Evaluation of Efficacy of Diode Laser in Treating Hypersensitive Dentin: A Pilot Study

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ABSTRACT

Aim and objective: The aim and objective of this study was to evaluate the potential of diode laser (DL) to treat dentinal hypersensitivity.

Materials and methods: Twelve specimens were obtained from dentin disks of 2 mm thickness prepared from the cementoenamel junction (CEJ) portion of six extracted human third molar teeth. The specimens were divided into two groups of six specimens each. All specimens received treatment with 1% citric acid to remove the smear layer. Specimens in group I (control) received no further treatment. Group II (DL) specimens received irradiation with 810 nm DL at an output power of 0.8 W for 10 seconds in noncontact mode. The specimens were prepared and observed under a scanning electron microscope. The diameters of tubules were analyzed.

Results: The mean tubular diameter of the samples belonging to group I (control) was 3.90 ± 0.63 μm. The mean tubular diameter of the samples belonging to group II (DL) was 2.11 ± 0.35 μm. Diode laser specimens showed a statistically significant reduction in the dentinal tubular diameter when compared to the control group.

Conclusion: In this study, DL was shown to effectively reduce the dentinal tubular diameters, thereby highlighting its potential to be used in the treatment of dentinal hypersensitivity.

Clinical significance: Diode laser shows promise in vitro, in reducing dentinal tubular diameters. And therefore could be used in the treatment of dentinal hypersensitivity, clinically. However, further in vivo studies need to be conducted to establish the efficacy of DL.

Keywords: Dentin hypersensitivity, Diode laser, Scanning electron microscope.


INTRODUCTION

Dentin hypersensitivity (DH) is a painful response of the tooth to external stimuli characterized by acute, non-spontaneous, short- or long-lasting pain that appears suddenly in a specific location, which cannot be attributed to any other dental pathology.¹ Dentinal hypersensitivity is usually caused due to an exposed dentin surface which responds to thermal, evaporative, tactile, osmotic, or chemical stimuli.²

It is one of the most commonly encountered problems that cause discomfort and pain in cervical region of exposed dentin. There is significant variation in prevalence from cross-sectional studies ranging from 3 to 57%. Studies conducted on periodontal patients show figures in the range of 72–98%.³ Various causes have been attributed to DH including occlusal wear and tear, aggressive toothbrushing leading to abrasion, parafuncional habits like bruxism, erosion from acidic beverages, and exposure of root surfaces due to gingival recession.⁴

Many theories have been proposed to explain the sensitivity of dentin. An early theory was the dentin receptor mechanism theory, which suggests that dentin sensitivity is due to direct stimulation of sensory nerve endings present in the dentin. This theory was discarded when studies showed absence of nerves in outer dentin. Another theory—the odontoblast transducer mechanism proposed by Rapp suggests that odontoblasts act as receptors which mediate changes through the synaptic junctions with nerves.⁵

The hydrodynamic theory was proposed in 1900,⁵,⁶ which is the most widely accepted theory. The hydrodynamic theory hypothesizes that any pain-producing stimuli would increase the fluid flow in the dentinal tubules. This causes a change in pressure across the dentin structure, which activates the A-delta fibers at the junction of pulp and dentin or in the dentinal tubules. Therefore, dentinal hypersensitivity depends on the presence of open dentinal tubules and a vital pulp, although it is still unclear how flow of fluid in the dentinal tubules stimulates the nerve endings.³

Studies show the presence of a greater number and wider tubules on hypersensitive dentin compared to normal dentin. The difference in diameter may be the more important factor since the dynamic fluid flow in the tubules is proportional to the radius. This plays an important role in the therapeutic strategies.³

The primary aim of managing DH is the control of pain and discomfort. This can be achieved by two means: Occluding the open dentinal tubules or interfering with the nerve impulse transmission. Occluding agents block the open dentinal tubules, preventing any external stimuli from causing the movement of dentinal fluid in the dentinal tubules which leads to decreased pain. The second method

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to manage DH is to block the nerve response to pain-provoking stimuli. This can be achieved by diffusion of certain ions.  

