Review Article

Effectiveness of Lasers in the Treatment of Dentin Hypersensitivity

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Abstract:

Dentin hypersensitivity (DH) is a relatively common painful condition among dental problems. Although many studies have been performed regarding the diagnosis and treatment of DH, dental practitioners are still confused about the definite diagnosis and treatment. The use of lasers as a treatment for dentin hypersensitivity was first introduced in 1985. Laser treatment in dentin hypersensitivity is an interesting and controversial issue and many investigations have been done on its mechanism of action, advantages, and unclear points. The present literature review tries to go over the definition, diagnosis, etiology, predisposing factors, various laser types in the treatment of DH alone or in combination with topical desensitizing agents. Since a certain treatment has not yet introduced for dentin hypersensitivity, a combination of laser therapy and topical desensitizing factors, can increase the success of the treatment compared with either treatments alone.

Keywords: dentine hypersensitivity; laser therapies; Nd-YAG lasers; dentin desensitizing agents

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Introduction

The term Dentin hypersensitivity (DH) has been used to describe a specific condition that is defined as pain arising from exposed dentin (1). Typically the pain in DH is brief, sharp, well-localized in response to thermal, evaporative, tactile, osmotic, or chemical stimuli which cannot be referred to any other form of dental defect or pathology (2). DH is probably a symptom complex, rather than disease and a persisting problem that affects as many as one of seven dental patients (1).

Dentin hypersensitivity is a prevalent oral problem, affecting more than 40% of adults worldwide and more than 40 million people in the United States (3). Some studies have reported prevalence levels as high as 68% (4). The cervical area of teeth is the most common site of hypersensitivity (5). Studies have reported that premolars are most commonly affected by dentin hypersensitivity (6). However, another study found that mandibular incisors were most commonly affected and determined that most hypersensitive areas were found on the facial surface of teeth (7).

Dentin Hypersensitivity Mechanism

Theories about the transmission of pain stimuli in DH suggest that pain is amplified when the dentinal tubules are open to the oral cavity (5).

Many theories have been used to explain the mechanisms of DH. The most widely accepted theory is classic—hydrodynamic mechanism proposed by Brännström and Astron (8).

In this mechanism, sudden movements of fluid in the dentinal tubules are believed to deform mechano-sensitive nerve fibers close to the odontobastic layer (1).

A variety of stimuli can result in pressure change traverse the dentin, resulting in stimulation of intra-dental nerves (2).

Dentin Hypersensitivity is the result of activation of A-δ nerve fibers located in the dentinal tubules (1). A-δ fibers are probably activated by the hydrodynamic mechanism. Therefore, their activation is directly associated to the presence of opened or occluded tubules.

However, hypersensitivity sometimes remains in spite of the effective blocking of the tubules, suggesting that other mechanisms contribute to nerve activation instead of or in addition to the hydrodynamic mechanism (9).

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 (10). The odontoblastic processes mechanism proposed by Rapp et al. suggested that odontoblasts act as receptor cells, mediating changes in the membrane potential of the odontoblasts via synaptic junctions with nerves (11).

The sensitivity of dentin has a direct correlation with the size and patency of the dentinal tubules. Absi and colleagues discovered that hypersensitive teeth have an increased number of patent tubules and wider tubules than those of non-sensitive teeth (12).

Etiology and Predisposing Factors of DH

The relationship between DH and the patency of dentin tubules in vivo has been established, and the occlusion of the tubules seems to decrease that sensitivity (5).

There are many varieties of potential causes for dentin sensitivity. The loss of enamel and removal of cementum from the root with exposure of dentin, however, is a major contributing factor (2). Enamel loss is usually due to a combination of two or more of these factors:

Attrition: is mechanical wear of the incisal or occlusal surface usually associated with occlusal functions 2,5.Attrition also includes proximal contact area because of physiologic tooth movement (5). Excessive or parafunctional habits, such as bruxism, may result in extreme

pathologic wear and increased sensitivity (2).

Abrasion: is abnormal tooth surface loss resulting from direct friction forces between the teeth and the external objects; or from frictional forces between contacting teeth components in the presence of an abrasive medium. Toothbrush abrasion is the most common example which is usually seen as a sharp, V-shaped notch in the gingival portion of the facial aspect of the tooth (5). Tooth brushing by itself, however, has minimal effect on enamel and even with toothpaste the effect is minimal on both enamel and dentin. The combination of tooth brushing and erosive agents results in loss of tooth structure (2).

Erosion: is the wear or loss of tooth surface by chemico-mechanical action (5). 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. 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 (13).

