The effect of non-surgical periodontal therapy on HbA1c levels: A systematic review

Paolo Boffano,1 Andrea Melle,1 Anna M. Agnone,1 Vincenzo Rocchetti1,2 Muhammad Ruslin3

Abstract

**Objective:** The purpose of the present article is to summarize the evidence present in systematic regarding the non-surgical periodontal therapy effects on HbA1c levels.

**Method:** Our study considered the consultation of the bibliographic databases “Medical Systematic Analysis and Retrieval System”. The research was carried out by using the keywords: “Diabetes mellitus” AND “Periodontal disease” AND “Scaling and root planing” AND “Non-surgical periodontal treatment” AND “Glycosylated hemoglobin”. Our research resulted in 18 articles published between 1995 and 2018.

**Results:** All studies have shown that periodontal indices have improved after non-surgical periodontal therapy, while the effects on metabolic control are controversial. Some authors find no influence on metabolic variables, others report an improvement. Some studies have used both topical and systemic antibiotics in combination with causal periodontal therapy, observing an improvement in metabolic compensation.

**Conclusion:** The approach to diabetic patient with periodontitis must be multidisciplinary with close collaboration between periodontists, dental hygienists and endocrinologists.

**Keywords:** Diabetes, Glycosylated hemoglobin, Periodontal disease, Periodontitis, Scaling

DOI: 10.15562/jdmfs.v8i3.1648

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**Introduction**

Considering all the existing correlations between diabetes and periodontal disease, the latter is considered the sixth complication of diabetes.1,50 The American Diabetes Association (ADA) has divided diabetes mellitus based on its etiology into: Type 1 Diabetes, characterized by β-cells destruction; Type 2 Diabetes, characterized by insulin resistance; Other Specific Types (genetic defects, pancreatic diseases, endocrinopathies) and Gestational Diabetes Mellitus, characterized by glucose intolerance in pregnancy.7 Diabetes diagnostic criteria include fasting glycemia ≥126 mg/dL or 7 mmol/L, with plasma sampling performed at least after 8 hours of fasting; HbA1c ≥6.5%; glycemia ≥200 mg/dL, 120 min after consuming 75 g of glucose.11

Furthermore, with the presence of symptoms the diagnosis could be carried out with random glycemia ≥200 mg/dL, regardless of recent food intake.11 Diabetes complications are divided into microvascular complications, which include retinopathy, nephropathy, neuropathy and atherothrombotic macrovascular complications.13

The World Health Organization (WHO) has approved the use of glycated hemoglobin (HbA1c) as a screening test on people at high risk of developing diabetes mellitus, above all, as a test for predicting the risk of microvascular complications.12

Systematic shows that diabetic subjects have a higher grade of gingival inflammation and more severe form of periodontitis, characterized by a greater loss of connective tissue and alveolar bone,
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Table 1  Review of the literature