Conventional therapeutic strategies for DH include dentifrices containing strontium salts, potassium nitrate, sodium fluoride, or monofluorophosphate, topical desensitizing agents like fluoride salts, potassium nitrate, oxalate, calcium phosphates and arginine, iontophoresis, adhesives, and resins. An ideal desensitizing agent should occlude the dentinal tubules without affecting the pulp, should be painless, easy to use, fast acting, and permanently effective, and should not cause discoloration of teeth. 

The use of laser for the treatment of DH for the first time was reported by Matsumoto et al., in 1985. The lasers used for the treatment of DH are divided into two groups:

- Low-level lasers: Helium–neon lasers and gallium/aluminum/arsenide (GaAlAs) diode lasers (DLs).
- Middle output power lasers: Neodymium– or erbium-doped yttrium-aluminum garnet (Nd:YAG or Er:YAG) and carbon dioxide lasers (CO₂ lasers).

The mechanism of action of laser on DH is thought to be the laser-induced thermal effects on dentin leading to blocking or narrowing of the dentinal tubules. Among the family of lasers, DLs under certain parameters show less damage to the root surfaces and minimal temperature rise on the irradiated area, making them a more promising and a safe option.

This study is aimed to evaluate the efficacy of DL application in the treatment of hypersensitive dentin, in vitro. The aim of the present study is to evaluate the efficacy of DL in occluding dentinal tubules when viewed under scanning electron microscope (SEM).

**Materials and Methods**

The present in vitro study was conducted in Department of Periodontics, College of Dental Sciences, Davangere.

**Collection of Tooth Specimens**

Six impacted healthy third molar teeth extracted for surgical reasons were collected after informed consent from patients aged 25–30 years, from the Department of Oral and Maxillofacial Surgery, College of Dental Sciences, Davangere.

**Selection Criteria**

**Inclusion Criteria**

- Third molar tooth indicated for the extraction due to impaction
- Teeth with intact crowns and root surfaces
- Teeth unaltered by extraction procedure.

**Exclusion Criteria**

- Teeth which had any periapical lesion before extraction
- Teeth affected by dental caries
- Teeth with developmental anomalies
- Teeth from patients with dental fluorosis.

**Procedural Steps**

The extracted teeth were immediately washed in running tap water and were stored in bottles containing 5% phosphate-buffered saline solution for <1 month until required for experiment.

**Dentin Disk Specimen Preparation**

Dentin disks of 2 mm thickness were prepared from the coronal portion of the tooth just above the level of cementoenamel junction (CEJ) using a hard tissue microtome.

The dentin disks were polished with sequential grades of silicon carbide paper of various grit sizes (400, 600, 800, and 1,000) to create a standardized smear layer. These polished disks were then cut into two parts using a diamond disk bur, so that one part can be allotted to each group. This ensured that the specimen for both control and test group were obtained from the same tooth.

After allocating the dentin specimens to the two groups, they were mounted on a glass slide using a cyanoacrylate adhesive. Once mounted, these specimens were ultrasonicated in distilled water for 2 minutes to remove the residual smear layer. Then, the specimens were etched in 1% citric acid for 60 seconds to simulate hypersensitive condition. The specimens were stored in artificial saliva during the experimental period.

**Experimental Groups and Treatments**

Twelve dentin specimens were taken and divided into two groups.

**Group I (Control)**

Specimens treated with 1% citric acid and receiving no further treatment.

**Group II (DL)**

Specimens are treated with 1% citric acid followed by DL (810 nm) irradiation (Picasso, AMD lasers, USA) in continuous wave, noncontact mode with an output power of 0.8 W for 10 seconds. The optical fiber tip (tip diameter — 300 μm) was held 1–2 mm away from the irradiated surface and moved at a speed of approximately 1 mm/second.

**Laser Parameters**

Wavelength = 810 nm, power = 0.8 W, time = 10 seconds, continuous wave, noncontact mode, tip diameter = 300 μm, maximum power density = 6.07 W/cm².

