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# LIPID PROFILE, UREA, AND URIC ACID SERUM LEVELS IN SUBJECTS WITH DIABETES AND\OR PERIODONTITIS COMPARED TO CONTROLS.

Abdulsalam Tawfeeq Salih Alsamarai\*, Amina Hamed Ahmed, Wesam Suhail Najem,
Abdulghani Mohamed Alsamarai

Tikrit University, Tikrit, Iraq.

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\*Corresponding Author Abdulsalam Tawfeeq Salih Alsamarai Tikrit University, Tikrit, Iraq.

#### **ABSTRACT**

**Background:** Diabetes mellitus and periodontitis are two conditions that characterized with metabolic, inflammatory and immunologic changes that may lead to serious complications, including infection and cardiovascular diseases. **Aim:** To determine the predictive value of lipid profile, glucose, urea and uric acid in these two conditions alone or in combination in comparison with control. **Study Design:** Prospective case control study. **Materials and Methods:** A total of 226 subjects recruited from Endocrinology Unit, Samara General Hospital during the period from 1<sup>st</sup> October 2014 to end of October

2015. Their age ranged from 32 to 75 years. The study population depending on their clinical and laboratory findings divided in to **non-** diabetic without periodontitis as control, diabetic, periodontitis without diabetes, and diabetic with periodontitis groups. Blood samples collected from subjects and serum isolated and tested for lipid profile, blood urea and sugar, and serum uric acid using enzymatic methods. **Results:** All tested markers with the exception of HDL were significantly higher in diabetic group and those with periodontitis and diabetic than in control, while HDL was significantly lower in both groups than in control. There was no significant differences in all markers between periodontitis and control groups; in between periodontitis with diabetic and diabetic group. However, there was significant higher value in those with diabetes and periodontitis as compared to subjects with periodontics. HDL was inversely correlated to cholesterol, triglyceride and LDL in control group, while this pattern of correlation not demonstrated in the 3 patients groups, indicating abnormal metabolic changes. **Conclusion:** Both diabetes and periodontitis as present alone or in combination were associated with lipid profile, blood urea, and uric acid disturbances. This finding clarify

the importance of monitoring both conditions for the achievement of good prognosis and prevent complications.

#### INTRODUCTION

Diabetes mellitus is a chronic metabolic disease with sequences of accumulative complications. To date, all the treatment approaches are palliative and all the therapies are to control the glycaemia and prevent complications<sup>[1]</sup>. Periodontal disease is an inflammatory and multifactorial disease that affect the connective tissue attachment and supporting bone around the teeth whose initiation and progression depends on the presence of virulent microorganisms capable of causing disease<sup>[2,3]</sup>. Periodontal disease is etiology is still without clear cut and this may lead to delay in diagnosis<sup>[3]</sup>. Variable biomarkers are used for diagnosis and monitoring of diabetes and periodontitis.<sup>[4,5]</sup>

Both diabetes and periodontitis may present together and the patients are prone to develop more and severe complications and both conditions may interact in a bidirectional effects. Thus this study was conducted to determine the predictive value of lipid profile, glucose, urea and uric acid in these two conditions alone or in combination in comparison with control.

#### MATERIALS AND METHODS

**Study design**: Prospective case control study.

#### **Study population**

A total of 226 subjects recruited from Endocrinology Unit, Samara General Hospital during the period from 1<sup>st</sup> October 2014 to end of October 2015. Their age ranged from 32 to 75 years. The study population depending on their clinical and laboratory findings divided in to the following groups:

**Group I**: (50) subjects who were non-diabetic without periodontal disease (apparently healthy) as a control group.

**Group II**: (56) subjects who were non-diabetic with periodontal disease.

**Group III:** (120) diabetic subject who were subdivided into:

- Diabetics with periodontitis: (60) subjects.
- Diabetics without periodontitis: (60) subjects.

The study was approved by the Ethical Committee of Tikrit University College of Medicine and informed consent taken from each participant before their inclusion in the study.

#### Sample collection

Blood samples collected from subjects attended to Endocrine Unit in Samarra General Hospital after an overnight fasting in plain tube in the absence of any anticoagulants, and serum had been harvested by allowing the sample to clot within 30 minutes then centrifugation for 10 minutes at 500 rpm, the sera supernatant of serum and saliva were divided into aliquots and stored at -20 until assayed.

#### **Exclusions criteria**

Patients with acute or chronic illnesses apart from diabetes mellitus has been excluded from the study.

#### Methods

Fasting blood sugar, serum total cholesterol, serum triglycerides, serum High density lipoprotein, blood urea and serum uric acid were determined using enzymatic method kits purchased from Biolabo, France.

#### Statistical analysis

Data were translated into codes using a specially designed coding sheet, and then converted to computerized database. An expert statistical advice was sought and statistical analyses were done using SPSS (Statistical Package for Social Science) version 22. Frequency distributions for variables were done first and plotted on histograms which fail to show distribution of normality and confirmed by using the chi square test. As no assumption about the normality of distribution of study variables was made, nonparametric methods were used to assess the statistical significant of associations. The statistical significant of difference in means of a quantitative continuous variable between two groups was assessed by Mann-Whitney test, while between more than two groups Kruskall-Wallis test was used. P value less than 0.05 level of significant was considered significant.

#### **RESULTS**

#### Comparison between Diabetic group and Control group.

# Fasting blood sugar, Blood urea and Uric acid.

The means value of fasting blood sugar was significantly (P=0.000) higher in Diabetic group (203.87 ±81.99 mg/dl) compared to Control group (92.4±14.98 mg/dl). Also the blood urea and Uric acid were significantly (P=0.000), (P=0.000) higher in Diabetic group (28.27±3.79)

mg/dl),  $(4.81 \pm 0.86 \text{ mg/dl})$  compared to Control group  $(18.6\pm 2.76 \text{ mg/dl})$ ,  $(3.25\pm 0.55 \text{ mg/dl})$  respectively, Table 1 and Fig 1.

# Lipid Profile Parameters.

The mean values of Serum Cholesterol, Serum Triglycerides, Low density lipoprotein (LDL) were significantly (P=0.000), (P<0.018), (P=0.000) higher in Diabetic group (234.03  $\pm$ 58.13 mg/dl), (213.9 $\pm$ 105.26 mg/dl), (149.37 $\pm$ 55.45 mg/dl), compared to Control group (173.3 $\pm$ 20.09mg/dl),(128.9 $\pm$ 43.83mg/dl),(77.6 $\pm$ 21.33mg/dl) respectively. While the mean value of Serum High density lipoprotein (HDL) was significantly (P=0.000) lower in Diabetic group (41.17 $\pm$ 11.86 mg/dl) compared to Control group (66.9 $\pm$  11.76 mg/dl), Table -2.

Table 1: Mean Fasting blood sugar, Blood urea, and Uric acid in Diabetic group compared to Control group.

Variable		c group -60	Control group N=50		P-value
	Mean	SD	Mean	SD	
Fasting Blood Sugar (mg/dl)	203.87	81.99	92.40	14.98	0.000
Blood urea (mg/dl)	28.27	3.79	18.60	2.76	0.000
Uric acid (mg/dl)	4.81	0.86	3.25	0.55	0.000

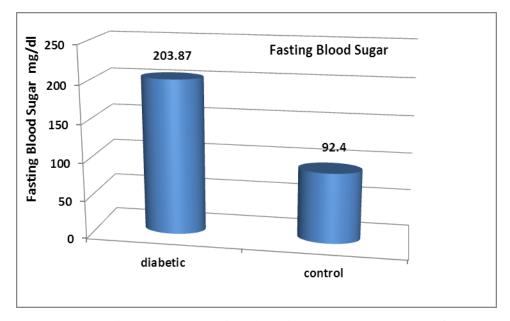


Figure.1: Mean Fasting blood sugar in Diabetic group compared to Control group.