<table>
<thead>
<tr>
<th>Diabetes type</th>
<th>Type of study</th>
<th>Study population</th>
<th>Follow up</th>
<th>Therapy</th>
<th>Initial HbAc1(%)</th>
<th>Post-therapy HbAc1(%)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>DM1</td>
<td>Randomized controlled</td>
<td>23 patients</td>
<td>2 months</td>
<td>Group 1 = scaling and root planning</td>
<td>9.8</td>
<td>10.4</td>
<td>NA</td>
</tr>
<tr>
<td>DM1</td>
<td>Clinical</td>
<td>36 patients</td>
<td>2 months</td>
<td>Group 2 = no therapy</td>
<td>9.7</td>
<td>9.5</td>
<td>NA</td>
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<tr>
<td>DM1</td>
<td>Cohort</td>
<td>40 patients</td>
<td>4 months</td>
<td>Group 1 = scaling and root planning</td>
<td>8.18</td>
<td>8.28</td>
<td>NA</td>
</tr>
<tr>
<td>DM2</td>
<td>Clinical</td>
<td>72 patients</td>
<td>10 months</td>
<td>Group 2 = no therapy</td>
<td>6.5</td>
<td>6.7</td>
<td>NA</td>
</tr>
<tr>
<td>DM2</td>
<td>Randomized controlled</td>
<td>44 patients</td>
<td>3 months</td>
<td>Group 1 = scaling and root planning extraction</td>
<td>4.3</td>
<td>4.7</td>
<td>p=0.0001</td>
</tr>
<tr>
<td>DM2</td>
<td>Cohort</td>
<td>20 patients</td>
<td>6 months</td>
<td>Group 1 = scaling and root planning</td>
<td>7.45</td>
<td>7.16</td>
<td>NA</td>
</tr>
<tr>
<td>DM2</td>
<td>Randomized controlled</td>
<td>60 patients</td>
<td>3 months</td>
<td>Group 1 = scaling and root planning doxycyclin</td>
<td>7.64</td>
<td>7.71</td>
<td>p=0.44</td>
</tr>
<tr>
<td>DM2</td>
<td>Clinical</td>
<td>45 patients</td>
<td>3 months</td>
<td>Group 1 = scaling and root planning</td>
<td>7.51</td>
<td>7.45</td>
<td></td>
</tr>
<tr>
<td>DM2</td>
<td>Randomized controlled</td>
<td>60 patients</td>
<td>6 months</td>
<td>Group 1 = scaling and root planning (poorly controlled diabetes)</td>
<td>9.96±1.45</td>
<td>9.77±1.15</td>
<td>p=0.427</td>
</tr>
<tr>
<td>DM2</td>
<td>Cohort</td>
<td>75 patients</td>
<td>12 months</td>
<td>Group 1 = scaling and root planning (mild periodontitis)</td>
<td>7.00±0.93</td>
<td>6.74±0.81</td>
<td>p=0.039</td>
</tr>
<tr>
<td>DM2</td>
<td>Randomized controlled</td>
<td>28 patients</td>
<td>6 months</td>
<td>Group 1 = scaling and root planning</td>
<td>7.87±0.74</td>
<td>7.16±0.69</td>
<td>p&lt;0.01</td>
</tr>
<tr>
<td>DM2</td>
<td>Randomized controlled</td>
<td>475 patients</td>
<td>6 months</td>
<td>Group 1 = scaling and root planning; 0.12% chlorhexidine mouthrinse</td>
<td>7.84±0.65</td>
<td>7.99</td>
<td>p=0.50</td>
</tr>
<tr>
<td>DM2</td>
<td>Randomized controlled</td>
<td>60 patients</td>
<td>3 months</td>
<td>Group 1 = scaling and root planning; 0.12% chlorhexidine mouthrinse</td>
<td>7.68±0.63</td>
<td>7.10±0.64</td>
<td>p&lt;0.004</td>
</tr>
</tbody>
</table>

Porphyromonas gingivalis was found in diabetic subjects compared to healthy patients. In particular, a higher prevalence of have been isolated in diabetic patients. In periodontal disease, as collagen is the main structural protein in the periodontium. Causing microangiopathy (typical of diabetes), as wall thickening and narrows the lumen destruction. The altered collagen synthesis also contributes to wound healing alterations with a progression of as AGE modified collagen leads to a vessel wall thickening and narrows the lumen destruction. The pro-inflammatory response resulting in tissue destruction. A greater number of periodontal pathogens also contribute to wound healing alterations. AGP modified collagen leads to a vessel wall thickening and narrows the lumen destruction. The pro-inflammatory response resulting in tissue destruction.
## A SYSTEMATIC REVIEW

<table>
<thead>
<tr>
<th>Diabetes type</th>
<th>Type of study</th>
<th>Study population</th>
<th>Follow up</th>
<th>Therapy</th>
<th>Initial HbA1c (%)</th>
<th>Post-therapy HbA1c (%)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>DM2</td>
<td>Randomized controlled</td>
<td>126 patients</td>
<td>4 months</td>
<td>Group 1 = scaling and root planning Group 2 = home oral hygiene</td>
<td>9.0±2.3</td>
<td>8.4±1.9</td>
<td>p=0.09</td>
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<tr>
<td>DM2</td>
<td>Randomized controlled</td>
<td>56 patients</td>
<td>3 months</td>
<td>Group 1 = scaling and root planning Group 2 = no therapy</td>
<td>8.49±1.50</td>
<td>8.47±0.89</td>
<td>p=0.347</td>
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<tr>
<td>DM2</td>
<td>Randomized controlled</td>
<td>30 patients</td>
<td>3 months</td>
<td>Group 1 = scaling and root planning doxycyclin Group 2 = no therapy</td>
<td>8.13±0.67</td>
<td>7.31±0.60</td>
<td>p&lt;0.0001</td>
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<tr>
<td>DM2</td>
<td>Randomized controlled</td>
<td>100 patients</td>
<td>6 months</td>
<td>Group 1 = scaling and root planning Group 2 = no therapy</td>
<td>8.09±0.76</td>
<td>8.16±0.74</td>
<td>p&lt;0.05</td>
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<tr>
<td>DM2</td>
<td>Randomized controlled</td>
<td>30 patients</td>
<td>3 months</td>
<td>Group 1 = scaling and root planning: 0.20% chlorhexidine mouthrinse 10% povidone-iodine Group 2 = no therapy</td>
<td>7.87±2.56</td>
<td>8.06±2.72</td>
<td>p&lt;0.001</td>
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Increased bleeding on probing and tooth mobility with consequent tooth loss.5,13