**Scanning Electron Microscope Analysis**

The specimens were placed in 2.5% glutaraldehyde in 0.1 M phosphate buffer (pH 7.4) for a minimum of 24 hours. Following washing and dehydration through a graded alcohol series, they were mounted on SEM stubs. Mounted specimens were air dried for 48 hours and sputter coated with 30–40 nm of gold using a gold sputtering machine (BAL-TEC, SCD-500). Finally, the specimens were examined using a SEM (JEOL, JSM IT300) operating at an accelerated voltage of 15 kV. The parameters were tubular diameter and occlusion. The area at the center of each specimen was scanned so as to obtain tubules in a circular cross section. Representative photomicrographs were obtained at 1500x (Figs 1 and 2).

**Measurement of Tubular Diameters**

The diameter of tubules was measured using IMAGE J analysis software. All the tubules in each photomicrograph were measured for diameter. The largest diameter across each tubule was measured to minimize the error caused to the tubules cut obliquely. The calibration was based on the scale bar on the photomicrographs. The measurements obtained from the software were converted to microns based on the scale bar of the photomicrograph.

The data obtained were statistically analyzed using SPSS software. Multiple group comparison was performed using one-way ANOVA for parametric data. A “p value” of 0.05 or less was considered as statistically significant.
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**RESULTS**

A total of 12 dentin specimens from 6 periodontally healthy teeth were taken in this *in vitro* study to evaluate the effect of DL 810 nm on dentin etched with 1% citric acid for 1 minute.

**Tubular Diameter**

- The mean tubular diameter of the samples belonging to group I (control) was 3.90 ± 0.63 μm (Table 1).
- The mean tubular diameter of the samples belonging to group II (DL) was 2.11 ± 0.35 μm (Table 1).
- Intergroup comparison revealed that there was statistically significant difference in tubular diameters between group I and group II (*p* = 0.001) (Table 1).

**DISCUSSION**

In the present study, DL 810 nm was used at an output power of 0.8 W for 10 seconds in continuous, noncontact mode. Umana et al. showed that these parameters were able to block the dentinal tubules. Hence, these parameters can be considered harmless to pulp, and may be effective in the treatment of DH.

Three wavelengths (780, 830, and 900 nm) of DLs have been used for the treatment of DH. According to studies using the DL at 830 nm, the reduction in DH is caused by blockade of the depolarization of C-fibers. Diode laser irradiation at a maximum power of 60 mW does not affect the enamel or dentin surface physically, but a small fraction of the laser energy is transmitted through enamel and dentin to reach the pulp.

A wavelength of 800 and 980 nm is poorly absorbed in water and hydroxyapatite crystals. This allows propagation, scattering, or diffused transmission of the laser energy throughout the dentin, and leads to thermal effects on the dentin. The energy absorbed by the dentin causes an increase in temperature and leads to a melting effect to block the dentinal tubules.

For this study, 810-nm wavelength was selected, which is the most commonly used laser wavelengths in dental practice, especially in periodontics. Continuous wave, noncontact mode was used to allow easier scanning of the entire dentinal surface.

The mean tubular diameter of the samples belonging to group I (control) was 3.90 ± 0.63 μm. The mean tubular diameter of the samples belonging to group II (DL) was 2.11 ± 0.35 μm. Umana et al. showed that 810 nm DL irradiation at 0.8 to 1 W, continuous mode, speed: 1 mm/second for 10 seconds can lead to melting effect on dentin and narrowing of dentinal tubules. In the current study, the tubular diameters observed in group II (DL) were significantly less when compared to group I (control) which appears to corroborate the findings of Umana et al.

**CONCLUSION**

In this study, DL was shown to effectively reduce the dentinal tubular diameters, thereby highlighting its potential to be used in the treatment of dentinal hypersensitivity. Within the limits of the study, it can be concluded that specimens treated with 810 nm DL showed significantly reduced dentinal tubular diameter than the control group. Reduction in the number of dentinal tubules per unit area and decrease in dentinal tubular diameter could reduce dentinal fluid flow and in turn reduce hypersensitivity.

**CLINICAL SIGNIFICANCE**

Diode laser shows promise *in vitro*, in reducing dentinal tubular diameters. And therefore could be used in the treatment of dentinal hypersensitivity, clinically. However, further *in vivo* studies need to be conducted to establish the efficacy of DL.
REFERENCES


