

Periodontal Diseases in Diabetics: Relationship, Prevention, and Treatment

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Summary

Schulze A, Busse M. Periodontal Diseases in Diabetics: Relationship, Prevention, and Treatment. *Clinical Sports Medicine International (CSMI) 2008, 1(2): 1-4.*

Diabetes represents a risk factor for periodontitis. Poor glycemic management and prolonged duration of the disease are associated with severe periodontal disease, commonly called the sixth complication of diabetes. The systemic inflammatory response generated by inflamed periodontal tissue worsens insulin resistance and promotes diabetes. This evidence points to a vicious cycle of diabetes and periodontitis exacerbating each other, which ultimately brings the diabetics to the attention of oral health practitioners. The primary and secondary prevention of periodontal disease is an important diabetic task; an aggressive management of oral health and regular check-ups in diabetic patients may diminish the inflammatory milieu's effects on diabetes control. Diabetics' therapy of periodontal disease is a long-term venture, requiring a modification of the customary periodontal treatment applied to non-diabetics. The use of bacterial analysis is an important tool for the extended management of periodontal disease in diabetics.

Key words: diabetes, periodontal therapy in systemic diseases, periodontitis and diabetes control, insulin resistance.

Introduction

The prevalence of diabetes mellitus is more than twice as high in patients with periodontitis compared to healthy subjects. Periodontal disease may contribute to systemic inflammation, worsening insulin resistance and diabetes due to the generation of inflammatory cytokines (6). A large body of information exists, showing that prevention, early diagnosis and consequent treatment of periodontal disease may have a major impact on the control of diabetes. Severe periodontal disease causes attachment loss, alveolar bone loss and tooth loss, extending beyond the local level to produce systemic effects. It exacerbates the inflammatory milieu, increases insulin resistance, and potentially worsens cardiovascular disease. Severe periodontitis is a risk factor for early death due to ischemic heart disease or renal dysfunction independent of diabetes, increasing the risk 3.2 fold as compared with those with none, mild, or moderate periodontitis (10).

Looking at our own data we calculated that the prevalence of periodontitis was 64% in diabetic subjects (130 patients on oral medication). 42% of these had a severe periodontitis and 4% had lost all their teeth due to periodontal disease. 14% of this group had received a periodontal treatment, 25 % of them without success. 50% of these post-treatment patients still had a severe periodontitis. In another group of 130 diabetics receiving insulin therapy, moderate and severe periodontal disease was seen in 14% and 52% respectively. 14% of these patients had lost all teeth due to periodontitis. 17% had a periodontal treatment, with a 26% success rate. 50% still had a severe periodontitis after the professional treatment. In a group of 50 low-grade diabetics without any medication at all, a periodontal disease was found in 40%. 27% revealed a severe periodontal disease.

Periodontitis and its effects on diabetes

Periodontal diseases are bacteria-induced infections affecting the periodontium and resulting in the loss of tooth attachment.

Porphyromonas gingivalis plays a central role in periodontal disease. *P. gingivalis* can invade endothelial

cells and is a potent signal for monocyte and macrophage activation. Thus, once established in the tissue, this chronic infection complicates diabetes control and increases the occurrence and severity of microvascular and macrovascular complications. The presence of the

periodontal pathogen bacteria thus increases the risk of stroke, ischemic heart disease and atherosclerosis (2).

Other relevant periodontal pathogen bacteria are: actinobacillus actinomycetem comitans, treponema denticola, fusobacterium nucleatum, prevotella intermedia, eikenella corrodens und camphylobacter rectus. These periodontal pathogen bacteria cause a chronic local low-grade inflammation and contribute to systemic inflammation. This is reflected by higher circulating levels of inflammatory markers such as C-reactive protein, IL-6 and $TNF\alpha$, the latter being responsible for worsening insulin resistance and diabetes (8). Exacerbating immune reactions lead to self-destruction of periodontal tissue (Fig.1)

