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AVALIAÇÃO DA EFICIÊNCIA DE AGENTES ANTI-HIPERESTÉSICOS NO TRATAMENTO DA HIPERESTESIA DENTINÁRIA

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RESUMO

A hiperestesia dentinária é um fenômeno sensorial complexo e de difícil solução na clínica odontológica. Apesar da grande diversidade de tratamentos propostos, ainda não existe uma terapia considerada ideal para eliminar essa situação desconfortável. Esta dissertação, constituída por dois artigos científicos, teve por objetivos: (1) revisar criticamente a literatura disponível sobre os principais aspectos relacionados à etiologia e ao tratamento da hiperestesia dentinária; (2) avaliar clinicamente a eficiência de agentes anti-hiperestésicos no tratamento da hiperestesia dentinária. No estudo 1, a literatura científica pertinente ao assunto foi analisada através dos resultados de investigações clínicas e laboratoriais pesquisadas usando a base de dados *medline* e busca manual de referências citadas em artigos científicos. No estudo 2, um total de 164 dentes, provenientes de 30 pacientes com diagnóstico de hiperestesia dentinária moderada ou severa, foi dividido aleatoriamente em três grupos e avaliado clinicamente de acordo com o tratamento administrado: aplicação de laser de arseniato de gálio-alumínio (AsGaAl), aplicação de gel de oxalato de potássio a 3% e aplicação de gel placebo. As aplicações dos tratamentos foram realizadas em intervalos semanais, durante o período de quatro semanas consecutivas e o grau de sensibilidade foi mensurado para cada dente através de uma escala visual analógica em resposta aos estímulos tátil (sonda exploradora) e evaporativo (jato de ar) antes da primeira aplicação (*baseline*), imediatamente após e três meses após a última aplicação dos tratamentos. Os dados foram submetidos à análise estatística pelo teste de Kruskal-Wallis ($p=0,05$) e o grau de redução da hiperestesia dentinária foi avaliado para cada um dos períodos observacionais em relação ao *baseline*. A análise crítica apresentada no artigo 1 mostrou que a literatura apresenta diversos tipos de tratamento para a hiperestesia dentinária, que variam desde procedimentos simples, que podem ser executados pelo próprio paciente, até procedimentos complexos, que envolvem a combinação de diferentes terapias. Os resultados do artigo 2 demonstraram que a aplicação dos tratamentos propostos, inclusive o placebo, proporcionou redução estatisticamente significante, imediata e mediata, da sensibilidade em resposta aos estímulos tátil e evaporativo ($p<0,05$). No entanto, não houve diferença estatisticamente significante entre os três grupos estudados, independentemente do estímulo aplicado, tanto na avaliação imediata quanto na mediata ($p>0,05$). Em conclusão, os resultados desses estudos indicam que o conhecimento acerca do mecanismo de ocorrência da hiperestesia dentinária e dos agentes anti-hiperestésicos disponíveis é indispensável para a elaboração de um tratamento eficiente. Além disso, concluiu-se que os três tratamentos utilizados no estudo clínico são eficientes para a redução da hiperestesia dentinária e que existe grande influência do efeito placebo na redução da sensibilidade dolorosa.

Palavras-chave: Sensibilidade da Dentina. Oxalatos. Lasers. Efeito Placebo.

ABSTRACT

Dentinal hypersensitivity is a complex sensorial condition which can cause considerable concern in the dental office. Despite the large number of different proposed kinds of treatment, there is no product or therapy reported in literature that could be considered ideal to eliminate this uncomfortable situation. The aim of this study, comprised by two manuscripts, was: (1) to critically review the literature related to the main evidences about the etiology and the management of dentinal hypersensitivity; (2) to evaluate the clinical performance of different dentine desensitizers in the treatment of dentinal hypersensitivity. In study 1, the scientific literature related to the issue was analyzed through the results of laboratory and clinical investigations searched using medline and manual tracing of references cited scientific papers. In study 2, a total of 164 teeth, from 30 patients with clinical diagnosis of moderate or severe dentinal hypersensitivity, were randomly divided into three groups and clinically evaluated according to the desensitizing treatment under study: gallium-aluminun-arsenide (GaAlAs) laser therapy, 3% potassium oxalate application and placebo gel application. Treatment sessions were performed at seven-day intervals for four consecutive weeks and the degree of sensitivity in response to tactile (probe) and evaporative (air blast) stimuli was assessed according to a visual analogue scale at baseline, immediately after and three months after the last treatment session. Data scores were submitted to Kruskal-Wallis statistical analysis ($p=0.05$) and were analyzed by dentinal hypersensitivity reduction for each observational moment in relation to baseline. The critical review presented in study 1 showed that the reviewed literature points out several treatment modalities ranging from simple procedures, which can be performed by the patient him/herself, to complex procedures that involve the combination of therapies. The results of study 2 demonstrated that the treatment of dentinal hypersensitivity performed with both active and control groups produced statistically significant reduction of pain in response to evaporative and tactile stimulation immediately after and three months after treatment ($p<0.05$). No significant differences among the three groups could be detected in both immediate and mediate evaluations irrespective of the applied stimulus ($p>0.05$). In conclusion, the results of these studies suggest that knowledge about the available desensitizing products and the factors involved in the mechanism of the dentinal hypersensitivity is indispensable in order to perform an effective treatment. Besides, it could be concluded that the three treatments performed in the clinical study were effective for treating dentinal hypersensitivity and that the placebo effect plays an important role in sensitivity reduction.

Key words: Dentin sensitivity. Oxalates. Lasers. Placebo effect.

SUMÁRIO

1. INTRODUÇÃO GERAL

A hiperestesia dentinária, também denominada hipersensibilidade dentinária cervical, consiste em uma resposta exagerada da dentina quando exposta a estímulos sensoriais (táteis, térmicos, evaporativos, químicos ou osmóticos), que normalmente não causariam resposta em um dente normal (1). É caracterizada como uma sensação dolorosa, do tipo localizada, transitória e aguda, que pode variar em intensidade desde um leve desconforto a uma dor extrema (2,3). Dependendo do grau de intensidade, a hiperestesia pode afetar a alimentação, a ingestão de líquidos, a respiração, a capacidade de efetivo controle da placa bacteriana pelos pacientes e até mesmo provocar mudanças emocionais que alteram o estilo de vida do indivíduo (1).

Esta condição tem sido considerada uma das queixas mais freqüentes nos relatos de odontalgia e representa um problema constante na clínica odontológica (4,5). Estudos de prevalência, realizados em diversas partes do mundo, indicam que a hiperestesia dentinária afeta entre 10% e 30% da população em geral (4,6-11), sendo os dentes mais comumente acometidos os caninos e pré-molares (12-14). Adicionalmente, haverá uma crescente demanda de pacientes incomodados com esta situação desconfortável, considerando o aumento da expectativa de vida dos indivíduos, que mantêm por mais tempo os seus dentes na cavidade bucal (1,2,4).

Várias teorias têm sido propostas para explicar a ocorrência da hiperestesia dentinária (15-17). No entanto, o complexo mecanismo no qual fibras nervosas, localizadas próximas à camada odontoblástica, são influenciadas por estímulos, que atuam na superfície da dentina exposta, ainda não é completamente elucidado (17-20). Atualmente, a teoria da hidrodinâmica é a hipótese mais aceita na literatura para interpretar a ativação destas fibras em conseqüência da transmissão de estímulos sensoriais (4,17,21,22).

Em condições normais, a dentina é permeada por diversos canalículos que se apresentam completamente protegidos do meio externo pela presença do esmalte ou do cemento dentário (22,23). No entanto, esta cobertura pode ser facilmente perdida em conseqüência de processos de abrasão, erosão, abfração ou pelo desnudamento da superfície radicular por recessão gengival ou procedimentos periodontais e determinar a exposição dos túbulos dentinários ao meio bucal (13,22,24,25).

De acordo com os princípios da hidrodinâmica, a sensação dolorosa percebida após a aplicação de um estímulo sobre a dentina exposta ao meio bucal é conseqüência do

deslocamento rápido do fluido presente dentro dos túbulos dentinários (15,16). Essa movimentação do fluido atua, portanto, como um sinalizador da presença de estímulos na superfície dentinária (18,26). Dependendo da natureza do estímulo, o fluido dentinário pode ser facilmente deslocado em direção à polpa ou em sentido contrário e determinar uma variação na pressão intra-pulpar capaz de estimular fibras nociceptivas, localizadas próximas à camada odontoblástica (15,16). A pressão aplicada sobre as terminações nervosas pode produzir deformação na membrana dessas fibras, abertura dos canais de sódio e a conseqüente despolarização da membrana (26,27). Após a ativação, sinais nociceptivos são transmitidos para o complexo do núcleo trigeminal localizado na medula, que desempenha um papel fundamental na tranferência desses sinais para regiões do córtex cerebral associadas com a percepção da dor (26).

