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Estimation of Salivary Osteocalcin, α-Amylase and Total Protein Levels and Periodontal Health Status in Type II Diabetic Patients and Non Diabetic (A Comparative study)

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Abstract

Background: Diabetes and periodontitis are complicated chronic diseases with an established bidirectional correlation of many researches. Diabetes is stimulus inflammation and effects the osteoclast activity and in balance in bone turnover, which might increase vulnerability to the progress of chronic periodontitis. Diabetes is happen alterations in salivary flow rates and influence salivary composition and function. We thus evaluated saliva samples for levels of Osteocalcin, α- Amylase and total protein in diabetics and non-diabetics. The current study aims to determine the periodontal health status in the chronic periodontitis patients with and without poorly or well controlled type 2 diabetes mellitus, measure the salivary levels ofOC, α-Am and TO and then correlate between these clinical periodontal and biochemical in each study and control groups. **Materials and Methods:** Eighty patients with T2DM, males and females, were recruited for the study, with an age range of (35-50) years were divided into four groups, (20 subjects each): two groups had well or poorly controlled Type 2Diabetes Mellitus both of them with chronic periodontitis, two non-diabetic groups: one of them with healthy periodontium and systemically healthy (Control),and the other with chronic periodontitis group. From all subjects five ml of unstimulated whole salivary samples were collected from all of the participants, then the samples were centrifuged and the supernatants were collected and kept frozen until the biochemical analysis to measure OC, α – Am and TO concentrations. Clinical periodontal parameters (plaque index, gingival index, bleeding on probing, probing pocket depth and clinical attachment level) were recorded for all subjects at four sites per tooth except for the third molars. **Results:** Patients had chronic periodontitis with poorly controlled Type 2Diabetes Mellitus demonstrated the highest median, mean and ± SD (standard deviation) values of all clinical periodontal parameters and highest increase in levels of salivary OC, α-Am and TO, in addition to the highly significant differences among the study and control groups regarding biochemical parameters and clinical parameters. Highly significant strong positive correlations were revealed between OC, α-Am with TO and among of them with all clinical periodontal parameters, while highly significant strong negative correlations with PPD for the chronic periodontitis with poorly controlled Type 2Diabetes Mellitus group in OC,α-Am and TO. There was a non-significant weak correlation between all clinical periodontal parameters and OC, α-Am and TO existed with CAL in chronic periodontitis with poorly controlled Type 2Diabetes Mellitus group, correlation with PPD in chronic periodontitis with well controlled Type 2 Diabetes Mellitus group and correlation with GI existed in chronic periodontitis group; as well as, OC correlated with PLI existed in chronic periodontitis group. Finally, the study found that the correlation between salivary OC, α-Am and TO were highly significant strong positive in each of the study and control groups. **Conclusion:** Patients with poor glycemic control had more severe periodontal tissue break down with increase in levels of OC, α- Am and TO than well controlled type 2 diabetic patients and non-diabetic patients all of them with chronic periodontitis. So, these biochemical markers which are used assessment for periodontal tissue destruction .These provide chances to permit practitioners for early diagnosis, better prognosis and efficient management of periodontal diseases and type 2 diabetes mellitus.

Keywords: Periodontitis, Type 2Diabetes Mellitus, salivary Osteocalcin, α-Amylase and Total protein.

Introduction

DM is a multisystem disorder considered as a relatively or absolutely inadequacy of insulin secretion and/or associated resistance to the metabolic action of insulin on target tissues[1]. T2DM which is susceptible tooral complications such as periodontal disease (PD), dry mouth and abscesses [2]. Periodontitis is an inflammatory lesion that is attended by soft tissue impairment and bone resorption in the tooth supportive structures.

It has a multifactorial etiology and the distinguishing tissue destruction is mediated essentially by the abnormal immune response of different inflammatory periodontal diseases has been well recognized[3]. Osteocalcin is a calcium-connecting protein of bone and is the greatest plentiful non collagenous protein in mineralized tissues. Osteocalcin is synthetic mostly by osteoblasts, odontoblasts and hypertrophic chondrocytes and it has an imperative role in bone formation and turnover. It has also been exposed to promote bone resorption, and stimulate differentiation of osteoclast progenitor cells[4]. The α-Amylaseis represent the imperative digestive enzyme in the human salivary fluid, formed chiefly by parotid gland[5]. Also, it appears to play a function in maintaining mucosal immunity[6]. It was recommended that the amylase assistance against streptococcal bacterial adherence, which inhibits more proliferation on colonization of bacteria and may benefit regulate normal bacterial flora in the mouth[6]. Salivary total protein is a vigorous constituent of salivaand responsible for greatest of the functions of it[1]. There has been a relationship between T2DM, PD and several markers in saliva [7]. These have motivated us to perform the current study, in order to evaluate the salivary *(*OC, α-Am and TO)levels in T2 diabetic patients with CP to determine the effect of the glycemic control on their levels and the extent of the periodontal destruction.