The increasing pocket depth and root surface infection, bacterial impact and invasion of periodontal tissue, alveolar bone loss and local and systemic immune reactions all have negative effects on diabetes. The association between diabetes and periodontal disease may be due to numerous physiological phenomena seen in diabetes, such

as impaired (immune) resistance, vascular changes, altered microflora, and abnormal collagen metabolism. This tends to support the higher incidence and severity of periodontitis in diabetic patients. The association between gene sets (for example IL1-genotype) and periodontitis has been discussed with conflicting conclusions (4,7). There is a direct causal or modifying relationship in which poor glycemic control results in more severe periodontitis (4). The worsening effects of diabetes on periodontal inflammation are well known. Studies also indicate a correlation between periodontal inflammation on diabetic balance and insulin resistance syndrome (3,6,8). This evidence points to a vicious cycle in which diabetes and periodontitis exacerbate one another. Periodontal treatment may improve diabetes control measured as a reduction in glycated hemoglobin (9). Because of this relationship, the diabetes specialist should put an emphasis on oral health and its control as an integral part of diabetes treatment.

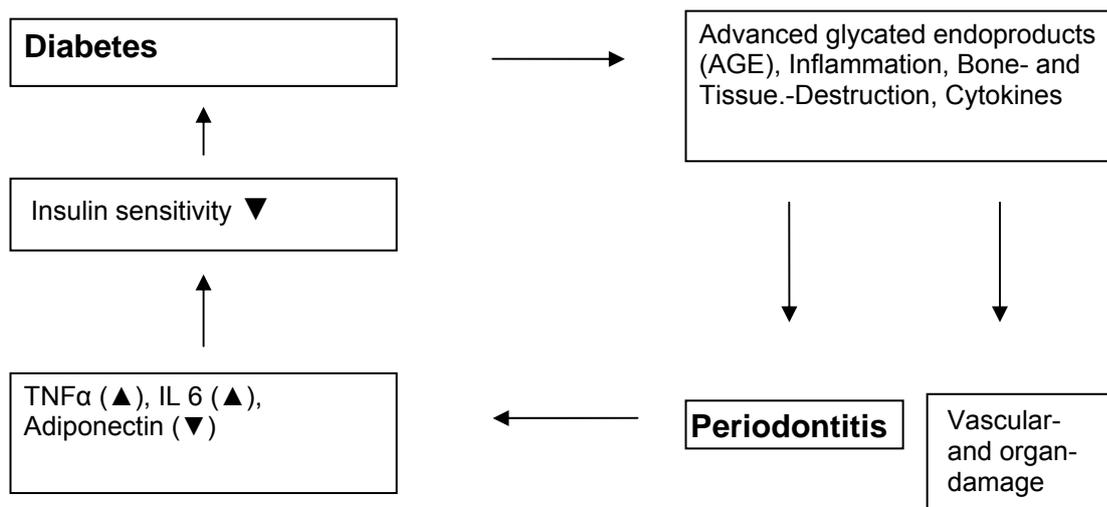


Fig. 1: Association of Diabetes and Parodontitis

Diagnosis of periodontal disease

Diabetes is a disease that frequently causes multiple comorbidities. Periodontal disease and other oral pathologies (gingivitis, candidiasis, oral malignancies etc.) are frequent complications of diabetes. The diagnosis and control of periodontal disease with its adverse effects on insulin resistance through the generation of inflammatory cytokines can have a major impact on diabetes control and diagnosis. In Germany, the incidence of periodontitis in diabetic subjects is not sufficiently recognized by specialized health professionals. In fact it is part of the diabetes specialist's obligation to monitor oral health. He or she should provide for the early diagnosis of periodontitis using simple screening methods suitable for the office. If a

patient shows signs of periodontitis, a specialist for oral diseases, a periodontologist, should perform further diagnostics and take therapeutic steps. Almost all subjects with diabetes require oral and dental evaluation and many of them will develop an oral pathology in the course of their disease that requires intervention.

