INTERLEUKIN-6 IN SALIVA & GCF LEVEL AFTER SRP IN CHRONIC PERIODONTITIS WITH & WITHOUT TYPE 2 DIABETES MELLITUS

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ABSTRACT

Background: Diabetes and periodontitis are common chronic diseases and epidemiological evidence implies a two-way relationship between the two diseases. Cytokines in saliva and gingival crevicular fluid (GCF) increased in chronic periodontitis with and without type 2 diabetes mellitus (T2DM) are associated with increased inflammation of periodontal tissue. Type 2 diabetes mellitus can worsen the periodontal condition, while periodontitis can also worsen glycemic control. Scaling and root planing (SRP) can improve glycemic control and reducing mediators of inflammatory cytokines such as interleukin-6 (IL-6).

Aim: To analyze comparison clinical parameters (Papillary Bleeding Index, Gingival Index, Pocket Depth, Clinical Attachment Loss), IL-6 level in saliva and GCF before and 2 weeks after SRP in chronic periodontitis patients with and without T2DM.

Methods: The study design was pre and post-test with samples of 15 subjects with chronic periodontitis with T2DM (CP + T2DM) and 15 subjects with chronic periodontitis (CP).

Results: There was a significant reduction in clinical parameters, IL-6 level in saliva and GCF after SRP in periodontitis patients with and without T2DM. Only PBI has a significant difference in clinical parameters changes after SRP between CP and CP+T2DM group. No significant difference in IL-6 level in saliva and GCF after SRP between chronic periodontitis patients with and without T2DM.

Conclusions: SRP was effective in improving clinical parameters and inflammatory parameters. IL-6 in saliva and GCF can be consider as monitor marker which can be act as a valuable tool in diagnostic potentials for the early detection of periodontal diseases.

KEYWORDS: Scaling and Root Planning; Interleukin-6; Chronic Periodontitis; Type 2 Diabetes Mellitus

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INTRODUCTION

Periodontal diseases is a common oral health problem with a fairly high prevalence in Indonesia, about 95,21% population at ≥ 30 years of age in Indonesia has periodontal diseases (National Basic Research of Indonesia in 2013).¹Chronic periodontitis is an inflammatory diseased characterized by inflamed gingiva, bleeding on probing, increased probing depth, slow progression of connective tissue destruction and alveolar bone resorption. Systemic

and environmental factor such as diabetes mellitus (DM) can affect the progression and severity of periodontitis.^{2,3}

National Basic Research of Indonesia in 2013 stated there were 6,9% proportion of Indonesia population or about 12 million people suffered from diabetes mellitus.⁴ Diabetes mellitus is a metabolic disorder characterized by elevated levels of glucose in the blood and has been considered a risk factor for periodontitis.^{5,6} The relationship between periodontal disease and diabetes mellitus is two way, meaning both can influence the severity to the other.⁵

The microbial challenge provokes an inflammatory response in the periodontal tissues that involves a network of cytokines functioning in synergy.⁵ Cytokines are water-soluble glycoproteins secreted by hematopoietic and non-hematopoietic cells in response to infection. The primary function of cytokines is intercellular signaling. An inflammatory cytokine is induced during inflammatory responses and is associated with the onset and/or progression of the disease. Cytokines play an important role in the host response to the periodontal film and they are pivotal to the pathogenesis of periodontal disease and may be used as diagnostic markers.^{7,8}

Interleukin-6 (IL-6) may promote the degeneration of inflamed periodontal tissues. IL-6 is an inflammatory cytokine that is secreted by monocytes, lymphocytes or endothelial cells at sites of tissue injury and it provokes the release of neutrophils and platelets from the bone marrow into the blood circulation as a part of acute and chronic inflammatory reactions.⁷ IL-6 stimulates osteoclast activation, thus, bone resorption and facilitates T-cells differentiation.⁵

IL-6 is an important mediator of host response to tissue injury and infection. It plays a major role in differentiation of B cell and also enhances proliferation of T cells and bone resorption. The correlation between level of IL-6 and the severity of the coincident inflammation is significant. The levels of IL-6 may correlate to stable with the severity of periodontal disease.⁷