Table -2: Mean Cholesterol, Triglycerides, High density lipoprotein (HDL) and Low density lipoprotein (LDL) in Diabetic group compared to Control group.

Variable		c group =60	Control group N=50		P-value
	Mean	SD	Mean	SD	
Cholesterol (mg/dl)	234.03	58.13	173.30	20.09	0.000
Triglycerides (mg/dl)	213.90	105.26	128.90	43.83	0.018
HDL (mg/dl)	41.17	11.86	66.90	11.76	0.000
LDL (mg/dl)	149.37	55.45	77.60	21.33	0.000

# Comparison between Diabetic with periodontitis group and control group. Fasting blood sugar, Blood urea and Uric acid.

The means values of Fasting blood sugar, Blood urea and Uric acid were significantly (P=0.000), (P=0.000), (P=0.000) higher in Diabetic with periodontitis group (219.3  $\pm$ 115.93 mg/dl), (28.33 $\pm$ 2.76 mg/dl), (4.93  $\pm$ 1.03 mg/dl) compared to control group (92.4 $\pm$ 14.98 mg/dl), (18.6 $\pm$ 2.76 mg/dl), (3.25 $\pm$  0.55 mg/dl) respectively, Table 3 and Fig. 2.

Table 3: Mean Fasting blood sugar, Blood urea, and Uric acid in Diabetic with periodontitis patients compared to Control group.

Variable	periodont	Diabetic with periodontitis group N=60 Control group N=50		_	
	Mean	SD	Mean	SD	
Fasting Blood Sugar (mg/dl)	219.3	115.93	92.40	14.98	0.000
Blood urea (mg/dl)	28.33	2.76	18.60	2.76	0.000
Uric acid (mg/dl)	4.93	1.03	3.25	0.55	0.000

#### Lipid Profile Parameters.

The means of serum Cholesterol was significantly(P<0.006) higher in Diabetic with periodontitis group (221 ±49.77 mg/dl) compared to Control group (173.3±20.09 mg/dl). Also Triglycerides and Low density lipoprotein (LDL) were significantly (P<0.014), (P=0.000) higher in Diabetic with periodontitis group (187±66.6 mg/dl), (132.1±45.05 mg/dl) compared to control group (128.9±43.83 mg/dl), (77.6± 21.33 mg/dl) respectively. While High density lipoprotein (HDL) was significantly (P=0.000) lower in Diabetic with

periodontitis group ( $36.14\pm10.09$  mg/dl) compared to Control group ( $66.9\pm~11.76$  mg/dl), Table 4.

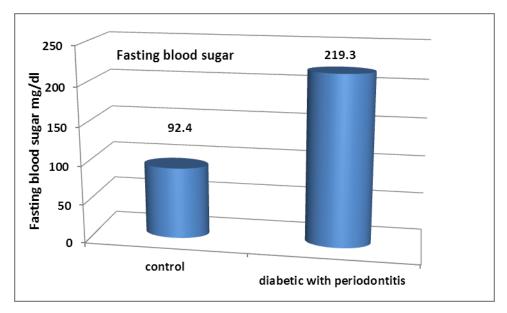


Figure.2: Mean Fasting blood sugar in Diabetic group with periodontitis group compared to Control group.

# Comparison between Control group and Periodontitis group.

#### Fasting blood sugar, Blood urea and Uric acid.

There was no significant differences between the means of Fasting blood sugar, Blood urea and Uric acid in Control group compared to Periodontitis group, Table 5.

#### Lipid Profile Parameters.

The means of Serum cholesterol was significantly(P=.000) higher in Control patients (173.3  $\pm 20.09$  mg/dl) compared to periodontitis group (120.14 $\pm 39.93$  mg/dl). High density lipoprotein (HDL) was significantly (P<0.024) higher in Control group (66.9 $\pm 11.76$  mg/dl) compared to Periodontitis group (51.60 $\pm$  19.26 mg/dl). Also LDL serum mean value was significantly (P< 0.02) higher in control group(77.6 $\pm 21.33$ ) as compared to those with periodontitis(64.79 $\pm 32.69$ ), Table 6.

Table 4: Mean Serum Cholesterol, Triglycerides, High density lipoprotein (HDL) and low density lipoprotein (LDL) in diabetes with periodontitis.

Variable	periodont	ic with itis group =60	Control group N=50		P-value	
	Mean	SD	Mean	SD		
Cholesterol (mg/dl)	221.00	49.77	173.30	20.09	0.006	
Triglycerides (mg/dl)	187.00	66.60	128.90	43.83	0.014	
HDL (mg/dl)	36.14	10.09	66.90	11.76	0.000	
LDL (mg/dl)	132.10	45.05	77.60	21.33	0.000	

# Comparison between Diabetic group and Periodontitis group.

# Fasting blood sugar, Blood urea and Uric acid.

The means value of Fasting blood sugar was significantly (P=0.000) higher in diabetic group (203.87  $\pm 81.99$  mg/dl) compared to periodontitis group (90 $\pm 5.16$  mg/dl). Also blood urea and uric acid were significantly (P=0.000), (P=0.000) higher in diabetic group (28.27 $\pm 3.79$  mg/dl), (4.81  $\pm 0.86$  mg/dl) compared to periodontitis group (19.29 $\pm 2.2$  mg/dl), (3.05 $\pm$  0.43 mg/dl) respectively, Table 7 and Fig.3.

Table -5: Mean Fasting blood sugar, Blood urea and Uric acid in Periodontitis group compared to Control group.

Variable	Control group N=50		Periodor	P- value	
	Mean	SD	Mean	SD	
Fasting Blood Sugar (mg/dl)	92.40	14.98	90.00	5.16	0.637
Blood urea (mg/dl)	18.60	2.76	19.29	2.2	0.505
Uric acid (mg/dl)	3.25	0.55	3.05	0.43	0.325

#### Lipid Profile Parameters.

The means of Serum Cholesterol was significantly (P=0.000) higher in diabetic group (234.03 ±58.13 mg/dl) compared to periodontitis group (120.14±39.93mg/dl). Also serum triglycerides and low density lipoprotein (LDL) were significantly (P=0.000), (P=0.000) higher in diabetic group (213.90±105.26 mg/dl), (149.37±55.45 mg/dl) compared to periodontitis group (120.36±19.14 mg/dl),(64.79± 32.6 mg/dl) respectively. While High

density lipoprotein (HDL) was significantly lower(P<0.015) in diabetic group(41.17±11.86) compared to periodontitis group (51.60±19.26), Table -8.

Table -6: Mean Serum Cholesterol, Triglycerides, High density lipoprotein (HDL) and Low density lipoprotein (LDL) in Periodontitis group compared to Control group.

Variable	Control gro	oup N=50	Periodont N:	P- value	
	Mean	SD	Mean	SD	
Cholesterol (mg/dl)	173.30	20.09	120.14	39.93	0.000
Triglycerides (mg/dl)	128.90	43.83	120.36	19.14	0.574
HDL (mg/dl)	66.90	11.76	51.60	19.26	0.024
LDL (mg/dl)	77.60	21.33	64.79	32.59	0.02

Table -7: Mean Fasting blood sugar, Blood urea and Uric acid in Diabetic group compared to Periodontitis group.

