Biochemical analysis and periodontal health status in type 1 and type 2 diabetes (Comparative study)

Salah Mahdi Ibrahim, B.D.S. [(1)]
Leka'a M. Ibrahim, B.D.S, MSc. [(2)]

ABSTRACT

Background: Diabetes mellitus is a group of metabolic disorders with one common manifestation: hyperglycemia. This study aimed to evaluate the periodontal health status and cera reactive protein (C.R.P) in saliva in type1 and type 2 diabetic patients and compared with healthy subjects.

Material and method: Total samples composed of eighty participants, the samples were divided to study group (60 diabetic patients) which include type 1 and type 2 diabetic patients and control group (20 healthy subjects).They were non-smokers male patients of age range 25-55 years old. Periodontal health status was estimated by measuring plaque index (PL.I), Gingival index (G.I), Bleeding on probing (BOP), Probing pocket depth (PPD) and Clinical attachment level CAL).Five mls of un stimulated whole saliva was collected for estimation of C.R.P.

Result: The result showed that the mean C.R.P in saliva was higher in study group compared to control group.No significant difference in PL.I and significant difference in G.I, BOP, PPD and CAL between type 1 and type 2 of diabetes .There was weak correlation between clinical periodontal parameters and biochemical parameters.

Conclusion: salivary C.R.P may be involved in the interaction of periodontitis in type 1 and type 2 diabetic patients.

Keywords: Diabetic, C.R.P, periodontal disease, (J Bagh Coll Dentistry 2012; 24(sp. Issue 1):80-84).

INTRODUCTION

Diabetes mellitus (DM) is a disease in which levels of blood glucose, also called blood sugar, are above normal. People with diabetes have problems converting food to energy **(1).**

People develop diabetes because the pancreas does not make enough insulin or because the cells in the muscles, liver, and fat do not use insulin properly, or both. As a result, the amount of glucose in the blood increases while the cells are starved of energy [(1)].

The two main types of diabetes are called type 1 and type 2. A third form of diabetes is called gestational diabetes.

Glycosylated haemoglobin (HbA_{1C}) is used to monitor treatment in patients with diabetes mellitus. Measurements of glycated hemoglobin have commonly been used to monitor the glycemic control of persons already diagnosed with diabetes mellitus [(2)].

Individuals, microbial challenges may overcome host defaces, leading to microbial extension into the sub gingival epithelium. This leads to the development of a gingival pocket and heralds the facilitation in growth of Gram-negative bacterial species. It has been well established that idividuals with diabetes have an increased risk of developing periodontal disease compared with non-diabetics, and that they experience more severe and more rapid progression of the disease**(3).**

The prevalence of diabetes mellitus is more than twice as high in patient with periodontitis compared to healthy subjects. Periodontal disease may contribute to systemic inflammation, worsening insulin resistance and diabetes due to the generation of inflammatory cytokines [(4)].

Poorly controlled subjects with diabetes displayed more gingival bleeding sites compared with those of subjects with diabetes with good or moderate control [(5)].

In the presence of similar plaque levels, poorly controlled subjects with type 1 diabetes of long duration (e.g. mean of 16.5 years) displayed more severe attachment and alveolar bone loss [(6)].

The presence of similar plaque scores, young subjects with type 1 diabetes of long duration suffered from more severe gingival inflammation and periodontal tissue destruction compared with subjects with diabetes of short duration [(7)] or non-diabetic subjects[(8)] .

Subjects with type 2 diabetes with periodontal disease suggested that the prevalence of severe periodontal disease increased with decreasing glycaemic control [(9)].

C-reactive protein is one of the best know members of a group of acute –phase proteins, which increase their concentration during certain inflammatory disorders. It has widely been used as a bio-marker of inflammation in the body .In recent years C.R.P has received a lot of attention because of its apparent ties to cardiovascular disease ,and it has also been linked to a number of other disease ,including hypertension ,diabetes ,cancer and autoimmune disorders **(10).**

(1) Master Thesis, College of Dentistry, University of Baghdad.
(2) Professor, Department of Periodontics, College of Dentistry, University of Baghdad.