Abfraction: May be a predisposing factor to DH (13). It has been proposed that the predominant causative factor of some cervical, wedge-shaped defects is a heavy eccentric occlusal force resulting in microfractures or abfractures. Such microfractures occur as the cervical area of the tooth flexes under such loads (5). Such fractures can cause the exposure of coronal dentin, and in more severe cases, coronal and root dentin (13).

Gingival recession: DH can be a major problem for periodontal patients, who frequently have gingival recession and exposed root surfaces (5). Abrasion also can cause gingival recession and as a result of the recession, the softer root surface is exposed. Exposed root surfaces could be also due to root prominence and thin overlying mucosa, dehiscences and fenestrations, frenum pulls, orthodontic movement, which causes a root to be moved outside its alveolar housing (2).

Physiological Causes: As age advances the number of teeth with root exposure is increased; results in root exposure, which may lead to DH (13).

Bleaching: 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 (13).

Periodontal Treatment: 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 (13).

Diagnosis of DH

There are many DH studies. Nevertheless, most dental professionals are confused about the diagnosis, etiology and mechanisms of DH (13). A good clinical history, clinical and radiographic examination and questions asked by the professional are essential to conclude a definitive diagnosis of DH.

Items which must be considered are: the pain (sharp, dull, or throbbing); how many teeth and their location; which part of the tooth elicits the pain; and the intensity of the pain (2). When symptoms are associated with exposed dentin, the diagnosis is DH. However when there is a specific etiologic factor causing the sensitivity, such as caries, fractures, leaking restorations, or recent restorative treatment, teeth with vital pulps may exhibit symptoms that are identical to DH. The definitive diagnosis is more difficult when clinical causes of reversible pulpitis are present in combination with exposed dentin. Hypersensitive teeth and inflamed pulps in many ways present the same symptoms (such as sensitivity to cold, air and heat) (1). Pulpal pain is usually more prolonged, dull, aching, and poorly localized and usually lasts longer than the applied stimulus (2).

Treatment—Self-Applied and Office Supplied

Dentin hypersensitivity is a common clinical condition that is difficult to treat because the treatment outcome is not consistently successful (5). Current techniques for treatment may be only transient in nature and results are not always predictable (2).

According to Grossman an ideal treatment for DH 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 (14). Two chief methods of treatment of dentin hypersensitivity are tubular occlusion and blockage of nerve activity (2).

Clinicians have used many materials and techniques to treat DH; including specific dentifrices (containing agents like calcium phosphate, potassium nitrate and oxalates), laser irradiation, dentin adhesives, antibacterial agents, resin suspensions (glass ionomer cement,), fluoride rinses and fluoride varnishes, dentinal adhesives, periodontal membranes (5,13). Desensitizing agents have been classified according to their mode of action or by their mode of administration. 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 (13). Self applied treatments to reduce sensitivity consist of materials that occlude dentinal tubules, coagulate or precipitate tubular fluids, encourage secondary dentin formation, or obstruct pulpal neural response (2).

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 (13).

Laser Treatment in Dentin Hypersensitivity

Laser therapy was first introduced as a potential method for treating dentinal hypersensitivity in 1985 (15). Since then, many studies have been done on laser applications for dentine hypersensitivity treatment and much information has been gathered.

Compared with conventional approaches, in-office DH laser treatment has some disadvantages

(ie, high cost, complexity of use, decreasing effectiveness over time, etc) that limit its clinical utility (16). In addition, the efficacy and mechanism of action of laser treatment for DH therapy are very controversial (17). The possibility of a placebo effect must be taken into consideration, especially as patient reports were positive immediately after laser treatment (18). According to a systematic review which was done by Sgolastra et al. laser therapy can reduce DH-related pain, but the evidence for its effectiveness is weak, and the possibility of a placebo effect must be considered (17).

This effect consists of a complex mixture of physiologic and psychological interactions, depending considerably on the doctor-patient relationship, with both parties needing to believe that the treatment is valuable and desiring to obtain relief of symptoms. On the other hand, since the mechanisms involved are multiple and unclear, questions arise regarding reproducibility and safety of this technique (18).

Studies have addressed the safety of using laser for treating DH, with some authors specifically analyzing the possibility of laser-induced pulp damage. One such study found that if the temperature increase within the pulp remains below 5°C, then no pulp damage is evident. This thermal threshold is generally not

exceeded when the energy and power settings of the laser remain within reported ranges (18).

A systematic review of the literature which surveyed the effectiveness of laser therapy and topical desensitizing agents in treating dentine hypersensitivity (done by HE et al.), indicates the likelihood that laser therapy has a slight clinical advantage over topical medicaments in the treatment of dentine hypersensitivity (38).