Materials and Methods

The human sample consists of 80 patients with T2DM, males and females, with age range of (35-50) years. The collection of The subjects recruited for the study were patients attending the diabetic department of Madinat AL-Hussan Medical Hospital, as well as, patients from the dental specialized center in Karbala city. The subjects were divided into four groups:

A. CP with poorly controlled T2DM (CP+pT2DM): consisted of 20 males and females with CP and HbA1c > 9%.
B. CP with well controlled T2DM (CP+wT2DM): consisted of 20 males and females with CP and HbA1c < 7%.
C. Systemically healthy with chronic periodontitis (CP): consisted of 20 males and females. CP in patients was defined as the presence of minimally four sites with PPD ≥ 4 mm and clinical attachment loss of (1-2) mm or greater[8].
D. Systemically healthy with healthy periodontium (Control): consisted of 20 males and females apparently systemically healthy and with clinically healthy periodontium, this was defined by gingival index (GI) scores <0.5[9], without periodontal pockets or clinical attachment loss. This group represents a base line data for the levels of salivary OC, α-Am and TO. Inclusion criteria: males and females with T2DM (diabetic for 5 years) on oral hypoglycemic therapy only, at least 20 teeth present [10]. While, the exclusion criteria included:, T1DM and T2DM administering insulin, smoking and alcohol consumption, presence of systemic diseases other than T2DM, presence of nephropathy, retinopathy and diabetic foot, patients who've undergone periodontal treatment or administrated medications (anti-inflammatory, anti-microbial and anti-depressants) in the three months prior to the study and Pregnant, lactating and menstruation cycle. From all subjects five ml of unstimulated whole salivary samples were collected from all of the groups at 9-12 a. m. [11]. Then the samples were centrifuged at 4000 rpm for 15 min. and frozen at - 20 ◦ C. Clinical periodontal parameters examination was performed after collecting the salivary samples by using the Michigan O periodontal probe on four surfaces (mesial, buccal, labial, distal and lingual, palatal) of all teeth except the third molar. These included:

1. Assessment of Soft Deposits by the Plaque Index System (PLI) [12].
2. Assessment of Gingival Inflammation by the Gingival Index System (GI)[9].
3. Assessment of Gingival Bleeding on Probing (BOP) [13].
4. Assessment of Probing Pocket Depth (PPD).
5. Assessment of clinical attachment level (CAL).

For the purpose of biochemical analysis of salivary of OC, which was done by Enzyme Linked Immuno Sorbent Assay (ELISA) technique by using kit manufactured by (Shanghai Yehua, China).

Biochemical analysis of salivary of α-Amylase, which was done by Enzyme Linked Immuno Sorbent Assay (ELISA) technique by using kit manufactured by (Shanghai Yehua, China). For Total protein analysis we used kit manufactured by, Spinreact, Espana), which was done by biuret Colorimetric method, concentrations were determined by measuring the absorbance at540 nm by the spectrophotometer.

Descriptive statistics in the form of median value and inferential statistics in the form of Kruskal-Wallis H test, Mann-Whitney U test and Pearson Correlation were used in this study. The levels are accepted as significant (S) at (0. 05≥Pvalue>0. 01), highly significant (HS) at P-value ≤ 0. 01 and non-significant (NS) at P-value > 0. 05.

Results

Clinical Periodontal Parameters Analysis:

The highest median mean and ± SD (standard deviation) values of the clinical periodontal parameters were recorded in CP+pT2DM, followed by CP+wT2DM then CP group and control group in terms of plaque index, gingival index,, the score 1 bleeding on probing, probing pocket depth and clinical attachment loss (Table -1). Inter study groups comparisons regarding all clinical periodontal parameters revealed, HS differences between Control group and all of the study groups, between CP+ pT2DM with CP + wT2DM and CP groups; as well as, between CP+wT2DM with CP group (Table -2).