As elevated C.R.P values are always associated with pathological changes, the C.R.P assay provides useful information for diagnosis, therapy and monitoring of inflammatory process and associated disease [11].

Sick people admitted to a hospital had average salivary C.R.P levels 25 times higher than healthy people .salivary C.R.P may largely reflect local inflammation in the mouth, but some serum CRP can enter saliva through gingival tissue ,especially if periodontal disease is present[12] .

Ongoing research is investigating the possibility that salivary C.R.P can be used to monitor inflammation in other parts of the body [13].

Periodontal infection results in higher C.R.P in type 2 diabetes patient. This elevated inflammatory factor may exacerbate insulin resistance and increase the risk for great vessels complications of diabetes mellitus [14] .

The present study aimed to evaluate the periodontal health status in type 1 and type 2 diabetic patients and compared with healthy subjects. Also to evaluate the level of C.R.P in saliva in type1 and type 2 diabetic patients and compared with healthy subjects.

MATERIALS AND METHOD

Total samples composed of eighty participants, the samples were divided to study group (60 diabetic patient) and control group (20 healthy subjects).They were non-smokers male patients of age range 25-55 years old.

Study group sample included:

Group 1 (G1): Fifteen patients type 1 diabetes with good control, HbA1c less than 7.5%.

Group 2(G2): Fifteen patients type 1 diabetes with poor control, HbA1c more than 7.5%.

Group 3(G3): Fifteen patients type 2 diabetes with good control, HbA1c less than 7.5%.

Group 4(G4): Fifteen patients type 2 diabetes with poor control, HbA1c more than 7.5%.

The control group sample (G 5) included 20 healthy subjects without any history for any systemic disease.

Periodontal health status was estimated by measuring following clinical parameters:

Plaque index (PL.I)[15]

Gingival index (G.I)[16]

Bleeding on probing (BOP)

Probing pocket depth (PPD)

Clinical attachment loss (CAL)

Five to six mls of un-stimulated (resting) whole saliva was collected before the clinical examination. The collected saliva was centrifuged at 3000 r.p.m for 15 minutes, clear supernatant saliva kept frozen and store at -20^{o}C for 1 week until C.R.P determination by using avitex aso kit for semi-quantitative determination of C.R.P in saliva.

RESULTS

The result showed that the mean C.R.P in saliva was higher in study group compared to control group. It was highest in group 4 which was 17.454 mg/ml ±7.802 and lowest in group 5 which was 8.421 mg/ml ±3.464 as shown in table-1-

The means of plaque index were higher in group 2, 4 compared with group 1,3,5. It was 2.12± 0.655 in group 2 and 2.07±0.688 in group4, while in group 1, 3, 5 they were 1.45±0.529, 1.9±0.584 and 1.32±0.512 respectively (table -1-).

The mean of gingival index in group 2,3,4 were higher compared with group 1,5. It was 1.97 0.636 in group 2, 1.9±0.672 in group 3 and 1.87±0.748 in group 4 while in group 1 it was 1.27±0.562 and in group 5 the mean was 1.22±0.588 (table -1-) .

The comparison of PL.I, G.I and salivary CRP were highly significant for all groups as shown in table -2-

Descriptive statistics for bleeding on probing were described in table (3).it was clearly that the number of bleeding sites in type 1 and type 2 of diabetes were higher than healthy subjects

The comparison of bleeding on probing showed that there was highly significant difference for all groups as in table-4-

Table -5- showed the probing pocket depth was increase in scale 1, 2, 3 in group 1, 2, 3 compared with group 5 while scale 0 was increased in group 5 in compared to other groups.

The clinical attachment loss showed increase in scale 2, 3, 4 in group 1, 2, 3, 4 compared with group 5 while scale 1 increase in group 5 compared to other groups as described in table -6-

There was weak correlation between clinical periodontal parameters and biochemical parameters for all groups as shown in table-7-

DISCUSSION

The level of CRP in saliva was found to be significantly higher in diabetic groups (type1) and (type 2) than of control group, the level of CRP in saliva highly in groups with periodontitis and increase the percentage of ppd and attachment loss.