And finally, according to some studies, laser treatment seems to be transient, however, and the sensitivity returns in time (19).

The mechanism of recurrence is unknown. As laser effects are considered to be due to the effects of sealing of dentinal tubules, nerve analgesia or placebo effect. The sealing effect is considered to be durable, whereas nerve analgesia or a placebo effect is not (20).

Mechanism of Laser Treatment

The mechanisms involved in laser treatment of dentine hypersensitivity are relatively unknown (18). The laser, by interacting with the tissue, causes different tissue reactions, according to its active medium, wavelength and power density and to the optical properties of the target tissue (22). In order for a laser to actually alter the dentin surface, it has to melt and resolidify the surface. This effectively closes the dentinal tubules. This does not occur. It is felt that laser treatment reduces sensitivity by coagulation of protein without altering the surface of the dentin (2). Pashley (19) 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. indicates that the reduction in DH could be the result of alteration of the root dentinal surface, physically occluding the dentinal tubules (41).

According to Myers & McDaniel's study laser energy interferes with the sodium pump mechanism, changes the cell membrane permeability and/or temporarily alters the endings of the sensory axons (18).

The immediate analgesic effect in the treatment of dentine hypersensitivity with diode laser was reported by Brugnera Júnior et al. Based on this study the laser interaction with the dental pulp causes a photobiomodulating effect, increasing the cellular metabolic activity of the odontoblasts and obliterating the dentinal tubules with the intensification of tertiary dentine production (46).

Effectiveness of Various Laser Types in The Treatment of DH

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]

2- Middle output power (Carbon Dioxide Laser (CO_2), 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) (18,21).

Desensitization seems to depend mostly on the type of laser therapy adopted (39).

The mechanism causing a reduction in hypersensitivity is most unknown but is thought that the mechanism for each laser is different. In case of low output power lasers, a small fraction of the lasers energy is transmitted through enamel or dentin to reach the pulp tissue (20).

Low-power laser therapy is an appropriate treatment strategy to promote biomodulatory effects, minimize pain and reduce inflammatory processes. Its use has been widely accepted and approved due to satisfactory results reported in the literature. In contrast, 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 (39).

Low Output Power Lasers

He-Ne laser: The first use of He-Ne laser for the treatment of dentine hypersensitivity was reported by Senda et al. (1985), then, consecutively by several other investigators (42). Irradiation modes were two types: pulsed (5 Hz only) and continuous wave (CW) mode. The laser tip has to be placed as close as possible to the tooth surface in noncontact mode.

The mechanism involved is mostly unknown. According to physiological experiments, He-Ne laser irradiation does not affect peripheral A-delta or C-fiber nociceptors, but does affect electric activity. Treatment effectiveness rates of He-Ne laser ranges from 5.2%–100% based on different studies (18,21,22).

GaAlAs laser: The first use of this laser in dentin hypersensitivity treatment was reported by Matsumoto et al. and later by others (43). Three wavelengths (780,

830, and 900 nm) of GaAlAs have been used for the treatment of dentine hypersensitivity (18). The laser tip has to be placed as close as possible to the tooth surface in noncontact mode.

It is assumed that this type of low output power lasers mediates an analgesic effect related to depressed nerve transmission. According to physiological experiments using the GaAlAs laser at 830 nm, this effect is caused by blocking the depolarization of C-fiber afferents (118,21,22). Treatment effectiveness rates range from 53.3%–94.2% for the GaAlAs laser at 1-month follow-up (18).

Middle Output Power Lasers:

CO_2 Laser: This laser was first used for the treatment of dentine hypersensitivity by Moritz et al. (1996) (24). Output powers of 1 to 2 W with CW or pulse mode can be recommended. Sometimes an air blast should be used for preventing the tooth pain induced with the laser irradiation. The laser tip has to be kept on the tooth or gingival surface at a distance of 10 to 20 cm and has to be scanned as quickly as possible on the tooth or gingival surface in order to avoid thermal damage to the tooth or gingival surface (21). Treatment effectiveness ranges from 59.8 to 100%. CO_2 laser effects on dentin hypersensitivity are due to

the occlusion or narrowing of dentinal tubules. There have been no reports on nerve analgesia by CO_2 laser irradiation. Using the CO_2 laser at moderate energy densities, mainly sealing of dentinal tubules is achieved, as well as a reduction of permeability (18,21).

Nd:YAG Laser: The first use of this laser for the treatment of dentine hypersensitivity was reported by Matsumoto et al. (1985) (25). A power output from 0.3 to 2 W is usually used. Continuous or pulsed wavelength is used and the number of the pulses is from 10 to 20 Hz (18,21).