Variable	Diabetic group N=60		Periodor	P-value	
	Mean	SD	Mean	SD	
Fasting Blood Sugar (mg/dl)	203.87	81.99	90.00	5.16	0.000
Blood urea (mg/dl)	28.27	3.79	19.29	2.2	0.000
Uric acid (mg/dl)	4.81	0.86	3.05	0.43	0.000

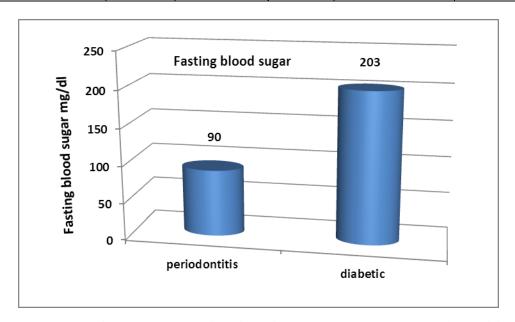


Figure.3: Mean Fasting blood sugar in Diabetic group compared to Periodontitis group.

Table -8: Mean Serum Cholesterol, Triglycerides, High density lipoprotein (HDL) and Low density lipoprotein (LDL) in Diabetic group compared to Periodontitis group.

Variable	Diabetic group N=60		Periodont N=	P-value	
	Mean	SD	Mean	SD	
Cholesterol (mg/dl)	234.03	58.13	120.14	39.93	0.000
Triglycerides (mg/dl)	213.90	105.26	120.36	19.14	0.000
HDL (mg/dl)	41.17	11.86	51.60	19.26	0.015
LDL (mg/dl)	149.37	55.45	64.79	32.6	0.000

# Comparison between Diabetic with periodontitis group and Periodontitis group. Fasting blood sugar, Blood urea and Uric acid.

The means of Fasting blood sugar was significantly (P=0.000) higher in diabetic with periodontitis group (219.30  $\pm$ 115.93 mg/dl) compared to periodontitis group (90 $\pm$ 5.16 mg/dl). Blood urea was significantly (P=0.000) higher in diabetic with periodontitis group (28.33 $\pm$ 2.76 mg/dl) compared to periodontitis (19.29 $\pm$ 2.2 mg/dl). Uric acid was significantly (P=0.000) higher in diabetic with periodontitis group (4.93  $\pm$ 1.03 mg/dl) compared to periodontitis group (3.05 $\pm$ 0.43 mg/dl), Table -9 and Figure 4.

Table -9: Mean Fasting blood sugar, Blood urea, and Uric acid in Diabetic with periodontitis group compared to Periodontitis group.

Variable		Diabetic with periodontitis group N=60		Periodontitis group N- 1	
	Mean	SD	Mean	SD	
Fasting Blood Sugar (mg/dl)	219.30	115.93	90.00	5.16	0.000
Blood urea (mg/dl)	28.33	2.76	19.29	2.2	0.000
Uric acid (mg/dl)	4.93	1.03	3.05	0.43	0.000

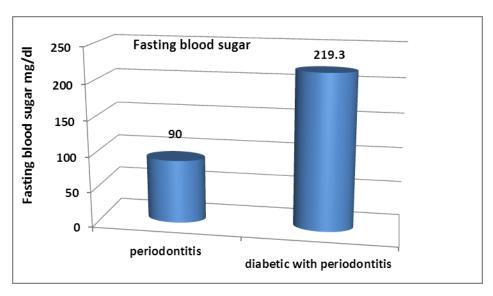


Figure.4: Mean Fasting blood sugar in Diabetic with periodontitis group compared to Periodontitis group.

#### Lipid Profile Parameters.

The means of Serum Cholesterol was significantly (P=.000) higher in Diabetic with periodontitis group (221 ±49.77 mg/dl) compared to Periodontitis group (120.14±39.93 mg/dl). Serum Triglycerides was significantly (P=0.000) higher in Diabetic with periodontitis group (187±66.6 mg/dl) compared to Periodontitis group (120.36±19.14 mg/dl). High density lipoprotein (HDL) was significantly (P<0.001) lower in Diabetic with periodontitis group (36.14± 10.09 mg/dl) compared to Periodontitis group (51.6±19.26 mg/dl). Low density lipoprotein (LDL) was significantly (P=0.000) higher in Diabetic with periodontitis group (132.1±45.05 mg/dl) compared to Periodontitis group (64.79± 32.6 mg/dl), Table -10.

Table (4-10): Mean Serum Cholesterol, Triglycerides, High density lipoprotein (HDL) and Low density lipoprotein (LDL) in Diabetic with periodontitis group compared to Periodontitis group.

Variable	periodonti	Diabetic with periodontitis group N=60		Periodontitis group N=56		
	Mean	SD	Mean	SD		
Cholesterol (mg/dl)	221.00	49.77	120.14	39.93	0.000	
Triglycerides (mg/dl)	187.00	66.60	120.36	19.14	0.000	
HDL (mg/dl)	36.14	10.09	51.60	19.26	0.001	
LDL (mg/dl)	132.10	45.05	64.79	32.6	0.000	

# Comparison between Diabetic group and Diabetic with periodontitis group.

# Fasting blood sugar, Blood urea and Uric acid.

There was no significant differences between the means of fasting blood sugar, blood urea and Uric acid in diabetic group compared to Diabetic with periodontitis group, Table -11.

#### Lipid Profile Parameters.

There was no significant differences between the means of lipid profile parameters between diabetic group compared to Diabetic with periodontitis group as shown in table -12.

#### Correlation Study.

# Correlation within Control group.

# Correlation between lipid profile parameters.

A correlation study between Serum Cholesterol, Triglycerides, High density lipoprotein (HDL), and Low density lipoprotein (LDL) within the Control group show a significant positive correlation between serum Triglycerides with serum Cholesterol. Also there was a significant negative correlation between (HDL) with both serum triglycerides and serum(LDL) within control group as shown in table -13, figures .5, 6 and .7.

#### Correlation between Fasting blood sugar, Blood urea and Uric acid.

A correlation study between Fasting blood sugar, Blood urea Uric acid within the Control group show a significant positive correlation between Fasting blood sugar and uric acid, Table -14.

Table -11: Mean Fasting blood sugar, Blood urea, and Uric acid in Diabetic group compared to Diabetic with periodontitis group.

Variable		c group =60	Diabetic with periodontitis group N=60		P-value	
	Mean	SD	Mean	SD		
Fasting Blood Sugar (mg/dl)	203.87	81.99	219.30	115.93	0.554	
Blood urea (mg/dl)	28.27	3.79	28.33	2.76	0.938	
Uric acid (mg/dl)	4.81	0.86	4.93	1.03	0.646	

# Correlation within Periodontitis group.

# Correlation between lipid profile parameters.

A correlation study between Serum Cholesterol, Triglycerides, High density lipoprotein (HDL), and Low density lipoprotein (LDL) within the Periodontitis group show a significant negative correlation between serum Cholesterol and serum Low density lipoprotein (LDL), Table -15 and figure .8.

Table -12: Mean Serum Cholesterol, Triglycerides, High density lipoprotein (HDL) and low density lipoprotein (LDL) in Diabetic group compared to Diabetic with periodontitis group.

Variable	Diabetic N=		Diabetic with periodontitis group N=60		P-value	
	Mean	SD	Mean	SD		
Cholesterol (mg/dl)	234.03	58.13	221.00	49.77	0.355	
Triglycerides (mg/dl)	213.90	105.26	187.00	66.6	0.242	
HDL (mg/dl)	41.17	11.86	36.14	10.09	0.178	
LDL (mg/dl)	149.37	55.45	132.10	45.05	0.191	

#### Correlation between Fasting blood sugar, Blood urea and Uric acid.

A correlation study between Fasting blood sugar, Blood urea and Uric acid within the periodontitis group didn't show any significant positive or negative correlation, Table .16.