This was accepted and in agreement with loos et al 2000[4], who reported elevated CRP levels among those with periodontitis .

CRP in saliva detected as result of systemic inflammation result elsewhere in the body as well as systemic inflammation induce by periodontitis [17].

The level of CRP in saliva was found to be significantly higher in diabetic groups (type2) than diabetic (type 1).

It was in agreement with Aspriello and Piemontese in 2010[18].

No significant difference in PL.I between type 1 and type 2 of diabetes and there was asignificant difference between type 1 and control group and there was ahighly significant difference between type2 and control group.

This was in agreement with result of study done by Raghad [19] ,who found that there was a highly significant difference between type2 and healthy subjects ; Bjelland and Bray [20] who found a significant difference between uncontrol diabetic and non –diabetic.

And disagree with Khader [21] who found no significant difference between type 2 diabetic and control group ; Giovanni [22] who found no significant difference between type1 diabetic and control group .

Nevertheless, there are several indications from this analysis that diabetes is associated with poorer oral health. Among adults aged 35–57 years, several indicators of periodontal disease were elevated at least two-fold. This is an important age group, first because periodontal tissue destruction at multiple sites at this age may be indicative of a poor prognosis for subsequent tooth loss due to periodontal disease. It is also at this age that Type 2 diabetes frequently develops, yet remains undetected, and potentially poorly controlled. In contrast, the lack of effect seen in those aged under 35 may be due to them having Type-1 diabetes, which is more likely to be well-controlled than Type-2 diabetes. Diabetics showed evidence of more plaque and almost double the degree of gingival inflammation compared to non-diabetics.

The periodontal disease typically begins with reversible inflammation of the gingival tissues in response to dental plaque. In susceptible individuals, microbial challenges may overcome host defences, leading to microbial extension into the subgingival epithelium. This leads to the development of a gingival pocket and heralds the facilitation in growth of Gram-negative bacterial species. It has been well established that individuals with diabetes have an increased risk of developing periodontal disease compared with non-diabetics, and that they experience more severe and more rapid progression of the disease.

For G.I there was a significant difference between type 1 and type 2 diabetes, and no significant between type 1 and control group and comparison between type 2 and control group was significant .This was accepted by Giovani Salvi,2005[22] who found no significant difference between type1 diabetic and control group; Ragaad,2006[19] who found a significant difference between type 2 diabetics and healthy subjects .

But disagree by al-saidy in 1996[23], who found ahigher gingival index scores in diabetics than non diabetics; khader,2010[21] ,also found no significant difference between type2 diabetic and control group.

According to our study, metabolic control play an important role in analysis of the relationship between periodontal disease and diabetes mellitus.

The worsening effects of inflammation are also indicate a correlation between periodontal inflammation on diabetic balance and insulin resistance syndrome**[4].**

This evidence points to a vicious cycle in which diabetes and periodontitis exacerbate one another.periodontal treatment may improve diabetes control measured as a reduction in glycated hemoglobin.

The gingival bleeding was observed to increase as the level of metabolic control deteriorated[5] the reason for the increase bleeding in diabetic groups could be either due to inflammation or vascular changes in the gingival tissue ;this was in agreement with Ervasti et al.[5] who found that the inflammatory reactions are intensified during poor metabolic control ,as the same amounts of plaque induced more gingival bleeding in groups of diabetic compared to control subjects.

Significant diference was found between type 1 and type 2 diabetic .Type 1 had 5.9% of sites with probing depth $\geq$ 4mmn compare to 8.3% of sites in type 2 diabetics.

The increase pocket depth and root surface infection, bacterial impact and invasion of periodontal tissue, alveolar bone loss and local and systemic immune reaction all have negative effects on diabetes. the association between diabetes and periodontal disease may be due to numerous physiological phenomena seen in diabetes ,such as impaired (immune)resistance ,vascular change ,altered microflora,and abnormal collagen metabolism.this tend to support the higher incidence and severity of periodontitis in diabetic patients.there is direct causal or modifying relationship in which poor glycemic control results in more severe periodontitis[9] .

