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Dentin hypersensitivity and laser therapy

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ABSTRACT
Dentin hypersensitivity is considered an acute pain of short duration, resulting from the exposure of open dentinal tubules to the oral environment in response to mechanical, thermal, chemical and osmotic stimuli. Among the various treatments available, hypersensitivity can be treated with specific dentifrices, fluoride, desensitizers, adhesive systems and also through laser therapy. Laser therapy has been increasingly used for therapeutic purposes in dentistry, with properties such as photobiostimulation, photobiomodulation, analgesia and anti-inflammatory action. The objective of this work was to demonstrate, through a literature review, the scientific basis and clinical studies that support the use of lasers for the treatment of cervical dentin hypersensitivity. For its elaboration, articles were selected in the databases: PubMed, Virtual Health Library (Medline/LILACS/BBO), Science Direct, applying the following search descriptors in Portuguese "hypersensitivity", "lasertherapy", "dentinal tubules", " pain", "non-carious cervical lesions", in English "Dentin hypersensitivity", "Non-carious cervical lesion" and "laser". Based on the reading of titles and abstracts, and considering the following inclusion criteria: literature review articles, systematic review, randomized clinical trials and laboratory studies reporting on the use of laser published in the period from 2015 to 2021. The excluded works were unavailable articles in full, monographs, studies with incomplete methodology, dissertation and thesis.

Keywords: Hypersensitivity, Lasertherapy, Dentinal tubules, Pain, Non-carious cervical lesions.

1 INTRODUCTION

Dentin hypersensitivity (DH) is one of the most common complaints of patients in dental clinics, it is usually described as a short-lived non-spontaneous acute pain resulting from the exposure of open dentin tubules to the oral environment in response to mechanical, thermal, chemical and osmotic stimuli. [1,2,3]

The buccal surface of the teeth is most commonly involved in patients with dentin hypersensitivity and occurs most frequently in the maxillary canines and premolars, followed by incisors and molars. HD is defined as a pathological process of multifactorial etiology and can occur as a consequence of a process of caries, abrasion, erosion, abfraction, traumatic toothbrushing and periodontal disease, resulting in loss of tooth structure in the cervical region.[4.5]

Zeola et al. (2019) in a systematic review and meta-analysis concluded that the prevalence of HD is between 11 and 33%, being considered a pathology of painful symptomatology with lower treatment success rates.[6]

Several theories seek to explain the mechanism by which a stimulus is transmitted from the dentin to the pulp, however, the most widely accepted theory is that of hydrodynamics, which was proposed in 1964 by Brännström, in which he proposes that the dentinal tubules are filled with fluids, and when exposed to external stimuli, they can be; physical, chemical, osmotic, pressure or temperature; They induce the movement of this fluid, promoting the contraction or distension of the extensions of odontoblasts, activating the nerve endings located near the dentin-pulp, generating pain throughout the tubules.[1,4,7]

Thus, it is necessary to make a correct diagnosis, identifying the etiological factors involved, understanding the multifactoriness of HD and its risk factors, which include traumatic toothbrushing, gingival recession due to periodontal therapy, parafunctional habits, individual behaviors and diet, for the elaboration of a personalized planning, aiming to manage the etiological factors for a correct intervention and prevention of future damages.[6.7]

The treatment of dentin hypersensitivity should aim to define the causal factors before the methods for controlling and treating the disease are selected. The painful symptomatology of dentin hypersensitivity may regress without treatment, by remineralization by saliva or by the formation of reactive dentin, and dentin permeability may decrease spontaneously.[1,6,7]

Treatment can be based on dentin desensitization, with the use of fluoride (toothpastes), gingival covering, dental adhesives (restoration), endodontic treatment using laser therapy, with low-level laser (LBI) and high-intensity laser (LAI).[1.7]

In cases of loss of less than 1 mm of tooth structure, desensitization is indicated. Desensitization also occurs by natural processes such as reparative dentin, sclerotic dentin, and dental calculus formation on the dentin surface.[8]

Desensitizing agents can act with neural or obliterating action. The neural mechanism acts in the desensitization of nerve fibers, with potassium being the only chemical desensitizing agent of neural action, which acts by promoting an increase in the concentration of potassium ion in the odontoblastic endings, reducing the ability to conduct the sensory stimulus of the nerve fibers that would promote pain.[6.8]