A literatura apresenta diversas formas de tratamento para a hiperestesia dentinária, que variam desde procedimentos simples a terapias complexas (3-5,21,22,28-56). Levando-se em consideração que o mecanismo da hidrodinâmica é o mais aceito para explicar a sensibilidade dolorosa em resposta a estímulos sensoriais, os agentes anti-hiperestésicos que reduzem a permeabilidade da dentina através da obliteração dos túbulos dentinários parecem ser os mais adequados para a diminuição dos sintomas (19,57,58).

Historicamente, agentes anti-hiperestésicos, como óleo quente, arsênico, nitrato de prata e formaldeído, foram utilizados para o alívio da sensação dolorosa (20,31). Atualmente, outras opções, tais como: a aplicação tópica de oxalato de potássio ou a irradiação da superfície dentinária com laser, surgiram e têm-se mostrado eficientes na redução da sensibilidade (3,5,29,31-34,38,40,42-44,48,51-53). No entanto, ainda não existe um produto considerado ideal no tratamento da hiperestesia dentinária (5,7,32,37) e, em geral, a escolha do agente anti-hiperestésico para o completo alívio da sensibilidade dolorosa é uma decisão difícil na clínica odontológica (4,14,24,25).

Nesse contexto, o conhecimento gerado pelos diversos estudos laboratoriais e clínicos torna-se fundamental para o direcionamento dos profissionais em busca de um tratamento eficiente (32,59). Entretanto, variações no planejamento e nos procedimentos experimentais utilizados nestas investigações freqüentemente produzem resultados contraditórios e difíceis de serem comparados (59-62). Dessa maneira, é fundamental que novos estudos clínicos bem conduzidos sejam desenvolvidos no sentido de elucidar ou comprovar estratégias terapêuticas eficientes para o tratamento da hiperestesia dentinária.

2. PROPOSIÇÃO

Os objetivos do presente trabalho foram:

a) revisar criticamente a literatura disponível sobre os principais aspectos relacionados à etiologia e ao tratamento da hiperestesia dentinária;

b) avaliar clinicamente a eficiência do gel oxalato de potássio a 3% e do laser de arseniato de gálio-alumínio (AsGaAl) no tratamento da hiperestesia dentinária, utilizando-se um gel placebo como controle.

3. CAPÍTULO 1

MANAGEMENT OF DENTINAL HYPERSENSITIVITY: A CRITICAL REVIEW

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ABSTRACT

Dentinal hypersensitivity is a complex sensorial condition which can cause considerable concern in the dental office. Despite the large number of available treatment modalities, there is no current desensitizing agent considered ideal to manage this uncomfortable situation. The choice of the right therapy is very intricate and requires a complete understanding of the mechanism by which a stimulus applied on the exposed dentine surface can influence the nerve fibers and produce the hyperesthesia. In this review, the main evidences regarding the etiology and the management of dentinal hypersensitivity are critically analyzed through the results of laboratory and clinical investigations.

Key words – Dentinal hypersensitivity; hyperesthesia; desensitizing agents.

INTRODUCTION

Dentinal hypersensitivity is an exaggerated response of exposed dentine when in contact with thermal, evaporative, tactile, osmotic or chemical stimuli (1). It is characterized by a localized, transient and sharp pain which may range from mild discomfort to extreme pain (1,2). Depending on its intensity, the hyperesthesia can affect eating, drinking and breathing, hinder the ability to control dental plaque effectively and, sometimes, it may even result in emotional changes that alter lifestyle (1).

This painful condition of the dentine is considered the most frequent complaint among reported odontalgias (3). Prevalence studies concerning the dentinal hypersensitivity indicate that it affects 10 to 30 percent of the general population (2, 4-8). Moreover, with teeth being retained for longer periods, there will be increasing demand by patients involved in this uncomfortable situation (1,2,4,9,10).

Several theories have been proposed to elucidate the mechanism of dentinal hypersensitivity (4,11-14). The Hydrodynamic Theory is considered to be the most widely accepted to explain the relationship between the presence of stimuli on the dentine surface and the nerve activation (11,12,14-20). According to its concept, the dentine exposure to the oral environment and the patency of the dentinal tubules are the main factors associated with the development of dentinal hypersensitivity (2,11,18,21). Dentine exposure usually occurs as a result of enamel loss by erosion, abrasion, abfraction or denudation of root surfaces as a result of gingival recession or periodontal procedures (1,3,4,17,22,23).

The reviewed literature presents a large number of treatment modalities for the management of dentinal hypersensitivity (3,4,9,10,14,16,17,19). Local application of desensitizing agents either by a dental professional or by the patient him/herself at home is the commonest attempt to eliminate or reduce the painful condition (4). Nevertheless, there is no

product that comprises all the qualities required for it to be designated as the ideal dentine desensitizer (3,4).

Therefore, a critical analysis with regard to the development of this hyperesthesia and its therapeutic approach is indispensable in order to suggest an effective treatment and control its incidence. The purpose of this review was to critically analyze the main evidences related to the etiology and management of dentinal hypersensitivity reported in laboratory and clinical investigations.

LITERATURE REVIEW

1. Peripheral and central mechanisms of dentinal pain

The mechanism of the dentinal pain reaction in response to a variety of stimuli is very complex and not yet clearly understood (15,17,24,25). Several theories have been proposed to explain this mechanism in the last few years (4,11-14,26).

According to the Transducer Theory, the odontoblastic processes can be directly excited by a variety of sensorial stimuli and release neurotransmitters which are responsible for conducting impulses to the nerve endings (4,15,24). The Neural Theory is based on the hypothesis that an applied stimulus can directly influence the nerve terminals within the dentinal tubules through direct communication with nerve fibers from the dental pulp (4,11,15,24). However, there was no solid evidence capable of supporting the statements of these theories (4,11,14,24).

The most widely accepted hypothesis about how the stimuli influence nerve fibers is the Hydrodynamic Theory (11,12,14-20). It states that the painful sensation that arises from exposed dentine after sensorial stimulation is a result of rapid fluid movement inside the dentinal tubules (11,12,14,17-19). The presence of tube-like structures in hypersensitive dentine plays an important role in maintaining the patency of the tubules which may prevent physicochemical processes from occluding the tubules, thereby maintaining the fluid flow across dentine structure. It was observed that biopsies from hypersensitive regions exhibited hollow, tube-like structures within the lumina of dentinal tubules in approximately 75.8% of the tubules, whereas in biopsies of non-sensitive areas of the same teeth, these tube-like structures were only seen in about 20.4% of the tubules. (27). According to the hydrodynamic mechanism, the fluid movement inside the tubules elicited by hydrodynamic stimuli is

thought to serve as a fluid transducer, signaling the presence of stimuli at the outer opening of the dentinal tubules (25,26). Depending on the nature of the applied stimulus, the fluid within the tubules can be easily displaced in either an inward or outward direction and determine a variation in the intra-pulpal pressure that stimulates nociceptive nerve fibers located on the pulpal side of the dentinal tubules (11,12). The pressure on the nerve endings of the subodontoblastic plexus produces deformation on the nerve membranes and opens the channels permeable to sodium (13). Due to the concentration gradient and negative charge in extracellular fluid, these ions enter the fiber by means of these channels and depolarize the membrane (13). Following nerve activation, nociceptive signals are transmitted to the trigeminal nuclear complex located in the medulla. This site plays an important role in processing and transferring these signals to higher brain regions. The perception of pain is thought to occur primarily within the cerebral cortex (26) (Figure 1).

Nevertheless, the complete mechanisms of odontogenic pain are complex and incompletely understood (28). The pain system can undergo dramatic changes in response to certain peripheral stimuli, leading to the development of two hallmark features of many clinical pain conditions: allodynia and hyperalgesia (26). Hyperalgesia is defined as an increase in the perceived magnitude of a painful stimulus and allodynia is defined as a reduction in pain threshold so that previously non-noxious stimuli are perceived as painful (26,28,29,30).

