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Original Research Article

Bite Force and Its Correlation with Periodontal Status and Salivary IL-1β Levels: A Comparative Study.

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Abstract: ***Background***: Chronic Periodontitis It is a multifactorial disease where significant elements incorporate not just the presence of pathogenic microscopic organisms and the immune systm . The Objectives: 1. Detect the clinical parameters of patients (plaque index- plI-, gingival index-GI-, probing pocket depth –ppd) 2. Evaluate maximum bite force by bite force sensor (loadstarsensor) in newton USA 3. Measuring the salivary biomarkers concentration (Human IL-1 by ElISA Kit). Materials &Method: Fourty subjects with age range (40-50) are participated in the present study which concluded 20 normal &20 with periodontal disease, samples taken from specialized health center,saliva samples after collected transferred to lab for centerfugated and detection of IL-B by ELISA, and the same time of collection bite force measured for each patient by specialed device for U/L arch R-L side,bleeding on probing also detected ,then compered the results. Results: age group of 45-50 of highly percentage of patients than the other with higher percentage of male however no significant association was found concerning all variables with both groups. The mean periodontal pocket PD1 score for the periodontitis group was not significantly higher than that of the non-periodontia group ($p>0.05$). The examination of the data showed that the periodontitis group had much lower maximal biting force measurements ($p \leq 0.05$). For Interleukin-1, the converse happened, with differences that were statistically significant ($p \leq 0.05$). In which bite force measurement for right side (-0.287) with periodontal pocket 2 in periodontitis patient. ***Conclusion***: The percentage of residual alveolar bone among all periodontal clinical characteristics is a reliable indicator of bite force and mobility.

Keywords: IL-1B, Bite Force, Periodontitis.

INTRODUCTION

Chronic periodontitis

One of the most prevalent long-term bacterial infections that damages the tooth's supporting tissues and causes tooth loss and attachment apparatus destruction is chronic periodontal disease (1). There are genetic variables associated with chronic periodontitis (CP) that affect the various disease phases. It is yet unclear, though, if these genetic predispositions could affect the relationships between these conditions. The hallmark of periodontitis is host-mediated inflammation linked to microbes, which damages periodontal supporting tissues. It is regarded as one of the main reasons why adults lose their teeth (2).

The bacteria and/or microbiota linked to the disease have been investigated as red-complex bacteria (Porphyromonas gingivalis, Treponema den4cola, and Tannerella forsythia) in chronic periodontitis. The polymicrobial infection causes the inflammation of periodontal disease. (3). Tannerella forsythia was suggested as a potential keystone pathogen among chronic periodontal bacteria that cause illness. *Tannerella forsythia*, is an anaerobic, Gram-nega3ve microorganism closely related to chronic periodontal disease. Different factors such as diabetes, smoking, gene3c predisposi3on, poor dental hygiene, fixed orthodon3c appliances can cause dysbiosis in the bacterial community (4). The goals of periodontal therapy are to lower the amount of bacteria and inhibit inflammation. The two main elements of non-surgical periodontal therapy (SRP) are subgingival debridement and oral hygiene instruction (OHI). Periodontal therapy has a high success rate in enhancing clinical parameters such as plaque index (PI), bleeding on probing (BOP), probing pocket depth (PPD), clinical attachment level (CAL), and, ultimately, decreased tooth loss. Risk factors may have an impact on treatment results. These can include smoking, obesity, poor oral hygiene, tooth-related parameters, compliance, and non-modifiable variables like heredity or acquired disorders (5).

A crucial indicator of how well the masticatory system is working is the biting force. The ability to bite is derived from the muscles of the jaw elevator involved in mastication. Certain features of craniomandibular biomechanics, including as muscular contraction and adaptation to the texture and density of food, modify the biting force that is generated (6).

Bite force is a gauge of the functioning of the masticatory system, and dentists frequently assess a patient's bite force. The primary purpose of this is to evaluate the therapeutic advantages of prosthetic devices through an understanding of mastication mechanics (7).

Maximum biting force, or MBF, is an objective indicator of chewing that takes into account the conditions of the stomatognathic system and provides information on tooth occlusion, muscle efficiency, and the development of masticatory function (8).

