

Evaluating the Effects of Non-surgical Periodontal Treatment on Masticatory Function in Patients with Aggressive Periodontitis: A Preliminary Study

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ABSTRACT

Introduction: Tooth loss is considered among the most common problems in patients with aggressive periodontitis. The loss of teeth heavily affects chewing ability and the neuromuscular control of mastication. Performing basic periodontal treatments have positive effect on the perseverance of teeth and subsequently the chewing ability.

Aim: To evaluate the effect of non-surgical periodontal treatment on the objective measures of masticatory function in patients with aggressive periodontitis.

Materials and Methods: Twenty patients (18-35 years of age) with generalised aggressive periodontitis were studied. Patients with 20 or more teeth in the oral cavity and normal occlusion on molar teeth were selected from the Department of

Periodontics, Faculty of Dentistry, Damascus University. Scaling and root planning alongside adjunctive chemical therapy (Ciprofloxacin+Metronidazole) was performed and bite-force registrations and Electromyography (EMG) records were determined on two occasions with three month intervals. Statistical analysis was done using paired samples t-test, independent samples t-test and Pearson's correlation coefficient.

Results: The overall improvement in the periodontal status following the non-surgical treatment was accompanied by a significant increase in bite-force (Newton) from 317.22±49.56 to 320.34±49.09, and a significant increase in EMG values from 347.01±51.81 to 351.74±50.65.

Conclusion: Periodontal treatment demonstrated a positive effect on bite-force and EMG values after non-surgical periodontal treatment.

Keywords: Bite-force, Mastication, Neuromuscular control

INTRODUCTION

Mastication is defined as an intermittent, rhythmic, semi-automatic movement in which a group of specified oro-facial structures (the masticatory muscles, the tongue, and the temporo-mandibular joint) act together to coordinate jaw movements and the direction of food morsels between teeth, preparing it for swallowing [1-3]. In order to achieve this precise and well-coordinated process, masticatory jaw movements are modulated by sensory inputs from receptors located in the oro-facial complex [3]. The most important receptors are the periodontal mechano-receptors, which are located in the periodontal ligament. The Periodontal Mechano-receptors (PMR) provide important sensory inputs to the central nervous system regarding the amplitude and direction of force, and the spatial orientation of the food morsels in the mouth [4,5]. The loss of such vital sensory information decreases the oral fine motor control, leading to an impaired masticatory function [6]. Aggressive periodontitis is a multifactorial, severe, and rapidly progressing inflammatory disease, caused by a group of specified microorganisms affecting the periodontal tissues, causing progressive bone loss and a marked increase in the probing depth [7]. These events can cause the loss of PMRs and subsequently an impaired masticatory function [8].

Previous studies assessing maximum voluntary bite-forces among patients with reduced periodontal tissue support due to chronic periodontitis, reported significant differences in the biting ability of such subjects compared to healthy controls [8,9]. Little is known about the control of mastication during the active phase of periodontitis and how its basic treatment would affect the PMRs responsible for the neural control of mastication.

The evaluation of masticatory function includes both subjective (questionnaires) [10] and objective measures such as assessing the electrical activity of the masticatory muscles and bite-

force registrations [11,12]. Masticatory function in subjects with chronic periodontitis was assessed previously in several studies [9,13,14], but no study was ever conducted on subjects with aggressive periodontitis.

The aim of the present study was to evaluate the effect of non-surgical periodontal treatment on the objective measures of masticatory function in patients with generalised aggressive periodontitis.

MATERIALS AND METHODS

The present quasi-experimental study followed a one group pre-test post-test design, which was approved by the ethics committee (N.1320) at Damascus university. Study was conducted at the Periodontology Department, Damascus University, from December 2017 till December 2018. Sample size was determined using Gpower 3.1 software. A minimum sample size of 15 patients was required with an alpha level of 0.05 and a power of 85%.

Twenty patients diagnosed with generalised aggressive periodontitis were enrolled in the present study with the following inclusion criteria: presence of 20 or more teeth, normal occlusion on molar teeth on both sides, no open bite or posterior cross-bite, generalised aggressive periodontitis diagnosed according to Armitage's 1999 classification [15] and molar teeth with probing depth of 6 or 7 mm.

The exclusion criteria were: systemic diseases, pregnancy, temporomandibular disorders, orthodontic appliances, fixed or implant supported prosthesis, teeth with pain (pulpitis), and the need for surgical periodontal treatment.

Non-surgical periodontal treatment was the treatment of choice in our study. The first step in the treatment of Generalised Aggressive Periodontitis as with most forms of periodontitis, is a cause-related treatment phase aimed at the reduction and/or elimination of the pathogenic microflora [16].