Laser is widely used in numerous therapies in dentistry, as a healing agent for ulcers, mucosal lesions, dental surgeries, and pain management.[2,9,10]

For the treatment of dentin hypersensitivity, one can use both LAI, which acts by promoting dentin sealing by obliteration of dentin tubules, and LBIs, which act through analgesic, anti-inflammatory and biostimulatory effects of the dental pulp, leading to the formation of reactive dentin.[2.10]

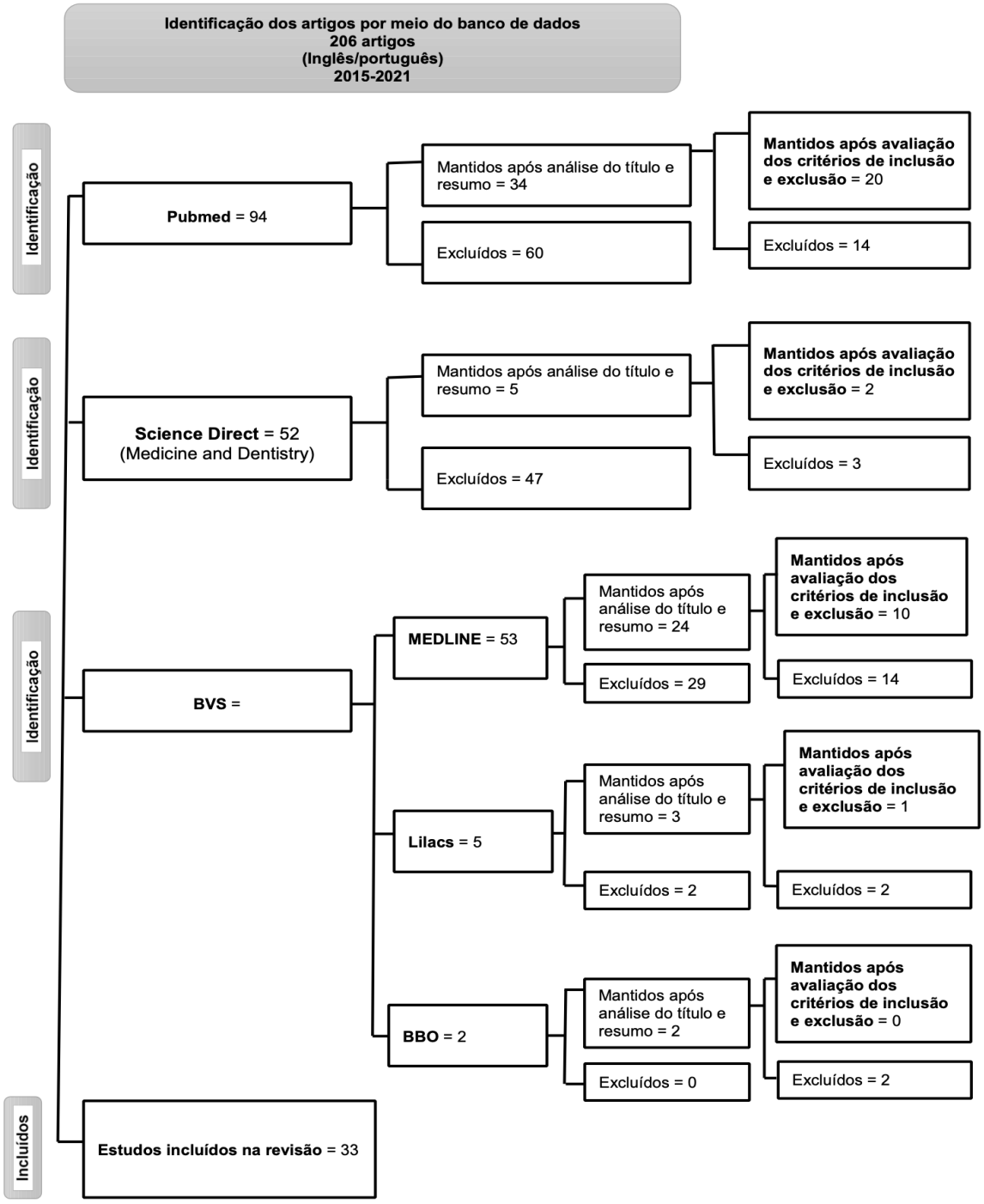
LBIs have been increasingly used in dentistry, considered physical agents of neural action that act on nerve endings, increasing the patient's pain threshold.[7,9,10]

2 OBJECTIVE

To demonstrate, through a literature review, the scientific basis and clinical studies that support the use of lasers for the treatment of cervical dentin hypersensitivity.