During mastication, the loading forces produced by the masticatory muscles are controlled by periodontal ligament mechanoreceptors (9).

The threshold level at which mechanoreceptor function is lowered may so result from decreased periodontal support. Biting force variations could result from this circumstance (10). The polypeptide interleukin-1 has a multiplicity of functions and roles in inflammation, immunology, tissue disintegration, and tissue homeostasis. After activation, it is produced by a variety of cell types, including monocytes, macrophages, T lymphocytes, fibroblasts, vascular cells, brain cells, and skin cells. IL-1 is essential to the pathogenesis of periodontitis because it controls bone resorption and the host's inflammatory response. Although other cell types can also make it, monocytes and macrophages are the main producers. IL-1α and IL-1β are the two subtypes of IL-1. The genes responsible for producing IL-1 have thus attracted the greatest interest lately as possible indicators of the advancement of periodontal disease (11).

MATERIAL AND METHOD:

Between July and December, 2023 among periodontitis patients aged 40–50 years who were matched in terms of their age and gender with non periodontitis patients (20 patients in each group). A sample of periodontitis patients was selected from the specialized dental health center in Babylon Governorate.

Inclusion criteria:

All the participants included in this study were apparently healthy, and did not use any systemic medicines, both gender was eligible. The study's protocol has been permitted by the research council of the Babylon Health Directorate / Ministry of Health. The assessment of periodontal condition, including the found or not found of periodontal pockets, was conducted using two periodontal

status indicators: periodontal pockets PD1 and PD2. A lightweight metallic probe called the Community Periodontal Index (CPI) was expressly constructed for this purpose (12). The ball tip of the probe should line up with the tooth root's surface anatomical arrangement when it was introduced. When a patient was being probed and experienced discomfort, it was likely that excessive force had been applied.

The entire extent of the gingival sulcus or pocket could be explored by carefully placing the probe tip into it. As close as possible to the point of contact with the third molar, for example, the probe was placed in the pocket at the second molar's distobuccal surface, parallel to the tooth's long axis. Then, using quick upward and downward movements along the buccal sulcus or pocket, the probe was softly pushed to the second molar's mesial surface. A similar procedure was applied to lingual surfaces, starting with the second molar's distolingual aspect.. Scores in your pocket: 0 indicates no condition, 1 indicates a pocket that is 4-5 mm, 2 indicates a pocket that is at least 6 mm, and 9 indicates an excluded tooth. X stands for missing tooth.

The present investigation employed the biting force sensor (Loadstarsensor) to measure the bite force (bite force sensor, loadstar sensor company, USA). The patients were measured while they were seated, facing forward, and standing up straight. For every patient, a single session was used to measure the maximal bite force using a portable bite force sensor that had to be held straight in relation to the floor (13).

After placing the device's sensor part on the right side's first molar region and asking the participant to bite as hard as they could for a short while, the maximum bite force for that side's first molar region was calculated in Newtons and displayed digitally (14). After two alternating runs of this bite measurement separated by two to three minutes, the greatest value was recorded.

Each participant was placed upright in a standard chair to measure the bite force. The USB Kit includes the sensor, a USB interface, and LoadVUE Pro software; all you need to do is plug the output cable's USB connector into a computer and launch the software. You can also place a disposable plastic cover over the sensor to keep saliva from destroying it and to keep it sterile for every user (12). Salivary enzymes were measured using the enzyme-linked immunosorbent assay (ELISA) kit, which measured interleukin-1β (IL-1β). In this study, 10 minutes between 9 and 11 a.m. were used to gather unstimulated saliva. In compliance with Munro guidelines (15), unstimulated salivary sample collection was done under standard operating settings. Salivary samples were then sent to a lab for analysis biochemically.

Detecting of IL-1 β:

After washing the unconnected components out of each well, the substrate solution was added. Both the hue of the Avidin HRP conjugate and the biotinylated detection antibodies would be blue. An enzyme-substrate interaction's color changes to yellow upon the addition of a sulphuric acid solution (15).