The following periodontal indices were measured by the same examiner at four sites per tooth using a periodontal probe (UNC-15): plaque index, gingival index, bleeding on probing, clinical attachment level, and probing depth.

Probing Pocket Depth (PPD) and Clinical Attachment Level (CAL) was registered for all teeth in the dental arch. However, bite-force registrations were performed only on molar teeth on both the right and left side. The periodontal treatment consisted of oral hygiene instructions, plaque control, and scaling and root planning. The treatment started right after the first session of clinical examination and physiological measurements. Mechanical treatment was attempted to be completed at the baseline visit. All patients received supra/sub-gingival full mouth debridement with manual scalers (CK6-U15), followed by scaling and root planning with manual instruments (Gracey cures) in deeper pockets as needed. Systemic antibiotics (ciprofloxacin 2 times a day and metronidazole two times a day for only 14 days) were prescribed as part of an adjunctive chemical therapy to the non-surgical periodontal treatment. Periodontal status was assessed prior to and three months after treatment. This was in accordance with a previous published study which revealed that the majority of both clinical and microbiological changes occurs in the 3 months following treatment [17].

Recordings of the masticatory muscles electrical activity was carried out using the sEMG (Micromed.S.p.a. viagiotto 2-31021 Mogliano Veneto-Treviso-Italy) which consists of two pairs of silver surface electrodes, an amplifier, and a software for analysing the EMG recordings (<http://www.micromed.eu/en-us/products/ID/6/categoryId/2/MYOQUICK--EMG-EP-Line>). The electrical activity from the bilateral masseter and anterior temporalis was recorded using two pairs of silver surface electrodes. First, the patient was seated in an upright position, and the skin corresponding to the studied muscles was prepared using alcohol cotton balls. The position of the surface electrodes was determinant by palpation while subjects performed maximal clenching. A reference electrode was placed on the subject's forehead.

Bite-force registrations were conducted using a force transducer (Elf+Flexi force- Tekscan 2008) to measure the maximal bite-force in newton. The bite-force registrations were performed only on molar teeth on both the right and left side. Acrylic occlusal disks (5 mm thick) covered the sensor to assess the bite-force at 10 mm mouth opening. In order to take all measurements at the same position, a dental impression material covered the bite-force transducer. The subjects clenched on the force transducer located between the first molars, and if missing the second molars were applicable. Maximum voluntary bite-force and muscles electrical activity were measured at the right and left sides in each subject. A verbal command was given for every subject to bite as hard as possible and to maintain clenching with maximal possible force for 5 seconds. In the present study, we did not record biting forces in healthy individuals, but according to studies in the literature, maximum voluntary molar bite-force in healthy individuals is 598.40 ± 247.91 [18].

For statistical purposes both PPD and CAL values were calculated based on the following: First, PD values of the buccal, mesial, distal and lingual sides of the right upper and lower molars were calculated and divided by 4. Subsequently, the mean of the upper and lower molar on the right side were added and then divided by 2. Finally, the result obtained represented the mean PD of the right side of the patient's mouth. In a similar fashion, the same was exactly done for the left side of each patient.

STATISTICAL ANALYSIS

The statistical study was conducted using the software package SPSS version 20.0 for windows. All results were considered significant at $p < 0.05$. The independent samples t-test was used to assess the differences in bite-force, EMG recordings, probing depth, and clinical attachment level before and after treatment, whilst Pearson's correlation coefficients were calculated for the

determination of correlations between the present variables.

RESULTS

Demographic Variables

A total of 20 patients (10 males/10 females) participated in the present study. The mean of the age was 28.6 ± 4.4 years.

Clinical Periodontal Parameters

Probing Pocket Depth (PPD): Probing pocket depth was measured at 4 sites for each tooth of the patient's dentition. The mean PPD was 3.45 ± 0.45 and 2.64 ± 0.24 before and after treatment respectively.

Clinical Attachment Level (CAL): Clinical attachment level was measured at 4 sites for each tooth of the patient's dentition. The mean CAL was 5.27 ± 1.07 and 4.12 ± 0.81 before and after treatment respectively.

Clinical parameters	Before	After	Mean difference	p-value
Gingival index	1.43 ± 0.39	0.84 ± 0.25	0.59	$< 0.001^*$
Plaque index	1.18 ± 0.37	0.69 ± 0.31	0.49	$< 0.001^*$
Bleeding on probing	0.49 ± 0.12	0.27 ± 0.08	0.22	$< 0.001^*$

[Table/Fig-1]: Mean values (\pm standard deviation) for plaque index, gingival index, bleeding on probing before and after 3 months of periodontal treatment ($p < 0.05$).

Mean values for plaque index, gingival index, bleeding on probing before and after 3 months of periodontal treatment [Table/Fig-1].

