VALUES OF SERUM C-REACTIVE PROTEIN AND SALIVARY IMMUNOGLOBULIN IGA IN DIABETIC PATIENTS: RELATION TO THE SEVERITY OF PERIODONTAL DISEASE

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ABSTRACT

Background: Altered immune response and improper neutrophils chemotaxis, phagocytosis, and adhesion might be the principal causative factor for increased susceptibility to periodontal pathogens and oral complications in diabetic patients. This study aimed to determine the relationship between C-reactive protein and salivary IgA with periodontal disease and the severity of dental caries with the glycemic control state of patients with diabetes mellitus.

Patients and Methods: This study was carried out on 91 subjects, 61 patients with diabetes mellitus, and 30 apparently healthy subjects (as a control group). The patient groups were regularly attended Duhok Diabetes Center, Duhok City, Kurdistan Region (Iraq) for diabetes management. The healthy controls were recruited from the staff and sub staff of Azadi Teaching Hospital. Fasting plasma glucose, HbA1c, and serum high sensitivity C-reactive protein (Hs-CRP) were measured. The whole saliva collection was performed by an unstimulated method for five minutes in a graduated test tube to recognize the salivary secretion rate and then stored at -20 ^{0}C for IgA estimation. Periodontal Index was used to determine the periodontal disease status. Each tooth was scored according to the condition of the surrounding tissues.

Results: Significantly higher Hs-CRP (9.2 vs 3.3 µg/ml), fasting plasma glucose (215.4 vs 98.9 mg/dl) and HbA1c (8.5 vs 4.9 %), (P < 0.001 for all parameters, together with lower hemoglobin (13.6 vs 14.5 gm/dl, P=0.03) levels were found in diabetic patients compared to control group, A significantly higher mean salivary IgA level in diabetic patients compared to controls (312.4 vs 177.3 mg/dl, P<0.001), associated with a significant high periodontal index (1.68 vs 0.81, P = 0.003).

Conclusion: Elevated serum Hs-CRP and salivary IgA in patients with diabetes mellitus as inflammatory response sequences raise inflammation potential in the periodontium. Further, the results confirm that periodontal index was associated with poor glycemic control.

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Keywords: Hs-CRP, IgA, Periodontal disease, Periodontal index

Epidemiological data support microvascular changes in diabetes mellitus (DM)[1]. The microvascular changes lead to reduced tissue blood flow, decreased oxygen diffusion, and a better environment for the growth of anaerobic bacteria and susceptibility to infection[2]. The possible association between oral and systemic health has been highlighted; evidence suggested a bi-directional relationship between systemic and periodontal disease. Oral manifestations in DM include high dental caries, xerostomia, glossitis, oral candidiasis, burning mouth, and gingival and periodontal disease[3,4]. The altered immune response and improper neutrophils

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chemotaxis, phagocytosis, and adhesion2 might be the principal causative factor for increased susceptibility to periodontal pathogens and oral complications[5]. Reduction in the quantity of salivary secretion or changes in its properties is responsible for oral and dental problems[6]. Salivary immunoglobulin A (s-IgA) antibodies could help oral immunity by preventing microbial adherence, neutralizing enzymes, toxins, and viruses. Studies have demonstrated a lower incidence of dental caries as a result of high s-IgA concentration, and low levels have been presented as a risk factor for upper respiratory infection, periodontal disease, and dental caries[7]. On the other hand, numerous studies in the last decade have shown that CRP is an independent predictor of risk for periodontal disease. This work was designed to determine the relationship between C-reactive protein, salivary IgA, and glycemic control with the severity of periodontal disease in diabetic patients.

PATIENTS AND METHODS:

This cross-sectional study was conducted at the Department of Physiology, College of Medicine, University of Duhok and Azadi Teaching Hospital in Duhok city, sixty-one patients with diabetes mellitus (45 with type 2 diabetes and 16 with type 1 diabetes) attended Duhok Diabetes Center, were selected randomly, (35) males and (26) females with ages ranged between 20-55 years. The healthy controls were recruited from the staff and sub-staff of the Azadi Teaching Hospital. Their age and sex-matched with the diabetic group; this group consisted of (19) males and (11) females and with ages ranged between 24- 51 years. All participants were informed about the study, and written consents were obtained before the study commenced. The study was approved by the local ethics committee of the Directorate of Health-Duhok. Exclusion criteria are patients with recent acute illness or upper respiratory tract infection in the past one week, history of chronic liver or renal disease, and those taking antibiotics or who underwent periodontal therapy one month before the study[7], pregnant females, and alcoholics.