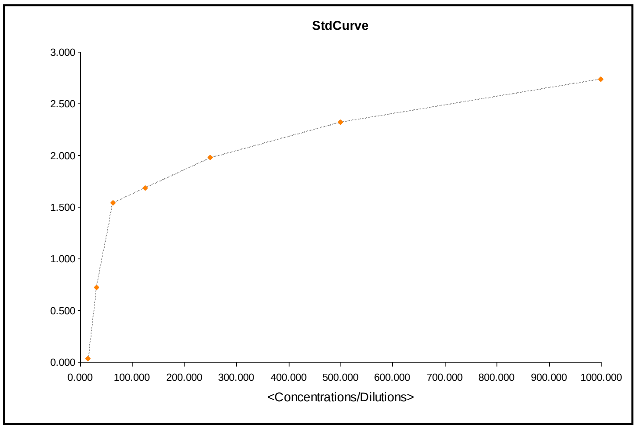

Figure (1): - IL-1B variations in saliva samples

RESULT:

The total sample was 40 healthy subjects divided into two groups equally (20 periodontitis and 20 non periodontitis) with age range 40-50 years. Table (1) shows the demographic data for both groups. In this table the age in years categorized into two groups with the older age group of 45-50 of highly percentage of patients than the other with higher percentage of male however no significant association was found concerning all variables with both groups. A seen in Table (1):

Table (1): Demographic description of the sample.

Variables		Study Groups				Statistic	
		Periodontitis		Non Periodontitis		Chi square	P value
		No.	%	No.	%		
Age (years)	40-44	8	40.00	7	35.00	0.107 NS	0.744
	45-50	12	60.00	13	65.00		
Gender	Male	11	55.00	12	60.00	0.102 NS	0.749
	Female	9	45.00	8	40.00		

Two indicators of periodontal conditions were used, periodontal pockets PD1and PD2. According to the data, the periodontitis group's mean periodontal pocket PD1 value was not significantly higher than that of the non-periodontitis group ($p>0.05$). The periodontal pocket PD2 showed the same image, however there was a statistically significant difference ($p\leq0.05$). Regarding bite force measurement, the data analysis revealed that the periodontitis group had significantly lower maximum bite force readings ($p\leq0.05$). Regarding Interleukin-1β, the opposite outcome was noted, with statistically significant differences ($p\leq0.05$), as shown in Table (2).

Variables	Study Groups				Statistic	
	Periodontitis		Non-Periodontitis		T test	P value
	Mean	Std. Dev.	Mean	Std. Dev.		
Periodontal pocket 1	1.800	1.207	1.467	0.273	1 .318	0.425
Periodontal pocket 2	0.916	0.727	0.414	0.200	2 .570	0.016*
Bite force	147.600	41.601	224.133	69.031	3 .678	0.001*
Interleukin-1β	119.466	13.11983	80.1087	23.958	2.915	0.007*

Table 2: Interleukin-1β, maximum biting force measurement, and mean value of periodontal conditions for the periodontitis group in comparison to the non-periodontitis group. *P≤0.05 indicates significant

When comparing the periodontitis group to the non-periodontitis group, Table (3) displays the correlation coefficients between Interleukin-1β and periodontal conditions. The periodontal pockets PD1 and PD2 among the periodontitis group showed significant positive trends ($p\leq0.05$). The same pattern was seen in the group without periodontitis, since the relationships were not statistically significant in either direction ($p>0.05$).

Study groups	Variables	Interleukin-1β	
		r	P value
Periodontitis	Periodontal pocket 1	0.264	0.041*
	Periodontal pocket 2	0.318	0.013*
Non-Periodontitis	Periodontal pocket 1	0.200	0.125
	Periodontal pocket 2	0.198	0.128

Table (3): The correlation coefficients between the periodontal conditions and Interleukin-1β for the group with periodontitis and the group without it are shown in Table (3). *P≤0.05, significant

Correlation coefficients between the maximum bite force measurement, periodontal conditions and Interleukin-1β for the periodontitis group compared with the non periodontitis group was revealed in Table (4). Among the periodontitis group, data analysis displayed that there were significant correlations in negative directions between maximum bite force measurement and periodontal pocket PD2 and Interleukin-1_{β} ($p\leq0.05$). The same picture was noticed for periodontal pocket PD1 with statistically not significant differences ($p>0.05$). Concerning the non periodontitis group, the same result was observed as the correlations were not significant in negative directions ($p>0.05$).