A chronic hyperglycemia condition leads the formation of AGEs, which contribute to the destruction of periodontal tissues.6,16

The glycation products action is carried out by binding to a membrane receptor called “receptor for advanced glycation end products” (RAGE) located on macrophage cells surfaces, which helps to activate the pro-inflammatory response resulting in tissue destruction.4,15,16

AGE modified collagen leads to a vessel wall thickening and narrows the lumen causing microangiopathy (typical of diabetes), as well as in the periodontium.9,25

The altered collagen synthesis also contributes to wound healing alterations with a progression of periodontal disease, as collagen is the main structural protein in the periodontium.9

A greater number of periodontal pathogens have been isolated in diabetic patients. In particular, a higher prevalence of Porphyromonas gingivalis was found in diabetic subjects compared to healthy patients.5,17,18

Peripheral blood monocytes of DM subjects produce high levels tumor necrosis factor-alpha (TNF-α) caused by porphyromonas gingivalis antigens compared to non-diabetic control subjects.19

The increase of TNF-α serum concentration can exacerbate periodontal disease through the stimulation of fibroblasts and osteoclasts, which can furthermore synthesize degradation enzymes (MMPs) and induce bone resorption.20,21

Diabetic subjects with periodontal disease with HbA1c >8% levels showed interleukin-1 β (IL-1β) levels in the crevicular fluid that were almost twice the amount compared to subjects with HbA1c <8% levels.19

Out of all the oral manifestations, hyposialia is among the most well-known (10% to 30%). Hyposialia is caused by a degeneration of the salivary glands, which causes glossodynia, ulcers, angular cheilitis, cavities and removable dentures instability which can cause soft tissues traumas and predispose to oral infections.3,24

Burning mouth syndrome (BMS) and dysgeusia can both signal the onset of diabetic peripheral neuropathy.14,24

HbA1c level higher than 12% is predictive of mycotic oral infections regardless dentures use.22,25

It has been shown that 52% of patients suffer from halitosis, which is the second most common oral complication in diabetic patients; however, a higher prevalence (76%) was observed among patients with uncompensated diabetes.22

A systemic spread of periodontal pathogens can cause bacteremia or endotoxemia, which induce an elevated inflammatory state and stimulate high levels of serum inflammatory markers.20 Loos has reported that the surface of the infected area in periodontal patients varies between 15-20 cm²; moreover, the presence of subgingival bacteria, which form highly organized colonies, represent a constant source of gram-negative bacteria.27,28

Lipoplysaccharide (LPS) is one of the main components of the outer cell membrane of gram-
negative bacteria; it is capable of stimulating macrophages and inducing the secretion of various inflammatory mediators (prostaglandins, interleukins, acid phosphatase). Pro-inflammatory cytokines can reach high concentrations in periodontal defects tissues, constituting a source through which these mediators continuously pour themselves into the circulation. The increased production of cytokines, including IL-6 and TNF-α, which interfere with the normal insulin signal transmission through the phosphorylation of the insulin receptor substrate (IRS-1) favoring the onset of insulin resistance and further propagating the state of chronic inflammation. Insulin resistance is the most important pathophysiological feature of prediabetes and DM2 stages.9,20

The purpose of the present article is to summarize the evidence present in systematic regarding the non-surgical periodontal therapy effects on HbA1c levels.