3 METHODOLOGY

Figura 1: Fluxograma da busca e seleção dos estudos



Fonte: Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 2021;372:n71. doi: 10.1136/bmj.n 71. Adaptado

4 LITERATURE REVIEW

Dentin hypersensitivity has a high prevalence in the adult population, affecting one in six patients, and is described as a short-lived non-spontaneous acute pain resulting from the loss of the protective material, tooth enamel, and exposure of the dentin tubules, promoting excessive sensitivity in the dentin in response to non-noxious stimuli, typically thermal, evaporative, tactile, osmotic or chemical, and which cannot be attributed to any other form of dental defects or disease.[2,11,12]

The incidence of HD peaks at 30-40 years, and female patients have the highest incidence of this pathology, and the most affected teeth are the canines and premolars, followed by the incisors and molars, and usually involves the buccal surfaces in the cervical region of the teeth.[3]

Pain is a sensory and emotional experience of discomfort, presenting a subjective and individual character, and dentin hypersensitivity affects the quality of life of patients and can lead to dietary and social limitations and psychological impact.[13] The affected teeth become sensitive to stimuli, the patient feels pain in daily activities such as eating, drinking, talking and brushing teeth.[13.14]

Pain results from the activation of specialized sensory receptors, the primary afferent nociceptors that are present in myelinated A-δ nerve fibers that correspond to mechanical and thermal stimuli (rapidly felt pain, pinpricking sensation, stinging).[13.14]

The A-δ fibers are activated by means of the hydrodynamic mechanism, represented by the flow of fluids inside the dentin tubules caused by external stimuli, histologically, the hypersensitive dentin has enlarged dentin tubules and in greater number per area, compared to the dentin absent of sensitivity, the dentin tubules when exposed, are vulnerable to cold, substances acidic or sweet, or just by the mechanical touch.[14th]

4.1 PAIN ASSESSMENT

In order to measure pain intensity, scales were developed to measure the variability of responses (verbal and non-verbal) to pain. The subjective pain scales aim to measure, qualify or evaluate the individual's behavior in the face of the painful experience. Among the subjective pain scales, the most used is the Visual Analogue Scale (VAS), considered the gold standard, with the advantage of reliability, simplicity, ease of use and versatility. [4,6,15]

The VAS scale is a one-dimensional instrument for the assessment of pain intensity, it consists of a horizontal line of 10 centimeters with the ends numbered from 0-10, at one end of the line is marked "no pain" and at the other "unbearable pain".[15.16]

To assess the intensity of the pain, the dentist should ask the patient to evaluate and mark on the line the pain present at the time of the consultation. The categorization of pain depends on the amplitude of the scale, on a scale of 0 to 10 points the criterion usually used is:

· 0-1 no pain; 1-3 mild pain; 3-7 moderate pain; 7-10 severe pain.

· 0-1 no pain; 1-3 mild pain; 3-6 moderate pain; 6-9 severe pain; 9-10; very pain.

The Schiff sensitivity scale is also used to assess the painful sensation of HD in the face of stimuli.[6] The Schiff scale has four levels of response to stimuli: (0) subject does not respond to air stimuli; (1) the subject responds to the air stimulus but does not require discontinuation of the stimulus; (2) subject responds to the air stimulus and requires discontinuation or flees from the stimulus; (3) subject responds to the stimulus, finds it painful, and requires discontinuation of the stimulus immediately.[6.15]

4.2 DIAGNOSIS

Accurately diagnosing HD is critical to determining an appropriate and effective treatment, and the primary goal of diagnosis is to identify and control etiologic factors. [4.17]

The initial diagnosis of dentin hypersensitivity is made through the patient's perception of the pain caused when the sensitized tooth is exposed to hot and cold stimuli, brushing, flossing, and chewing. [1.6]

The steps to conclude an accurate diagnosis include: thorough and detailed anamnesis followed by extraoral and intraoral clinical examination; complete periodontal examination; occlusal analysis, signs of parafunction, articulator mounting; analysis of salivary profile and parameters; analysis of eating habits; analysis of oral hygiene habits, and radiographic examinations.[17.18]

