Role of Diabetes Mellitus on Maintenance of Periodontal Disease

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Received: February 23, 2017; Published: May 22, 2017

Abstract

Periodontitis is defined as a chronic infectious disease caused by periodontal bacteria. Chronic periodontal inflammation results in alveolar bone loss and periodontal supportive tissue, eventually leading to tooth loss. Type 2 diabetes mellitus is mainly characterized by hyperglycemia because of impaired action of insulin. Though the etiology of the two diseases is different, number of reports have shown a close association between periodontitis and diabetes mellitus.

Keywords: Diabetes Mellitus; Periodontal Diseases; Advanced Glycation End Products; Glycemic Control

Introduction

Diabetes mellitus is one of the major risk factors for periodontitis, and, vice versa, periodontitis is considered to increase the risk of developing diabetes mellitus. The effect of periodontal intervention therapy on glycemic control has been examined for many years, and many studies to validate these effects have been reported. However, to the best of our knowledge, no studies reported the effect of improved glycemic control by glycemic intervention therapy on periodontitis. The aim of the present review was to examine whether improvement of glycemic control by glycemic intervention therapy affects periodontal disease in type 2 diabetic patients without treatment of periodontitis [1].

Effect of Diabetes Mellitus Control on Progression and Maintenance of Periodontal Disease

Many studies addressed the effect of improved periodontitis by periodontal intervention therapy on glycemic control, and recently, Teeuw, et al. carried out a meta-analysis by selecting five reliable studies among 639 intervention studies, and they found a significant decline of 0.4% in HbA1c after periodontal intervention therapy in type 2 diabetic patients.

Hence, it is reasonable to consider that in type 2 diabetic patients effective periodontal intervention therapy improves glycemic control. However, to our knowledge, no studies reported the effect of glycemic intervention therapy without periodontal treatment on periodontal status in type 2 diabetes.

An epidemiological study by Offenbacher, et al. showed that diabetes augmented the gingival inflammatory responses against plaque biofilm, and the diabetes prevalence was significantly higher in the gingivitis patients with high BOP lesions compared with low BOP lesions. Thus, the amelioration of diabetes may improve the gingival inflammatory responses. Gingival tissue recession is dependent on gingival biotype (thickness of the periodontal tissue) and resolution of the inflammation. In contrast, deep PPD lesions are the result of irreversible destruction of the gingival tissue attachment and/or periodontal ligament on the root surface. PPD restoration is the result seen clinically from the attachment gain of the gingival tissue on the root and/or the gingival tissue recession. The diseased root surface which is contaminated with bacteria plaque is debrided for the gingival tissue to gain the attachment. Therefore, diabetic treatment alone might not be enough to restore deep PPD though the recession of gingival tissue [1].

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It has been established that bacterial microflora at periodontal disease sites in diabetic patients are similar to the microflora at similar disease sites in non-diabetic subjects and thus, the causes of susceptibility and severity of periodontitis in type 2 diabetic patients come from host problems. Diabetic complications are a result of complex abnormalities on the host side because of chronic hyperglycemia. Inflammation, advanced glycation endproducts (AGEs), oxidative stress, microangiopathy and macroangiopathy are the factors that cause these abnormalities. We can consider that these abnormalities makes diabetic patients susceptible to bacterial infection in the periodontal tissue. Many studies have shown that effective periodontal treatment in type 2 diabetic patients improves glycemic control [1].

Periodontitis is a chronic, subclinical, inflammatory disease that increases the risk of onset and progression of diabetes mellitus and arteriosclerosis. Taken with the present and previous results, it is conceivable that glycemic treatment could be carried out simultaneously with periodontal treatments in the cases of type 2 diabetic patients with periodontitis; it might facilitate glycemic control and, furthermore, it might prevent and inhibit the development of arteriosclerosis.

Periodontitis is the important complication of diabetes mellitus and it varies significantly varies with the glycemic control. Higher frequency of periodontitis was reported in the measures of indices used, and shows significant difference in the controlled and uncontrolled group of diabetic patients. This finding correlates with that of many previous studies thus proposing that diabetes is a risk factor for periodontal disease. In other study, moderate disease was more common among the diabetics than the controls where pocket depth was considered as a marker of periodontal disease severity. No significant association between DM and periodontal disease was found by using the deep periodontal pockets as the clinical parameter for periodontal disease severity [2].

Among diabetic patients another characteristic of a population with poor oral hygiene is many missing teeth. This study found similarity between both the groups in this variable. In the diabetic patient group a comparative Saudi study showed that, 81% of patients had 9 – 20 missing teeth, while it is 19% in non-diabetic group. Other study done by Kawamura and co-workers showed that diabetic patients had 6.7 missing teeth compared to 4.3 teeth in the control group [2].