Periodontal treatment such as SRP is a common and non-invasive periodontal therapy (phase I) which has been reported in controlling periodontitis in diabetics. Several clinical benefits of SRP such as reduction in probing depth, bleeding on probing, and gain in clinical attachment.⁹ Numerous studies have reported the beneficial effect of SRP on periodontal clinical parameters and improve metabolic control in T2DM patients.^{9,10}

The risk and severity of chronic periodontitis can be predicted by evaluating the IL-6 level. In the future, IL-6 level can be used as important biomarker for the diagnosis, prognosis and to predict the outcome of periodontitis.⁵ Hence this study aims to to analyze comparison IL-6 level in saliva and GCF before and after SRP in chronic periodontitis patients with and without T2DM.

MATERIALS AND METHODS

The study was an experimental research pre and post-test only group design. This study was comprised of 30 subjects patients divided into 2 groups (15 CP and 15 CP + T2DM) who were treated at the Department of Periodontics, Faculty of Dentistry, Universitas Sumatera Utara and Department of Internal Medicine, Faculty of Medicine, H. Adam Malik General Hospital, Medan, Indonesia. A written inform consent was obtained from all the subjects enrolled in the study. Ethical clearance was given and approved by the Health Research Ethical Committee, Medical Faculty of Universitas Sumatera Utara, Indonesia (No: 551/TGL/KEPK FK USU-RSUP HAM/2018).

The inclusion criteria were patients with clinical diagnosis chronic periodontitis with at least over 1-year defined by loss of attachment ≥ 3 mm at least in 2 teeth and presence of at least 15 teeth per mouth, aged range 35-65 years. The

exclusion criteria were any subject / patient who had other systemic disease (osteoporosis, cardiovascular diseases, immunology disorder, rheumatoid arthritis, tumor), pregnant and breast-feeding, smokers, alcoholic, consuming vitamin, antibiotics, anti-inflammatory drug during previous 3 months, used of immune suppressive medication, phenytoin, cyclosporine, calcium channel blockers or any medication that could interfere in the periodontium or in response to periodontal therapy, who has undergone periodontal therapy for chronic periodontitis in last 3 months and using mouth-rinse.

Diagnosis and classification of study of sample were established based on the basis of clinical parameters. The patients underwent a full-mouth clinical examination by a single examiner periodontics specialist at six sites per tooth (third molar excluded) using the periodontal probe UNC-15. Clinical parameters included probing depth (PD), clinical attachment loss (CAL), papillary bleeding index (PBI), gingival index (GI) were measured at baseline and 2 weeks after periodontal treatment.

Saliva was taken by spitting method and collected in saliva pot. GCF was taken using a micropipette from the deepest pocket for 30s and put inside the Eppendorf tubes and both samples was then stored at -80°C . After removing baseline samples of biofilm, full-mouth SRP was performed in all sites with periodontal disease in a single session. Oral hygiene instructions were given by a periodontist. The samples were collected in the same site at baseline and 2 weeks after SRP and were analyzed using enzyme-linked immunosorbent assay (Human IL-6 ELISA kit, Elabscience, USA). The concentration of IL-6 was examined using ELISA assay kits, and the result was analyzed by a microplate spectrophotometer (Multiscan Go, Thermo Fischer Scientific, USA).

Statistical Analysis

The numerical descriptive data were inputted into the computer and all the data obtained from the study were processed and analyzed using SPSS 20.0. Data processing began with the descriptive statistical analysis and hypothesis testing. Descriptive statistic were used to present the data in the form of a frequency distribution of data center size, such as the average value (the value of mean) and in the form of a data spread size, such as standard deviation (SD). The normality of all the data group distribution was analyzed by using Shapiro-Wilk Test. All data passed the normality test were tested for significant differences by using Paired T-Test. Data that considerably deviated from the normal distribution were analyzed using Wilcoxon test. $P < 0.05$ was considered to indicate a statistically significant difference.

RESULTS

Data collection was conducted by clinical examination, questionnaires on oral hygiene habit, saliva and GCF sampling. The purposive sampling technique was performed on the subject of 15 chronic periodontitis patients with (CP + T2DM) and 15 chronic periodontitis patients without T2DM (CP).