In order to conclude the diagnosis, in addition to the HD test and complementary tests, it is essential to induce the characteristic transient acute pain by applying a stimulus to the affected tooth to mimic the patient's complaint or symptom.[17.18]

It is important that the dentist knows how to make a differential diagnosis, as there are other clinical situations thathave the same characteristics as dentin hypersensitivity. To do this, the professional must evaluate and investigate the patient's clinical history, compare teeth with and without symptoms, seeking to eliminate possible causes of pain.[2] Some conditions that may present with symptoms similar to HD are coronary fractures, defective or fractured restorations, enamel invaginations, occlusal trauma, dental preparation for restorations or restoration-induced pulp hyperemia, teeth whitening, cervical plaque, gingivitis, periodontal disease, and pulp pathologies.[19th]

Persistent, non-transient dental pain, different from that presented with HD, is usually a sign of inflammation in the dental pulp or periodontal tissues, as well as possible infection associated with a pathological condition, such as caries, traumatic tooth fracture, pulp exposure, pulp sclerosis, periodontal disease.[13.20 am]

HD has a multifactorial etiology that corresponds to a triad of factors related to dentin hypersensitivity, gingival recession and non-carious cervical lesions. The factors that modulate the formation and evolution of dentin hypersensitivity are abrasion, attrition, erosion and abfraction.[21]

Abrasion is the mechanical wear of the tooth structure due to the repeated friction of a foreign body (external factors), with inadequate brushing being one of the main causes. Erosion, on the other hand, is the superficial and substantial loss of hard dental tissues caused by chemical processes, not bacteriological.[21]

- Dental attrition: corresponds to mechanical wear that affects the incisal and occlusal surfaces related to occlusal hyperfunction and/or parafunctional habits such as clenching and bruxism.[21.22]

Abfraction are wedge-like lesions that arise from the presence of occlusal trauma, excessive masticatory force, or tooth clenching, leading to changes in tooth structure.[21.22]

In this sense, several studies have been carried out in recent years5,6,19,20, with the objective of analyzing the most effective treatment to combat dentin hypersensitivity, which can be treated with toothpastes, fluoride applications, desensitizers, restorative adhesive systems, and application of high and low power lasers.[22]

4.3 LASER THERAPY

Among the numerous therapies for HD, laser technology has been researched in the field of health, analyzing the interaction of light with the most diverse tissues, expanding its use as a therapy for dentin pain.[21] Laser can be integrated as an adjunct to conventional treatment therapy or used alone as an alternative mode in some pathologies.[10,14,23]

According to the wavelength of the laser and the dose applied, the application of the laser on dental tissue and adjacent tissues is differentiated, being classified as low power laser (LILT – low intensity laser treatment) or non-surgical, and high power (HILT – High intensity laser treatment) or surgical. [8.9]

High-intensity lasers, such as Nd:YAG, Er:YAG, Er, Cr:YSGG and CO2, are used to obliterate the embouchure of dentin tubules, act on dentin through phothermal effects by heating and melting the hydroxyapatite crystals of the dentin. When the dentin cools, it recrystallizes promoting the sealing of the dentin tubules.[3,9,14]

Low-level lasers such as GaAlAs or He-Ne produce rapid action with analgesic and anti-inflammatory effects through a photochemical process. Low-level laser photobiomodulation therapy acts at the molecular level, its neural mechanism of action promotes the change of the electrical potential of the cell membrane, activating the Na+ and K+ pumps, promoting an increase in the synthesis of adenosine triphosphate (ATP), the release of endorphins and the blocking of the depolarization of afferent C fibers, preventing the transmission of the pain stimulus.[3,9,14]

Lasers can be applied to different media (solid, liquid, or gaseous), resulting in different types of radiation and wavelengths.[4.23]

Due to these different types of wavelengths, which have specific properties, they can generate different interactions of transmission, absorption, dispersion or reflection, and the absorption is most commonly used in dentistry, because what is sought is the absorption of laser light by the target tissue, thus implying the achievement of the intended biological effect.[4.23]

There are different parameters that influence the treatment response, which must be observed for the application of the laser, namely: power (W), exposure time (seconds), energy density (J/cm2), energy per point (J), type of emission - pulsed or continuous, and number of irradiated points.[9]

In dentistry, the use of laser has intensified since the 1980s, when the effects of low-level laser in the treatment of patients with dentin hypersensitivity were analyzed.[2]