Methods

Our study considered the consultation of the bibliographic databases "Medical Systematic Analysis and Retrieval System". The research was carried out by using the keywords: "Diabetes mellitus" AND "Periodontal disease" AND "Scaling and root planing" AND "Non-surgical periodontal treatment" AND "Glycosylated hemoglobin".

Only articles with subjects with at least 4 non-contiguous sites with probing depth >5 mm and with at least 10 teeth were included. Our research resulted in 18 articles published between 1995 and 2018. PICO inclusion and exclusion criteria were adopted.

Results

All the analyzed studies have similar inclusion and exclusion criteria table 1. Subjects with at least 4 non-contiguous sites with probing depth >5 mm and with at least 10 teeth were considered to be affected by chronic periodontitis. All patients suffering from other concomitant pathologies, patients taking other drugs in addition to hypoglycemic drugs (oral or insulin), subjects who underwent periodontal therapy within the six months prior to enrollment in the protocols and patients in pregnancy / breastfeeding, were excluded.

The first 4 examined papers concern only periodontal disease patients with DM1. The assessment by PI (plaque index), GI (gingival index), PD, CAL, and BOP (bleeding on probing) indexes were recorded. In some studies, chlorhexidin mouthrinses were associated with scaling.

Discussion

In 1995 evaluated non-surgical periodontal therapy effect in a sample of DM1 patients. 23 patients, aged between 20 and 60 years, underwent blood sampling for HbA1c and fill out periodontal indices; 12 subjects (SG) were subjected to SRP (scaling and root-planing) and motivated to home oral hygiene care. A statistically significant improvement in periodontal values (p<0.01) was observed after 3 months, but no decrease in HbA1c values as in the control group consisting of 11 DMI patients not treated with SRP.20

In 1996 evaluated periodontal therapy effect in a sample of 18 patients with DM1 (SG), while the CG was formed by 18 subjects comparable by sex and age, but without any periodontal and metabolic alteration. The data concerning the periodontal indices, the clinical attachment level (CAL) and the HbA1c value were analyzed. The same values were re-evaluated 2 months after the non-surgical periodontal therapy. The results show a statistically significant improvement in the periodontal conditions of the subjects in the study group, while no statistically significant difference was observed in the HbA1c values.21

Similar results with a cohort of 40 patients; study group (SG=20) comprehending 7 with DM1 and 13 with DM2. With a range of HbA1c between 4.4 and 10.6 and moderate / severe periodontitis. The control group (CG=20) consisted of healthy patients, without metabolic and periodontal alterations. The SG underwent SRP, while the CG did not receive any therapy. The 4-month follow-up demonstrated a reduction in periodontal indices, but showed no impact on the level of metabolic compensation.22

In 2005, 60 patients with periodontitis and type 1 diabetes mellitus were divided into study group (SG=30) and control group (CG=30). 22 patients showed a good glycemic control (HbA1c <7%), 15 individuals had moderate control (HbA1c 7%-8%), while 23 subjects showed poor metabolic control (HbA1c >8%). The groups were subjected to SRP in association with 0.20% chlorhexidine mouthwashes for 12 weeks. In the study group (SG) was administered doxycycline 100 mg/day for 15 days as well. The variation of HbA1c values in SG was not statistically significant.23

Following studies only considered patients with type 2 diabetes mellitus. Stewart et al. in 2001 evaluated the effects of periodontal therapy (SRP) on a glycemic control in a sample of 72 patients with type 2 diabetes mellitus. The study group consisted of 36 patients, including 6 on diet therapy, 19 on oral hypoglycemic therapy and 11 on insulin therapy. The control group consisted of 36 patients with
type 2 diabetes mellitus; they were not subjected to periodontal therapy. All SG patients received non-surgical periodontal therapy and an extraction of compromised tooth elements. The control of the HbA1c values after 10 months from the causal therapy showed the reduction from 9.5% to 7.6% in SG.24

Statistically significant changes in CG were observed in the oral hypoglycemic group and in the insulin therapy group. During the same period of time also in the CG the levels of HbA1C decreased from 8.5% to 7.7%, this inconsistency is justified by the Authors with the simultaneous change of the therapeutic protocol in the reference center.25