Considering that dentinal pain in hypersensitive teeth is classically defined by an exaggerated painful response to innocuous sensory stimulus, the altered pain state of allodynia, which is also experienced during inflammation of pulpal or periradicular tissue, may be designated as an essential feature in the mechanism of dentinal hypersensitivity (29). Moreover, dentinal hypersensitivity is not an invariant sensation as pulpal inflammation may predispose a tooth to enhanced dentinal hypersensitivity by reducing the threshold for

activation presumably because of peripheral or central mechanisms (26). Although the development and maintenance of mechanical allodynia in teeth with pulpal inflammation has yet to be elucidated, some potential hypotheses have been suggested. These hypotheses propose that mechanical allodynia in teeth with pulpal inflammation could be generated by either sensitization of mechanoreceptors (pulpal or periradicular) or a result of central sensitization (29,30).

During pulpal inflammation, activation or sensitization of nociceptors can be obtained by the concentration or deposition of inflammatory mediators that leads tissue levels to be sufficiently high to permit binding and activation of the receptor (26). There are many mediators, receptors, channels, enzymes, and transcription factors involved in pain signaling pathways (26,31). Peripheral afferent fibers respond to mediators such as nerve growth factor by increasing protein synthesis of substance P and calcitonin gene-related peptide (CGRP) and by sprouting terminal fibers in the inflamed tissue (26). While glutamate, substance P and CGRP are released as excitatory neurotransmitters from central terminals of primary afferent sensory neurons, γ-amino butyric acid (GABA) and glycine are released from interneurons and bind to their receptors in the dorsal horn, and act as inhibitory neurotransmitters. Adenosine triphosphate (ATP) is believed to cause significant membrane Ca^2+ permeability and can elicit pain via ATP-gated ion channels of the P2X receptor family. There have been seven P2X subunits identified so far (31). Other important receptor involved in pain signaling pathway in the primary sensory neurons is the transient receptor potential (TRP). The vanilloid receptor TRPV1 is known as the receptor for capsaicin and shows high Ca^2+ permeability and is also activated by temperatures over 43ºC (32). TRPV1 can be sensitized by ATP and bradykinin, and the threshold for heat activation of the sensitised TRPV1 becomes as low as 30ºC. Additionally, several channels and receptors expressed in

nociceptive sensory neurons have been recently identified, thus many of these molecules and pathways are attractive therapeutic targets for the treatment of pain (31).

2. Management of dentinal hypersensitivity

The management of dentinal hypersensitivity should be first based on the correct diagnosis of the condition and its causative factors (33). In addition to the desensitizing approach, the treatment plan for dentinal hypersensitivity should include identifying and eliminating predisposing etiologic factors, such as endogenous or exogenous acids and toothbrush trauma, in order to prevent or minimize further damage on the exposed dentine surface (33,34). Otherwise, the treatment is likely to provide only short-term success (20).

In fact, the achievement of an effective treatment for dentinal hypersensitivity has been a challenge for clinicians over the years (4). Historically, several desensitizing agents have been used for treating this problem, including, hot oil, arsenic, silver nitrate and formaldehyde (23). Now, other types of treatment, such as oxalates and calcium phosphate solutions, have emerged and shown significant effectiveness in reducing the hyperesthesia (3,14,17,19,23,35-38).

Dentine desensitizers can be usually assigned to three large groups: the anti-inflammatory, therapeutic occlusive agents and those with effect on nerve fibers depolarization (19). Since the hydrodynamic mechanism is the most accepted to explain the dentinal pain, the products that interfere with dentinal permeability seem to be more appropriate for desensitization (3,6,23,24,26). The occlusion of dentinal tubules promotes reduction in dentinal permeability, and proportionally decreases the degree of pain (3,6,24,35,39). This occlusion can be obtained through protein precipitation, particles deposition, application of laser beams or restorative procedures (18,19).

There are several requirements for considering a therapeutic occlusive agent as being ideal (40). Among others, it should be effective for a long period, easy and practical to apply, well tolerated by the patient and not irritating to the pulp (3,40). However, no current agent incorporates all these requirements and, therefore, an ideal treatment technique still has to be developed (3,19,40). Considering the need for a better approach to the management of dentinal hypersensitivity, the products that produce obliteration of dentinal tubules (oxalates, fluorides, laser beams, calcium phosphate solutions) have been intensely tested through laboratory and clinical studies (3,10,14,17-19,23,24,36-38,41-49). Laboratory studies are summarized in Table 1 and clinical studies are summarized in Table 2.

3. Laboratory investigations

Most of laboratory investigations are based on measuring the permeability of dentine discs obtained from extracted teeth (23,24,41,44,48,50). In these studies, a hydrostatic pressure gradient is applied to the specimens and the bulk fluid movement occurring through dentine is quantified (23,24,41). The measurement of dentine hydraulic conductance provides a convenient measurement of dentine permeability and allows the researcher to evaluate *in vitro* the efficacy of desensitizing agents in obliterating dentinal tubules (23,24,41,50).

Greenhill, Pashley (24) evaluated the ability of some dentine desensitizers to reduce the rate of fluid flow through dentine discs. The specimens treated with 30% potassium oxalate presented the largest reduction (98.4%) in the hydraulic conductance of dentine. This probably happens as a result of the deposition of insoluble calcium oxalate crystals on the dentine surface that consequently control the permeability of the exposed dentine.

The reduction in dentine permeability after dentine surface treatment with potassium oxalate was also experienced by Pashley, Galloway (41) through the application of different potassium oxalate formulations – 30% neutral dipotassium oxalate and acidic 3% monopotassium-monohydrogen oxalate – in different experimental groups of dentine discs.

Pereira et al. (48) observed *in vitro* that potassium oxalate-based agents were able to reduce filtration to values similar to those obtained with a smear layer with a mean flow reduction of 83%. In this study, the reduction in hydraulic conductance obtained with fluoride gel ranged from 41.47 to 77.47%.

According to Santiago et al. (23), the formulations based on potassium oxalate are considered an excellent option for the treatment of the dentinal hypersensitivity. A decrease in dentine permeability of about 75% was obtained with different potassium oxalate formulations, which indicates the obstructive effectiveness of these products.

Suge et al. (44) observed reduction in the hydraulic conductance of dentine discs treated with calcium phosphate precipitation method and of dentine discs treated with potassium oxalate, sodium fluoride and strontium chloride. The treatment by calcium phosphate precipitation method produced immediate reduction in dentine permeability to 6%. This permeability remained low even seven days after the discs were immersed in artificial saliva. The potassium oxalate treatment reduced dentine permeability to 8%. However, in this case, the permeability gradually increased with the immersion time.

Other laboratory studies evaluated the effects of desensitizing agents on dentine discs by verifying the morphology of dentine treated with different substances by scanning electron microscopy (SEM) (16,18,43,46). In this context, Oda et al. (16) evaluated the formation of an impermeable layer on dentine surface when glutaraldehyde, potassium oxalate and fluoride were applied to dentine discs after acid etching. The results showed that no uniform impermeable layer was formed when glutaraldehyde and oxalate were used.

However, when the treatment with sodium fluoride was performed, this layer could be observed, but it was fragile and easily removed by washing the dentine surface. It must be noticed that, in this study, the specimens were not fractured and, therefore, the effects of the desensitizing agents were analyzed only at the dentine surface. In addition, the desensitizing agents were applied after acid etching and this may have limited the reaction of these agents with the dentine surface as a result of calcium and phosphate removal.

Arrais et al. (46) verified the occurrence of intra-tubular precipitation and sub-superficial tubule occlusion of these three desensitizing agents, by means of analyzing their corresponding effects on fractured specimens. The SEM examination demonstrated the precipitation of crystal-like deposits extending up to 15µm inside the tubules of dentine treated with potassium oxalate; formation of a 1µm thick layer that covered the surface and infiltrated into tubules of discs after application of glutaraldehyde; and deposition of precipitates that occluded the dentine surface with no attachment to the tubules walls after phosphate fluoride treatment.

Ishikawa et al. (43) observed longitudinally sectioned specimens to investigate the occlusion of dentinal tubules by dentine treatment with an acidic solution that contains both calcium and phosphate. The dentine discs showed tubules occluded by a calcium phosphate precipitate up to a distance of approximately 15µm from the surface after the treatment with the solution.

These findings are in agreement with the results of an *in vitro* investigation developed by Pereira et al. (18). In this study, impressions and resin replicas of three dentine discs treated with potassium oxalate, calcium phosphate or glutaraldehyde were obtained and submitted to SEM analysis. Partial occlusion of dentinal tubules by crystal precipitation, usually below the surface, was observed after treating the dentine disc with potassium oxalate. The disc treated with the calcium phosphate solution originated a thick smear of precipitated

amorphous calcium phosphate that covered most of the tubules. The impressions of both of these discs presented short silicone tags, indicating little penetration of the impression material into the tubules. The treatment with glutaraldehyde also resulted in partial obliteration of the dentinal tubules. However, in this situation, the corresponding impression showed that the impression material penetrated deeply into some open tubules.