Effect of Glycemic Control on Periodontal Status

The education level of these patients was low. Very poor oral hygiene only 16.3% of the included patients were brushing at least 2 times a day. Oral health behaviours among patients with type 1 and 2 DM were studied by Spangler and Konen. 97.9% and 99.3% of the diabetic patients brushes daily (controlled and uncontrolled diabetics respectively). Miswak and finger is also used as cleansing regime, but in our study only 6% were miswak users. Miswak is a traditional chewing stick which acts as a natural toothbrush. It has antibacterial effect and is as effective as tooth brush in removing dental plaque and decreasing gingivitis. A study from India revealed that chewing stems is believed to facilitate salivary secretion and, thereby help in plaque control, while some stems have an anti-bacterial actions as well for example Neem datum. Syrj and colleagues reported 50% rate for brushing twice daily [2].

Bartold and colleagues concluded that the incidence of severe periodontal disease is affected by the improved oral hygiene, and the continuous assessment of at risk patients and regular thorough subgingival debridement leads to the successful management of the disease. It was also concluded in two Finnish studies that perception of dental self-efficacy plays a decisive role and has a positive influence on compliance in relation to oral health behaviour. Smoking showed its effects on both glycemic control and periodontal health. We could not find significant difference between the two groups. In both groups, subjects smoked almost the same number of cigarettes per day i.e. 10.40 ± 7.76 (controlled diabetes) and 10.92 ± 8.6 (uncontrolled diabetes) [2].

In this, subjects who are unaware of their diabetic status could get an important clue towards diagnosis by dental examination. This study is restricted to subjects who were already diagnosed with type-2 diabetes. Borrell suggested screening for unidentified diabetics in the population based on their self-reported family history of diabetes, hypertension, hypercholesterolemia and periodontal status. Hence, diabetes is a disorder of importance to dentists and dental hygienists and to patients seen in the dental office as it has this bidirectional relationship with periodontitis [2].

### Table 1: Effect of degree of glycemic control on periodontal status [3].

<table>
<thead>
<tr>
<th>Reference</th>
<th>Country</th>
<th>Study design</th>
<th>Diabetes type</th>
<th>Age group</th>
<th>Effect</th>
<th>Non-DM comparison group</th>
<th>Evidence level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Karikoski and Murtomaa (2003)</td>
<td>Finland</td>
<td>Prospective</td>
<td>1, 2, other</td>
<td>Adults</td>
<td>0</td>
<td>No</td>
<td>II-2</td>
</tr>
<tr>
<td>Tervonen., et al. (2000)</td>
<td>Finland</td>
<td>Cross-sectional</td>
<td>1</td>
<td>Adults</td>
<td>1</td>
<td>Yes</td>
<td>III</td>
</tr>
<tr>
<td>Sandberg., et al. (2000)</td>
<td>Sweden</td>
<td>Cross-sectional</td>
<td>2</td>
<td>Adults</td>
<td>0</td>
<td>Yes</td>
<td>III</td>
</tr>
<tr>
<td>Tsai., et al. (2002)</td>
<td>USA</td>
<td>Cross-sectional</td>
<td>2</td>
<td>Adults</td>
<td>1</td>
<td>Yes</td>
<td>III</td>
</tr>
<tr>
<td>Lu and Yang (2004)</td>
<td>Taiwan</td>
<td>Cross-sectional</td>
<td>2</td>
<td>Adults</td>
<td>1</td>
<td>Yes</td>
<td>III</td>
</tr>
<tr>
<td>Campus., et al. (2005)</td>
<td>Italy</td>
<td>Cross-sectional</td>
<td>2</td>
<td>Adults</td>
<td>1</td>
<td>Yes</td>
<td>III</td>
</tr>
<tr>
<td>Chuang., et al. (2005)</td>
<td>Taiwan</td>
<td>Cross-sectional</td>
<td>2</td>
<td>Adults</td>
<td>0</td>
<td>No</td>
<td>III</td>
</tr>
<tr>
<td>Peck., et al. (2006)</td>
<td>South Africa</td>
<td>Cross-sectional</td>
<td>2</td>
<td>Adults</td>
<td>1</td>
<td>No</td>
<td>III</td>
</tr>
<tr>
<td>Jansson., et al. (2006)</td>
<td>Sweden</td>
<td>Cross-sectional</td>
<td>2</td>
<td>Adults</td>
<td>1</td>
<td>No</td>
<td>III</td>
</tr>
<tr>
<td>Arrieta-Blanco., et al. (2003)</td>
<td>Spain</td>
<td>Cross-sectional</td>
<td>1, 2</td>
<td>Mixed ages</td>
<td>0</td>
<td>Yes</td>
<td>III</td>
</tr>
<tr>
<td>Guzman., et al. (2003)</td>
<td>USA</td>
<td>Cross-sectional</td>
<td>1, 2*</td>
<td>Adults</td>
<td>1</td>
<td>No</td>
<td>III</td>
</tr>
<tr>
<td>Negishi., et al. (2004)</td>
<td>Japan</td>
<td>Cross-sectional</td>
<td>1, 2*</td>
<td>Adults</td>
<td>1</td>
<td>No</td>
<td>III</td>
</tr>
</tbody>
</table>

*Hierarchy of evidence based on classification scheme used (U.S. Preventive Services Task Force, 1996) where: I = evidence obtained from at least one properly randomized controlled trial; II-1 = evidence obtained from well-designed controlled trial without randomization; II-2 = evidence obtained from well-designed cohort or case-control analytic studies, preferably from more than one center or research group; II-3 = evidence obtained from multiple time series with or without the intervention. Dramatic results in uncontrolled experiments (such as the results of the introduction of penicillin treatment in the 1940s) could also be regarded as this type of evidence; III = opinions of respected authorities, based on clinical experience; descriptive studies and case reports; or reports of expert committees.

