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To cite this article: F. Kaya, F. Çağlayan, A. Dag, H. Kaya & C. Kaya (2006) The Investigation of Gingival Crevicular Fluid Prostaglandin E2 Level of the Type II Diabetes Mellitus Patients with Periodontitis, *Biotechnology & Biotechnological Equipment*, 20:2, 179-184, DOI: [10.1080/13102818.2006.10817363](https://doi.org/10.1080/13102818.2006.10817363)

To link to this article: <https://doi.org/10.1080/13102818.2006.10817363>



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Published online: 15 Apr 2014.



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# THE INVESTIGATION OF GINGIVAL CREVICULAR FLUID PROSTHOGLANDIN E2 LEVEL OF THE TYPE II DIABETES MELLITUS PATIENTS WITH PERIODONTITIS

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## ABSTRACT

*Diabetes mellitus (DM) is a risk factor for periodontal diseases and oral complications are frequently encountered in diabetic patients. In recent studies it was hypothesed that, the gingival crevicular fluid (GCF) prostoglandin E2 (PGE2) levels are higher in type I diabetic individuals than non diabetics. The aim of our study is to determine the GCF PGE2 levels of the individuals with type II diabetes mellitus and periodontitis. In addition it is aimed to investigate to correlation between the GCF PGE2 levels, the clinical parameters and the severity of the disease. 20 type II diabetes mellitus patient (group I), and 20 systemic healthy (group II), a total of 40 individual with periodontitis were examined. All individuals pocket depth (PD), plaque index (PI), gingival index (GI), and gingival bleeding index (GBI) scores were recorded to determine their clinical status. In addition to these, the volume of GCF was also determined from the same sampling side. The GCF PGE2 levels were determined by radioimmunoassay (RIA) method. The GCF PGE2 levels of I. and II. groups were determined sequentially as  $61.88 \pm 28.71$  and  $13.30 \pm 4.953$  pg/nm. The level of PGE2 level was determined significantly higher ( $p < 0.001$ ) in the group of diabetes mellitus patients than systemic healthy group. Any correlation was not determined between the GCF PGE2 level and clinical parameters among both of the groups. By comparing all oral clinical parameters it was established that there was significant differences between the groups ( $P < 0.001$ ). The PD and GCF amount of sampling site was also statistically different among the groups ( $p < 0.05$ ), but the difference of other parameter scores were not statistically important ( $p > 0.05$ ). The findings of this study confirms that diabetes mellitus is a risk factor for periodontal diseases and the correlation between GCF PGE2 level and the severity of the periodontal disease. Attracts attention for the GCF PGE2 level which could be used as a marker to determine the periodontal disease severity among type II diabetes mellitus patients.*

## Introduction

There are many studies about the correlation between diabetes and the forming of oral diseases. As there is no consensus the main opinion is; diabetes mellitus is a risk

factor for periodontitis and the frequent co-occurrence of oral complication in diabetes mellitus patients (1, 2, 3, 4-17). In recent epidemiological studies it was reported that diabetes increases the severity of severe

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periodontitis or present periodontal disease 2 or 3 times more (18, 19). Very severe gingival inflammations, deep periodontal pockets, rapid bone loss and periodontal abscess are frequently seen among diabetes mellitus patients with poor oral hygiene (1, 8, 20, 21).

In long term studies it was suggested that the recurrency level after periodontal therapy is higher in diabetic patients than non diabetics and the mean bone and attachment loss is also higher than non diabetics (22, 23).

In the study of Emrich et al on a Pima Indian population it is stated that the severity of the periodontal disease is more exaggerated in type II diabetes mellitus patients than non diabetics (24).

Many investigations were carried out to determine the frequency of severe inflammatory gingival diseases, the correlation between gram (-) LPS (lipopolysaccharide) stimulation and GCF PGE2 level among diabetes mellitus patients (12, 22, 25-28). PGE2 is the strongest mediator of inflammation and plays important roles in the pathogenesis of periodontal diseases (29).

In recent studies type I diabetics and non diabetics were compared and it was found that the monocytic PGE2, IL-1B and TNF-A levels in GCF were 2-3 times more in diabetics than non diabetics. By the light of these data, it was suggested that; diabetic patients shows exaggerated inflammatory response when compared to non diabetics (12, 27).

In our investigation we could not find any literature focused on type II diabetic patients GCF PGE2 levels and its probable relation with clinical parameters. The aim of this study is to determine the GCF PGE2 level of type II diabetes mellitus patients with periodontitis. In addition it is aimed to investigate to correlation between the GCF PGE2 levels, the clinical parameters and the severity of the disease.

## Materials and Methods

### Study Population

20 individual clinically diagnosed as type II diabetes mellitus with periodontitis (mean age  $42.10 \pm 4.972$ ) and 20 systemic healthy with periodontitis (mean age  $38.45 \pm 2.605$ ) a total of 40 women who applied to Dicle University Medicine Faculty Endocrinology Department and Dentistry Faculty were included to our study. By selecting the patients care was taken for the presence of periodontitis, not to have any systemic problem except diabetes mellitus type II, to have minimum  $\geq 5$ mm PD and minimum 15 teeth, not to have an antibiotic, anti-inflammatory and periodontal therapy in last 6 months.