In 2005 evaluated the possible effects of periodontal therapy on metabolic compensation in a study of 44 subjects with type 2 diabetes mellitus and periodontitis divided equally into two groups (SG=22; CG=22) made up of patients with HbA1c values between 6% and 8%. SRP therapy was performed only in the SG. The 3-month follow-up showed an improvement in periodontal parameters and a decrease in glycated hemoglobin of about 10.94% compared to the base level in the SG (p<0.0001).26

In 2006 on a sample of 20 periodontal patients, consisted of the study group (SG=10) diagnosed with type 2 diabetes mellitus and the control group (CG=10) non-diabetic. SRP therapy was performed only in the SG, with subsequent supportive therapy 3 months later. The 6-month follow-up showed a statistically significant reduction in HbA1C in SG at both 3 and 6 months, as well as in periodontal values.27

In 2009 comprised 45 periodontal patients, including 30 diagnosed with type 2 diabetes mellitus, 15 (SG1A) poorly controlled diabetics (Hb1Ac ≥ 7%) and 15 patients (SG1B) with good metabolic control (Hb1Ac < 7%). The control group (CGp) consisted of 15 periodontal patients without metabolic alterations. Patients with at least 15 teeth, with 4 sites of PD ≥ 5 mm and CAL ≥ 3 mm were included. After the collection of periodontal indices and radiographic examinations (RX OPT), SRP was performed in the whole cohort. The result at the follow-up (3 months) showed a periodontal improvement in the group of patients with uncompromised diabetes (SG1A) and a statistically insignificant result for the Hb1Ac values (p = 0.427). In the second group, a statistically significant reduction in periodontal and Hb1Ac values (p<0.002) was reported. In the CGp, only the significant reduction of periodontal parameters was highlighted.10

A cohort of 60 patients with moderate to severe periodontal disease and type 2 diabetes mellitus divided into study group (SG=30) subjected to non-surgical periodontal treatment and in the control group (CG=30) where only supragingival oral hygiene was performed. Periodontal parameters and HbA1c values were evaluated at 1, 3 and 6 months. At follow-up, improvements in periodontal parameters were observed in SG. HbA1c levels decreased more in SG than in CG (0.72% vs 0.13%; p<0.01) regardless of the other variables (age, sex, BMI, smoke).27

In 2012 included 75 patients with type 2 diabetes mellitus (Hb1Ac 7.3 ± 0.94) divided into two study groups according to the grade of periodontal disease: 21 patients (SG1) with moderate periodontitis (n < 2 elements with CAL ≥ 6 mm and < 1 tooth with PD ≥ 5 mm) and 54 patients (SG2) with severe periodontitis (> 2 elements CAL ≥ 6 mm and > 1 tooth with a PD ≥ 5 mm). Non-surgical periodontal therapy was performed in both groups, and oral hygiene sessions were planned at 3, 6, 9 and 12 months apart. The 12-month follow-up showed an improvement in periodontal parameters, a slight decrease in Hb1Ac values (SG1= 6.74 ± 0.81; SG2= 7.22 ± 1.02) (p= 0.039).28

In 2012 recruited 28 periodontal patients with type 2 diabetes mellitus, the cohort was divided into two study groups of 14 subjects each. In SG1 SRP was performed, while in SG2 SRP was performed with 2% subgingival minocycline supplement (once administration week for one month). After 6 months, a similar reduction in HbA1c values was observed in both groups. Regarding PD, there was a reduction in both groups (p = 0.0473) with better results in the one with applications of minocycline (SG2).29

In 2013 considered a sample of 475 patients diagnosed with periodontitis and type 2 diabetes mellitus (Hb1Ac 7%-9%). The study group (SG= 240) received SRP associated with 0.12% chlorhexidine mouthwash for 2 weeks. The control group (CG= 235) received only the motivation for home oral hygiene. After 6 months, a slight decrease in HbA1c levels was recorded but not statistically significant (p= 0.50).30

In 2013 examined on a sample of 60 subjects aged between 35 and 45 years on oral hypoglycemic therapy divided randomly into 3 groups: first study group (SGa= 20) received SRP combined with 0.12% chlorhexidine mouthwash, second group study (SGb= 20) only rinses with 0.12% chlorhexidine mouthwash and control group (CG= 20) no therapy. After 3 months from the SRP, the levels of HbA1c, fasting glucose, depth 6 of the probing pocket (PD), gingival index (GI), plaque index (PI) were measured. It is noted that after SRP, the values of PD, fasting glycemia and HbA1c improved (p = 0.004) in the SGa and SGb groups, while in the CG they were not statistically significant.31

