

Effect of Argon Laser on Enamel Demineralization around Orthodontic Brackets: An In Vitro Study

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Abstract

Objective This study was designed to evaluate the effect of argon laser irradiation on development and progress of enamel demineralization around orthodontic brackets.

Materials and Methods: Fifty caries-free, intact human premolars were randomly assigned to one of the following five equal groups: Groups 1 (control) and 2: The brackets were bonded using conventional halogen light for 40s and argon laser for 10s, respectively. Teeth in group 3 were lased with argon laser for 10s before bracket bonding with halogen light. Group 4 was the same as group 3 except that brackets were also bonded with argon laser. In group 5 samples were bonded conventionally, immersed in an artificial caries solution for two days and then irradiated for 10s with argon laser.

All samples were subjected to demineralization by artificial caries solution for 10 days. After bracket removal, samples were buccolingually sectioned and evaluated by polarized light microscopy. Decalcified lesion depth in each section was measured by a trained examiner in a blind fashion. Data were analyzed in SPSS 14 using one-way ANOVA and Tukey's HSD post hoc test.

Results: The control group showed the greatest mean lesion depth while group 5 revealed the lowest. The laser-treated groups had significantly lower mean lesion depth compared with the control group ($P<0.05$) except for group 4 ($P=0.192$).

Conclusion: Argon laser irradiation for 10s before or during bracket bonding can increase caries resistance of intact and demineralized enamel.

Key words: Orthodontics; Argon laser; Demineralization; Enamel

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INTRODUCTION

Enamel demineralization or development of white spot lesions during orthodontic treatment presents a significant problem in orthodontic patients [1]. According to Noel, ortho-

dontic brackets bonded to enamel surface complicate the removal of food debris and result in the accumulation of plaque around the brackets; consequently bacterial acids dissolve the inorganic portion of enamel [2].

This dissolution can cause white spot formation in as little as 4 weeks [1].

Several methods have been implemented to prevent or reduce enamel decalcification during orthodontic treatment including fluoride application in various forms, enamel sealants, rigorous oral hygiene and modified appliance designs [1]. Dental laser also has a potential for prevention of white spot lesions by increasing the hard tissue resistance to caries [3]. The narrow band of wavelength of argon laser (centered around 470nm)[4,5] and its intense, monochromatic, coherent and collimated light make it a superior light source for quick and effective composite polymerization [2,6] occurring at 460-480nm [7]. It has been reported that argon laser irradiation on enamel surface reduces the demineralization depth around orthodontic brackets by 30-50% [2]. More recent studies have shown that argon laser irradiation of enamel reduces the susceptibility of enamel to demineralization by up to 50% [4]. Although the enamel surface can be lasered before bracket placement, application of argon laser for curing the bonding agent during bracket placement along with the irradiation of the adjacent enamel may simultaneously confer demineralization resistance. Many studies have tried to investigate the effect of argon laser irradiation on enamel demineralization [8-11]; but there is an important clinical question remained unanswered: " Does the argon laser have any therapeutic effect on demineralized enamel?" In other words, the effect of laser treatment on initial demineralization around orthodontic brackets has not yet been investigated.

This study evaluated the effect of argon laser irradiation on development and treatment of initial decalcified lesions around orthodontic brackets.

MATERIALS AND METHODS

Fifty caries-free human premolars extracted for orthodontic reasons were collected and stored in 10% formalin. Patients were aware

that their extracted teeth would be used in a study. The criteria for tooth selection were: intact buccal enamel with no developmental defects, no cracks, no caries or white spot lesions, no exposure to any pretreatment chemical agents such as hydrogen peroxide and no damage by the extraction forceps.

After debridement of soft tissue residues with a razor blade, the teeth were cleaned and polished with non-fluoridated pumice paste and prophylactic rubber cups. Samples were randomly assigned to one of the following five groups (n=10):

(1) Conventional bonding (control group):

The buccal surface of each tooth was etched for 15s with 37% phosphoric acid (3M, Unitek, CA, USA), rinsed with water for 20s and dried with oil free air stream for 10s, giving the enamel a chalky white appearance. Brackets (SS Standard 018 Slot, Dentaurum, Germany) were bonded with Transbond XT (3M, Unitek, CA, USA) according to the manufacturer's instructions, using Quartz Tungsten halogen (QTH) light curing unit (Faraz Dentin halogen light, Faraz Mehr, Isfahan) for 40s (20s from each of the mesial and distal surfaces). The intensity of the light source was verified before curing each specimen and recorded to be 450 mW/cm², using a radiometer (Apoza, Apoza Enterprise Co. Ltd., Taiwan).