Data collection: all subjects completed a pre-tested questionnaire, which included anthropometric data and a diabetes record. Body mass index and duration of the diabetes were recorded. History was taken to select healthy subjects (controls) and to exclude those with a known personal or family history of diabetes mellitus or the presence of any of the mentioned abnormalities (exclusion criteria).

HbA1c% was estimated using a commercial kit (StanbioGlycohemo-globin Pre-fil procedure no. P350), a qualitative colorimetric determination of glycol-hemoglobin in whole blood. Estimation of Serum Hs-CRP level using AccuBind Hs-CRP ELISA kit, product code: 3125-300 (Netherlands), was used for quantitative estimation of serum Hs-CRP. The reaction between various CRP antibodies and native CRP forms a sandwich complex that binds with streptavidin-coated to the well. After equilibrium is attained, the antibody-bound fraction is separated by decantation or aspiration. The enzyme activity presents on the surface of the well quantitated by reaction with a suitable substrate to produce color that is directly proportional to the native antigen concentration. A dose response curve was used to ascertain the concentrations of CRP in the samples. The

quantitative determination of salivary IgA was measured by turbidimetry method using Vital Diagnostics IgA Turbidimetry kit (ACCC 16-010). The reference range provided by the manufacturer (70–400 mg/dl).

Assessment of the HbA1c, CRP, and IgA

The Assessment HbA1c (%) at the universal diagnostic decision of the cut-off value of (48mmol/mol; 6.5%) was considered:

Normal= (< 5.7 %) Prediabetes= (5.7%-6.4%) Diabetes= (≥ 6.5%)

And for glycemic control HbA1c (%) in diabetic patients was considered: Good control (< 6.5 %) Fair control (> 6.5% – < 7.5%) Poor control (> 7.5 %)[8].

The assessment of CRP and IgA was done by using the calculated cut-off value formula:

Cut off value = mean + 3 SD[9]. The clinical cut-off value for Hs.CRP = 5.1 (µg/ml) and for salivary IgA = 211.3 mg/dl. The result of higher than 5.1 (µg/ml) for Hs.CRP and 211.3 mg/dl for IgA provided prognostic information as a risk values for periodontal diseases.

Salivary, Oral and Dental Assessment

- Assessment of Salivary Secretion Rate (SSR) was determined by measuring the amount of unstimulated saliva produced (collected) in a given period of time[10].

- The Salivary Flow Index (SFI) for unstimulated saliva was used, and subjects were classified into 3 categories[11]: Normal: 0.25-0.35 ml/min., Low: 0.1-0.24 ml/min., Very low (Hyposalivation): less than 0.1 ml/min.

- Assessment of Periodontal Index (PI) or Score: Periodontal Index determines the periodontal disease status. Each tooth is scored according to the condition of the surrounding tissues.

On examination, each tooth is assigned a score (from zero to eight) using the following criteria[12].

0: Negative. Neither overt inflammation nor loss of function caused by the destruction of supporting tissue is noted.

1: Mild Gingivitis: overt inflammation in the free gingiva is present, does not circumscribe the tooth.

2: Gingivitis: Inflammation surrounds the tooth; there is no apparent break in the epithelial attachment.

6: Gingivitis with pocket formation. The epithelial attachment of the gum to the tooth is broken. There is no interference with normal function. The tooth is not loose or drifting.

8: Advanced destruction with loss of function. The tooth may be loose or drifting. It may sound dull on percussion and maybe depressible in the socket. The scores for each tooth are added, and the total divided by the number of teeth examined. Scores can be interpreted as follows:

0.0-0.2: Clinically normal supportive tissues.

0.3-0.9: Simple gingivitis.

1.0-1.9: Beginning destructive periodontal disease.

2.0-4.9: Established destructive periodontal disease.

5.0-8.0: Terminal periodontal disease.