[Table 1] shows that statistically significant reduction of all clinical parameters 2 weeks after SRP in both groups. Both groups demonstrated reduced PBI after 2 weeks and improvement in oral hygiene.

[Table 2] show that changes in PBI 2 weeks after SRP in CP group has greater value than CP+T2DM and it is statistically different between both groups. No statistically differences in clinical parameters between both groups after SRP.

[Table 3] shows a statistically significant decrease in IL-6 level in saliva and GCF 2 weeks after SRP in chronic periodontitis with and without T2DM group.

[Table 4] also shows no significant difference changes in the mean IL-6 level in saliva and GCF at baseline and 2 weeks after SRP between chronic periodontitis with T2DM compared with those without T2DM group.

DISCUSSIONS

In the present study, CP+T2DM and CP groups were selected and treated by full mouth SRP to verify whether there were changes in clinical parameters and IL-6 level in saliva and GCF between both groups and baseline and 2 weeks after periodontal treatment. The average age in the chronic periodontitis group with T2DM (52.27 ± 7.73) years, meanwhile the chronic periodontitis group without T2DM (49.07 ± 10.38) years (Table 1).

The present study showed a significant reduction in all clinical parameters (PBI, GI, PD and CAL) after periodontal treatment in chronic periodontitis with and without T2DM groups (Table 1). Aziz AS et al. showed a significant reduction in clinical parameters on GI, PD and CAL in patients with periodontitis with and without T2DM before and after SRP.⁹ Meanwhile Camargo et al suggested that significant reduction in PBI, GI, PD and CAL in periodontitis group with and without T2DM 3 months after SRP.⁵ Zekeridou et al. showed a reduction in PBI and GI after 1 month and 3 months SRP.¹¹ Various studies above have reported improvement in periodontal clinical parameters in both groups after periodontal treatment which is in accordance with our study. The result of the present study only changes in PBI had a significant difference at baseline and 2 weeks after SRP between both groups (Table 2).

Reduction of clinical parameters after SRP was greater in CP group compared to CP+T2DM group probably due to the presence of systemic inflammation (low grade chronic systemic inflammation) in CP+T2DM group. Clinical parameters were re-evaluate again 2 weeks according to the time of the soft tissue healing re-epithelization process, reduction in inflammation in connective tissue and the formation of a long junction formation after periodontal treatment.^{12,13} Significant pocket depth reduction (initial stage) occurred 1 week after root planning and reduced further (secondary stage) at 3 weeks. Initial pocket reduction was associated with significant gingival recession, whereas secondary pocket reduction was associated with significant gain of clinical attachment.¹³

The present study showed that there was statistically significant reduction in IL-6 level in saliva and GCF after SRP in chronic periodontitis with and without T2DM group (Table 3). No significant difference in the mean IL-6 level in saliva and GCF at baseline and 2 weeks after SRP between chronic periodontitis with T2DM compared with those without T2DM group (Table 4).

Goutoudi et al. found that the mean of IL-6 level in GCF in chronic periodontitis group was 11.38 ± 4.73 pg/ml, and 6 weeks after SRP increased 33.56 ± 10.54 pg/ml.¹⁴ According to Alwan, the mean of IL-6 GCF level in chronic periodontitis group were 9.76 ± 2.98 pg/ml while those in the healthy group were 3.13 ± 1.71 pg/ml.¹⁵ Talbert et al. suggested that serum IL-6 level in chronic periodontitis patients with T2DM at baseline were 2.8 ± 3.9 pg/ml and after SRP were 6.0 ± 9.0 pg/ml.¹⁶ In this study the mean IL-6 level in GCF in the chronic periodontitis group without T2DM were 317.41 ± 58.89 pg/ml while in the periodontitis group with T2DM were 727.57 ± 121.34 pg/ml.