The use of these low-level lasers provides significant pain relief after application, in addition to the production of tertiary dentin by the odontoblasts, resulting in a decrease in fluid movement in the dentin tubules and dentin permeability.[8,24,25]

The wavelength leads to an increase in the level of excitability of the free nerve endings, thus reducing pain. Currently, the most widely used (non-surgical) lasers are diode lasers, with wavelengths ranging from 600 to 700 nm (visible red range) and 700 to 950 nm (infrared range of the electromagnetic spectrum). [11.4]

Considering that low-power lasers are not based on increasing temperature, it should not exceed 1º Celsius, and the power should not exceed 500 mW, providing a cellular bioregulation action (anti-inflammatory, healing, myorelaxing and analgesic effects).[11]

Table 1 shows the laser therapy protocols found in the literature.

Table 1 – Laser therapy protocol applied to the treatment of Hypersensitivity

Author year	N	Protocol Used	Stimulation	Evaluation/method	Follow-up period
Pantuzzo, 2020	28 (10)	GaAlAs Laser, Diode (810–830 nm, power 0.5–4.5 W), for 60s.	Probe & Air Jet	EVA	After 15min, 7 days.
Maximiano, 2018	70 (143/ 124/127)	Nd:YAG laser (1 W, 10 Hz, 85 J/cm2) 4 irradiations for 15 seconds with an interval of 10 seconds. Irradiation (performed twice in the mesial-distal and occlusion-gingival directions.	Probe & Air Jet	EVA	After 5 min, 1 week and 4 weeks
Chebel, 2018	78 (39/39)	Laser Nd:YAG (60 mJ, 2 Hz, 0.64 W, 35.8 J/cm2, 4 repetitions for 20 s). Distance from exposed surfaces, 6mm.	Probe & Air Jet	EVA	1week,1,3,6 months
Osmari, 2018	76 (19/19/19/19)	Diode Laser (810–830 nm, power 0.5–4.5 W)	Air Jet	EVA	Immediate, 15, 30 and 60 days.

Ozlem, 2018	17 (100)	Nd:YAG ((1 W, 10 Hz 100 mJ pulse energy (35.8 J/cm2) inmesiodistal direction for 20 s to each tooth for three times. 10s Interval	Air Jet	EVA	30 min, after 7, 90 and 180 days
Lopes, 2017	G2 (117) n=13	Laser Photon Lase (DMC) 3 points irradiation in the buccal portion and 1 apical 30mW, 10 J/cm2.9 s by 810 nm, with threesessions with an interval of 72 h.	Probe & Air Jet	EVA	5 min (post 1), 12 months (post 2) and 18 months (post 3)
Lopes, 2017	G3 (117) n=13	Laser Photon Lase (DMC) power of 100 mW, 40 J/cm2, and 11 s at each point (dose of 1.1 J per point) three sessions with an interval of 72 h.	Probe & Air Jet	EVA	5 min (post 1), 12 months (post 2) and 18 months (post 3)
Lopes, 2017	G6 (117) n=13	Laser Nd: YAG 120 µ s, 100 mJ, 85 J/cm2, in contact, 1 W power and 10 Hz repetition rate. 4x 15s/ 10s interval	Probe & Air Jet	EVA	5 min (post 1), 12 months (post 2) and 18 months (post 3)
Lopes, 2017	G8 (117) n=13	LPLD + Nd:YAG laser/parameters described in groups G2 and G6, 3 sessions.	Air Jet	EVA	5 min (post 1), 12 months (post 2) and 18 months (post 3)
Pandey, 2017	45 (15)	Picassa Diode Laser (810–830 nm, power 0.5–4.5 W), 60s.	Probe & Air Jet	EVA	1,2 and 3 weeks.
Suri, 2016	60 (30/30)	Laser GaAIAs (980nm, 2W power, 2x 20s)	Probe & Air Jet	EVA	24 hours, 1 week, 1 and 2 months.