(2) Laser bonding:

Brackets were bonded the same as in group 1 but to cure the bonding agent, low fluence argon laser was applied (6.9 J/cm², 0.270 watts, 5-millimeter beam size, power density of approximately 1080 mW/cm², 10-miliseconds pulse width, 593 AP Line tunable systems, Mellas Griot, USA) for 10s, 5s from each side of mesial and distal. Laser beam covered 5 mm of tooth surface occlusogingly (2 mm above the occlusal edge of bracket).

(3) Prelased and conventional light cure bonding:



Fig 1. Demineralized enamel lesion

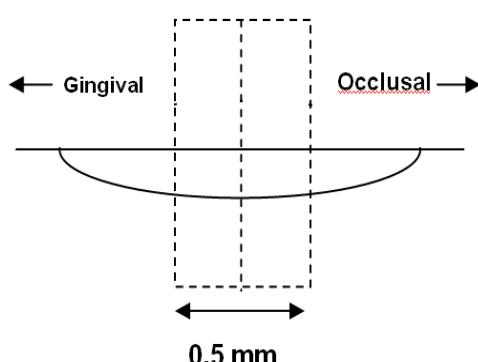


Fig 2. Template measuring lesion depth

The teeth were irradiated with argon laser for 10s and then the brackets were bonded as in group 1 with conventional halogen light.

(4) Prelased and bonding with argon laser:

The teeth were irradiated with argon laser for 10s and then the brackets were bonded as in group 2 using argon laser.

(5) Conventional bonding and laser treatment of artificial demineralization:

After bonding the brackets with conventional halogen light (the same as in group 1), samples were immersed in artificial caries solution consisting of 2.2 mM/L Ca²⁺, 2.2 mM/L PO₄²⁻ and 0.50 ppm fluoride at a pH of 4.3 at room temperature for two days and then were irradiated for 10s by argon laser. After bonding the brackets, all teeth were painted with a thin coat of acid resistance varnish covering all surfaces of the teeth except for the coronal area of the brackets. This enabled isolation of an area of demineralization. In the present study, we used coronal area of brackets because forceps forces during extraction may result in fracture in gingival area of the brackets.

All samples were stored in de-ionized water and were then subjected to demineralization. The specimens were immersed in artificial caries solution with constant circulation for 10 days.

After 10 days, the presence or absence of demineralization was judged by visual inspection of the teeth. The appearance of frosty white enamel, when dried, was considered as the presence of demineralization; which was positive for all samples. Brackets were then removed and after mounting the samples in epoxy resin, the teeth were sectioned buccolingually with a water-cooled diamond disk along the long axis of the tooth by a hard tissue microtome (SP1600, Leica, Germany). The 90-100 μm thick sections were oriented longitudinally on glass cover slides. The sections were then embedded in water (refractive index 1.33) for evaluation under polarized light microscopy (SZX 12 Olympus, Japan). The samples were digitally photographed with a magnification of 100× (Figure 1). Lesion depth for each section was measured as the average of three representative measurements from the surface of the lesion to the base of it (occlusal, middle, gingival). A template was used to measure a standardized area of each lesion. The template had 0.5 mm width and three lines, a central line with two side lines (Figure 2) [2].

The gingival line of the template was positioned 0.5 mm occlusal to the upper border of bracket. One examiner performed all the measurements in a blind fashion and after 1 week the measurements were repeated. The intra-class correlation was 0.99.

All statistical analyses were performed after assessing the data for normal distribution according to Kolmogorov-Smirnov test with SPSS software (version 14.0 for Windows; SPSS, Chicago, IL). P<0.05 was considered statistically significant.

RESULTS

Individual lesion depth measurements are summarized in Table 1 based on their location (occlusal, middle and gingival) for the 5 study groups.

The control group had the highest mean lesion depth while group 5 had the lowest. Based on the one-way ANOVA, there was a significant difference between groups in the mean lesion depth (P<0.05).

Tukey's HSD post hoc test revealed that the laser-treated groups had significantly lower mean lesion depth compared with the control group (P<0.05), except for group 4 (P=0.44). On the other hand, the lesion depth in groups 1 and 4 was significantly greater than in other experimental groups (Table 2).

Table 1. The mean lesion depth in three areas (μm)

Group	Coronal	Middle	Gingival	Total mean
1	135.45±(32.08)	143.25± (14.82)	145.75± (15.64)	141.48± (15.89)
2	105.50± (36.77)	101.00(38.52)	93.25± (39.74)	99..91± (35.05)
3	93.38± (28.12)	88.38± (26.70)	101.38± (29.86)	94.37± (24.48)
4	125.13±9(36.93)	122.13± (43.66)	131.13± (44.14)	126.12± (40.27)
5	76.38± (46.15)	78.00±46.57)	80.13± (35.61)	78.16± (20.53)

Table 2. Comparison of the mean lesion depth of experimental groups using Tukey's HSD test