Table -13: Correlation between Serum Cholesterol, Triglycerides, High density lipoprotein (HDL) and Low density lipoprotein (LDL) within Control group.

		Cholesterol	Triglycerides	HDL	LDL
Cholesterol	r- value	1	.768**	.480	.206
Cholesteroi	P- value		.010	.160	.568
Triglycerides	r- value	.768**	1	687 <sup>*</sup>	364
	P- value	.010		.028	.302
HDL	r- value	790**	687*	1	638*
	P- value	.007	.028		.047
IDI	r- value	.206	364	638*	1
LDL	P- value	.568	.302	.047	

<sup>\*\*.</sup> Correlation is significant at the 0.01 level (2-tailed).

<sup>\*.</sup> Correlation is significant at the 0.05 level (2-tailed).

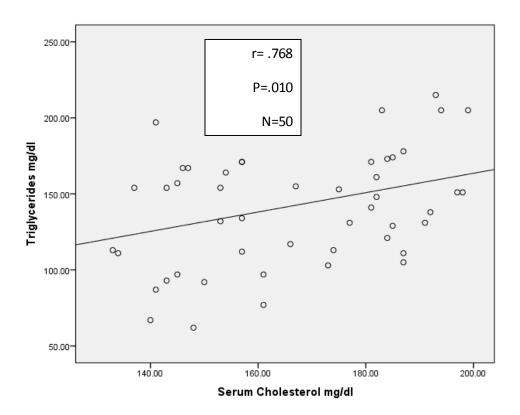


Figure.5: Correlation between Serum Cholesterol and Serum Triglycerides within Control group.

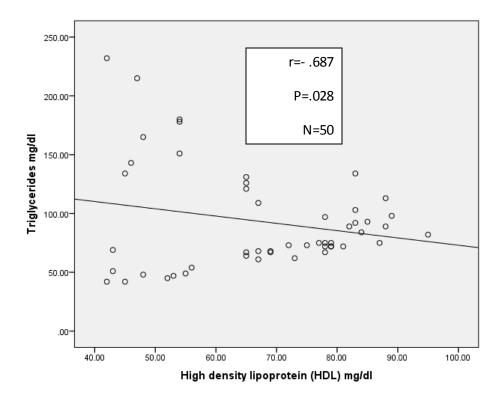


Figure. 6: Correlation between Serum Triglycerides and Serum High density lipoprotein (HDL) within Control group.

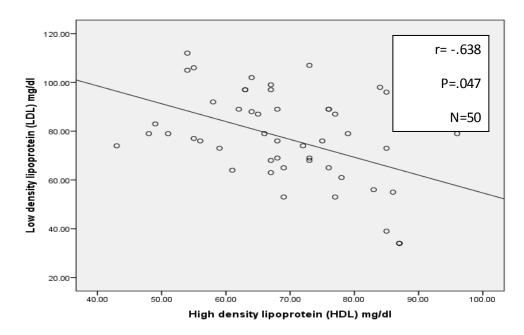


Figure .7: Correlation between Serum Low density lipoprotein (LDL) and Serum High density lipoprotein (HDL) within Control group.

# Correlation within Diabetic group.

#### Correlation between lipid profile parameters.

A correlation study between serum Cholesterol, Triglycerides, High density lipoprotein (HDL), and Low density lipoprotein (LDL) within the Diabetic group show a significant positive correlation between serum Cholesterol and serum low density lipoprotein (LDL), Table.17 and figure .9.

Table -14: Correlation between Fasting blood sugar, Blood urea Uric acid within the control group.

Variable		Fasting blood sugar	Blood urea	Uric acid
E 4 11 1	r- value	1	.084	.596
Fasting blood sugar	P- value		.817	.05
Blood urea	r- value	084	1	.362
Dioou urea	P- value	.817		.305
Uric acid	r- value	596	.362	1
	P- value	.05	.305	

#### Correlation between Fasting blood sugar, Blood urea and Uric acid.

A correlation study between Fasting blood sugar, Blood urea and Uric acid within the Diabetic group didn't show any significant positive or negative correlation as shown in table (4-18).

Variable		Cholesterol	Triglycerides	HDL	LDL
	r-value	1	.114	.451	.898**
Cholesterol	P-value		.698	.106	.000
	r-value	.114	1	.291	.055
Triglycerides	P-value	.698		.312	.852
	r-value	.451	.291	1	.106
HDL	P-value	.106	.312		.718
	r-value	.898**	.055	.106	1
LDL	P-value	.000	.852	.718	

Table -15: Correlation between Serum Cholesterol, Triglycerides, High density lipoprotein (HDL) and Low density lipoprotein (LDL) within Periodontitis group.

#### Correlation within Diabetic with periodontitis group.

#### Correlation between lipid profile parameters.

A correlation study between serum Cholesterol, Triglycerides, High density lipoprotein (HDL), and Low density lipoprotein (LDL) within the Diabetic with periodontitis group show a significant positive correlation between Serum Cholesterol with both serum Triglycerides and serum low density lipoprotein (LDL), Table -19, figures .10 and .11.

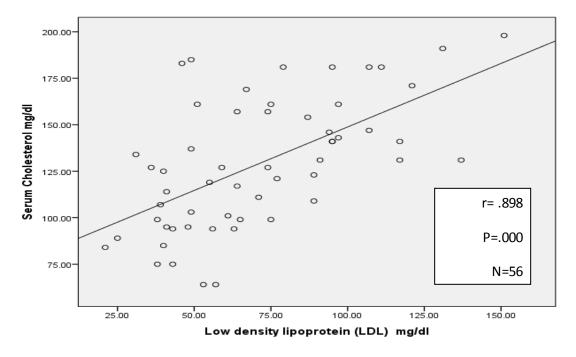


Figure 4.8: Correlation between Serum Low density lipoprotein (LDL) and Serum Cholesterol within Periodontitis group.

<sup>\*\*.</sup> Correlation is significant at the 0.01 level (2-tailed).

# Correlation between Fasting blood sugar, Blood urea and Uric acid.

A correlation study between Fasting blood sugar, Blood urea and Uric acid within the Diabetic with periodontitis group didn't show any significant positive or negative correlation, Table -20.

Table -16. Correlation between Fasting blood sugar, Blood urea and Uric acid within the periodontitis group.

Variable		Fasting blood sugar	Blood urea	Uric acid
Fasting blood sugar	r- value	1	027	021
Fasting blood sugar	P- value		.927	.943
Blood urea	r- value	027	1	.278
Dioou urea	P- value	.927		.335
Uric acid	r- value	021	.278	1
Offic acid	P- value	.943	.335	

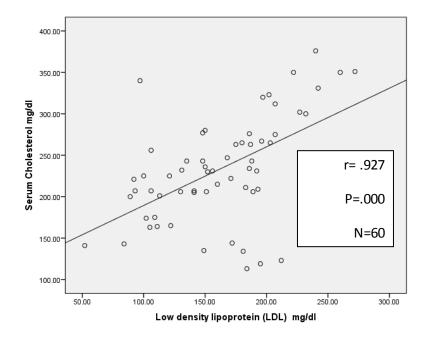


Figure. 9: Correlation between Serum Low density lipoprotein (LDL) and Serum Cholesterol within Diabetic group.