Source: Prepared by the authors

5 DISCUSSION

Dentin hypersensitivity is one of the most common painful clinical conditions reported by patients in the clinical routine of dental offices, with multifactorial etiology. It is characterized by acute pain associated with great discomfort and with a negative impact on the quality of life of the affected individual.[1,6,19]

Despite being a widely studied topic, HD continues to be a complaint with prevalence ranging from 3% to 98% in the adult population for different samples.[6] This discrepancy is due to some differences, such as study design, inclusion and exclusion criteria, diagnostic approach, as well as participants' habits and diet.[14.30 am]

Low-level laser treatment in patients with dentin hypersensitivity has been widely used by dentists due to its efficacy, ease of application, relatively low cost, and painless to the patient.[28th]

Laser therapy has an immediate effect on the relief of pain sensitivity, photo-biomodulation in the dental pulp, promoting a delayed effect, which corresponds to the obliteration of the dentin tubules resulting from the increase in the cellular metabolic activity of the odontoblasts, which intensifies the production of tertiary dentin, leading to the reduction of dentin permeability and fluid movement within the dentin tubules, promoting analgesia for the patient.[2]

Sgreccia et al.[30] (2020) evaluated in their randomized clinical trial the efficacy of potassium oxalate gel (Oxa-Gel BF) and GaAIAs (gallium and aluminum) laser in the treatment of HD in 74 patients (389 NCCL) and concludedthat after four applications of low-level laser (GaAlAs) with the 100 mW laser programming protocol, with a wavelength of 808 nm, and standardized energy at 60 J/cm2, the laser was able to eliminate patient discomfort in 52.0% in the tactile stimulus test and 74.7% in the evaporative stimulus test.[30th]

Pantuzzo et al.[14] (2020) conducted a study with 28 individuals and demonstrated efficacy in reducing dentin hypersensitivity by 36% in laser treatment when compared to fluoride treatment, which was 9.5%, after 15 minutes of exposure. And after 7 days, the diode laser treatment was also more effective, with a 37.5% reduction in HD, while the fluoride treatment led to a 31.6% reduction.[14th]

Maximiano et al.[26] (2019) conducted a randomized, double-blind, placebo-controlled clinical trial in which they evaluated the efficacy of the immediate and long-term effects (1 and 4 weeks, respectively) of the Nd:YAG laser and a paste-containing sodium calcium phosphosilicate in the treatment of dentin hypersensitivity in 70 patients. In their study, 23 patients were allocated to the placebo control group (16 with "severe" pain and 7 with "moderate" pain), in group 2 the prophylactic paste of sodium and calcium phosphosilicate (15% CSF) was used in 23 patients (17 with "severe" pain and 6 with "moderate" pain)) and group 3 was used a high-power Nd:YAG laser (1064nm) in 24 patients (17 with "severe" pain and 7 with "moderate" pain). [26th]

The results showed that both the Nd:YAG laser and the prophylactic paste with 15% FCS used in dentin desensitization were efficient in reducing the pain level of the volunteers, both immediately and in 4 weeks.[26th]

Similar results were observed by Chebel et al.[25] (2018) when evaluating the efficacy of Nd:YAG laser compared to MI varnish, the study involved 54 teeth in 12 patients who had dentin hypersensitivity. A split-mouth design was used, the 54 teeth were divided into 27 pairs. Twenty-seven teeth received treatment with Nd:YAG laser, and the 27 contralateral teeth received application of MI varnish composed of 5% sodium fluoride, casein, phosphopeptide, amorphous calcium and phosphate varnish.[25th]

The Schiff sensitivity scale was used to assess pain with air jet stimulation, and the visual analogue scale was used for tactile stimulation tests with the explorer probe and thermal tests.[25th]

The results showed that there was no significant difference between the two treatments, Nd:YAG laser and MI varnish. Both treatments were effective and reduced dentin hypersensitivity immediately and after the 6-month treatment.[25th]

Ozlem et. al.[16] (2018) conducted a study to evaluate the efficiency of a glutaraldehyde-containing agent (Gluma) and Nd:YAG, Er, Cr:YSGG lasers alone and in combination with the desensitizer Gluma in the treatment of dentin hypersensitivity in 17 patients (100 teeth).[16]

The results showed that the reduction in HD was lower in the gluma group than in the Er, Cr:YSGG laser groups, but it was similar in the Nd:YAG laser groups. Er, Cr:YSGG lasers with or without application of the Gluma desensitizer is the most effective modality in the treatment of dentin hypersensitivity.[16]

A randomized longitudinal study conducted by Lopes et al.[28] (2016) compared the different hypersensitivity treatment protocols in 32 patients (117 teeth) for a period of 12 to 18 months. The therapies used included the low-level Photon Lase (DMC) laser, the high-power Nd:YAG laser and the Gluma Desensitizer, a desensitizer based on the HEMA monomer (hydroxyethylmethacrylate) and glutaraldehyde, and finally the association between the laser and the desensitizing agent.[28th]