When using Nd:YAG laser irradiation, the use of black ink as an absorption enhancer is recommended, to prevent deep penetration of the Nd:YAG laser beam through the enamel and dentin and excessive effects in the pulp. Without Chinese black ink, the laser tip has to be held over the tooth surface at a distance of approximately 10 to 20 cm. When Chinese black ink is used, the laser tip has to be kept close to the tooth or gingival surface in noncontact mode, and has to be scanned as quickly as possible over the tooth or gingival surface in order to avoid thermal damage to the tooth or gingival surface (21). Treatment effectiveness ranged from 5.2 to 100%. The mechanism of Nd:YAG laser effects on dentine hypersensitivity is thought to be the laser-induced occlusion or narrowing of dentinal tubules (26,27) as well as direct nerve analgesia [28]. Nd:YAG and CO_2 lasers effectively cause occlusion of dentinal tubules (18,21).

Er:YAG Laser: This type of laser is suitable for caries treatment but endodontic and periodontic applications have also been studied. It was first used for DH therapy by Schwarz et al (44). The parameters of Er:YAG laser irradiation for DH therapy are 1 W and 10-12 Hz for less than 60 s. In order to prevent damage to the tooth and gingival surface, the distance of the laser tip to tooth surface has to be kept more than 10 cm. The laser tip has to be quickly scanned across the tooth or gingival surface to prevent laser damage on the tooth or gingival surface (21). The reduction in DH at 6 months with the Er:YAG laser reportedly ranges from 38.2%–47%; however, few data are available for this laser type, which has only recently been introduced. [18, 29] However there are a lot of ambiguous points in mechanism of Er:YAG as this laser is absorbed by hydroxyapatite's water molecules, which can cause dentin surface ablation and is opposite to the sealing of the dentinal tubules [30]. According to Kimura, Nd:YAG laser is more effective than Er:YAG laser (21).

Er,Cr:YSGG Lasers: The Er,Cr:YSGG laser has been shown to be effective for soft-tissue surgery as well as for cutting enamel, dentine and bone. However, there is limited knowledge on the effects of this laser on DH and few studies have been published concerning the clinical aspects of the desensitizing effect obtained with the Er,Cr:YSGG laser. The output power used for DH therapy varies from 0.25 to 0.5 W (39). Yilmaz et al. demonstrated that the single application of Er,Cr:YSGG laser has shown efficacy in rapid DH reduction compared with placebo treatment. This effect has become apparent immediately, and it remained stable for a 3-month examination period (40). Based on the results of a Short-term clinical evaluation which compared the effects of Er:YAG and Er,Cr:YSGG lasers on DH, the Er,Cr:YSGG laser at a power of 0.25 W showed the best performance in the clinical evaluations (39).

Combination of Laser Treatment with Chemical Agents

Since dentine hypersensitivity has multifactorial

etiology and generally more than one factor is found associated and active in this painful manifestation; therefore, more than one treatment method should be associated to desensitize the dentine to satisfactory levels.

There have been some reports on combination use of laser irradiation with chemical agents. The combination use of laser irradiation with chemical agents such as sodium fluoride and stannous fluoride can enhance treatment effectiveness by more than 20% over that of laser treatment alone (31,32,33).

Kumar and Mehta reported that the combination of Nd: YAG laser and 5% NaF varnish seems to show an impressive efficacy when compared to each treatment alone (34).

Hsu et al. evaluated the combined occluding effects of fluoride-containing dentin desensitizer and Nd-YAG laser irradiation on human dentinal tubules. The occluding agent was thus *burned into* the dentinal tubules, and could neither be dissolved by vitamin C solution nor removed by brushing. Therefore, they concluded that the FDTOA combined with Nd-YAG laser irradiation burns the occluding agent into the dentinal tubules increasing the duration of desensitizing effect (35).

Goharkhay et al., reported that CO_2 laser irradiation through a layer of stannous fluoride causes a highly resistant layer on sensitized dentin. This layer induced by physical and chemical bonding mechanisms, provides a superior defense against external stimuli (36).

The combined use of the GaAlAs laser (830 nm wavelength) with fluoridation enhances treatment effectiveness by more than 20% over that of laser treatment only. In an in vitro study, most dentinal tubule orifices were occluded after treatment by Nd:YAG laser irradiation followed by topical sodium fluoride (31,33).

Kue and colleagues used dicalcium phosphate-bioglass in combination with Nd:YAG laser in treatment of DH. According to their study, this combination sealed dentinal tubules to a depth of 10 mm (37).

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