The results of the present study were consistent with Azis et al⁹ which showed a statistically significant reduction in IL-6 level after SRP in the chronic periodontitis with and without T2DM groups.⁹ D'Aiuto et al. reveals that periodontal

treatment can decrease inflammatory mediators due to inflammatory mediators such as IL-6 and improve clinical parameters. Pro-inflammatory cytokines, including IL-6, are inflammatory mediators that cause tissue destruction and induce connective tissue degradation and alveolar bone resorption. SRP is effective to eliminate bacterial plaque and remove calculus so as to reduce inflammatory mediators such as IL-6.⁶

In contrast, Zekeridou et al. suggested a significant increase in IL-6 level in the chronic periodontitis group after SRP.¹¹ Talbert et al. also showed an increase in serum IL-6 level that were not significant 3 months after SRP.¹⁶ The differences in the results of this present study with previous studies may be due to differences in the timing of the present study where the evaluation of this study was only carried out for 2 weeks. Increased IL-6 level suggest that shifting in the balance between the activities of pro-inflammatory and anti-inflammatory cytokines during the periodontal inflammation can affect the intensity and duration of inflammation.⁷ The concentration of IL-6 level is significantly higher in healthy areas than in the diseased area, while after periodontal treatment IL-6 level will increase significantly. This may be due to reduced gingival sulcus volume followed by the success of periodontal therapy. It has been stated that gingival sulcus total amount of cytokines may be more representative of disease conditions compared to its concentration.^{14,15}

Talbert et al. suggested that the difference in the results of these studies may be due to an increase in systemically stress caused by bacteria from plaques and calculus that enter the flow direction after SRP were carried out, so that there was an increase in inflammatory mediators.¹⁶ In addition, differences in the sampling area might also influence the differences in the results of the study, in which the samples were taken from saliva and gingival sulcus while in Talbert's taken from blood serum. According to Majeed et al., the selection of GCF samples as biomarkers due to their proximity to periodontal tissue which minimizes the possibility of reflecting responses from other inflammatory processes in the body.¹⁷ However, this present study showed a significant decrease in mean IL-6 level in saliva and GCF in the chronic periodontitis with T2DM and without T2DM group. This suggests that IL-6 level in saliva and GCF can act as biomarkers / indicators for diagnosis and predicting outcomes of periodontal treatment in chronic periodontitis and T2DM patients.

Interleukin-6 is an important cytokine involved in the regulation of host responses to tissue injury or infection. It is produced by various cells such as monocytes, fibroblasts, osteoblasts and endothelial cells in response to inflammatory challenges. IL-6 plays an important role in B cell differentiation and T-cell proliferation, while IL-6 synergistic with IL-1 β induces bone resorption. However, it can also increase TIMP production, suppress the expression of IL-1 and induce synthesis of IL-1Ra and release TNF receptors that show anti-inflammatory properties.¹⁴ In cases of periodontitis, pro-inflammatory cytokines, such as IL-6, are produced in response to bacterial plaque. Mengel et al. and Buhlin et al. suggested that periodontitis was associated with an increase in IL-6 level associated with disease severity. The persistent increase in IL-6 in cases of DM can have an effect on the liver and stimulate the formation of acute-phase proteins, which cause dysregulation from lipid metabolism associated with type 2 diabetes.⁶ Duarte et al. also showed higher IL-6 level in chronic periodontitis patients with T2DM that contributed to periodontal tissue damage.¹⁸ This was consistent with the results of this study which showed IL-6 level higher in periodontitis patients with T2DM both at baseline and after SRP.

Periodontal infections trigger the release of pro-inflammatory cytokines both in the area of periodontal infection that occurs and throughout the endothelial system.¹⁹ In the case of periodontitis, pro-inflammatory cytokines are produced in response to bacterial plaque. Pro-inflammatory cytokines, including IL-6, are inflammatory mediators that cause tissue destruction and induce degradation of connective tissue and resection of alveolar bone, causing loss of attachment levels

and an increase in pocket depth.⁷ IL-6 level in saliva increase with the severity of periodontitis. Pro-inflammatory cytokines are sensitive to pathogenic infections and can indicate the severity of periodontitis.²⁰