Table -17: Correlation between Serum Cholesterol, Triglycerides, High density lipoprotein (HDL) and Low density lipoprotein (LDL) within Diabetic group.

Variable		Cholesterol	Triglycerides	HDL	LDL
Chalagtaral	r-value	1	.299	.064	.927**
Cholesterol	P-value		.109	.737	.000
Triglygorides	r-value	.299	1	263	008
Triglycerides	P-value	.109		.160	.968

HI)I,	r-value	.064	263	1	060
	P-value	.737	.160		.753
11.1)1.	r-value	.927**	008	060	1
	P-value	.000	.968	.753	
**. Correlation is significant at the 0.01 level (2-tailed).					

Table -18. Correlation between Fasting blood sugar, Blood urea Uric acid within the Diabetic group.

Variable		Fasting blood sugar	Blood urea	Uric acid
Facting blood sugar	r- value	1	.005	.253
Fasting blood sugar	P- value	30	.978	.177
Blood urea	r- value	.005	1	.018
Dioou urea	P- value	.978		.925
Uric acid	r- value	.253	.018	1
Offic acid	P- value	.177	.925	

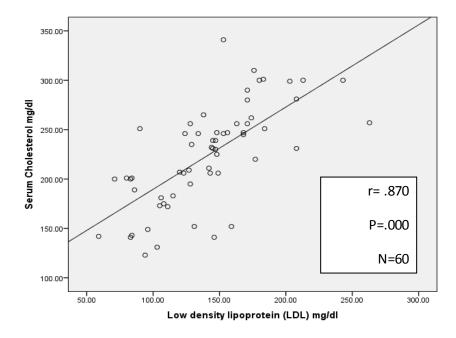


Figure.10: Correlation between Serum Low density lipoprotein (LDL) and Serum Cholesterol within Diabetic with periodontitis group.

Table -19: Correlation between Serum Cholesterol, Triglycerides, High density lipoprotein (HDL) and Low density lipoprotein (LDL) within Diabetic with periodontitis group.

Variable		Cholesterol	Triglycerides	HDL	LDL
Chalagtaral	r-value	1	.466**	.225	.870**
Cholesterol	P-value		.010	.232	.000
Triglycerides	r-value	.466**	1	.106	.173
	P-value	.010		.577	.359

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HDL LDL	r-value	.225	.106	1	211
	P-value	.232	.577		.263
	r-value	.870**	.173	211	1
	P-value	.000	.359	.263	
**. Correlation is significant at the 0.01 level (2-tailed).					

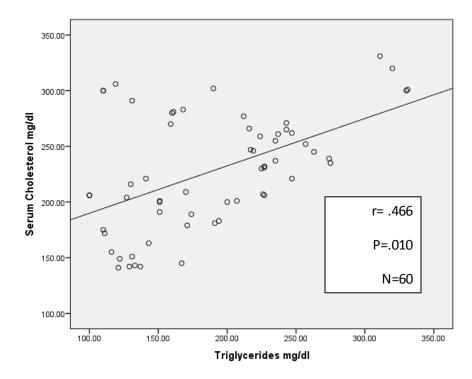


Figure.11: Correlation between Serum Triglycerides and Serum Cholesterol within Diabetic with periodontitis group.

Table -20. Correlation between Fasting blood sugar, Blood urea Uric acid within the Diabetic with periodontitis group.

Variable		Fasting blood sugar	Blood urea	Uric acid
Fasting	<b>blood</b> r- value	1	.273	117
sugar	P- value		.144	.538
Blood urea	r- value	.273	1	.004
	P- value	.144		.983
Uric acid	r- value	117	.004	1
	P- value	.538	.983	

# **DISCUSSION**

Diabetes is a chronic metabolic disease which is with bad prognosis if not well controlled through treatment and monitoring. The present study indicated a highly significant differences in some biomarkers in patients with diabetes as compared to non-diabetic subjects. Unfortunately, the mean of glucose in diabetic group was not well controlled and

exceeded the standard normal value of 100 mg/dl for fasting and 140 mg/dl post-meal value<sup>[6]</sup>. In addition, blood urea mean value was highly significant higher in diabetic subjects than in control non-diabetic individuals. However, the mean value still lower than normal upper limit value of 40 mg/dl. Furthermore, uric acid mean value was significantly higher in diabetic individuals than in control group, however, the mean value still lower than standard normal upper limit value of 7.2 mg/dl<sup>[7]</sup>.

The higher level of blood urea and serum uric acid in this study cohort may be attributed to metabolic changes that occurs as a sequences of uncontrolled diabetes. Blood urea is a predictive biomarker of renal dysfunction and thus their increase in our study cohort indicated an association between diabetes and nephropathy<sup>[8]</sup>.

This study shows that serum cholesterol, triglycerides, and LDL mean values were significantly higher in diabetic subject than in non-diabetic controls. While serum HDL mean value was significantly lower in diabetic group than in non-diabetic controls. However, serum HDL in diabetic group still within the accepted limit of  $\geq 40$  mg/dl.

Khare  $et\ al^{[9]}$  concluded from their study that mean serum uric acid was higher in diabetic subject than in control and it was more in those with long history as compared to newly diagnosed cases. However, Al-Rawi  $et\ al^{[10]}$  reported lower mean concentration of serum uric acid as compared to control, but the difference was not significant. Other studies indicated an increased serum/plasma levels in subject with diabetes as compared to non-diabetic control [9,11,12].

Ashakiran  $et\ al^{[13]}$  suggest that the trend of serum uric acid levels in diabetic patients with complication differ from that without complications. They found that serum uric acids was significantly lower in diabetic patients without retinopathy as compared to controls group and diabetic with retinopathy.

Bhole  $et\ al^{[14]}$  in a prospective study suggest that high serum uric acid is a risk factor for type 2 diabetes development. In addition Baldwin  $et\ al^{[15]}$  in animal model found that hyperuricemia lead to proiflamatory endocrine imbalance which may attribute to cell surface morphological changes in vascular smooth muscles and adipose tissue and insulin resistance.

The present study shows a high mean serum level of uric acid and blood urea indicating that they were with predictive value biomarkers for diabetic renal complication.

Presence of periodontitis with diabetes in the same individual did not effected significantly the level of fasting blood sugar, blood urea, and uric acid as this study indicated. However, periodontitis was without significant effect on fasting blood sugar, blood urea, and uric acid when compared to matched control. Thus periodontitis alone with low impact on the serum level of these three above mentioned biomarkers. The present study finding indicated that diabetes alone, or in combination with periodontitis leads to significant increase in mean serum levels of blood urea and serum uric acid. Thus the local disease such as periodontitis may affect serum levels of blood urea and uric acid, but with lower effect as compared systemic disease.

The total cholesterol increased 17% of the standard international control and 35% of this study non-diabetic control. While triglycerides increased 66% of this study control ad 43% of the international standard. The LDL was much higher for both this study control(92%) and international standard. The HDL reduced 39% in comparison to this study control and 43% to the international standard. The above data collectively indicated that diabetic patients included in this study was with not well controlled diabetes. Previous studies<sup>[16-21]</sup> reported increase in serum / plasma total cholesterol, LDL, and triglycerides and decrease level of HDL. The present study findings and that reported in literatures suggest that dyslipidemia was associated with diabetes and may represent a risk factor for cardiovascular disease in subject with diabetes.