Any periodontal treatment was not performed to sampling site not to effect the present periodontal status. Also any order was not given to change their oral care. All individuals were informed about the study and their approval were got.

### Clinical Evaluation And Periodontal Examination

Clinical evaluation and GCF sampling procedures were made by one experienced clinician. To determine the patients periodontal status all mouth and sampling site plaque index (30)(PI), gingival index (31)(GI), pocket depth (PD), gingival bleeding index (32)(GBI) scores were determined by using a Williams probe and recorded. All measurements were performed on all teeth at 6 side which are distobuccal, buccal, mesiobuccal, distolingual, lingual and mesiolingual sides and expressed and recorded as milimeter.

### Getting Gingival Crevicular Fluid (GCF)

GCF samples were got with specifically manufactured paper ribbons (periopaper®) by the method of Rudin et al. (33). To prevent the contamination of the sampling material with saliva, in upper jaws anterior teeth, the method is limited with vestibular sides. Care was taken into account to the pocket depth (PD) to be  $> 5$  mm.

The sampling method was performed al-

TABLE 1

Data of full mouth and sampling site clinical parameters

	PD (mm)	PI	GI	GBI	GCF (mg)	PGE <sub>2</sub> (pg/ml)
I. group	2.496±0.459 <sup>#</sup>	2.314±0.452	1.545±0.289	%80.01±21.80	4.000±1.429*	61.88±28.71*
	3.831±0.577*	2.257±0.634	1.757±0.361	%86.83±21.07		
II. group	2.114±0.203	1.508±0.242	1.183±0.241	%47.78±11.16	1.530±0.551*	13.30±4.953*
	3.404±0.517	2.049±0.663	1.557±0.459	%84.16±23.24		

<sup>#</sup> Full mouth; \* Sampling site; Mean±Standart Deviation

ways in morning among all individuals. Before sampling method was performed, the site was isolated with cotton rolls, plaques were eliminated and the teeth surface were dried with gentle air spray. The periopapers were placed in the entrance of sulcus by the help of o pressel. After waiting 30 minutes the periopapers were weightened on a sessitive scale and placed into eppendorph tubes. The tubes were kept at -20C untill analysis method was performed. All individuals sampling sites were recorded. Care was taken not to cause bleeding during sampling method, but if happens the samples were not included to the study. The GCF samples were taken at first not to effect the fluid flow and volume. Just before the analysis method was performed 1000ml sterile NaCl (9mg/ml) was added in the tubes and GCF was seperated at 3000g and +5C for 20 min.

#### Laboratory Procedures

The analysis method of PGE<sub>2</sub> in GCF was performed in Dicle University Nuclear Medicine Department. The PGE<sub>2</sub> activity was determined by using commercial PROSTOGLANDIN E<sub>2</sub> [<sup>125</sup>I] RIA KIT\*. In the analysis method the guide of the kit was followed.

#### Statistical Analysis

'Student t test' was used to compare the groups. To compare full mouth clinical scores and sampling site clinical scores 'Two pairs difference signficancy test' was used. To compare the GCF volume and its probable relations with clinical parameters 'simple correlation analyse' was used. Statistically signficancy was taken from p<0.05 point.

## Results and Discussion

The mean and standart deviation scores of diabetes mellitus and systhemic healthy patients are shown in **Table 1**. All the full mouth parameters were significantly different among all the groups (PD; p<0.01, PI, GI and GBI; p<0.001). Among sampling sites values, while there was no statistical important difference between PI, GI, GBI scores (p>0.05) the PD score difference was statistically signficiant among all the groups (p<0.001). In the topic of GCF PGE<sub>2</sub> the 'Student t Test' was used to compare the groups and the difference between the groups found statistically signficiant (p<0.001).

In **Table 2** 'Simple Correlation Analyse' results in which the sampling site clinical parameters were compared are shown. In the diabetic group; important correlations were determined between PI and GBI, and between GI and GBI (sequentially p<0.05 and p<0.01). In the systhemic healty group; between PI and GBI, GI and GBI, GBI and GCF signficiant correlations were determined (p<0.01).

The PGE<sub>2</sub> level in GCF and its relation with sampling site parameters are shown in **Table 3**. the realation between PGE<sub>2</sub> level and sampling site clinical parameters were evaluated and were not found signficiant statistically (p>0.05).