- Assessment of Decayed, Missed, and Filled Teeth (DMFT) Index or Score

To arrive at a DMFT score for an individual subject's mouth, three values must be determined the number of teeth with carious lesions, the number of extracted teeth, and the number of teeth with fillings

or crowns[13,14]. Participants were divided into three groups according to their DMFT score:

Group A: DMFT score 0-6
Group B: DMFT score 7-12
Group C: DMFT score13 and more.

STATISTICAL ANALYSES

All data were analyzed using the Statistical Package for Social Science (SPSS) version 22.0; independent *t*-test and one-way ANOVA test were used to examine statistical differences between groups.

RESULTS

Table 1, shows the baseline characteristics of the studied individuals. No significant differences were found with respect to age and BMI between the diabetic and healthy control groups. Significantly higher fasting plasma glucose, Hs-CRP, and HbA1c levels were observed in diabetic patients compared to healthy controls (P=0.010 for all parameters). Regarding salivary IgA and periodontal index, a significantly higher mean IgA and the periodontal index was found in the diabetic group compared to the healthy control group (*P*<0.001, *P*=0.003, respectively). No significant differences were found with respect to salivary secretion rate and DMFT score between the two groups A higher periodontal index (1.86 vs. 1.19) and DMFT score (9.0 vs. 6.6) was observed among patients with type 2 DM compared to patients with type I DM, but the difference was not significant. No significant differences were found with respect to Hs-CRP, salivary secretion rate, and salivary IgA levels between both types of diabetes.

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Table 1: Baseline characteristics of diabetic patients and healthy controls

Characteristics	Control Group Mean ± SE, N=30	DM Patient Group Mean ± SE, N=61	*P*-value
Age (years)	38.9 ± 1.5	40.4 ± 1.3	NS*
Body Mass Index (kg/m2)	26.7 ± 0.74	28.5 ± 0.72	NS
Serum Hs-C Reactive Protein (µg/ml)	3.3 ± 0.6	9.2 ± 1.2	<0.001
Fasting blood sugar (mg/dl)	98.9 ± 1.4	215.4 ± 10.7	< 0.001
HbA1c (%)	4.9 ± 0.04	8.5 ± 0.3	<0.001
Hemoglobin (gm/dl)	14.5 ± 0.3	13.6 ± 0.2	0.03
Salivary secretion rate (ml/min.)	0.34 ± 0.028	0.29 ± 0.023	NS
Salivary IgA (mg/dl)	177.3 ± 11.3	312.4 ± 15.5	< 0.001
Periodontal index	0.81 ± 0.13	1.68 ± 0.2	0.003
DMFT score	7.73 ± 0.61	8.36 ± 0.58	NS

*** NS= non-significant**

The relationship between Hs-CRP, salivary IgA, and periodontal parameters with glycemic control in people with diabetes and healthy controls are presented in table 2. As shown, diabetic patients with poor glycemic control had significantly higher periodontal index than that in those with good glycemic control (*P*=0.003)

Table 2: Comparison of Selected Parameters According to Glycemic Control State in Diabetic Patients

Parameters	Glycemic Control State			*P*-value
	Good Control N = 12	Fair Control N = 10	Poor control N = 39	ANOVA
Serum Hs-C Reactive Protein (µg/ml)	7.7 ± 2.6	8.7 ± 3.6	9.8 ± 1.5	NS
Salivary secretion rate (ml/min)	0.29 ± 0.06	0.33 ± 0.05	0.27 ± 0.03	NS
Salivary IgA (mg/dl)	313.6 ± 25.9	272.8 ± 20.6	322.1 ± 22.3	NS
Periodontal index	1.02 ± 0.21	1.1 ± 0.28	2.03 ± 0.28	0.04
Poor control vs Fair control:			P = 0.035	
Poor control vs Good control:			P = 0.003	
DMFT score	8.83 ± 1.1	10 ± 1.0	7.8 ± 0.8	NS

NS: Non-significant

To determine which of the selected or periodontal parameters was significantly associated with periodontal index level in diabetic patients, we performed ANOVA analysis between the periodontal index and Hs-CRP, salivary secretion rate, salivary IgA, and DFMT score. The results are presented in table 3. As shown by ANOVA analysis, salivary secretion rate was significantly higher in the group with low periodontal index (0-0.2) compared with those with periodontal index 2 or more (*P*=0.032).