Physiological Parameters

Bite-force and EMG recordings: Bite-force and EMG values were tested before and after 3 months of treatment. The mean bite-force was 317.22 ± 49.56 and 320.34 ± 49.09 before and after treatment, respectively. The mean EMG was 347.01 ± 51.81 and 351.74 ± 50.65

Physiological and clinical parameters	Before	After	Mean difference	p-value
Probing depth (mm)	3.45 ± 0.45	2.64 ± 0.24	0.81	$< 0.001^*$
Clinical attachment level (mm)	5.27 ± 1.07	4.12 ± 0.81	1.15	$< 0.001^*$
Bite-force (N)	317.22 ± 49.56	320.34 ± 49.09	3.12	$< 0.001^*$
EMG (V)	347.01 ± 51.81	351.74 ± 50.65	4.73	$< 0.001^*$

[Table/Fig-2]: Mean values (\pm standard deviation) for PPD, CAL, Bite-force and EMG before and after 3 months of periodontal treatment (Paired samples t-test) ($p < 0.05$).

before and after treatment, respectively. Significant differences in mean PPD, CAL, bite-force, and EMG were found before and after the non-surgical periodontal treatment [Table/Fig-2].

Physiological parameters	Time point	Correlation coefficient (Pearson's) and p-value	EMG	PPD	CAL
Bite-force	Before	p	0.004*	0.520	0.081
		r	0.617	0.153	0.400
	After	p	0.004*	0.090	0.503
		r	0.613	-0.389	0.159
EMG	Before	p	-	0.625	0.051
		r	-	0.116	0.443
	After	p	-	0.277	0.199
		r	-	-0.255	0.300

[Table/Fig-3]: Correlation between variables before and after treatment (Pearson's correlation coefficient).

There was a significant positive correlation between bite-force and EMG both prior to and following treatment ($p < 0.05$). No correlation was found between the clinical (PPD\CAL) and the physiological (Bite-force\EMG) both prior to and following treatment ($p < 0.05$) [Table/Fig-3].

DISCUSSION

PPD and CAL were improved significantly after 3 month of treatment, indicating the importance of combined non-surgical/adjunctive chemical therapy in the treatment of subjects with aggressive periodontitis. A significant improvement in bite-force and EMG records was observed after 3 month of conservative treatment. An explanation of this is based on the belief that periodontal destruction and inflammation affects the neural control of mastication because of the impairment and change in the sensory function [19]. Periodontal inflammation affects the threshold level of the PMRs [20] and subsequently the neurological control of masticatory actions. These results are in accordance with previous studies in which inflammation affected the threshold level of PMRs [20,21]. Bite-force and EMG records were significantly correlated before and after treatment, these findings are in coordination with previous studies [22,23]. A study by Pereira LJ et al., found an elevation in bite-force and the electrical activity of the masticatory muscles without being statistically significant. The discrepancy found between the previous study and our investigation is due to the difference in the time period when the measures were re-taken. In our study, the assessment of bite-force and EMG was implemented after 3 month of the treatment, whereas the assessment of these measures in the study of Pereira LJ et al., was carried out after 45 days of the non-surgical periodontal treatment, and this time period could not be enough to promote an immediate response from the mechano-receptors in the periodontal ligament. Another possible reason is the different bite-force registration methods employed in our study and the study by Pereira et al., 2012. It is possible that PMRs function differently when applying forces at increased vertical dimension or at intercuspal position [14].

PPD and CAL values did not correlate with both of bite-force and EMG values. This result can be attributed to the assumption that the remaining PMRs are enough for the proper feedback mechanisms necessary for the oral fine motor control. The periodontal ligament contains two different groups of mechanosensitive nerve fibers. The first group consists of cell bodies located in the trigeminal ganglion and their fibers extend from the marginal gingiva to the apex. Whereas the second group consists of cell bodies located in the mesencephalic nucleus and their fibers are mainly present in the apical part of ligament. Thus, periodontal tissue destruction could cause impairment to at least a group of the periodontal mechano-receptors [24]. This is in accordance with previous studies that found no correlation between these different variables [14,19]. A discrepancy in results between the present study and a study by Takeuchi N et al., was found. This variation in results might be explained by methodological differences. In the present investigation, bite-force was measured only in the molar region whilst in the study by Takeuchi N et al., biting force was measured using pressure sensitive sheets in full arch [9].

LIMITATION

The limitation of the present investigation was the lack of previous studies assessing the effects of non-surgical treatment on bite-force and EMG in patients with aggressive periodontitis, hence limiting our ability to make direct comparison with previous data in the literature.

CONCLUSION

This study showed that non-surgical periodontal treatment has a positive effect on the objective measures of the masticatory function. Further studies with larger sample size should be done to evaluate the effect of reduced periodontal tissue support on the masticatory function.

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