4. Clinical studies

The clinical trials on dentinal hypersensitivity are studies of great interest to produce scientific evidence about the effectiveness of therapeutic procedures (3,10,14,17,19,36-38,41,45,47,50). However, the lack of standardization of the methods used for the clinical treatment can yield contradictory findings and lead to results that are difficult to compare (51). Furthermore, the physiologic and the emotional aspects are important factors that influence these studies, thus the patient's response is very subjective and largely dependent upon the individual's pain threshold (4,50).

Pillon et al. (14), through a controlled clinical study, verified that a single application of a 3% potassium oxalate gel immediately after subgingival scaling and root planing resulted in greater reduction in dentinal hypersensitivity when compared with placebo gel application on the homologous contralateral teeth. This was observed 7, 14 and 21 days post-treatment. The highest percentage reduction for test (81%) and control (34.7%) groups was observed 21 days after the application.

Kishore et al. (36) evaluated the effectiveness of strontium chloride, potassium nitrate, sodium fluoride and formalin solutions for the treatment of dentinal hypersensitivity. It was verified that only the strontium chloride, the sodium fluoride and the formalin solutions significantly reduced the symptoms.

Pereira et al. (17) studied the immediate and mediate clinical responses of hypersensitive teeth after the application of three different potassium oxalate-based formulations (experimental groups) and after the application of a placebo gel (control group). The degree of sensitivity was assessed before (baseline), after four applications at seven-day intervals (immediate results) as well as after 6 months and 1 year (mediate results). The results demonstrated that all the substances applied, including the placebo, irrespective of the time period, resulted in an immediate reduction in dentinal hypersensitivity, which could also be observed after 6 months and 1 year.

The advent of laser technology and its growing use in dentistry included an additional therapeutic option for the treatment of dentinal hypersensitivity (3,9). The lasers used for this purpose may be divided into two groups: the low output power (low-level) lasers and the middle output power lasers (19,40).

Low-level laser therapy causes a photobiomodulating effect that increases the cellular metabolic activity of the odontoblasts and obliterates dentinal tubules as a result of the intensification of tertiary dentine production (52). On the other hand, the middle output power laser application promotes occlusion of these tubules through melting the dentine structure (38).

Ladarlado et al. (3) compared the therapeutic effects of a 660nm wavelength red diode laser and an 830nm wavelength infrared diode laser on the treatment of adult individuals with dentinal hypersensitivity and observed a higher level of desensitization with the 660nm red diode laser.

Corona et al. (19) evaluated the use of low-level laser therapy and the sodium fluoride varnish application for the treatment of dentinal hypersensitivity. Both treatments were effective in decreasing the painful condition. Nevertheless, the laser therapy showed improved results for treating teeth with a higher degree of sensitivity.

Schwarz et al. (37) observed the effects of an Er:YAG laser and an aqueous solution (22.5% polyurethane-isocyanate; 77.5% methylenechloride) on hypersensitive dentine. The therapies resulted in significant reduction in discomfort immediately after and 1 week post-treatment. After 2 months, the discomfort in the group treated with the desensitizing solution increased up to 65% and even up to 90% after 6 months, whereas the effect of the laser remained at the same level achieved immediately after the treatment. Compared with an untreated control group, both treatment forms resulted in a significant reduction in the hyperesthesia at both examination periods.

Lan et al. (38) evaluated the morphologic changes of hypersensitive dentine after Nd:YAG laser irradiation by taking an impression of the dentine surface before and after laser treatment and then examining it by SEM. The presence of protrusive rods on the impression indicates the penetration of the material inside dentine structure and, therefore, it is a measure of open dentinal tubules. The impression obtained after the laser treatment showed no protrusive rods, in comparison with the presence of numerous rods before the irradiation. Because of protrusive rods on the impression material is related to open tubules in hypersensitive areas, these authors supported the hypothesis that Nd:YAG laser irradiation can be used to seal exposed dentinal tubules of hypersensitive teeth.

Some clinical studies have reported the influence of the placebo effect on the treatment of dentinal hypersensitivity (10,17,42,45,47). This effect is described as a complex physiological and psychological interaction that depends to a large extent on the relationship between the patient and the professional (10,40). A positive and motivated emotional behavior may activate the central system pain inhibition, which controls the painful stimulus of the periphery by releasing endorphins. In studies on dentinal hypersensitivity, the trust in the professional and the will to obtain relief undoubtedly contribute to this effect (17).

Another possible phenomenon which can occur in clinical trials is the Hawthorne effect (10,50). It is related to non-intervention procedures, such as frequent examinations, improved oral hygiene or compliance with the treatment regimen. In dentinal hypersensitivity clinical studies, better oral hygiene may allow greater saliva access to patent dentinal tubules and, therefore, may enhance tubule occlusion through the deposition of salivary calcium, phosphate and proteins (10). In addition, it must also be taken in consideration the possibility that, in clinical investigation that evaluate different desensitizing agents, the saliva could dissolve and carry the agents under study, leading to the desensitization of non-treated teeth (carryover effect) (50).

The positive response with the use of the placebo was verified by Lier et al. (45). The results of a test group, treated with Nd:YAG laser, and a control group, in which the laser device was positioned but not activated, were compared and a similar reduction in the painful sensation was obtained in both groups.

Gentile et al. (47) observed reduction in dentinal hypersensitivity in 32 patients distributed into two groups: a treated group, exposed to six laser applications (gallium-aluminum-arsenide diode laser), and a control group that received applications of a curing light, as placebo. There was no statistically significant difference in pain reduction between treated and control groups between the beginning and end of treatment.

Similar results were obtained by Pereira et al. (17) after the application of three different potassium oxalate-based formulations in patients of experimental groups and after the application of a placebo gel in a control group. All the experimental groups and the placebo group resulted in an immediate statistically significant reduction in dentinal hypersensitivity, which continued after the periods of 6 months and 1 year.

The placebo effect was also reported by Cooley, Sandoval (42) in a clinical study that involved 28 individuals with hypersensitive teeth in two different areas of the mouth. The

teeth in one of these areas were treated with distilled water and the teeth in the other location received treatment with potassium oxalate. The results demonstrated an initial decrease in sensitivity in both groups. However, it was observed that the sensitivity of the water-treated teeth remained approximately the same for three months, while the sensitivity of the oxalate-treated teeth progressively increased over this period.

The results revealed by both laboratory and clinical research are extremely important to support the development or improvement of therapeutic procedures (3,10,14,16,-19,23,24,36-38,41-48,50). Consequently, the critical analysis of different studies with regard to current desensitization methods is of great interest to provide essential information and direct clinicians towards an effective treatment of dentinal hypersensitivity.

CONCLUSION

It could be concluded that dentinal hypersensitivity is a complex condition and its management in the dental office is very intricate. The reviewed literature points out several treatment modalities ranging from simple procedures, which can be performed by the patient him/herself, to complex procedures that involve the combination of therapies. Therefore, knowledge about the available desensitizing products and the factors involved in the mechanism of the dentinal hypersensitivity is indispensable in order to perform an effective treatment.