Table 3: Comparison of Selected Parameters According to Periodontal Index in Diabetic Patients

Parameters	Groups of Periodontal Index				*P*-value
	A (0–0.2) N = 9	B (0.3–0.9) N = 12	C (1–1.9) N = 22	D (≥ 2) N = 18	ANOVA
Serum Hs-CRP (µg/ml)	9.8 ± 1.8	8.7 ± 2.3	9.2 ± 2.2	9.4 ± 2.5	NS
Salivary secretion rate (ml/min.)	0.39 ± 0.06	0.28 ± 0.06	0.29 ± 0.03	0.22 ± 0.03	0.032
Group A vs Group D,				P = 0.013	
Salivary IgA (mg/dl)	286.1 ± 22.2	318.9 ± 26.6	320.6 ± 28.9	311 ± 34.3	NS
DMFT score	7.3 ± 1.5	9.3 ± 1.2	7.7 ± 0.9	9.1 ± 1.3	NS

NS: Non-significant

Diabetic patients were divided into three groups according to their DMFT score (A, B, and C). All variables were further analyzed in ANOVA analyses; only salivary IgA level was significantly different between these groups (*P* = 0.039)

as shown in table 4, and it was significantly higher in group C (DMFT score 13 and more) compared to group A (DMFT score 0–6).

Table 4: Comparison of Selected Parameters According to DMFT Score in Diabetic patients

Parameters	Group of DMFT Score			*P*-value
	A (0 – 6) N = 25	B (7 – 12) N = 25	C (≥ 13) N = 11	ANOVA
Serum Hs-C Reactive Protein (µg/ml)	7.6 ± 1.7	10.5 ± 2.1	10.0 ± 3.2	NS
Salivary secretion rate (ml/min.)	0.25 ± 0.03	0.29 ± 0.03	0.39 ± 0.06	NS
Salivary IgA (mg/dl)	274.5 ± 17.8	324.7 ± 27.1	370.5 ± 39.7	0.039
Group A vs Group C		P = 0.016		
Periodontal index	1.76 ± 0.25	1.5 ± 0.3	2.15 ± 0.65	NS
DMFT score	4.3 ± 0.37	9.3 ± 0.33	15.5 ± 0.87	<0.001

NS: Non-significant

DISCUSSION:

This study showed significantly higher Hs-CRP and salivary IgA levels in diabetic patients compared with levels in healthy controls. The results confirm a relationship between the periodontal index and glycemic control, as the mean periodontal index in diabetic patients with poor glycemic control was about 2 times higher than that in the good control group, as well as between DMFT and salivary IgA level., as the mean DMFT score was about 3 times in participants with severe dental carries than that with mild dental carries. It has been reported that inflammatory processes are indicated by high Hs-CRP, but data concerning the relation of Hs-CRP levels with the severity of periodontal disease in diabetic patients are scarce. General categories of risk factors associated with the development of periodontitis include genetic, environmental (e.g., tobacco use), and acquired risk factors, e.g., DM and cardiovascular diseases. The elevation in Hs-CRP levels in diabetic patients might be attributed to the enhanced inflammatory process and its complications rather than the severity of the periodontal index. Moreover, CRP is not synthesized locally by the periodontal tissues because CRP-mRNA has not been isolated from periodontal tissue[15]. In patients with DM who are susceptible to periodontitis, the above inflammatory process will eventually extend apically and laterally to involve deeper connective tissues and alveolar bone. The possible explanation is that Hs-CRP and their positive correlation reflect activation of the immune system. In this study, the periodontal index was significantly higher in diabetic patients, with an increase of about two-fold compared with the control group. Many studies have found a higher prevalence of the periodontal disease among diabetic patients than among healthy controls[4,11]. Large epidemiological studies have shown that diabetes increases the risk of alveolar bone loss approximately three-fold compared with non-diabetic individuals. The mechanisms by which diabetes influences the periodontium are similar to the pathophysiology of classic