*Diabetes type: 1 = type 1 diabetes mellitus; 2 = type 2 diabetes mellitus; 1,2 = both subjects with type 1 and type 2 diabetes mellitus included; GDM = gestational diabetes mellitus; 9 = diabetes type not specified and not clearly ascertainable from other information in the report; *= diabetes type not specified but ascertained by reviewers from other information in the report or from other sources, such as direct communication with the authors.

*Effect: 1 = subjects with poorer glycemic control had poorer health than the comparison group(s); 0 = no difference in the periodontal health status between subjects with poorer glycemic control and comparison group(s).

*Diabetes types are 1 and 2 for all but one subject who had drug-induced diabetes mellitus.

### Periodontal Disease and its Effects on Glycemic Control

The chronic challenge of the periodontal pathogens may provide a constant source of proinflammatory cytokines that may be associated with tissue insulin resistance and poor glycemic control in subjects with diabetes [4,5]. In inflamed periodontal tissues, there are increased levels of inflammatory mediators associated with tissue destruction, including tumor necrosis factor alpha (TNF-α), interleukin 6 (IL-6), interleukin 1-α (IL-1α), prostaglandin E2, and matrix metalloproteinases [5]. In addition to the local destruction, this inflammation involves increased permeability of the capillaries leading to potential portals to the systemic circulation for the inflammatory mediators as well as the products of the bacterial infection (Figure 1).

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**Citation:** Krishna Kripal, et al. “Role of Diabetes Mellitus on Maintenance of Periodontal Disease”. *EC Dental Science* 1.52 (2017): S38-S43.
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Figure 1: Increased number of neutrophils, monocytes, macrophages infiltrates the tissue to release more divert cytokines and prostaglandins that exacerbate the inflammatory response and periodontal tissue destruction.

These mediators have been reported to be important in pathways for the pathogenesis of insulin resistance, coronary heart disease, and, more recently, diabetes [6]. Based on this theory, it was hypothesized that the successful control of periodontal infection could improve the clinical signs of periodontitis as well as the metabolic control of DM [4]. Various investigations [5-11] demonstrated that scaling and root planing (SRP), associated or not with antibiotics, yielded clinical benefits in subjects with diabetes, including a reduction in probing depth (PD), bleeding on probing (BOP), and suppuration and a gain in clinical attachment level (CAL). Simultaneously, these interventional studies [5-15] assessed the potential effects of different types of periodontal therapy on glycemic control in subjects with diabetes, as measured by glycated hemoglobin (HbA1c) levels. Some investigations suggested that improvements in periodontal condition positively affect metabolic control, whereas other studies [6,13,14] did not find this beneficial effect. Al-Mubarak and colleagues [16] recently conducted a study to evaluate the effectiveness of scaling and root planing (SRP) and adjunctive chemotheraphy (doxycycline hyclate, 20 mg) on gingival health and glycemic control in diabetic subjects with chronic periodontitis. Three hundred and forty-six type 1 and 2 diabetic subjects with chronic periodontitis were randomized into four test groups: Group 1 received one session of SRP at the baseline visit and placebo tablets twice/day, started at the baseline visit, for 3 months, Group 2 received one session of SRP at the baseline visit, and doxycycline hyclate (20 mg, twice/day) started at the baseline visit for 3 months, Group 3 received two sessions of SRP, first at the baseline visit and second at the 6 months, with placebo tablets twice/day started at the baseline visit and 6-month visit, for 3 months at each visit, and Group 4 received two sessions of SRP, first at the baseline visit and the second at the 6-month visit, and doxycycline hyclate 20 mg twice/day, started at the baseline visit and the 6-month visit, for 3 months at each visit. To evaluate glycated hemoglobin (HbA1c) Venous blood samples were collected; dental measurements were also included. They found that HbA1c showed significant improvement (P < 0.05) only for those subjects with glycated hemoglobin ≤ 8.8% within each group, as well as when subjects were combined. Statistically significant improvements for most of the dental parameters at follow-up visits (P < 0.05) compared to the baseline is shown by all groups. We concluded that that SRP and adjunctive therapy may significantly decrease glycated hemoglobin levels for subjects with HbA1c ≤ 8.8% [10].

Finally, more systematic studies in diverse groups of populations is warranted to support existing evidence that treating periodontal infections can contribute to glycemic control management and possibly to the decrease of the burden of diabetes complications.

Conclusion

Poor oral hygiene and poor glycemic control has strong negative impact on the periodontal health, which is evident by raised scores on indices scale of gingival, periodontal, plaque and calculus. Poor metabolic control of diabetes further compounds the effect of inflammation of gingiva and leads to loss of attachment.

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Bibliography


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*Volume 1 Issue S2 May 2017*
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