass ionomer cements, as well as varnishes and dentinal adhesives work as fillings, sealing the entrances of the open dentinal tubules and blocking sensitivity by the formation of a sealing covering. Nevertheless, a restorative material must only be used when there is a loss of dental structure (69,80-83).

Powell, Gordon and Johnson (83) found significantly diminished post-operative sensitivity to all the stimuli when using only a restorative material, glass ionomer, or resin composite, or a combination of resin composite with glass ionomer lining. After six months, the reduction in sensitivity of the lesions to air was between 57 and 78%; to heat 80%: and to cold between 57 and 76%.

However, there are controversies as regards the option to restore non-carious cervical lesions. For the majority of professionals, restorations would be indicated in cases

when the structural integrity of the dental element is compromised, pulp is exposed, the aspect of the lesion makes it difficult to manufacture a denture, or esthetics is compromised.

More invasive therapies, such as restorations, dental pulp removal, etc, can be the treatment of choice if attempts to achieve pain remission with a more conservative procedure fail (84).

Laser Treatment

Laser therapy has been recommended by Kimura et al. (85) to treat DH with effectiveness between 5.2% and 100%, depending on the type of laser and parameters used. According to the authors, lasers are more effective than other treatments, although the effectiveness diminishes in severe DH.

The mechanism of laser treatment for DH is not well explained (85), although Pashley (47) suggests that it may occur through coagulation and protein precipitation of the plasma in the dentinal fluid or by alteration of the nerve fiber activity. The study by McCarthy et al. (86) indicates that the reduction in DH could be the result of alteration of the root dentinal surface, physically occluding the dentinal tubules.

Recent Progress in the Treatment of DH

A new proposal presented by Gandolfi et al. (87) is the application of a calcium silicate paste derived from Portland cement, which was shown to be effective in tubular occlusion and reduction of dentin permeability, and may be indicated for the treatment of DH.

Table 2 presents a surprisingly large number of information in the published literature regarding products for treatment of DH.

From a review of the literature, it is noted that an effective treatment must be preceded by proper diagnosis established after the exclusion of any other possible causes of the pain. It is important to manage the cases efficiently, quickly and permanently.

The availability of a wide variety of treatments could be an indicator that there is still no ideal desensitizing agent for the treatment of DH, or that it is difficult to treat, irrespective of the options of available treatments. Even with the large number of studies published, it was still not possible to reach a consensus about a product that represents the gold standard in the treatment of DH.

Table 2 Summary of treatment strategies for DH

1. Nerve desensitization
Potassium nitrate
2. Cover or plugging dentinal tubules
a. Plugging dentinal tubules
Ions/salts
Aluminum
Ammonium hexafluorosilicate
Calcium hydroxide
Calcium carbonate
Calcium phosphate
Calcium silicate
Dibasic sodium citrate
Fluorosilicate
Potassium oxalate
Silicate
Sodium monofluorophosphate
Sodium fluoride
Sodium fluoride/stannous fluoride combination
Stannous fluoride
Strontium acetate with fluoride
Strontium chloride
Protein precipitants
Formaldehyde
Glutaraldehyde
Silver nitrate
Strontium chloride hexahydrate
Zinc chloride
Phytocomplexes
Rhubarb rhaponicum
Spinacia oleracia
Fluoride iontophoresis
b. Dentine sealers
Glass ionomer cements
Composites
Dentinal adhesives
Resinous dentinal desensitizers
Varnishes
Sealants
Methyl Methacrylate
c. Periodontal soft tissue grafting
d. Lasers
e. Homeophatic medications
Plantago maior
Propolis

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