microvascular and macrovascular complications. The present study showed a significantly higher periodontal index in people with diabetes with poor glycemic control compared to those with good and fair control; this finding is consistent with previously reported data[4,8]. Moreover, it has been shown that periodontal disease and DM have a two-way relationship; periodontal disease may be a risk factor for the development of type2 DM 4: additionally, diabetic patients with the periodontal infection have worse glycemic control compared with diabetics subjects without periodontal disease[15]. Salivary Secretion Rate (SSR) in both studied groups was not significantly different, and in both groups, SSR were within normal reference values; the average value for SSR of whole saliva in healthy individuals is about 0.3 ml/min[16]. In this study, subjects spit out the saliva into the test tube once a minute, which may have influenced the flow rate of unstimulated saliva. Panchbhai et al. [17] and Al-Zahawi *et al.* [18] showed that poorly controlled type II DM has no influence on salivary output; however, Harrison and Bowen[19] concluded that patients with poorly controlled type I DM had the significantly lower flow of the whole saliva compared to well-controlled and healthy subjects. Al-Maroof RH (2010) in line with our finding, he was found that the high concentration of the total proteins level in diabetic patient could be related to the increase in IgA and amylase levels in IDDM, or may be due to the decrease in the flow rate since, total proteins has an inverse relationship with salivary flow rate. [20]

Results of the present study revealed a highly significant increase in salivary IgA in diabetic patients. This finding is in agreement with that of Bhuyan *et al.* [21]. Sardari F *et al.* [22] found that diabetic patients had higher salivary IgA levels compared to non-diabetic individuals. The possible mechanism responsible for increased salivary IgA was sought to the increased severity of periodontal disease in diabetic patients (evidenced by increased periodontal index). In periodontitis, the subgingival plaque accumulation results in an acute inflammatory response with infiltration of lymphocytes and plasma cells and a possible outpouring of immunoglobulins. Data concerning the relation of salivary IgA with DMFT score and dental caries in diabetics, particularly in type II DM, are limited. It has been reported that the bacteria S. mutants is the primary causative agent of dental caries in human beings. The duration of DM has no influence on salivary IgA level, while others found higher salivary IgA in long-duration patients with type I DM [21, 23] and they attributed this increase to decreased SSR due to presence of negative correlation between SSR and IgA level; however, in this study SSR was not changed significantly in diabetic patients; also no correlation was found between SSR and salivary IgA levels.

CONCLUSION

The results confirm that Serum Hs-CRP and salivary IgA levels are significantly elevated in diabetic patients. These findings may have attributed to the activation of the immune system and inflammation with excess production of pro-inflammatory mediators. As expected, higher levels of the periodontal index are associated with poor glycemic control. Therefore, a primary

prevention model of reducing blood glucose and inflammation by means of proper oral hygiene and glycemic control may be beneficial in diabetic patients.

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پوختە

پەیوەندیا چەوانیا زاڵکرنێ لسەر شەکرا خوینێ، نەخوشیا دەوروبەرێن ددانی و قوربوونا ددانا دگەل گلوبیولینێ بەرگرییێ خوزییێ أ لجەم نەخوشێن شەکرێ

پێشەکی

نەخوشیا شەکری ڤاکتەرەکێ هاریکارە بو تێکچونا تەندروستیا دەڤی، گرێدان دناڤبەرا نەخوشیا شەکرێ و گوهرینێن نەخوشیان دناڤ دەڤیدا یا هاتیە دیارکرن دراپورتێن گرێدایی نوژداریا ددانافە، هاتیە بەر چاڤکرن کو دگەل نەخوشیا شەکری نەخوشێن دەوروبەرێن ددانی زێدە دبن و دئێتە هژمارتن وەك ئالوزیا شەشێ بو نەخوشیا شەکرێ.

بۆ شێوەیەکێ گشتی کارێن خوزییێ ئەگەرا هەی لسەر تەندرستیا دەڤی، گلوبیولینێ بەرگرییێ خوزییێ أ کار دکەت وەك هێلا ئێکێ بو بەرگریکرنێ ژ میکروبێن نەخوشیا ئەوێن داگیرکرنێ و تێپەربوونا پانیا دکەن و بەلکی پاراستنێ ژ نەخوشیێن دەوروبەرێن ددانی بکەت. ئەڤ ڤەکولینە هاتە نەخشەکرن ژبەر گرنگیا گلوبیولینێ بەرگرییێ خوزییێ أ وەك فاکتەرەکێ بەرەڤانییێ ژ مێڤاندارى و هەروەسا کێماسیا زانینا لسەر رولێ گلوبیولینێ بەرگرییێ خوزییێ أ دژی نەخوشیێن دەوروبەرێ ددانا لجەم نەخوشێن شەکرێ.