The level of pain sensitivity of each patient was analyzed by VAS using cold air stimuli and an exploratory probe, before and after the treatments. The authors concluded that after 6 months of evaluation, all treatments performed were efficient in reducing dentin hypersensitivity.[28th]

Lopes et al.[28] (2016) also point out that among high-power lasers, the Nd:YAG laser is considered a gold standard for the treatment of HD because it has been shown to have the ability to obliterate dentin tubules, by fusion and resolidification of the dentin, without pulp lesions or fissures in the irradiated dentin, when used with an appropriate protocol. [28th]

Pandey et al.[2] (2017) in their randomized study compared the efficacy of 5% potassium nitrate (KNO3) toothpaste, low-level laser therapy, and LLLT together with the application of 5% KNO3 toothpaste in the treatment of dentin hypersensitivity. The treatments were performed in weekly sessions, with a total duration of 3 weeks.[2]

The authors concluded that low-level laser alone was more effective in reducing long-term HD pain when compared with 5% potassium nitrate (KNO3). According to this study, there was no additional benefit to using the toothpaste with 5% potassium nitrate along with low-power laser.[2]

According to the authors, the reduction in HD can be attributed to the property of LLLT to act on cellular biostimulation by promoting the increase in mitochondrial ATP production, increasing the threshold of free nerve endings, providing an analgesic effect and stimulating the pulp to produce secondary dentin. Thus, it is expected that the action of the laser will not be immediate, since the formation of this newly formed dentin requires a certain amount of time. This makes the action of the laser gradual and the patient perceives its action in the long term.[2]

Suri et al.[29] (2016), conducted a comparative clinical study that evaluated the efficacy of fluoride varnish, GaAlAs 980 nm diode laser, and the association between laser and fluoride varnish. The study involved 120 teeth in 30 patients with HD assessed by tactile and air jet stimuli measured by the visual analogue scale (VAS). The most effective results were obtained when patients were treated by the association between varnish and high-intensity laser.[29th]

Although clinical studies have shown very favorable results for the use of laser therapy in the treatment of HD, its clinical management has been a challenge for dental professionals, due to the multifactoriness of the disease. Dental surgeons (DC) need to diagnose and act in the control of the cause, it is necessary to have a detailed anamnesis, obtain information on occupational habits, diet diary, gastroesophageal diseases, eating disorders, anxiety-related diseases , temporomandibular disorders and medications of daily use.[2,6,33]

Dentin hypersensitivity has been shown to be extremely related to people's individual habits and lifestyle, excessive and traumatic brushing, poor oral hygiene, gum recession due to periodontal therapy, eating and anxiety disorders, high-stress routine, parafunctional habits.[22.26]

Thus, teeth are exposed to various chemical and physical challenges, leading to an increased prevalence of non-carious cervical lesions and cervical dentin hypersensitivity. For the efficacy of the treatment, the DC must act to control the pain and the evolution of these lesions.[26th]

It is necessary to guide the patient to remove the risk factors, recommend the removal of excess acid in the diet, advise on the correct brushing techniques, educate the patient to control the force of brushing, recommend that brushing be performed 30 minutes after meals, if it is identified that the patient suffers from an anxiety disorder, it should be referred to a professional.[26.33]

6 FINAL THOUGHTS

Laser therapy treatment in patients with dentin hypersensitivity is a promising and effective alternative for pain [control.1,4]

Due to the lack of universally accepted guidelines for differential diagnosis, and the diversity of irradiation methodologies and parameters used in different studies, it is necessary to carry out randomized and controlled clinical investigations to standardize protocols with the use of laser and its correct clinical application in the treatment of dentin hypersensitivity.[2]

The dental surgeon must be able to make an accurate diagnosis, aiming to achieve success in the treatment, for this it is necessary to perform a thorough clinical examination showing whether or not there is the presence of carious lesions, fractured and maladapted restorations, fractured teeth, enamel cracks, lesions that have communication with the pulp chamber, enamel invagination and pulp pathologies, since these conditions are confused due to the similar symptomatology of dental pain.[14,17,19]

Since HD is largely the result of erosive/abrasive tooth wear or dentin exposure related to gingival recession, laser therapy should be coupled with strategies for the management of HD that include oral hygiene education and instruction on brushing techniques, behavioral management, and elimination of predisposing factors.[4]

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