Table 1: Descriptive statistics of the Clinical Periodontal Parameters

Variables	Groups	N	Media	Mean	S. D.	X^2	p-value
PI	Control	20	0. 42	0. 43	0. 08	72. 216	0. 000 HS
	Periodontitis	20	1. 15	1. 16	0. 08		
	Well control D. M.	20	2. 27	2. 28	0. 12		
	Poor control D. M.	20	2. 76	2. 70	0. 21		
GI	Control	20	0. 55	0. 50	0. 24	67. 757	0. 000 HS
	Periodontitis	20	1. 08	1. 02	0. 17		
	Well control D. M.	20	1. 99	1. 83	0. 36		
	Poor control D. M.	20	2. 27	2. 07	0. 40		
BOP	Periodontitis	20	27. 3	26. 84	3. 91	21. 731	0.000 HS
	Well control D. M.	20	28. 7	29. 69	3. 04		
	Poor control D. M.	20	37. 7	35. 91	5. 62		
PPD	Periodontitis	20	4. 43	4. 55	0. 49	43. 095	0. 000 HS
	Well control D. M.	20	5. 31	5. 42	0. 45		
	Poor control D. M.	20	6. 35	6. 27	0. 37		
CAL	Periodontitis	20	2. 6	2. 72	0. 34	50. 060	0. 000 HS
	Well control D. M.	20	3. 73	3. 69	0. 35		
	Poor control D. M.	20	4. 66	4. 71	0. 29		

Table2:Comparison between each two groups in clinical parameters

Groups	Test	PI	GI	BOP	PPD	CAL
Control vs. Periodontitis	Mann-Whitney U	0	14. 5	-	-	-
	p-value	0. 000HS	0. 000HS			
Control vs. well control D. M.	Mann-Whitney U	0	0	-	-	-
	p-value	0. 000HS	0. 000HS			
Control vs. poor control D. M.	Mann-Whitney U	0	0	-	-	-
	p-value	0. 000HS	0. 000HS			
Periodontitis vs. well control D. M.	Mann-Whitney U	0	5	109. 5	41	17
	p-value	0. 000HS	0. 000HS	0. 014HS	0. 000HS	0. 000 HS
Periodontitis vs. poor control D. M.	Mann-Whitney U	0	0	53	4	0
	p-value	0. 000HS	0. 000HS	0. 000HS	0. 000HS	0. 000 HS
Well control D. M. vs. poor control D. M.	Mann-Whitney U	27	82	73. 5	26. 5	2
	p-value	0. 000 HS	0. 001HS	0. 001 HS	0. 000HS	0. 000 HS

Biochemical Parameters Analysis:

The biochemical analysis (table-3) of the salivary OC, α-Am and TO revealed that the highest concentration was in CP+wT2DM, followed by CP+pT2DM then CP and finally the Control. Furthermore, highly significant differences in the median values of the salivary OC, α-Am and TO concentrations revealed among the study and control groups at $p < 0.01$. The results of the comparisons for all pairs of the study and control groups in (table-4) about biochemical parameters levels revealed: highly significant differences between Control group and all of the study groups, between CP+pT2DM and CP+wT2DM; as well as, between CP+wT2DM with CP group. Finally, the comparisons between CP+pT2DM with CP groups.

Table 3: Descriptive statistics of biochemical analysers

Variables	Groups	N	Media	Mean	S. D.	X^2	p-value
Osteocalcin	Control	20	8. 46	8. 32	3. 05	74. 074	0. 000 HS
	Periodontitis	20	25. 99	24. 75	4. 27		
	Well control D. M.	20	38. 90	39. 26	1. 98		
	Poor control D. M.	20	55. 60	58. 73	8. 64		
α Amylase	Control	20	713. 32	654. 15	265. 56	74. 074	0. 000 HS
	Periodontitis	20	1220. 36	1235	144. 50		
	Well control D. M.	20	1737. 12	1725. 96	147. 59		
	Poor control D. M.	20	2574. 03	2584. 33	349. 94		
Total protein	Control	20	210	206	17. 59	74. 193	0. 000 HS
	Periodontitis	20	260	264	18. 18		
	Well control D. M.	20	340	339	24. 47		
	Poor control D. M.	20	525	512	76. 27		

Table 4: Comparison between each two groups of biochemical analysers

Groups	Test	Osteocalcin	α Amylase	Total proteins
Control vs. Periodontitis	Mann-Whitney U	0	0	0
	p-value	0. 000 HS	0. 000HS	0. 000HS
Control vs. well control D. M.	Mann-Whitney U	0	0	0
	p-value	0. 000HS	0. 000HS	0. 000HS
Control vs. poor control D. M.	Mann-Whitney U	0	0	0
	p-value	0. 000HS	0. 000HS	0. 000HS
Periodontitis vs. well control D. M.	Mann-Whitney U	0	0	0
	p-value	0. 000HS	0. 000	0. 000
Periodontitis vs. poor control D. M.	Mann-Whitney U	0	0	0
	p-value	0. 000HS	0. 000HS	0. 000HS
Well control D. M. vs. poor control D. M.	Mann-Whitney U	0	0	0
	p-value	0. 000HS	0. 000HS	0. 000HS