مەبەست: ڤەکولین لسەر رێژا گلوبیولینێ بەرگرییێ خوزییێ أ دگەل نەخوشیێن دەوروبەرێ ددانی، قوربوونا ددانا و چەوانیا زاڵکرنێ لسەر شەکرا خوینێ لجەم نەخوشێن شەکرێ.

کەس وڕێکێن ڤەکولینێ

ئەم ڤەکولینا بەلاڤ ەاتە ئەنجامدان ب پشکداریا 61 نەخوشێن شەکرێ و 31 کە سێن ساخلەم. هەمی کە سان بە شداری بوون لدویڤ هندەك بنەمایێن دیارکری. هەمی بە شداربوونا فورمێن تایبەت پرکرن کو تێدا پیڤەرێن رێژا مروڤی و تومارا نەخوشیا شەکرێ هەبوون. رێژا چێبونا خوزییێ هاتە تومارکرن، پیڤەرێن نەخوشیا دەوروبەرێن ددانی و قوربوونا ددانا (قوربون، هەلکێ شان و داگرتن)، نمونێن خوزییێ و خوینێ هاتنە وەرگرتن، بەشەك ژ خوینێ هاتە دانان دناڤ تیوبێن تایبەت. ب نەهێلانا هشکبونا خوینێ و هاتە هنارتن بو پیڤانا بیڤەرێن خوینێ و هیموگلوبین گرێدایی ب شەکرێ ڤە. بەشێ دووێ یێ خوینێ و خوزییێ هاتنە سنترفیوج کرن لژێر پلا گەرماتیێ 4 پلە. پا شی سیروم و خوزیا بۆ سەرڤە یا پاقژ هاتنە هەلگرتن لژێر پلا گەرماتیێ 28 پلە دبن سفرێ هەتا دەمێ شلوڤەکرنێ بو پیڤەرێن بایوکیمیاوی.

پیڤەرێن بایوکیمیاوی ئەوێن هاتینە وەرگرتن: 1- تیراتیا گلوبیولینێ بەرگرییێ خوزییێ أ 2- تیراتیا HS – CRO د سیرومیدا.
3- تیراتیا شەکرا خوینێ، HbA1c (٪) + الهیموکلوبین الکلایکوزی 4- تیراتیا چەوراتیا خوینێ.

ئەنجام

ئەنجاما دیارکر بلندبونەکا بەر چاڤ دناڤەندا تیراتیا پروتینێ هەولدایی د سیرومیدا و ژمارا خروکێن سپی بێن خوینێ لجەم نەخوشێن شەکرێ بەراورد دگەل کە سێن ساخلەم (هەمان ژمارە) لدویڤ ئێکدا. ژمارا خروکێن سپی لجەم نەخوشێن کونترول لسەر شەکرا خوینێ نەهاتیە کرن بلندتر بو ژوان نەخوشێن کونترول لسەر شەکرا خوینێ کرى (9.2 مایکروغرام/ مل و 8.2 × 10 3/ملم3 بەراورد بـ 3.3 مایکروغرام/ مل و 6.9 × 10 3/ملم3) لدویڤ ئێکدا.

پیڤەرێ نەخوشیا دەوروبەرێن ددانی لجەم نەخوشێن شەکرێ بلندتر بو (1.68) بەراورد دگەل کە سێن ساخلەم (0.81) (P=0.003)، هەروەسا بلندتر بو لجەم نەخوشێن شەکرێ ئەوێن کونترول لسەر نەخوشیا شەکرێ نەهاتیەکرن بەراورد دگەل ئەوێن کونترولەکا پەسەند یان باش لسەر شەکرا خوینێ کرى (هەمان ژمارە) لدویڤ ئێکدا.