Study groups	Variables	Bite force	
		R	P value
Periodontitis	Periodontal pocket 1	-0.155	0.236
	Periodontal pocket 2	-0.287	0.026*
	Interleukin-1_{β}	-0.308	0.017*
Non-Periodontitis	Periodontal pocket 1	-0.186	0.154
	Periodontal pocket 2	-0.217	0.096
	Interleukin-1_{β}	-0.192	0.142

Table (4) shows the correlation coefficients between the periodontal conditions, maximal biting force measurement, and interleukin-1β for the group with periodontitis and the group without. *P≤0.05 indicates significant

DISCUSSION:

According to the study's findings, individuals were not able to produce their optimum biting force when they had decreased periodontal tissue support, as seen by higher attachment loss and decreased residual bone support. Similar outcomes were observed in patients with reconstructed crossarch bilateral end abutment bridges in Laurell and Lundgren's study. Our results are not consistent with those of the study by Kleinfelder and Ludwig (16), they proposed that there was no correlation between the area of the periodontal ligament and the maximum bite force in teeth that were not splinted, indicating that even fewer periodontal neural receptors might be sufficient for the proper input for mechanisms that limit the pressures used when biting and chewing.

The temporomandibular joint and/or pulp

mechanoreceptors can potentially affect the generation of the maximal biting force in the current investigation since a more comfortable bite force recording device was utilized, one that makes direct contact with the tooth surface area.

The average maximal bite force measured across all subject categories assessed in this study is 340 N. These trials yielded mean peak biting force values ranging from 176 N to 738 N (17). These findings are consistent with the study. The bite-force feature should be regarded as a critical criterion for evaluating people with dental or periodontal diseases, particularly those who have bruxism and oral parafunctions that may be impeded by high tooth stress. A bite's force increases with the posterior transducer's placement within the dental arch (18).

A jaw-lever mechanism made of metal has helped to clarify this. Further resistance to better withstand the bite force is provided by larger root surface area and the periodontal ligament around multirooted roots (19). Masticatory muscles provide loading forces during mastication, which are regulated by the periodontal ligament's mechanoreceptors. Therefore, the threshold level of mechanoreceptor activity may be lowered by diminished periodontal support (20). Alterations in the biting function may result from this illness. Individuals lacking connection exhibited reduced sensory function, according to Williams et al. Healthy subjects' ability to bite was shown to be much higher than that of people with chronic periodontitis, according to Faster et al. (21).

These results are in line with another study that found a clear correlation between lower periodontal support and lower biting intensity. The disparity in periodontal disease severity among the studies could be the cause of the discrepancy. According to Cheng et al., (22) sensory performance would be impacted by an inflammatory disturbance of the periodontal ligament, leading to a loss of control over an excessive amount of biting power. Numerous morphologic and physiological parameters, including age, gender, temporomandibular joint diseases, periodontal health of the teeth, and dentition status, can affect bite force (23). Considering these limitations, all other elements were regarded as constants, with the exception of the teeth's periodontal condition; as a result, the biting force varied to varying degrees (24).

To assess the variation in biting force values prior to and during periodontal therapy, more bite force measurement research is required, with a bigger sample size and a longer follow-up period. A correlation with IL-1B was discovered, which is in line with what Mohamadit et al. found (25). Furthermore, there was a relationship between the incidence of periodontitis and IL-1B. Therefore, our findings are consistent with those of Zeng et al., who discovered that IL-1B carriage and prevalence were elevated in patients with chronic periodontitis (26).

Ethical approval:
The patient's verbal and analytical consent was acquired before the sample was taken. Document number 115 states that on July 18, 2023, a local ethics commission examined and approved the study protocol, subject data, and consent form.

CONCLUSION:

1. Compared to females, men have a noticeably higher bite force value.
2. There was no discernible change in the bite force value between the participants with skeletal class II malocclusion and Angle's class I malocclusion The bite force is influenced by the vertical facial morphology, with a larger value for hypodivergent faces compared to hyperdivergent faces.
3. Individuals were not able to produce their optimum biting force when they had decreased periodontal tissue support, as seen by higher attachment loss and decreased residual bone support. Additionally, there was a correlation between the IL-1B and the prevalence of periodontitis. Our results stated that patients with chronic periodontitis had increased IL-1B carriage and prevalence.

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