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FIGURE 1

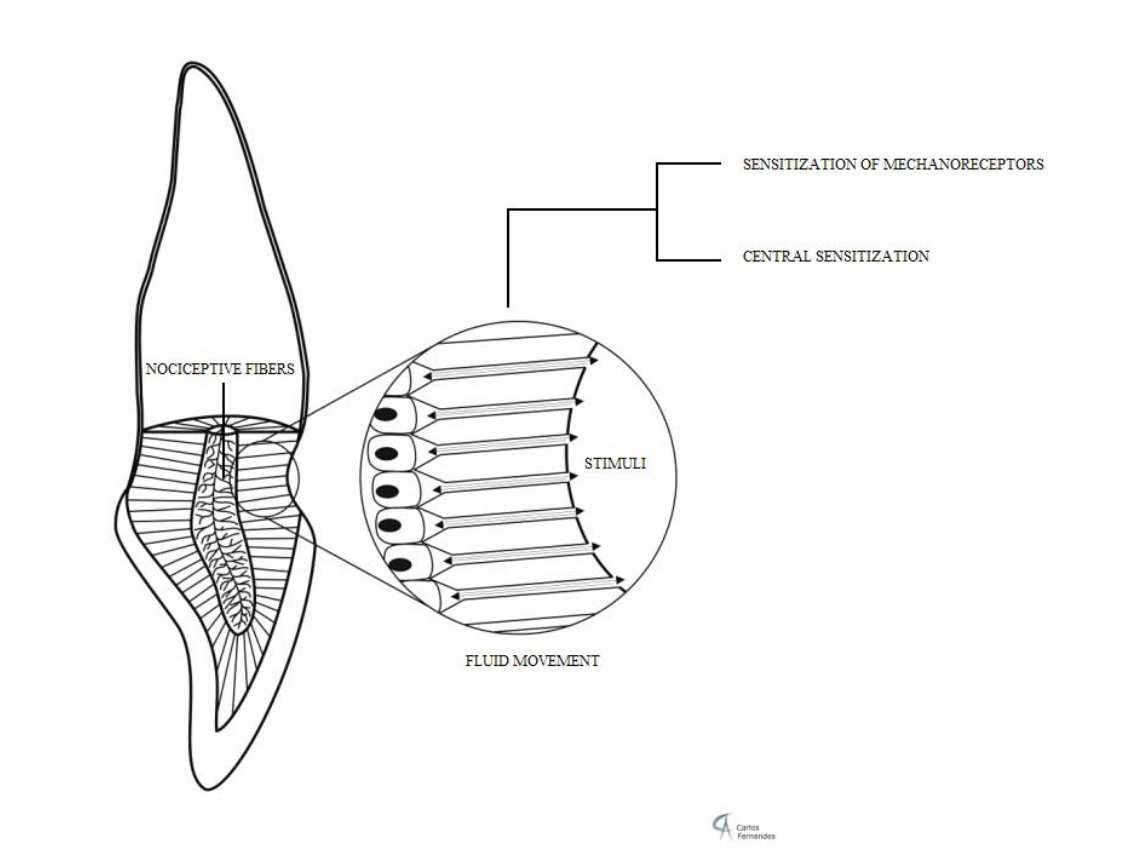

Figure 1. Peripheral and central mechanisms of dentinal pain and the fluid movement after the application of hydrodynamic stimuli at exposed dentine surface.

TABLE 1

Table 1. Summary of laboratory studies on dentinal hypersensitivity.

Authors	Dentine desensitizer	Assessment
Greenhill, Pashley 1981	30% potassium oxalate	HC
Pashley, Galloway 1985	30% dipotassium oxalate; 3% monopotassium-monohydrogen oxalate	HC
Pereira et al. 2005	Potassium oxalate-based agents; fluoride gel	HC
Santiago et al. 2006	Potassium oxalate formulations	HC
Suge et al.1995	Calcium phosphate; potassium oxalate, sodium fluoride, strontium chloride	HC
Oda et al. 1999	Glutaraldehyde; potassium oxalate; fluoride	SEM
Arrais et al. 2004	Glutaraldehyde; potassium oxalate; fluoride	SEM
Ishikawa et al. 1994	Solution containing both calcium and phosphate	SEM
Pereira et al. 2002	Potassium oxalate; calcium phosphate; glutaraldehyde	SEM

HC -Hydraulic conductance and SEM - Scanning electron microscopy.

TABLE 2

Table 2. Summary of clinical studies on dentinal hypersensitivity

Authors	Dentine desensitizer	Assessment
Pillon et al. 2004	3% potassium oxalate gel; placebo gel	VAS
Kishore et al. 2002	Strontium chloride; potassium nitrate; sodium fluoride; formalin solutions	VRS
Pereira et al. 2001	Potassium oxalate-based formulations; placebo gel	VRS
Ladarlado et al. 2002	660nm wavelength diode laser; 830nm wavelength diode laser	NS
Corona et al. 2003	GaAlAs laser; sodium fluoride varnish	VRS
Schwarz et al. 2002	Er:YAG laser; aqueous solution	VRS
Lan et al. 2004	Nd:YAG laser	SEM
Lier et al. 2002	Nd:YAG laser; control group	VAS
Gentile et al. 2004	GaAlAs laser; curing light	VAS
Cooley, Sandoval 1989	Potassium oxalate; distilled water	Q

VAS - Visual Analogue Scale; VRS - Verbal Rating Scale; NS - Numeric Scale; SEM - Scanning electron microscopy and Q - Questionnaire.

4. CAPÍTULO 2

CLINICAL EVALUATION OF A 3% POTASSIUM OXALATE GEL AND A GaAlAs LASER FOR THE TREATMENT OF DENTINAL HYPERSENSITIVITY

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Running title: Clinical evaluation of desensitizing agents.

One-sentence summary: Both active and control groups under study provided similar immediate and mediate reduction in dentinal hypersensitivity.

ABSTRACT

Background: The present study aimed at verifying the immediate and mediate clinical performance of a low-level Gallium-Aluminum-Arsenide (GaAlAs) laser and a 3% potassium oxalate gel for the treatment of dentinal hypersensitivity, using a placebo gel as a control.

Methods: A total of 164 teeth from 30 patients with clinical diagnosis of moderate or severe dentinal hypersensitivity were selected for this controlled clinical trial. The teeth were randomly divided into three groups according to the desensitizing treatment under study (GaAlAs laser, 3% potassium oxalate gel and placebo gel) and treatment sessions were performed at seven-day intervals for four consecutive weeks. The degree of sensitivity in response to evaporative and tactile stimuli was assessed according to a visual analogue scale at baseline, immediately after and three months after the fourth application. The results were analyzed by dentinal hypersensitivity reduction for each observational moment in relation to baseline, for each group separately.

Results: Both active and control groups resulted in statistically significant reduction of dentinal hypersensitivity in response to evaporative and tactile stimulation immediately after and three months after treatment. No significant differences among the three groups could be detected in both immediate and mediate evaluations irrespective of the applied stimulus.

Conclusions: The treatments under study had similar effectiveness and both active and placebo treatments were effective for treating dentinal hypersensitivity.

KEY WORDS: dentin sensitivity; oxalates; lasers; placebo effect.

INTRODUCTION

Dentinal hypersensitivity is an exaggerated response to sensory stimulus that usually causes no response in a normal, healthy tooth.[1] It is perceived as a localized, rapidly developing and transient pain and it is associated with dentine exposure to the oral environment.[1,2] Dentine exposure can result from enamel loss by erosion, abrasion, abfraction or denudation of root surfaces as a result of gingival recession or periodontal procedures.[3-10]

Prevalence studies indicate that dentinal hypersensitivity affects 10 to 30 per cent of the general population.[2,3,11-14] Moreover, with teeth being preserved and retained for longer periods, there will be increasing demand by patients involved in this uncomfortable situation.[1-3] It is considered the most frequent complaint among reported odontalgias.[15] Depending on its intensity, the hyperesthesia can affect eating, drinking and breathing, hinder the ability to control dental plaque effectively and, sometimes, it may even result in emotional changes that alter lifestyle.[1,5]

The hydrodynamic theory is the most widely accepted hypothesis to explain how stimuli applied on dentine surface influence nerve fibers.[3,4,5,10,16-20] It states that the painful sensation that arises from exposed dentine after sensorial stimulation is a result of rapid fluid movement in either an inward or outward direction inside the dentinal tubules.[16,17] The fluid displacement within the tubules determines variation in the intra-pulpal pressure that stimulates nerve endings located at the pulp/dentin interface and, therefore, results in generation of pain impulses.[16,17,20]

Historically, several desensitizing agents have been used for treating this problem, including, hot oil, arsenic, silver nitrate and formaldehyde.[8,21] Now, other types of treatment, such as oxalate gels and application of laser beams, have emerged and shown significant effectiveness in reducing the hyperesthesia.[8,10,15,20-44]

Potassium oxalate formulations have been intensely tested through laboratory and clinical studies.[7,8,21,22,26,29,32,34,35,38-41,44] The application of oxalates on hypersensitive surfaces mainly results in precipitation of insoluble calcium oxalate crystals that obliterate patent dentinal tubules and consequently control the permeability of exposed dentine.[8,21,22,26,34,35,37-39,45] Reduction in fluid movement within dentine by the crystal deposition, according to the hydrodynamic mechanism, proportionally decreases the degree of pain.[8,26,35,39,45]

An additional therapeutic approach for dentinal hypersensitivity came up with the advent of laser technology and its growing use in dentistry.[15,23-25,28,30,31,33,42] The low-level laser therapy can occlude dentinal tubules by increasing the cellular metabolic activity of odontoblasts which promotes intensification on tertiary dentine production.[46]

Review of literature presents a great number of treatment modalities for dentinal hypersensitivity, which suggests that none of them is totally efficient.[3,4,7,23,28,45,47] There is no current desensitizing agent considered ideal to manage this complex sensorial condition.[5,12,15,23,28] Clinical trials have supported different standpoints and the results have proved to be mostly contradictory.[45,48,49] Besides, the placebo effect has to be taken into consideration for its significant role often reported in clinical investigations.[7,21,24,31,47]

Therefore, reports on controlled studies with emphasis on the effectiveness of desensitizing agents are important and well-recognized.[6,23] The purpose of this study was to assess, at different examination periods, the clinical performance of a low-level gallium-aluminum-arsenide (GaAlAs) laser and a 3% potassium oxalate gel for treating hypersensitive teeth, using a placebo gel as a control.