In 2014 examined 126 periodontal patients with type 2 diabetes mellitus divided into two groups: SG=...
66 and CG= 60. The mean baseline HbA1c value was 9.0 ± 2.3 in the SG and 8.4 ± 2.0 in the CG. There were no statistically significant differences in HbA1c values between the study and control groups 4 months after causal therapy.26

In 2019 recruited 56 subjects diagnosed with type 2 diabetes mellitus on oral hypoglycemic therapy and suffering from periodontitis. The SG (=28) received SRP and motivation, the CG (=28) only motivation for home oral hygiene. At the 3-month follow-up, improvements in periodontal indices were recorded in SG, but there were no statistically significant changes in HbA1c levels (p=0.347).27

In 2015, 30 patients were recruited and randomly divided into two groups. The SG (=15) received SRP followed by systemic doxycycline (100 mg/day for 14 days), while the CG (=15) received no periodontal and drug treatment. All periodontal parameters, serum levels of TNF-α and HbA1c (p<0.0001) were significantly decreased in the SG group, no significant changes were observed in the CG.28

In 2015 selected a cohort of 100 subjects with type 2 diabetes mellitus, divided into two groups: a study group (SG=50) subjected to SRP, and the control group (CG=50) with equivalent characteristics but not subjected to SRP. After 6 months of observation, regression analysis demonstrated the improvement of the periodontal status regardless of the glycemic improvement. SRP therapy improved glycemic control in SG (p<0.05).29

From 2018 on 30 periodontal disease subjects with type 2 diabetes mellitus divided into two groups. Patients in the study group (SG= 15) were subjected to SRP with subgingival irrigation based on 10% povidone iodine in combination with 0.20% chlorhexidine mouthwash for 5 days. In the control group (CG=15), however, no treatment was carried out other than the motivation for home oral hygiene. The observation time was 3 months, the results showed the statistically significant reduction (p<0.001) of the HbA1c levels in the SG; on the contrary, in the CG no significant differences emerged. Although there is a wide heterogeneity in the population analyzed in terms of age, type of diabetes, different therapies, the analysis of these works leads to the conclusion that there is an association between periodontal disease and diabetes regardless of the type of diabetes. All studies have shown that periodontal indices have improved after non-surgical periodontal therapy, while the effects on metabolic control are controversial. Some authors find no influence on metabolic variables, others report an improvement.30

Some studies have used both topical and systemic antibiotics in combination with causal periodontal therapy, observing an improvement in metabolic compensation.31

A review of the randomized clinical trials highlighted the improvement of periodontal indices in all treated patients, a reduction in HbA1c levels was revealed especially in patients with decompensated diabetes, but without reaching statistical significance.32

In particular, the variations in HbA1c after nonsurgical periodontal therapy on patients with type 1 diabetes mellitus were very limited, since the effect of insulin therapy covers the possible variations due to causal therapy.10,31,32

**Conclusion**

It can be suggested to perform a primary prevention program for diabetic subjects without periodontitis in order to motivate the patient, to make him aware of the essential importance that home oral hygiene assumes for the achievement and maintenance of the oral health, and to illustrate him hygienic devices and related application methods. A diabetic subject suffering from periodontitis may undergo periodontal therapy (initially, non-surgical type, and subsequently maintenance). Diabetic patient may not be treated only with NSPT or SPC but may need Periodontal surgery according to specific needs. Failure of reaching pocket closure in a diabetic patient may not only lead to peridontal deterioration but also may contribute to poor glycemic control. After NSPT is delivered Periodontal surgery may be indicated to achieve pocket closure even in a diabetic patient. Furthermore the recent EFP guidelines in Periodontal therapy for stage I-III periodontitis stated that SPC interval should be personalized. While a recall frequency between 3 and 6 months seems to be effective in the majority of cases, there is not enough evidence to suggest a specific interval to maintain peridontal health. For this reason, reviewing the currently available and ever-changing international systematic, in reference to randomized clinical trials and systematic reviews, the approach to diabetic patient with periodontitis must be multidisciplinary with close collaboration between periodontists, dental hygienists and endocrinologists.

**Acknowledgment**

None.

**Conflict of Interest**

The authors report no conflict of interest.

**References**
References