Group	vs	Group	Mean difference	Std. error	Sig.
1	2		41.56	8.17	0.00*
1	3		47.10	8.17	0.00*
1	4		15.35	8.17	0.44
1	5		63.31	8.17	0.00*
2	3		5.54	8.17	0.97
2	4		-26.2	8.17	0.04 *
2	5		21.75	8.17	0.13
3	4		-31.75	8.17	0.00 *
3	5		16.2	8.17	0.40
4	5		-47.95	8.17	0.00 *

*Significant at the 0.05 level

Dependent variable: Mean lesion depth

DISCUSSION

A number of studies have shown that argon laser can be used to prevent enamel demineralization by altering its crystalline structure [13]. The argon laser has been more effective for prevention of caries than Nd:YAG laser, showing a smaller demineralization area in the enamel reported by Tavares et al [14]. Hicks et al [15,16] in their study reported that enamel exposure to argon laser at an energy level of 250 mW/cm^2 for 10s caused a 31-35% reduction in enamel demineralization compared with visible light (similar to group 3 of this study). Our study showed that irradiation of enamel with argon laser before bonding the brackets led to significantly lower mean lesion depth in comparison with the control group ($P<0.05$). This was also true when applying argon laser for bonding the brackets, as seen in group 2 ($P=0.01$) causing approximately 30% reduction in the mean lesion depth. In an *in-vitro* study in 2003, Noel et al. found that bonding the brackets with 10s of argon laser exposure (250 mW/cm^2) reduced the average lesion depth by 22% compared with the control group [2].

Using argon laser after or during bracket bonding may result in enamel surface changes making it resistant to demineralization.

Several mechanisms have been suggested for the anti-caries effects of argon laser. Low-energy argon laser treatment can considerably alter the surface morphology while maintaining an intact enamel surface [17]. The most likely mechanism for caries resistance is creation of micro-spaces in lased enamel. Penetration of acid into the enamel can result in the release of calcium, phosphorous and fluoride ions during demineralization. In lased enamel, the micro-spaces trap the released ions and act as mineral reservoirs within the enamel structure. Thus, lased enamel has an increased affinity for calcium, phosphate and fluoride ions [2, 18, 19]. It was reported that the enamel surface morphology became smoother after the argon laser treatment; in addition, the mo-

netite phase (CaHPO_4) which is a phase of CaP with a high aqueous solubility at the physiological pH compared to other phases of CaP (for example calcium hydroxyapatite)[20] in enamel crystals decreased in the lased cases leading to increased crystallinity and greater resistance to demineralization [21].

In our study, no statistically significant difference was found between groups 2 and 3 ($P=0.977$). To date, there is no study comparing the effect of argon laser exposure before and during the bonding of brackets on caries resistance; however, the results of this study showed that argon radiation on enamel surface before or during bracket bonding did not have a significant effect on the enamel surface.

An interesting finding of this study was that there was no significant difference between group 4 (prelased and bonded with argon laser) and the control group ($P=0.44$). Although group 4 received 20s of argon irradiation, its mean lesion depth was not significantly different compared to the control group. A larger sample size might have revealed differences in exposure time more precisely.

In the study by Blankenau et al [22], low power argon laser irradiation decreased enamel demineralization around orthodontic brackets. But it seems that there is not a dose-dependent linear relationship between laser irradiation and enamel demineralization. Thus, a future study is required to assess the relationship between the radiation dose and demineralization resistance.

Recent studies have shown that low-fluence argon laser exposure for very short periods of time enhances caries resistance in the absence or presence of topical fluoride agents, in both enamel and root surfaces [23]. However, the effect of argon laser irradiation on progression of initial decalcified lesions is unknown. A unique characteristic of our study was to investigate the effect of argon laser on both decalcified and sound enamel. A new finding of this study was that lasing the enamel with an initial decalcified lesion (group 5) had a sig-

nificant difference with the control group ($P=0.00$) and revealed a 45% reduction in the mean lesion depth. This procedure can be beneficial for patients with white spot lesions who seek orthodontic treatment. Future clinical trials can better elucidate this effect. Blanckena et al [22] also reported that in their pilot study, *in vivo* natural caries formation was significantly affected by a single exposure to low-fluence argon laser irradiation. The percentage of patients who experience some degree of white spot formation during orthodontic treatment ranges from 49.6% to 64% [2]. Thus, these patients could benefit from this effect of laser more than others. It should be noted that argon laser machine is too big and expensive for routine private clinical use; but research centers and universities can provide this opportunity for referral patients to benefit from its advantages. There are some limitations to this study; for example, placing the teeth in artificial caries solution does not exactly replicate the conditions in the oral cavity where de- and remineralizations occur. Future studies are required to assess the use of argon laser in the clinical setting.

CONCLUSION

According to the results of this *in vitro* study, irradiation of argon laser for 10s before or during the bonding of brackets can increase caries resistance of intact and demineralized enamel. In contrast, increasing the exposure time to 20s did not enhance resistance to demineralization.

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