MATERIALS AND METHODS

A total of 164 teeth from 30 patients (7 males and 23 females, aged between 24 and 68 years) with clinical diagnosis of moderate or severe dentinal hypersensitivity were selected and enrolled for the study on March 2007. To participate in the trial, patients should present good oral hygiene and at least three hypersensitive teeth, especially canines and pre-molars. Patients who presented severe systemic and/or psychological diseases, constant use of analgesic and/or anti-inflammatory drugs or allergic response to dental products were excluded. Besides, during the previous six months, the individuals should not have used any desensitizing agent and/or have been submitted to periodontal surgery or scaling. The selected teeth should not present carious lesions, defective restorations, cracks or fractures, premature contact, prosthesis or orthodontics appliances, periodontal pockets, mobility or evidence of pulpits. Participants were informed about the purpose and design of the investigation and signed an appropriate informed consent form. Standard dentifrices, toothbrushes and instructions were given to all subjects. The research protocol was approved by the Committee of Ethics in Research of Federal University of Ceará (protocol number 36/07).

The experimental model was split-mouth, controlled and double-blinded. The equipment and substances used in the study included a low-level GaAlAs diode laser device,[‡] a 3% potassium oxalate gel[§] and a placebo gel[‖] containing the same composition of the potassium oxalate gel except for the active substance. Both gels had the same color and texture characteristics. The 164 teeth (23 incisors, 20 canines, 77 pre-molars, 44 molars) were randomly divided into three groups according to the desensitizing treatment under study: laser treatment, potassium oxalate gel treatment and placebo gel treatment. This randomization was performed by placing all the selected teeth in a list and assigning its treatment according to a predefined sequence: (1) laser; (2) potassium oxalate gel; (3) placebo gel. Moreover, the three

different treatments were designated to the same patient to permit data correlation regarding his/her sensitivity threshold.

The degree of sensitivity was determined for each tooth in response to tactile (probe) and evaporative (air blast) stimuli. The probe stimulus was applied under slight manual pressure in mesiodistal direction on the cervical area of the tooth. The air blast was performed with an air syringe for one second at the distance of 1cm of the tooth surface. Dentinal hypersensitivity was assessed by patient's indication of the amount of pain related to each tooth, immediately after each stimulus, according to a visual analogue scale (VAS). The VAS was 10cm long and, on the left and right ends, contained an indication of "no pain" and "severe pain", respectively. The participants were instructed to place a mark on this 10cm line that corresponded to the pain severity of each tooth elicited by the hydrodynamic stimuli. The sensitivity patterns were recorded at baseline, immediately after and three months after treatment by an examiner previously calibrated for applying the stimuli. Neither the examiner nor the patients knew which type of treatment corresponded to each tooth.

The treatments were applied under relative isolation by one experienced operator other than the examiner. The diode laser device was used on contact mode with the following parameters: continuous emission, 30mW output power, wavelength of 660nm and 4Jcm^{-2} energy density. Laser beam was applied with the laser tip positioned perpendicularly to the tooth surface at four points to the apex (apical point) and the cervical area (mesio-buccal, disto-buccal and lingual points) of the tooth. Potassium oxalate gel was applied according to manufacturer's instructions: passive application, using a brush, for 2 minutes. During this period, the laser device was positioned, but not activated. The placebo gel application followed the same procedure. The treatment was repeated at seven-day intervals for four consecutive weeks. Three months after the fourth treatment session, patients were recalled for reassessment of dentinal hypersensitivity.

Statistical Analysis

The mean values of the clinical parameters were calculated for the three groups according to the different stimuli. Scores obtained immediately after and three months after the fourth treatment session were considered, respectively, the immediate and the mediate results of the treatment. The data was submitted to Kruskal-Wallis test with significance level of 5% (p=0.05) and analyzed by dentinal hypersensitivity reduction for each observational moment in relation to baseline.

RESULTS

A total of twenty-four patients completed the three-month study period, which corresponded to 80% of the patients and teeth involved in the investigation. No complications such as detrimental pulpal effects or allergic reactions were observed during this period. All teeth remained vital after treatment, with no adverse reactions reported or clinically detectable complications.

Table 1 shows the VAS scores (means and standard deviations) recorded after application of each of the stimuli for active and control groups at baseline, immediate and mediate periods. No statistically significant differences in VAS scores existed between the three groups at baseline following application of any of the stimuli ($p>0.05$). All groups provided significant overall reduction in dentinal hypersensitivity in response to evaporative and tactile stimulation ($p<0.05$). As presented in Table 1, the three treatment forms resulted in statistically significant reduction of VAS scores between baseline and immediate results ($p<0.001$). This could also be observed between baseline and mediate results ($p<0.05$). Additionally, the laser treatment tended to result in greater reduction of sensitivity between immediate and mediate results when considering the evaporative stimulation ($p<0.05$), whereas the effectiveness of the oxalate and placebo treatments remained at the same level achieved immediately after treatment ($p>0.05$). However, statistically significant differences were not detected among the three groups at both immediate and mediate results irrespective of the stimulus ($p>0.05$).

The pain severity elicited by the two different stimuli irrespective of the treatment is presented in Table 2. The VAS scores recorded at baseline demonstrate that the evaporative stimulus was more effective for detecting sensitivity ($p<0.05$). Three months after treatment,

percent reduction of sensitivity in response to tactile and evaporative stimuli was 65.5% and 62.5%, respectively, compared to baseline scores.

DISCUSSION

The present study evaluated the clinical performance of an oxalate gel and a GaAlAs laser, using placebo gel as control, for treating hypersensitive teeth. The degree of pain was recorded following tactile and evaporative stimulation, according to a VAS, in the following periods: baseline, immediate and mediate. It was observed that the three groups were very similar with respect to the degree of pain recorded at baseline in response to any of the stimuli. During the period of this study, all treatments resulted in statistically significant reduction in discomfort immediately after and three months after treatment. However, statistical analysis demonstrated no significant differences among the three groups in the decrease of dentinal pain observed in the immediate and mediate periods irrespective of the stimulus.

Potassium oxalate formulations have become well accepted for treating dentinal hypersensitivity as many laboratory and clinical studies have supported its ability in obliterating dentinal tubules.[7,8,21,22,26,29,32,34,35,38-41,44] It has been demonstrated, by scanning electron microscopy, that topical application of oxalates on dentine discs results in precipitation of insoluble calcium oxalate crystals on dentine surface and inside patent tubules.[22,26,34,38,44] The crystal deposition reduces fluid movement within dentine and, consequently, decreases dentinal sensitivity according to the hydrodynamic principles.[8,26,35,39,45] Reduction in dentine hydraulic conductance over 75% after application of different potassium oxalate formulations on dentine discs surfaces has been observed in vitro by Greenhill and Pashley,[26] Pashley and Galloway,[35] Pereira et al.,[39] Santiago et al.[8] and Suge et al.[44] Previous clinical studies about potassium oxalate effects on dentinal hypersensitivity developed by Cooley and Sandoval,[21] Kishore et al.,[29] Merika et al.,[32] Pereira et al.[7] and Pillon et al.[40] also support the results of the present investigation. However, an extensive comparison

with the findings of these studies may be limited due to variations in clinical trial design and experimental procedures.[45,48,49]

Pereira et al.[7] obtained significant clinical reduction in sensitivity scores with application of three different potassium oxalate formulations and with application of a placebo gel (control group) immediately after, 6 months and 1 year after four treatment sessions at seven-day intervals. In addition to the occluding effects, potassium oxalate neural action was mentioned for explaining the relief on dentinal hypersensitivity observed immediately after treatment. Similarly to the present study, these authors used tactile and evaporative stimulation for eliciting pain sensation. According to Holland et al.,[49] these stimuli are recommended for quantifying dentinal pain in clinical trials as they are both physiological and controllable. Nevertheless, the degree of pain severity was recorded in accordance to a verbal descriptor scale, while in the present investigation a VAS was adopted. The VAS is considered an objective method for assessing dentinal pain in which each tooth can act as its own control.[49] It also offers the advantage of being a continuous scale and its use for assessing sensitivity degree in clinical studies has been intensely reported by many other investigators.[24,25,31,32,40,48,49] Besides, this method is considered preferable to the use of numerical rating or verbal descriptor scales as these may be restrictive and not offer enough descriptions that could be placed in a continuous order of pain severity.[45,48,49]