Therefore a basic understanding of periodontal diagnosis and management is strongly recommended for any diabetes professional:

1. Basic diagnostic indices for periodontitis are: the evaluation of gingival bleeding and swelling, recessions, attachment loss, pathological pockets (depth >4mm), loss of tooth insertion, pocket exudation, foetor, bleeding on probing with a special ball-point probe and alveolar bone loss assessed from radiographs..
2. A further diagnostic step is the determination of inflammation markers such as sCRP, TNF α , IL-6, IL-10.
3. For diabetic patients, the identification of subgingival bacteria, especially *p. gingivalis*, *p.*

intermedia, *actinobacillus actinomycetem comitans* and *t. forsythensis*, represents another important diagnostic tool. Consequent follow-up examinations should include a bacterial test to monitor subgingival bacterial levels which are best measured using a multiplex polymerase chain reaction.

Very useful and simple tools are informational flyers to help increase patient awareness of periodontal disease, its association with diabetes and its prevention and treatment.

Prevention of periodontitis

The following behavioural recommendations should be included in regular instructions for diabetic patients:

- No smoking
- Strict glycaemic control
- Professional counseling on the importance of oral health (self-response, self-questionnaire)
- Qualified instruction in home oral care and the importance of soft tooth brushes
- Thorough oral cleaning at least twice a day – after breakfast and before bedtime – for more than 3 minutes
- Daily interdental cleaning (floss, tooth picks, interdental-brushes)

- Tongue-brushing
- Proper tooth brush handling - daily disinfection of the tooth brush, drying, alternate use of two brushes, no natural bristles!
- Partner treatment - if only one suffers from periodontitis special care should be taken to avoid transfer of bacteria through shared dishes, cups, forks; no kissing allowed during acute phases (?)
- Antibacterial and anti-inflammatory ingredients in toothpaste and mouth rinses, for example
- chlorhexidine (anti-inflammatory), triclosan and thymol (anti-inflammatory))

Periodontal treatment of diabetic patients

Evidence shows that the mechanical removal of subgingival concretions and diseased tissue does not result in a complete elimination of periodontal infection. Consequently there is no effect on diabetes control or a reduction in glycated hemoglobin. Studies incorporating systemic antibiotics as adjuncts to mechanical debridement show a reduction of *p. gingivalis* and a concomitant reduction in glycated haemoglobin (in non-diabetics?). The evidence supports the view, that treatment of chronic periodontal infection is essential for the diabetic patient. Assessment of the infection status in diabetic patients (PCR, biochemistry) is fundamental for appropriate treatment decisions. First, the periodontal inflammation should be reduced even before scaling or root planing takes place. The following are a basic guideline:

1. Measurement of pocket depth using a ball-point probe
2. Extraction and identification of subgingival bacterial samples (paper points and sterile tubes, available from distributors or laboratories)
3. Germ reduction through the use of antibacterial mouthwash, pocket solutions, subgingival application of metronidazol gel, chlorhexidine gel (applied with mouth guards several times a week).

The elimination of periodontal infection with these antibacterial adjuncts results in a reduction of the periodontal pathogen bacteria to low or even nondetectable levels.

4. A follow-up extraction and identification of subgingival bacteria samples (same extraction sites)
5. Scaling and/or root planing accompanied by antibiotic treatment
6. Maintenance of intensive oral care and additional use of mouthwash, controlled with regular PCR analysis
7. Recall

Unlike non diabetic subjects, the modalities of treatment in poorly controlled diabetic patients often do not result in a complete reduction of inflammation and/or a healthy periodont. Treatment decisions often involve compromises. Even though an inactive periodontal pocket is the main goal of periodontal treatment, diabetes-induced problems may go so far as to cause failure of treatment. On the other hand an aggressive management of oral hygiene and health control including early bacterial detection may be an efficient way to decrease periodontal inflammation and positively affect diabetes management. The diabetes specialist may be the first to perform basic diagnostics as given in points 1-

4 and 6. as well as obtain samples for bacterial analysis using subgingival paper point probes. However, cooperation with a periodontologist will be necessary, since point 5 of the diagnostic regimen and all therapeutic work can only be performed by a specialized dentist.

A close cooperation between the diabetes specialist and the periodontologist is vital to manage the patient's periodontal problems and diminish the inflammatory milieu's detrimental effects on diabetes control and cardiovascular health. Combined, these two disciplines have a greater success in the diagnosis and control of diabetes and periodontitis.

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