The mean serum value of cholesterol, HDL, and LDL were significantly higher in control group than individual with periodontitis, while triglycerides higher in control group but not reach the significant level. This finding indicate that local disease may affect the concentration of lipid profile in patients with periodontitis. However, fasting blood sugar (FBS), blood urea, and uric acid mean serum were not significantly different between patients with periodontitis and control subjects. Gormat *et al*<sup>[21]</sup> reported that increased serum glucose was associated with significant increase in cholesterol, LDL, and triglicerides and decreased in HDL.

Diabetes and periodontitis are two pathological conditions occurred in human being with bidirectional relationship<sup>[22,23]</sup>. However, it was uncertain at which direction the effect was driven. The individuals with diabetes and periodontitis co-morbidities were significantly with higher mean serum value of FBS, blood urea, uric acid total cholesterol, triglycerides and

LDL. While HDL was significantly higher in control as compared to diabetic with periodontitis subjects.

The present study finding indicate that presence of periodontitis with diabetes in the same individual may lead to reduction in cholesterol, triglycerides, and LDL and increase in HDL.

Total cholesterol, triglycerides, and LDL mean serum values were higher in individuals with diabetes as compared to those with diabetes and periodontitis. While in subjects with periodontitis were with cholesterol, triglycerides, and LDL lower serum levels than in diabetic group and group of diabetes with periodontitis.

Although, periodontitis reduced mean serum levels of cholesterol, triglycerides, and LDL, however, diabetes was the pathological condition that affect serum levels of lipid profile. In addition, the present study findings indicated that the systemic diseases (e.g. diabetes) affected lipid profile levels more than local diseases(e.g. periodontitis), however, both diseases were with close association<sup>[19]</sup>. Diabetic xerostomia may complicated periodontitis condition and interfere with local hygiene, immunity, and oxidative stress<sup>[25-27]</sup>.

Comparison between subjects with diabetes and those with periodontitis show a significantly higher FBS, uric acid, cholesterol, triglycerides, and LDL mean serum levels in diabetic group. This finding indicated that periodontitis do affect the serum level of above mentioned biomarkers, however the effect of diabetes was more than that of periodontitis. In addition, mean serum HDL was significantly lower in diabetic patients as compared to those with periodontitis.

Subjects with diabetes and periodontitis show significantly higher FBS, blood urea, uric acid, cholesterol, triglycerides, and LDL than in individuals with periodontitis. However, the presence of periodontitis with diabetes was with minor effect on FBS, blood urea, uric acid and lipid profile mean serum values. This hypothesis was confirmed by the present study finding which indicate a non-significant differences in mean values of FBS, blood urea, uric acid and lipid profile between diabetic subject group and individuals with diabetes and periodontitis.

Previous studies indicated that diabetes is a risk factor for periodontitis<sup>[28-31]</sup>. Diabetes and periodontitis were reported since 1960s<sup>[23]</sup> and suggested by later performed studies<sup>[30,32,33]</sup>. In addition, in subject with uncontrolled diabetes, periodontitis was associated with more

complications than in those with controlled diabetes<sup>[34-38]</sup>. However, other studies not confirm such association<sup>[39-41]</sup>. Periodontitis demonstrated impact on diabetes and may increase frequently of diabetic complications<sup>[42]</sup>. Other studies reported that treatment of periodontal disease attributed to glycemic control in subject with diabetes and periodontitis<sup>[43-59]</sup>. Diabetes influenced periodontitis by exaggerated inflammatory response, immunological dysfunction, metabolic changes, and oxidant-anti-oxidant disturbances<sup>[60-77]</sup>.

As expected in control subjects, cholesterol was significantly positively correlated with triglycerides and negatively with HDL. However, the correlation between cholesterol and LDL was not significant. In addition HDL was negatively with triglycerides and LDL, but not reach the significant level for LDL.

In periodontal disease group, in contrast to control, a significant correlation demonstrated between cholesterol and LDL only. This may be due to that individuals with periodontitis are with inflammatory, microbial, and immunologic response that may contribute to dyslipidemia. The present study confirm the presence of dyslipidemia as compared to control group.

FBS, blood urea, and uric acid in periodontitis group don't show any significant correlation among each other. This finding may be expected as there was no significant differences in theses biomarkers between periodontitis group and control, indicating a slight changes in theses parameters.

In diabetic group, a significant positive correlation was found between cholesterol and LDL. While in group of diabetes with periodontitis, a positive significant correlation was achieved between cholesterol with triglycerides and cholesterol with LDL. The trend of correlation in diabetic group was similar to that found in periodontitis, while it was markedly different from that in control group and slightly different from that in diabetes with periodontitis group. These data collectively indicated that subjects with diabetes or periodontitis or diabetes with periodontitis are prone to develop dyslipidemia of variable magnitude from each group to others. These dyslipidemia must be considered as an important co-morbidities in diabetes and periodontal disease and may be a risk for cardiovascular diseases in such population.

FBS, uric acid and blood urea do not demonstrated any significant correlation among them in individuals with periodontitis, diabetes and those with diabetes and periodontitis. This finding

may be explained on the mechanisms complexity that associated with such inflammatory, immunologic, microbial and metabolic diseases.

Amarty *et al*<sup>[11]</sup> found that there was a significant correlation between uric acid and body mass index (BMI); other study<sup>[13]</sup>, show a significant correlation between uric acid and glucose; uric acid and cholesterol; and between uric acid and LDL in diabetic individuals.

In present study serum uric acid was significantly correlated with cholesterol and triglycerides in diabetic patients. While in subjects with diabetic and periodontitis, uric acid significantly positively correlated with serum cholesterol and negatively with HDL, while in control it was negatively correlated with triglycerides. Sarmah and Sharma<sup>[78]</sup> found a significant positive correlation between uric acid and total cholesterol, uric acid and triglycerides, and uric acid and LDL and negative correlation between uric acid and HDL.

In conclusion, both diabetes and periodontitis as present alone or in combination were associated with lipid profile, blood urea, and uric acid disturbances. This finding clarify the importance of monitoring both conditions for the achievement of good prognosis and prevent complications.

#### REFERENCES

- Indirakumari N, Vinutha. S, Chandrakala K. Study of Lipid Profile in Diabetes Mellitus Patients Who Were On Glibenclamide and Glimeperide. IOSR Journal of Dental and Medical Sciences (IOSR-JDMS) 2015; 14: 13-22.
- 2. Socransky SS, Haffajee AD. The bacterial etiology of destructive periodontal disease: current concepts. J Periodontol 1992; 63: 322-331.
- 3. Zhang L, Henson BS, Camargo PM, Wong DT. The clinical value of salivary biomarkers for periodontal disease. Periodontol 2009; 51: 25-37.
- 4. Pavankumar A, Jagdishreddy G, Raja Babu P. Biomarkers in Periodontal Disease. J Mol Biomark Diagn 2015; 6: 232. doi: 10.4172/2155-9929.1000232.
- 5. Tripathy BB, Chandalia HB, Das AK. RSSDI TEXTBOOK OF DIABETES MILLITUS. Vol 1 & 2. 2nd Edition, 2012, JAYPEE, India.
- 6. American Diabetes Association (ADA). What is a normal blood glucose level?. 2016; www.diabetes.org#sthash.8Y7rAcUG.dpuf. Accessed 30\9\2016.
- 7. Gabbey AE and Nall R. Uric acid test (blood analysis). Health line 2015. http://www.healthline.com/health/uric-acid-blood. Accessed 30\9\2016.