Pillon et al.[40] verified that a single application of a 3% potassium oxalate gel immediately after periodontal procedures resulted in an increasing percent reduction in dentinal hypersensitivity at 7 (29.4%), 14 (64.6%) and 21 days (81.0%) post-treatment. Conversely, in the present investigation, the effectiveness of the oxalate treatment observed three months after the application sessions tended to remain at the same level achieved immediately after treatment. The results obtained in the previous study may have been strongly influenced by the spontaneous decrease of dentinal pain due to periodontal

procedures. According to Orchardson et al.,[2] the sensitivity tends to self-heal in a period of three to four weeks that follows periodontal treatment. It must also be considered that in the investigation developed by Pillon et al.,[40] though a VAS have been used for estimating the pain severity, the evaluation of dentinal hypersensitivity was performed by asking each participant about pain sensation during routine activities and not in response to sensorial stimulation. Nevertheless, it is suggested that at least two hydrodynamic stimuli should be used for assessing dentinal pain in clinical trials and the least severe stimulus should be applied first.[45,49] In order to contribute to this standardization, the results of the present study were analyzed irrespective of the treatment and demonstrated more pronounced painful response to evaporative stimulus when compared to tactile stimulus and it was also confirmed by Gentile and Greghi[24]. This may be attributed to the relatively greater number of dentinal tubules that are potentially stimulated by an air blast compared to probe stimulus.[32,48] It must also be mentioned that the air blast may produce three combined different physical effects: evaporation, thermal effect and physical compression of the air to the dentinal fluid.

Recent studies on dentinal hypersensitivity are beginning to focus on the use of lasers to manage dentinal pain since it emerged as a promising treatment modality[15,23-25,28,30,31,33,42] This study demonstrated that the GaAlAs low-level laser therapy is an effective method for reducing the degree of sensitivity in response to hydrodynamic stimuli immediately after and three months after treatment. Similar results in which GaAlAs laser irradiation provided decrease in dentinal pain elicited by thermal, tactile and evaporative stimulation were further reported.[15,23-25,33] The clinical effect of low-level lasers on dentinal hypersensitivity relies upon an immediate analgesic effect, as a result of laser-induced changes in neural transmission networks, and a mediate obliteration of dentinal tubules by tertiary dentine, due to intensification in the metabolic activity of odontoblasts.[15,23,25,28]

Gerschman et al.[25] investigated the result of four applications of GaAlAs laser using the wavelength of 830nm to both the apex and the cervical area of hypersensitive teeth at one-week, two-week and eight-week intervals. It was observed that the decrease in pain severity became more evident over time and that at eight weeks the percent reduction in sensitivity recorded to probe and air stimuli, in accordance to a VAS, was 65% and 67%, respectively.[25] These results differ from the findings of Gentile and Greghi,[24] that using GaAlAs laser with the wavelength of 670nm in six applications with intervals from 48 to 72 hours, verified more accentuated reduction of pain to tactile instead of evaporative stimulus immediately after the last application. In the present study, laser treatment was performed with the wavelength of 660nm at seven-day intervals and the pain relief immediately after the fourth application was also more pronounced to tactile than to evaporative stimuli. However, it was observed that the sensitivity degree to evaporative stimulation continued to reduce between the immediate and mediate evaluation, whereas sensitivity to tactile stimulus remained at a similar level achieved immediately after treatment. Comparatively, laser irradiation was performed by Gerschman et al.[25] to both the apex and cervical area of the teeth, similarly to this study, while laser application by Gentile and Greghi[24] was directed only to the cervical area. It is claimed that application of laser beams to the cervical area affects A-delta fibers while irradiation on the apex area is related to C-fibers.[25] Although the dentinal pain is said to be sharp and fast (A-delta fibers) whereas pulpal pain is slow and dull (C-fibers), in many cases the symptoms and aetiology are mixed.[20,25]

Despite promising outcomes have been reported, there is still a need for clarification regarding the specification of laser irradiation parameters for dentinal hypersensitivity treatment since the tissue response may be different after applications performed with distinct active medium, wavelength, power density, emission mode or application method.[15,28] In this context, Ladalardo et al.[15] compared the effectiveness of four

applications of a 660nm and an 830nm wavelength performed with GaAlAs laser in the cervical area of hypersensitive teeth at seven-day intervals and concluded that the laser application with the former wavelength was more effective for desensitization than the latter. The present study supported the effectiveness of the GaAlAs laser used with the wavelength of 660nm for treating dentinal hypersensitivity.

Although the present investigation demonstrated decrease in sensitivity scores for both the oxalate and the laser groups, a significant immediate and mediate reduction was also observed for the control group treated with placebo gel, and no differences could be detected among the three groups during the period of this study. However, Gerschman et al.[25] and Pillon et al.[40], through controlled studies, verified significant higher decrease in dentinal hypersensitivity for active than placebo group and also observed that the differences between these groups increased further in the subsequent evaluations. On the other hand, Pereira et al.[7] and Gentile and Greghi[24], in accordance to the results of the present investigation, obtained no statistically significant difference between the results of active and control groups in reducing the pain condition of hypersensitive teeth. Moreover, positive results involving the use of placebo in clinical trials have also been reported by other investigators.[7,21,24,31,47] This may me attributed to the so called placebo effect. It is described as a complex physiological and psychological interaction that depends to a large extent on the relationship between the patient and the professional.[21,28,47] Besides, the patient's response to sensorial stimulation is very subjective and largely dependent upon the individual's pain threshold, what may influence the results obtained in clinical trials.[3,28,48,49]

It is possible that studies with longer observational periods could enhance the differences between active and placebo groups. Therefore further clinical investigations are needed in order to evaluate long-term stability of the positive results obtained with these desensitizing agents. It could be concluded that all treatment modalities under study (GaAlAs

laser, potassium oxalate gel and placebo gel) showed similar overall performance and provided statistically significant reduction in dentinal hypersensitivity immediately after and three months after treatment was performed.

FOOTNOTES

‡ Bio Wave LLLT Dual®, Kondortech Equip Odontológicos Ltda., São Carlos, SP, Brazil

§ Oxa-Gel®, Kota Import's Ltda., São Paulo, SP, Brazil

‖ ARTPELE Farmácia com Manipulação Ltda., Fortaleza, CE, Brazil

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TABLES

Table 1. Means and standard deviations of VAS scores recorded at baseline, immediate and mediate periods after application of each stimulus.

Stimulus	Period	Laser group	Oxalate group	Placebo group
evaporative	Baseline	6.20 ± 2.48 A,a	6.41 ± 2.43 A,a	6.30 ± 2.44 A,a
	Immediate	2.66 ± 3.22 B,a	2.79 ± 3.48 B,a	2.70 ± 3.28 B,a
	Mediate	2.11 ± 2.69 C,a	2.53 ± 3.03 B,a	2.46 ± 2.93 B,a
tactile	Baseline	3.85 ± 3.26 D,d	3.67 ± 3.31 D,d	3.68 ± 3.29 D,d
	Immediate	1.33 ± 2.54 E,d	1.06 ± 2.19 E,d	1.25 ± 2.50 E,d
	Mediate	1.28 ± 2.19 E,d	1.31 ± 2.35 E,d	1.29 ± 2.17 E,d

¶ Different uppercase letters in the same column indicate statistically significant differences for each stimulus ($p<0.05$) and different lowercase letters in the same row indicate statistically significant differences for each stimulus ($p<0.05$).

Table 2. VAS scores (means and standard deviations) of sensitivity elicited by tactile and evaporative stimuli irrespective of the treatment.

Period	Evaporative stimulus	Tactile stimulus
Baseline	6.30 ± 2.43 A	3.74 ± 3.27 B
Immediate	2.72 ± 3.31 C (56.8%)	1.21 ± 2.40 E (67.7%)
Mediate	2.36 ± 2.87 D (62.5%)	1.29 ± 2.22 E (65.5%)

Different uppercase letters indicate statistically significant differences ($p<0.05$). The values in parenthesis represent the percent reduction of sensitivity compared to baseline scores.