Correlations of Salivary OC, α-Am with TO levels with Clinical Parameters and with Each Other:

As can be seen in table 5, generally demonstrated the highly significant differences among the study and control groups regarding biochemical parameters and clinical parameters. Highly significant strong positive correlations were revealed between OC, α-Am with TO with all clinical periodontal parameters, while highly significant strong negative correlations with PPD for the CP+pT2DM group in OC. α-Am and TO. There was a non- significant weak correlation between all clinical periodontal parameters and OC, α-Am and TO existed with CAL in CP+pT2DM group,

correlation with PPD in CP+wT2M group and correlation with GI existed in CP group,as well as, OC correlated with PLI existed in CP group. Finally, highly significant strong positive correlations were found among OC, α-Am with TO levels in the saliva at each of the study and control groups (table-6).

Table 5: Relation between the periodontal parameters and biochemical analysers

Variables	Groups		Osteocalcin	α Amylase	Total proteins
PI	Control	R	0. 663	0. 645	0. 607
		p-value	0. 001HS	0. 002HS	0. 005HS
	Periodontitis	R	0. 430	0. 453	0. 466
		p-value	0. 058 NS	0. 045S	0. 038S
	Well control D. M.	R	0. 911	0. 870	0. 670
		p-value	0. 000HS	0. 000HS	0. 001HS
	Poor control D. M.	R	0. 900	0. 998	0. 989
		p-value	0. 000HS	0. 000HS	0. 000HS
GI	Control	R	0. 687	0. 677	0. 521
		p-value	0. 001HS	0. 001HS	0. 018HS
	Periodontitis	R	0. 317	0. 320	0. 227
		p-value	0. 174 NS	0. 168 NS	0. 336 NS
	Well control D. M.	R	0. 999	0. 964	0. 753
		p-value	0. 000HS	0. 000HS	0. 000HS
	Poor control D. M.	R	0. 901	0. 998	0. 989
		p-value	0. 000HS	0. 000HS	0. 000HS
BOP	Periodontitis	R	0. 636	0. 624	0. 554
		p-value	0. 003HS	0. 003HS	0. 011HS
	Well control D. M.	R	0. 998	0. 958	0. 757
		p-value	0. 000HS	0. 000HS	0. 000HS
	Poor control D. M.	R	0. 919	0. 888	0. 896
		p-value	0. 000HS	0. 000HS	0. 000HS
PPD	Periodontitis	R	0. 539	0. 538	0. 513
		p-value	0. 014HS	0. 014HS	0. 021HS
	Well control D. M.	R	0. 299	0. 240	0. 181
		p-value	0. 201 NS	0. 308 NS	0. 446 NS
	Poor control D. M.	R	-0. 761	-0. 748	-0. 767
		p-value	0. 000HS	0. 000HS	0. 000HS
CAL	Periodontitis	R	0. 754	0. 749	0. 718
		p-value	0. 000HS	0. 000HS	0. 000HS
	Well control D. M.	R	0. 645	0. 605	0. 855
		p-value	0. 002HS	0. 005HS	0. 000HS
	Poor control D. M.	R	0. 032	0. 017	-0. 017
		p-value	0. 895 NS	0. 945 NS	0. 943

Table 6: Relation among the enzymes and proteins in each group

Groups	Variables		α Amylase	Total proteins
Control	Osteocalcin	R	0. 998	0. 881
		p-value	0. 000 HS	0. 000HS
	α Amylase	R		0. 874
		p-value		0. 000HS
Periodontitis	Osteocalcin	R	0. 998	0. 962
		p-value	0. 000HS	0. 000HS
	α Amylase	R		0. 962
		p-value		0. 000HS
Well control D. M.	Osteocalcin	R	0. 959	0. 757
		p-value	0. 000HS	0. 000HS
	α Amylase	R		0. 683
		p-value		0. 001HS
Poor control D. M.	Osteocalcin	R	0. 917	0. 891
		p-value	0. 000HS	0. 000HS
	α Amylase	R		0. 989
		p-value		0. 000HS

Discussion

Clinical Periodontal Parameters Analysis:

In diabetic patients, the decrease in the volume of saliva and buffering capacity in addition to the variation in bacterial flora. Altogether, these factors produce greater accumulation of plaque[14]. Furthermore, the harmful effects of advanced glycation end products and receptor for advanced glycation end products (AGEs-RAGEs)interactions in the periodontium of diabetic patients that comprise: increase vascular permeability, impaired wound healing and vascular variations contribute to further periodontal destruction [15]. The DM alters periodontitis by deregulating the immune and inflammatory responses in the periodontium, further cytokines are accumulated in the gingival tissues which will give rise to further periodontal destruction[16]. Also, DM effects diminished function of the neutrophils and hyperactivity of macrophages and monocytes which will result in further devastation of the periodontium, thus diabetic patients have greater prevalence and extent of periodontal pockets [17]. Poor glycemic control, with the associated rising in AGEs[18], these certainly play a significant role in the susceptibility of diabetic patients to infections and damaging PD. There were augmented BOP, augmented tooth mobility and more loss of attachment as the individuals with diabetes are twice as possible to exhibit attachment loss as non diabetic individuals [19].

Biochemical Parameters Analysis:

The Osteocalcin is one of the greatest plentiful matrix proteins found in bones and the only matrix protein synthesized absolutely there. Small osteocalcin fragments are found in regions of bone remodeling and are in fact degradation products of the bone matrix, that is released outside cells into the gingival crevicular fluid (GCF) and saliva after destruction of periodontal tissue during periodontitis[20]In addition, unusual blood glucose control was a risk factor for bone loss. Reduced bone mass and augmented fracture rate were communal in diabetes, that attributable to reduced late-stage differentiation of osteoblasts and a decrease in osteoblast function. Also, advanced glycated end products (AGEs) had been associated to abnormal development of osteoblasts, that believed to enhance bone resorption and induce apoptosis. As well as, enzymatic cross-linkage of collagen fibers provided strength to bone, nevertheless AGE-induced non-enzymatic collagen cross-linkage caused increasing fracture risk[21]. The OC is produced by osteoblasts and is widely accepted as a marker of bone osteoblastic activity. OC, incorporated into the bone matrix, is released into the circulation from the matrix during bone resorption and, hence, is considered a marker of bone turnover, rather than a specific marker of bone formation [22].

In diabetic, the augmented basement membrane permeability of salivary glands, in diabetics causes augmented passage of proteins into the saliva, furthermore the sialosis in the parotid gland in T2 diabetics, therefore most of α-amylase existence synthesized in this gland, could product in differences in the salivary composition[2]. α-amylase is a main lipopolysaccharide binding protein of A. a. and P. gingivalis and interferes with bacterial adherence and biofilm formation [23], moreover performs a direct inhibitory influence on the growing of *Neisseria gonorrhoea and*[24]. Accordingly, the rise concentration of salivary α-amylase recommends it to be an imperative defense molecule chiefly for the innate immunity in the oral cavity [25]. The increased protein levels due to the inflammatory process that activates the sympathetic system to augment the synthesis and secretion of some proteins, the salivary glands may respond to periodontal diseases by enhanced synthesis of some acinar proteins, thereby increasing the protecting potential of saliva against the diseases [25]. Diabetes is related with microvascular complications, which may affect the salivary excretions. The rise in salivary protein values can possibly be attributed to more microorganism activity or proteins of periodontal tissue source[26].

Correlations of Salivary OC, α-Am with TO levels with Clinical Parameters and with Each Other:

In diabetic patients with chronic periodontitis, when the values of clinical periodontal parameters increase, this mean increase in the severity of PD, more destruction of alveolar bone, increase in the OC concentration due to the damaged products of alveolar bone, stimulated the osteoclast activity by inhibiting Osteoprotegerin OPG expression and in balance bone turnover, furthermore augmented levels of α – Amylase, in which the former preferred multiplying of both aerobic and anaerobic bacteria in plaque, whereas the last well-organized potential energy sources and permit the attachment of pathogenic bacteria therefore change the conformation of plaque. Accordingly, diabetics is a risk of having periodontitis compared to non-diabetics, therefore adults with diabetic had significantly greater prevalence of severe periodontitis, hence the gingival inflammation and hemorrhage are intensified, more prevalence and extent of pockets with twice as possible a non diabetics to have attachment loss [2]. No study that addresses the correlation between OC, α-Am with TO levels in saliva was performed before. The present study is revealed that a highly significant positive correlation between of them. The possible explanation is coincides with the fact that, the increased inflammation in the study groups was associated with increased OC, α-Am with TO levels in saliva.

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