جودایێن بەر چاڤ نەبوون د پیڤەرێ قوربوونا ددانا (قوربوون، هەلکێ شان و داگرتن) و رێژا چێبونا خوزییێ دناڤبەرا نەخوشێن شەکرێ و کە سێن ساخلەم. بەلێ ناڤەندا چێبونا خوزییێ کێمتر بو لجەم نەخوشێن شەکرێ ئەوێن پیڤەرێ نەخوشیا دەوروبەرێن ددانا بەرزهەین (هەمان ژمارە) هەروەسا پەیوەندیەکا نێگەتیڤ هاتە دیارکرن لناڤبەرا رێژا چێبونا خوزییێ و ناڤەندا پیڤەرێ نەخوشیا دەوروبەرێن ددانا (2.03 بەراورد بـ 1.1 و 1.02) (P=0.003 , P= 0.035) لدویڤ ئێك بلندبوونا بەر چاڤ هاتە دیتن ل ناڤەندا گلوبیولینێ بەرگرییێ خوزییێ أ لجەم نەخوشێن شەکرێ بەراورد دگەل کە سێن ساخلەم (P=0.032). ناڤەندا گلوبیولینێ بەرگرییێ خوزییێ أ بەرچاڤ بلندتر بو لجەم نەخوشێن شەکرێ ئەوێن پیڤەرێ قوربوونا ددانا بلند هەین (بلندتر ژ 13) بەراورد دگەل ئەڤێن پیڤەرێ قوربوونا کێم هەین (0

6) (وترکیز 370.5 بەراورد بـ 274.5 ملغم/ 100 مل). لدیف ئێکدا ژی، رەگەز، جگارەکێشان، کونترول لسەر شەکرا خوینێ، دەمێ دریژیا نەخوشیێ، جورە و ئالوزیێن شەکرێ هیچ ئاکام نەبوون لسەر ریژا ناڤەندا چێبوونا خوزییێ و گلوبیولینێ بەرگرییێ خوزییێ أ.

دەرئەنجام

تیراتیا گلوبیولینێ بەرگریێ خوزیێ أ و پیڤەرێ نەخو شیا دەوروبەرێن ددانا لجەم نەخو شێن شەکرێ یێن بلندبوون. تیراتیا گلوبیولینێ بەرگریێ خوزییێ أ بلندتر بوو لجەم نەخوشێن شەکرێ ئەوێن ریژەکا بلند هەین یا پیڤەرێ قوربوونا ددانا. کونترول لسەر شەکرا خوینێ و پیڤەرێ نەخوشیا دەوروبەرێن ددانا ئاکا نەبوون لسەر تیراتیا گلوبیولینێ بەرگرییێ خوزییێ أ.

الخلاصة

مستويات البروتين التفاعلي (ج) في مصل الدم والكلوبيولين المناعي اللعابي أ ممكن أن يعكسان شدة أمراضية ما حول الأسنان لدى مرضى داء السكر

خلفية البحث

يعتبرداءالسكري كعامل خطر مضر بصحة الفم, كانت العلاقة بين مرض السكري والتغيرات المرضية في تجويف الفم موضوع العديد من التقارير في أبحاث طب ا لأسنان . اقترحت الأدلة الطبية أن مرض السكري يترافق مع زيادة انتشار أمراض حول الأسنان وتعتبرمن المضاعفات الشائعة والسادسة في الترتيب لمرض السكري. يؤدي العاب خدمات ووظائف اللعاب تؤثر في صحة الفم بشكل عام , الكلوبيولين اللعابي المناعي نوع A يمكن أن يكون بمثابة خط أول للدفاع ضد الجراثيم التي تستعمر وتغزو الأسطح المخاطية، وربما تكون الواقية من أمراض حول الأسنان. وفقا لذلك ،تم تصميم هذه الدراسة استنادا إلى أهمية الكلوبيولين المناعي نوع أ في اللعاب كعامل دفاع عن جسم المضيف فضلا عن غياب الفهم الكامل لدور الكلوبيولين المناعي نوع أ في التسبب في أمراض حول الأسنان في مرضى السكري. تهدف الدراسة للتحقق بالعلاقة بين مستويات الكلوبيولين المناعي نوع أ في اللعاب مع أمراض حول السن ونخر الأسنان مع مدى السيطرة على مستويات سكر الدم في مرضى داء السكري.