Increase clinical attachment loss was significantly higher in the type 1 and type 2 diabetic compare to control group this was in agreement with Thorstensson et al [24], who found that more attachment loss in long duration of diabetic compare to short duration diabetic and non-diabetic.

Periodontal pocketing together with loss of clinical attachment is indicative of periodontal bone loss that is not influenced by pseudo pocketing caused solely by gingival inflammation or overgrowth. Furthermore, when the percentage of sites with both pocketing and attachment loss is computed, yielding an extent score, comparisons can be made between groups in a way that adjusts for differences in the number of teeth measured. For all ages combined, the extent of combined pocketing and attachment loss was twice as large in diabetics compared to non-diabetics.

In general there was weak correlation between clinical and biochemical parameters but no reported to compare with it.

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Table 1: The mean and SD of PL.I, G.I and CRP in saliva for all groups

Groups	PL.I	G.I	CRP in saliva
G1	1.45 ± 0.529	1.27 ± 0.562	12.60 ± 7.720
G2	2.12 ± 0.655	1.97 ± 0.636	16.20 ± 7.509
G3	1.9 ± 0.584	1.9 ± 0.672	14.727 ± 6.214
G4	2.07 ± 0.688	1.87 ± 0.748	17.454± 7.802
G5	1.32 ± 0.512	1.22 ± 0.558	8.421± 3.464

Table 2: Comparison of PL.I, G.I and CRP in saliva for all groups

Groups	f- test			P- value			Sig		
	PL.I	G.I	CRP	PL.I	G.I	CRP	PL.I	G.I	CRP
G.1	-460.267	-457.524	-39.097	0.000	0.000	0.000	HS	HS	HS
G.2									
G.3									
G.4									
G.5									

Table 3: Number and percentage of BOP for all groups.

Score	G1		G2		G3		G4		G5	
	No	%	No	%	No	%	No	%	No	%
0	632	37.1	324	23.4	466	38.3	412	25	1024	71
1	1053	62.9	1046	76.6	748	61.7	1218	75	415	29

Table 4: Comparison of BOP foe all groups.

Groups	Chi-square	P- value	Sig
G.1	21.270	0.000	HS
G.2			
G.3			
G.4			
G.5			

Table 5: Number and percentage of PPD per site and Chi square among all groups.

Scale	G.1		G.2		G.3		G.4		G.5		Chi-square	P-value
	No.	%	No.	%	No.	%	No.	%	No.	%		
0	1600	95	1298	93	1148	94.4	1470	90	1399	98	31.952	S
1	40	2.5	43	3.2	48	3.7	106	7.1	24	1.6	39.677	S
2	28	2	31	2.6	12	1.4	42	3	11	0.8	11	S
3	8	0.5	8	1	6	0.5	12	0.9	5	0.3	34.131	S

Table 6: Number and percentage of CAL per site and Chi- square among all groups

scale	Group1		Group2		Group3		Group4		Group5		Chi-square	p-value
	No.	%	No.	%	No.	%	No.	%	No.	%		
1	122	65.5	84	51.8	220	40.9	180	44.5	76	68.4	52.834	S
2	22	18	38	22.2	74	33.6	134	33.2	22	19.8	50.061	HS
3	12	9.8	28	17.2	42	19	58	14.3	11	9.9	52.834	S
4	4	6.7	12	8.8	14	6.5	32	8.0	2	1.9	16.094	S

Table 7: Intra group correlation between clinical periodontal parameter and CRP in saliva for all groups

G.1	CRP	PL.I 0.234	G.I 0.023	BOP -0.161	PPD -0.031	CAL -0.067
G.2	CRP	PL.I 0.194	G.I 0.157	BOP 0.000	PPD 0.073	CAL 0.193
G.3	CRP	PL.I 0.116	G.I 0.302	BOP 0.243	PPD 0.226	CAL 0.217
G.4	CRP	PL.I 0.065	G.I 0.081	BOP -0.183	PPD -0.107	CAL 0.059
G.5	CRP	PL.I -0.237	G.I 0.322	BOP 0.560	PPD 0.388	CAL 0.571