- 8. Mayo Clinic Staff. Blood urea nitrogen (BUN). Patients care and health information. http://www.mayoclinic.org/tests-procedures/blood-urea-nitrogen/home/ovc-20211239. Accessed 30\9\2016.
- 9. Khare Sh., Kumar J., Kansal A.To Study Serum Uric Acid In Type 2 Diabetes Mellitus Patient. IOSR-JDMS 2015; 14: 82-86.
- 10. Al-Rawi Kh., Saif Allah P., Al-Korwi E., Taleab Sh. Evaluation of vitamin C, uric acid, urea and creatinine levels in the blood of Type 2 diabetic Iraqi females. J University Anbar Pure Sci 2013; 7: 1-10.
- 11. Amartey N., NsiahK., Mensah F. Plasma Levels of Uric Acid, Urea and Creatinine in Diabetics Who Visit the Clinical Analysis Laboratory (CAn-Lab) at Kwame Nkrumah University of Science and Technology, Kumasi, Ghana. Journal of Clinical and Diagnostic Research. 2015; 9(2): BC05-BC09.
- 12. Feig D., Kang D. and Johnson R. Uric Acid and Cardiovascular Risk. N Engl J Med. 2008, 23; 359(17): 1811–1821
- 13. Ashakiran. S., N. Krishnamurthy, Navin S., Sandeep Patil. Behaviour of serum uric acid and lipid profile in relation to glycemic status in proliferative and non-proliferative diabetic retinopathy. Current Neurobiology 2010; 2(1): 57-61.
- 14. Bhole V, Choi J., Kim S., Vera M., Choi H. Serum Uric Acid Levels and the Risk of Type 2 Diabetes: A Prospective Study. The American Journal of Medicine, 2010; 123: 957-961.
- 15. Baldwin W. et al. Hyperuricemia as a mediator of the proinflammatory endocrine imbalance in the adipose tissue in a murine model of the metabolic syndrome. Diabetes. 2011 Apr; 60(4): 1258-69.
- 16. Stamouli M, Pouliakis A, Mourtzikou A, Skliris A, Panagiotou I, Mournianakis E and Totos G. Evaluation of the lipid profile in type 2 diabetes mellitus patients in Greece. Clin Lab. 2014; 60(10): 1593-600.
- 17. Syamala D., Indira Kumari and Pavan G. The effect of physical and atorvastatin action on lipid profile of sedentary and non sedentary alcohilic myocardial infarction patients. J Evolution Med Dent Sci 2015; 4: 1858-1862.
- 18. Sultana R. Impact of type 2 diabetes mellitus on lipid profile. Gomal Journal of Medical Sciences 2010; 8: 57-59.
- 19. Samatha P, Venka M, Siva V. Lipid Profile Levels in Type 2 Diabetes Mellitus from the Tribal Population of Adilabad in Andhra Pradesh, India. Journal of Clinical and Diagnostic Research. 2012; 6(4): 590-592.

- 20. Singh G. and Kumar K. A Study of Lipid Profile in Type 2 Diabetic Punjabi Population. Journal of Exercise Science and Physiotherapy, 2012; 8: 7-10.
- 21. Gormat N., Benmansour F., A. Hammas A. Lipid profile in type 2 diabetic and hypertensive population in Western Algeria. Annals of Biological Research, 2011; 2(4): 447-454.
- 22. Gumus P. and Bunduneli N. Diabetes mellitus and periodontitis: Sign of a bidirectional relationship. EMJ Diabet. 2013; 1: 30-36.
- 23. Sima C. and Glogauer M. Periodontitis in Patients with Diabetes-A Complication that Impacts on Metabolic Control. US Endocrinology, 2012; 8(1): 35-39.
- 24. Taylor JJ, Preshaw PM and Lalla E. A. A review of the evidence forpathogenic mechanisms that may link periodontitis and diabetes. J Periodontol 2013; 84(4 Suppl.): S113-S134.
- 25. Wei PF, Ho KY, Ho YP, Wu YM, Yang YH, Tsai CC. The investigation of glutathione peroxidase, lactoferrin, myeloperoxidase and interleukin-1beta in gingival crevicular fluid: implications for oxidative stress in human periodontal diseases. J Periodontal Res. 2004; 39(5): 287-293.
- 26. Arana C et al. Parameters of oxidative stress in saliva from diabetic and parenteral drug addict patients. J Oral Pathol Med. 2006; 35(9): 554-9.
- 27. Ben-Zvi I, Green Y, Nakhoul F, Kanter Y and Nagler RM. Effects of diabetes mellitus, chronic renal failure and hemodialysis on serum and salivary antioxidant status. Nephron Clin Pract. 2007; 105(3): c114-20.
- 28. Löe H, Periodontal disease. The sixth complication of diabetes mellitus, Diabetes Care, 1993; 16: 329–34.
- 29. Taylor GW, Borgnakke WS, Periodontal disease: associations with diabetes, glycemic control and complications, Oral Dis, 2008; 14: 191-203.
- 30. Soskolne WA, Klinger A, The relationship between periodontal diseases and diabetes: an overview, Ann Periodontol, 2001; 6: 91–8.
- 31. Firatli E, The relationship between clinical periodontal status and insulin-dependent diabetes mellitus. Results after 5 years, J Periodontol, 1997; 68: 136–40.
- 32. Mealey BL, Ocampo GL, Diabetes mellitus and periodontal disease, Periodontol 2000, 2007; 44: 127–53.
- 33. Demmer RT, Desvarieux M, Holtfreter B, et al., Periodontal status and A1C change: longitudinal results from the study of health in Pomerania (SHIP), Diabetes Care, 2010; 33: 1037–43.

- 34. Tsai C, Hayes C, Taylor GW, Glycemic control of type 2 diabetes and severe periodontal disease in the US adult population, Community Dent Oral Epidemiol, 2002; 30: 182–92.
- 35. Firatli E, The relationship between clinical periodontal status and insulin-dependent diabetes mellitus. Results after 5 years, J Periodontol, 1997; 68: 136–40.
- 36. Oliver RC, Tervonen T, Periodontitis and tooth loss: comparing diabetics with the general population, J Am Dent Assoc, 1993; 124: 71–6.
- 37. Novaes AB Jr, Gutierrez FG, Novaes AB, Periodontal disease progression in type II non-insulin-dependent diabetes mellitus patients (NIDDM). Part I Probing pocket depth and clinical attachment, Braz Dent J, 1996;7:65–73.
- 38. Seppälä B, Ainamo J, A site-by-site follow-up study on the effect of controlled versus poorly controlled insulin-dependent diabetes mellitus, J Clin Periodontol, 1994; 21: 161–5.
- 39. DePommereau V, Dargent-Paré C, Robert JJ, Brion M, Periodontal status in insulindependent diabetic adolescents, J Clin Periodontol, 1992; 19: 628–32.
- 40. Bridges RB, Anderson JW, Saxe SR, et al., Periodontal status of diabetic and non-diabetic men: effects of smoking, glycemic control, and socioeconomic factors, J Periodontol, 1996; 67: 1185–92.
- 41. Sastrowijoto SH, Hillemans P, van Steenbergen TJ, et al., Periodontal condition and microbiology of healthy and diseased periodontal pockets in type 1 diabetes mellitus patients, J Clin Periodontol, 1989; 16: 316–22.
- 42. Thorstensson H, Kuylenstierna J, Hugoson A, Medical status and complications in relation to periodontal disease experience in insulin-dependent diabetics, J Clin Periodontol, 1996; 23: 194–202.
- 43. Grossi SG, Skrepcinski FB, DeCaro T, et al., Treatment of periodontal disease in diabetics reduces glycated hemoglobin, J Periodontol, 1997; 68: 713–9.
- 44. Katagiri S, Nitta H, Nagasawa T, et al., Multi-center intervention study on glycohemoglobin (HbA1c) and serum, high-sensitivity CRP (hs-CRP) after local anti-infectious periodontal treatment in type 2 diabetic patients with periodontal disease, Diabetes Res Clin Pract, 2009; 83: 308–15.
- 45. Correa FO, Gonçalves D, Figueredo CM, et al., Effect of periodontal treatment on metabolic control, systemic inflammation and cytokines in patients with type 2 diabetes, J Clin Periodontol, 2010; 37: 53–8.
- 46. O'Connell PA, Taba M, Nomizo A, et al., Effects of periodontal therapy on glycemic control and inflammatory markers, J Periodontol, 2008; 79: 774–83.