Increased severity of periodontitis has been associated with the development of glucose intolerance, possibly due to increased inflammation which causes an increase in IL-6. The liver is an important target for IL-6 work which leads to an increase in the inflammatory response with disruption of work and insulin signaling and causes a decrease in insulin production. Patients with impaired insulin production cannot control IL-6 activation and increase inflammation induced by IL-6. Increased level of IL-6 have been identified in untreated chronic periodontitis patients. Persson suggested that an increase in the levels of pro-inflammatory cytokines in T2DM patients caused by periodontitis may exacerbate the inflammatory response in other organs that are usually affected in T2DM patients.¹⁹

CONCLUSIONS

In this study, we observed a significant reduction in clinical parameters (PBI, GI, PD, CAL) and IL-6 level in saliva and GCF after periodontal treatment in chronic periodontitis with and without T2DM. There was no significant difference in IL-6 level in saliva and GCF after SRP between chronic periodontitis patients with and without T2DM. Only PBI has significant differences in clinical parameters changes after SRP between chronic periodontitis with and without T2DM group. The SRP therapy was effective in improving clinical parameters and inflammatory parameters. Local production of IL-6 in saliva and GCF can be consider as monitor marker which can be act as a valuable tool in diagnostic potentials for the early detection of periodontal diseases.

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TABLES

Table 1: Distribution of Clinical Parameters at Baseline and 2 Weeks After SRP of Chronic Periodontitis With and Without T2DM Group

Clinical Parameters	CP + T2DM		P Value	CP		P Value
	Baseline	2 Weeks		Baseline	2 Weeks	
PBI	39,73 ± 20,65	9,80 ± 5,57	0,000*	51,47 ± 13,25	8,13 ± 2,62	0,000*
GI	1,03 ± 0,41	0,12 ± 0,10	0,000*	1,22(0,40-2,00)	0,22(0,08-0,50)	0,001 [†]
PD (mm)	4,11 ± 1,16	3,33 ± 1,15	0,000*	3,59(2,23-6,39)	2,95(1,63-5,11)	0,000 [†]
CAL (mm)	5,81 ± 1,76	4,83 ± 1,76	0,000*	5,61 ± 1,61	4,88 ± 1,44	0,000*

*Paired t-test ; [†]Wilcoxon test ; P<0.05 Significant Difference**Table 2: Differences in Clinical Parameters Changes at Baseline and 2 Weeks After SRP between Chronic Periodontitis With and Without T2DM Group**

Clinical Parameters	CP + T2DM	CP	P Value*
Δ PBI	29,93 ± 16,35	43,33 ± 12,57	0,018
Δ GI	0,76 ± 0,39	0,97 ± 0,32	0,125
Δ PD (mm)	0,77 ± 0,26	0,71 ± 0,23	0,324
Δ CAL(mm)	0,98 ± 1,05	0,73 ± 0,38	0,317

*Independent t-test ; Δ Difference between Baseline and After 2 weeks SRP; P<0.05 Significant Difference

Table 3: IL-6 Level in Saliva and GCF at Baseline and 2 Weeks After SRP between Chronic Periodontitis With and Without T2DM Group

Variable	CP + DMT2		P Value	CP		P Value
	Baseline	2 Weeks		Baseline	2 Weeks	
IL-6 saliva	153,21 (102,98- 266,27)	132,45 (84,4-220,74)	0,000*	138,43 ± 89,12	104,75 ± 69,14	0,000 [†]
IL-6 GCF	727,57 ± 121,34	619,06 ± 140,72	0,000 [†]	317,41 ± 58,89	239,03 ± 65,40	0,001 [†]

*Wilcoxon test ; [†]Paired t-test ; P<0.05 Significant Difference**Table 4: Differences in Changes IL-6 Level in Saliva and GCF at Baseline and 2 Weeks After SRP between Chronic Periodontitis With and Without T2DM**

Variable	CP + T2DM	CP	P Value*
Δ IL-6 saliva	32,08 ± 14,62	33,68 ± 19,98	0,166
Δ IL-6 GCF	108,51 ± 19,38	78,38 ± 6,51	0,882

*Independent t-test; Δ Differences between Baseline and 2 Weeks After SRP; P<0.05 Significant Difference