5. CONCLUSÃO GERAL

Da avaliação dos resultados obtidos neste trabalho, pode-se concluir que:

a) a literatura apresenta diversos tipos de tratamento para a hiperestesia dentinária, que variam desde procedimentos simples, que podem ser executados pelo próprio paciente, até procedimentos complexos, que envolvem a combinação de diferentes terapias. Dessa forma, o conhecimento acerca do mecanismo de ocorrência da hiperestesia dentinária e dos agentes anti-hiperestésicos disponíveis é indispensável para a elaboração de um tratamento eficiente;

b) os agentes anti-hiperestésicos utilizados no estudo clínico podem ser considerados eficientes para o tratamento da hiperestesia dentinária, uma vez que proporcionaram redução estatisticamente significante do grau de sensibilidade dolorosa em resposta aos estímulos tátil e evaporativo, nos períodos de avaliação imediato e mediato;

c) tratamentos simples e pouco dispendiosos, como a aplicação tópica de gel de oxalato de potássio, podem produzir resultados similares na diminuição dos sintomas da hiperestesia dentinária aos obtidos através de terapias mais complexas, que envolvem o manuseio de um aparelho de laser;

d) o tratamento da hiperestesia dentinária está sujeito a grande influência de fatores psicológicos do paciente e da natureza subjetiva da dor. Nesse contexto, a utilização de placebos em estudos clínicos que avaliam agentes anti-hiperestésicos é capaz de produzir redução significativa da sensibilidade dolorosa em resposta a estímulos hidrodinâmicos e, além disso, demonstrar resultados estatisticamente semelhantes aos observados com os tratamentos convencionais utilizados no presente estudo clínico;

e) o efeito anti-hiperestésico dos tratamentos utilizados no presente estudo clínico pode-se prolongar por três meses após o término do tratamento. No entanto, é importante que novas investigações clínicas sejam realizadas no intuito de avaliar a estabilidade dos resultados positivos obtidos com estes tratamentos, em longo prazo.

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ANEXO A – Aprovação do Comitê de Ética em Pesquisa

Universidade Federal do Ceará
Comitê de Ética em Pesquisa

Of. Nº 185/07 Fortaleza, 12 de março de 2007
Protocolo COMEPE nº 36/07
Pesquisador responsável: Alessandra Helen Magacho Vieira
Deptº./Serviço: Departamento de Odontologia
Título do Projeto: "Avaliação da eficácia de agentes anti-hiperestésicos no tratamento da hiperistesia dentinária"

Levamos ao conhecimento de V.Sª. que o Comitê de Ética em Pesquisa da Universidade Federal do Ceará – COMEPE, dentro das normas que regulamentam a pesquisa em seres humanos, do Conselho Nacional de Saúde – Ministério da Saúde, Resolução nº 196 de 10 de outubro de 1996 e complementares, aprovou o projeto supracitado na reunião do dia 08 de março de 2007.

Outrossim, informamos, que o pesquisador deverá se comprometer a enviar o relatório parcial e final do referido projeto.

Atenciosamente,

Dr. Fernando A. Frota Bezerra
Coordenador do Comitê
de Ética em Pesquisa
COMEPE/UFC

ANEXO B – Termo de consentimento livre e esclarecido

Você está sendo convidado(a) a participar, como voluntário, de uma pesquisa a ser realizada na Universidade Federal do Ceará. Após ser esclarecido(a) sobre as informações a seguir e caso aceite fazer parte do estudo, assine ao final deste documento, que está em duas vias. Uma delas é sua e a outra é da pesquisadora responsável. A qualquer momento, você poderá desistir de participar da pesquisa e retirar seu consentimento. Sua recusa não trará nenhum prejuízo em sua relação com a pesquisadora ou com a instituição. Em caso de dúvida, você pode entrar em contato com a pesquisadora responsável através do telefone e endereço que constam neste termo ou procurar o Comitê de Ética em Pesquisa da Universidade Federal do Ceará pelo telefone (85) 3366 8338.

1. DADOS DE IDENTIFICAÇÃO DO PACIENTE E/OU RESPONSÁVEL LEGAL

Nome do paciente:			
Documento de identidade nº:	Gênero:	Data de nascimento: ___/___/_____	
Endereço:	Cidade:		UF:
Telefones para contato:		CEP:	

Nome do responsável legal:			
Documento de identidade nº:	Gênero:	Data de nascimento: ___/___/_____	
Endereço:	Cidade:		UF:
Natureza (grau de parentesco, tutor, curador etc):			

2. INFORMAÇÕES SOBRE A PESQUISA (PROCEDIMENTOS, RISCOS E BENEFÍCIOS)

Pesquisadora responsável: Alessandra Helen Magacho Vieira.
Endereço: Rua Capitão Francisco Pedro s/n - Rodolfo Teófilo - Curso de Odontologia FFOE (UFC).
Telefone para contato: (85) 3366 8410
Título do Projeto: Avaliação da eficiência de agentes anti-hiperestésicos no tratamento da hiperestesia dentinária.

A hiperestesia dentinária é uma das mais antigas queixas de pacientes e tem sido um problema constante na clínica odontológica, representando um número cada vez maior de pessoas que procuram o consultório odontológico incomodadas com esta situação desconfortável. Neste estudo serão aplicados produtos para o alívio da hiperestesia dentinária, tratando-se de um procedimento corriqueiro em clínica odontológica e necessário ao bem estar da saúde do paciente. Os materiais utilizados encontram-se disponíveis no mercado e foram previamente estudados através de testes de comportamento físico e estudos prévios de biocompatibilidade, não demonstrando nenhum risco à integridade do ser humano.

Confidencialidade: Os registros individuais dos seus dados serão mantidos em sigilo (confidencial). As informações a respeito dessa pesquisa poderão ser publicadas em revista científica. Apenas os resultados envolvendo médias serão divulgados ou, em pequeno número e de forma ilustrativa, fotografias sem a sua devida identificação.

3. ASSINATURAS

Eu, _____, concordo em participar da pesquisa e declaro que fui devidamente informado(a) e esclarecido(a) sobre o tipo de pesquisa, os procedimentos nela envolvidos, assim como os possíveis riscos e benefícios decorrentes da minha participação. Foi esclarecido que posso retirar meu consentimento a qualquer momento, sem que isto leve a qualquer penalidade.

Fortaleza, ___ de _____ de 2007 _____

Sujeito da Pesquisa

Confirmamos a solicitação de consentimento, esclarecimentos sobre a pesquisa e aceite do sujeito em participar:

_____ _____

Pesquisadora Responsável Testemunha

Nome: Identidade nº:

ANEXO C – Modelo de ficha de identificação do paciente

nº _____ Data _____/_____/_____

1. Dados gerais do paciente

Nome _____

Endereço _____ nº_____ ap._____

Bairro _____ CEP _____ Fone _____

Cidade _____ Estado _____ Data nasc. ____/____/_____

Idade _____ Sexo ( ) Fem. ( ) Masc. Cor _____ Nacionalidade_____

Profissão _____ Local de trabalho _____

Endereço do Trabalho _____ Fone _____

Outras informações _____

Relato do paciente _____

Já fez algum tratamento ou cirurgia periodontal? _____ quando?_____

Se já fez, a dor existia antes deste tratamento? _____

Como aparece esta dor, qual o estímulo necessário (ar, alimentos doces ou gelados, ao escovar os dentes)? _____

Já fez algum tipo de tratamento para diminuir esta dor? _____

Se já fez, qual o tratamento utilizado? _____ quando?_____

Qual o tipo de sua dieta (frutas ácidas, refrigerantes)? _____

Como escova os seus dentes? _____

Quantas vezes ao dia? _____ Qual é o detifrício utilizado? _____

Qual o tipo de escova de dentes utilizada (dura, macia, extra-macia)? _____

Tem o hábito de ranger os dentes ou apertá-los durante o dia ou à noite? _____

Tem algum problema de saúde (diabetes, hipertireoidismo, xerostomia, problemas digestivos, acidez)?_____

Tem algum problema psicossomático como anorexia, bulimia e outros? _____

Faz uso de algum medicamento ou droga (psicotrópicos ou álcool)? _____

Tem alguma alergia a cosméticos e/ou produtos odontológicos? _____

Está em período de gestação ou amamentação? _____

2. Dentes:

Dente	Tipo de dor	Rel. oclusal	Fac. desg	Cárie	C. Period.	Rest.

Dor: **P** – provocada **E** - espontânea **M** - momentânea **I** - intermitente

Intensidade: **L** - leve **M** - moderada **S** - severa

Percussão: **V+** - vertical positiva **H+** - horizontal positiva

3. Presença de lesão cervical não cariosa:

Dente	Tipo de Lesão		Localização		Tamanho e Profundidade
	Cunha	Pires	Face	Região	

ANEXO D – Escala visual analógica