- 47. Sun WL, Chen LL, Zhang SZ, et al., Changes of adiponectin and inflammatory cytokines after periodontal intervention in type 2 diabetes patients with periodontitis, Arch Oral Biol, 2010; 55: 970–4.
- 48. Teeuw WJ, Gerdes VE, Loos BG, Effect of periodontal treatment on glycemic control of diabetic patients: a systematic review and metaanalysis, Diabetes Care, 2010; 33: 421–7.
- 49. Simpson T, Needleman I, Wild SH, Treatment of periodontal disease for glycaemic control in people with diabetes, Australian Dent J, 2010; 55: 472–4.
- 50. Kiran M, Arpak N, Unsal E, Erdogan MF, The effect of improved periodontal health on metabolic control in type 2 diabetes mellitus, J Clin Periodontol, 2005; 32: 266–72.
- 51. Jones JA, Miller DR, Wehler CJ, et al., Does periodontal care improve glycemic control? The Department of Veterans Affairs Dental Diabetes Study, J Clin Periodontol, 2007; 34: 46–52.
- 52. Rocha M, Nava LE, Vázquez de la Torre C, et al., Clinical and radiological improvement of periodontal disease in patients with type 2 diabetes mellitus treated with alendronate:a randomized, placebo controlled trial, J Periodontol, 2001; 72: 204–9.
- 53. Al-Mubarak S, Ciancio S, Aljada A, et al., Comparative evaluation of adjunctive oral irrigation in diabetics, J Clin Periodontol, 2002; 29: 295–300.
- 54. Rodrigues DC, Taba MJ, Novaes AB, et al., Effect of nonsurgical periodontal therapy on glycemic control in patients with type 2 diabetes mellitus, J Periodontol, 2003; 74: 1361–7.
- 55. Stewart JE, Wager KA, Friedlander AH, Zadeh HH, The effect of periodontal treatment on glycemic control in patients with type 2 diabetes mellitus, J Clin Periodontol, 2001; 28: 306–10.
- 56. Promsudthi A, Pimapansri S, Deerochanawong C, Kanchanavasita W, The effect of periodontal therapy on uncontrolled type 2 diabetes mellitus in older subjects, Oral Dis, 2005; 11: 293–8.
- 57. Darré L, Vergnes JN, Gourdy P, Sixou M, Efficacy of periodontal treatment on glycaemic control in diabetic patients: A meta-analysis of interventional studies, Diabetes Metab, 2008; 34: 497–506.
- 58. Stratton IM, Adler AI, Neil HA, et al., Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35): prospective observational study, BMJ, 2000; 321: 405–12.

- 59. Khaw KT, Wareham N, Bingham S, et al., Association of hemoglobin A1c with cardiovascular disease and mortality in adults: the European prospective investigation into cancer in Norfolk, Ann Intern Med, 2004; 141: 413–20.
- 60. Mealey BL, Rose LF, Diabetes mellitus and inflammatory periodontal diseases, Curr Opin Endocrinol Diabetes Obes, 2008; 15: 135–41.
- 61. Mealey BL, Oates TW, American Academy of Periodontology, Diabetes mellitus and periodontal diseases, J Periodontol, 2006; 77: 1289–303.
- 62. Manouchehr-Pour M, Spagnuolo PJ, Rodman HM and Bissada NF. Impaired neutrophil chemotaxis in diabetic patients with severe periodontitis, J Dent Res, 1981; 60: 729–30.
- 63. McMullen JA, Van Dyke TE, Horoszewicz HU, Genco RJ, Neutrophil chemotaxis in individuals with advanced periodontal disease and a genetic predisposition to diabetes mellitus, J Periodontol, 1981; 52: 167–73.
- 64. Graves DT, Liu R, Alikhani M, et al., Diabetes-enhanced inflammation and apoptosis impact on periodontal pathology, J Dent Res, 2006; 85: 15–21.
- 65. Salvi GE, Collins JG, Yalda B, et al., Monocytic TNF alpha secretion patterns in IDDM patients with periodontal diseases, J Clin Periodontol, 1997; 24: 8–16.
- 66. Lalla E, Kaplan S, Yang J, et al., Effects of periodontal therapy on serum C-reactive protein, sE-selectin, and tumor necrosis factor-alpha secretion by peripheral blood-derived macrophages in diabetes. A pilot study, J Periodontal Res, 2007; 42: 274–82.
- 67. Sima C, Rhourida K, Van Dyke TE, Gyurko R, Type 1 diabetes predisposes to enhanced gingival leukocyte margination and macromolecule extravasation in vivo, J Periodontal Res, 2010; 45: 748–56.
- 68. Gyurko R, Siquiera CC, Caldon N, et al., Chronic hyperglycemia predisposes to exaggerated inflammatory response and leukocyte dysfunction in Akita mice, J Immunol, 2006; 177: 7250–6.
- 69. He H, Liu R, Desta T, et al., Diabetes causes decreased osteoclastogenesis, reduced bone formation, and enhanced apoptosis of osteoblastic cells in bacteria stimulated bone loss, Endocrinology, 2004; 145: 447–52.
- 70. Liu R, Bal HS, Desta T, et al., Tumor necrosis factor-alpha mediates diabetes-enhanced apoptosis of matrix-producing cells and impairs diabetic healing, Am J Pathol, 2006; 168: 757–64.
- 71. White CB, Turner NS, Lee G-C, Haidukewych GJ, Open ankle fractures in patients with diabetes mellitus, Clin Orthop Relat Res, 2003; 414: 37–44.

- 72. Beam HA, Parsons JR, Lin SS, The effects of blood glucose control upon fracture healing in the BB Wistar rat with diabetes mellitus, J Orthop Res, 2002; 20: 1210–16.
- 73. Tonetti MS, D'Aiuto F, Nibali L, et al., Treatment of periodontitis and endothelial function, N Engl J Med, 2007; 356: 911–20.
- 74. Engebretson S, Chertog R, Nichols A, et al., Plasma levels of tumor necrosis factor-alpha in patients with chronic periodontitis and type 2 diabetes, J Clin Periodontol, 2007; 34: 18–24.
- 75. Lalla E, Lamster IB, Feit M, et al., Blockade of RAGE suppresses periodontitis-associated bone loss in diabetic mice, J Clin Invest, 2000; 105: 1117–24.
- 76. Takeda M, Ojima M, Yoshioka H, et al., Relationship of serum advanced glycation end products with deterioration of periodontitis in type 2 diabetes patients, J Periodontol, 2006; 77: 15–20.
- 77. Murillo J, Wang Y, Xu X, et al., Advanced glycation of type I collagen and fibronectin modifies periodontal cell behavior, J Periodontol, 2008; 79: 2190–9.
- 78. Sarmah D. and Sharma B. A correlative study of uric acid with lipid profile. Asian Journal of Medical Science, 2013; 4: 8-14.