There are many studies about the realation between diabetes mellitus and formation of oral diseases (24, 25). The research data establishes that the risk of formation of periodontitis is more in the diabetic individuals whose metabolic comtroll is poor

TABLE 2  
The 'Simple Correlation Analysis' results in which the sampling site clinical parameters were compared

	I. GROUP		II. GROUP	
	r	P	r	p
PI-GI	0.284	0.226	-0.227	0.336
PI-GBI	0.489	0.029*	-0.475	0.034*
PI-PD	0.001	0.997	0.022	0.926
PI-GCF	-0.115	0.630	-0.227	0.336
GI-GBI	0.842	0.000**	0.691	0.001*
GI-PD	-0.067	0.482	0.025	0.916
GI-GCF	-0.029	0.904	0.386	0.093
GBI-PD	0.122	0.607	-0.246	0.295
GBI-GCF	-0.207	0.380	0.593	0.006*
PD-GCF	0.044	0.854	-0.200	0.397

\* P<0.05, \*\* P<0.01

TABLE 3  
'The Correlation Analysis' results in which the GCF PGE<sub>2</sub> levels and sampling site clinical parameters were compared.

	I. GROUP		II. GROUP	
	r	p	r	p
PI- PGE <sub>2</sub>	-0.149	0.532	-0.134	0.574
GI- PGE <sub>2</sub>	-0.281	0.230	-0.394	0.085
GBI- PGE <sub>2</sub>	-0.064	0.787	-0.185	0.435
PD- PGE <sub>2</sub>	0.312	0.180	0.046	0.847
GCF- PGE <sub>2</sub>	0.182	0.443	-0.154	0.518

\* P<0.05

and the damage level is gigher (34, 35).

There are also many studies aimed to determine the periodontal status and the severity of the present periodontal disease in diabetes mellitus patients (7, 24, 36-38). It is proved by many researches that the GCF of type I idabetes mellitus patients includes more PGE<sub>2</sub> levels than non diabetics (12, 27, 29). But there is no study about the level of PGE<sub>2</sub> in the GCF of type II diabetes mellitus patients. This study is planned with the aim of determining the GCF PGE<sub>2</sub> level of type II diabetes mellitus patients, determining its probale relations with clinical parameters and estab-

lishing the probable differences among the groups.

Our study is performed on type II diabetes mellitus patients and systhemic healthy individuals who were all with periodontitis. Periodontal disease parameters and mediators in GCF are related primarily with the health of periodontium. Because of this, to determine the just the effects of diabetes all individuals included to the study were selected from periodontitis patients. The aim of standartically selected periodontitis patients is the impossibility of finding these group of patients without periodontitis especially in our population. To prevent our data from a probable cyclus rytım difference care was taken of the individuals to be in the 14. day of menstrual cyclus at the the of sampling.

All the clinical parameters were determined significantly higher in the diabetic group than the systhemic healthy group. With the knowledge of all our individuals included to the study were periodontitis patients and the diabetes patients higher scores evidence that diabetes is a risk factor (3, 7, 8, 10, 11, 13, 14, 15, 24). This data is in the same opinion with Emrich et al. who reported that the periodontal disease severity is more in diabetes mellitus patients than systhemic healthy individuals (24). The PD, PI, GI and GBI scores of sampling site were higher than full muoth scores in both of the groups. This data can be explained as we selected the sampling sites from the most damaged sites periodontally.

When the relations between the clinical parameters were observed it was determined that there was a strong and positive directed correlation between PI and GBI, GI and GBI among all the groups. It is accepted that bleeding after stimulation is the clinical sign of gingival inflammation (39). Because of the bacterial plaque is the main ethiologic factor of gingival inflammation, in our study the positive directed reletion between PI and GBI is an expected result. The GCF volume is determined signifi-

cantly higher in the diabetic group. Cima-soni suggested that there is a positive correlation between the gingival inflammation severity and GCF volume (40). In our study the because of the gingival inflammation severity was more in diabetic group, the high volume of GCF was also an expected result. In addition one of the main complications of diabetes is its effects on vascular system. The special vascular changes of diabetes is oftenly seen in very small arteries, arteriols, capillars and venules (41). The predisposition of the diabetic individuals to periodontitis especially to early onset form is explained by the impairment of capillar permeability (42). The high level of the GCF of the type II diabetes mellitus group in our study can be explained as capillar permeability impairment.

In the case of GCF PGE2 level, in our study it was determined that the healthy groups level is significantly lower than the type II diabetes mellitus group. This result is adjusted with Salvi et al. who suggested that the GCF PGE2 levels of type I diabetes mellitus patients with periodontitis were significantly higher than the non diabetic periodontitis patients (12, 27). In our study the probable relations between all the clinical parameters and the PGE2 level, but no statistical relation was not determined.

As a summary, in our study periodontal disease scores and GCF PGE2 levels are significantly higher than non diabetic patients. As it can be understood from our study, because of the type II diabetes mellitus is a risk factor the diabetic patients their periodontal controls should be performed regularly. By this way, oftenly asymptomatic periodontal diseases which is seen among diabetic individuals can be diagnosed in an easy way. In this study which we compared the two groups can not suggest any relation between PGE2 as a proinflammatory mediator and activity of periodontal disease. But our results can be thought as a different parameter which reflects the clinical periodontal status by

GCF PGE2 level. Our opinion is; long term studies in which among more type II diabetes mellitus patients included, GCF proinflammatory mediators and cytokin levels determined studies would be useful.

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