الاشخاص وطرق البحث

شارك في هذه الدراسة المقطعية 61 مريضاً يعانون من داء السكري و30 من الأصحاء ظاهريا والمتجانسين مع عينة البحث في العمر والجنس كمجموعة ضابطة للمقارنة. وخضع اختيار جميع المشاركين في الدراسة لمعايير البحث الادراجية .جميع المشتركين أكملوا استمارة الاستبيان التى شملت معطيات القياسات البشرية والتي تضمنت تجارب ماقبل التنفيذ،كذلك سجل لمرضى السكري. بعد الصوم ليلا تم قياس معدل افراز اللعاب بدون تحفيزومؤشر حول السن ومؤشر التسوس (نخر، قلع وحشوة الاسنان). تم اخذ عينات من اللعاب والدم الوريدي. تم وضع جزء من الدم في انابيب اختبار تحتوي على مانع تخثر و ارسلت لاحتساب صورة الدم الشاملة و قياس نسبة الهيموكلوبين المرتبط بالدم. الكمية الباقية من الدم وعينات اللعاب تم فصلها بواسطة الطرد المركزي وعند درجة حرارة 4 درجة مئوية. تم اخذ اللعاب الصافي من الشوائب ومصل الدم و تم خزنها في درجة حرارة 28 تحت الصفر لحين اجراء التحليلات الكيموحياتية.

تم اجراء التحليلات الكيموحياتية التالية:

مستوى الكلوبيولين المناعي أ في اللعاب

مستوى البروتين التفاعلي ج عالي الحساسية (C-Reactive Protein) في المصل.

مستوى السكر في الدم في حالة الصيام.

مستوى السكر التراكمي (%) HbA1c الهيموكلوبين الكلايكوزي

مستوى دهون الدم.

النتائج والمناقشة

اظهرت النتائج زيادة معنوية في معدل مستوى البروتين التفاعلي ج في المصل لدى مرضى السكري مقارنة بالاشخاص الاصحاء (9.2 مايكروغرام \ مل دم مقارنة ب 3.3 مايكروغرام \ مل) كذلك وجدنا زيادة معنوية لمؤشرامراض حول السن لدى مرضى السكري (1.68) مقارنة بالاصحاء (P=0.0030.81) (وكذلك كان اعلى لدى المرضى الذين يعانون من عدم السيطرة على مستوى السكر مقارنة بالمرضى ذو السيطرة المقبولة او الجيدة (2,03 مقارنة بـ1.1 و1.02) P= 0.003 (P= 0.035) بالتتابع.

لم يلاحظ وجود فروقات احصائية في مؤشر التسوس (نخر، قلع وحشوة الاسنان) ومتوسط إفراز اللعاب بين مرضى السكري ومجموعة السيطرة. بينما كان متوسط افراز اللعاب معنويا اقل في مرضى السكري الذين لديهم مؤشر حول السن عالي (P= 0.032) ووجد ارتباط سلبي معنوي بين متوسط إفراز اللعاب و مؤشر حول السن (P=0.03, r=-0.278). لوحظ زيادة معنوية في متوسط الكلوبيولين المناعي اللعابي أ لدى مرضى السكري مقارنة بالاصحاء (312.4 مقارنة بـ177.3 ملغم/

100مل) بالتتابع. متوسط الكلوبيولين المناعي اللعابي أ كان أعلى معنوياً في مرضى السكري الذين لديهم مؤشر تسوس الأسنان عالي (أكثر من 13 نقطة) مقارنة مع الذين لديهم مؤشر تسوس قليل (0–6) نقطة وتركيز 370.5 مقارنة بـ274.5 ملغم/ 100 مل (P= 0.016).

الاستنتاج

ازداد متوسط الكلوبيولين المناعي اللعابي أ و مؤشر حول السن لدى مرضى السكري. إن متوسط الكلوبيولين المناعي اللعابي أ وجد أعلى عند مرضى السكري الذين لديهم مؤشر تسوس الآسنان, بينما متوسط الكلوبيولين المناعي اللعابي A لم يتأثر بمدى السيطرة على مستوى السكر في الدم و مؤشر حول السن لدى مرضى السكري.