

Generalized Severe Periodontitis and Periodontal Abscess in Type 2 Diabetes: A Case Report

SUMMARY

The bidirectional relationship between periodontitis and diabetes mellitus can cause distinct oral symptoms that can impact the general health conditions of affected patients. The presented case report of a female diabetes type 2 patient with severe periodontitis and a periodontal abscess shows how interdisciplinary collaboration between the attending physician and dentist can significantly improve oral conditions and metabolic control.

Key words: diabetes type 2, oral symptoms, periodontitis

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CASE REPORT (CR)

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Introduction

Diabetes mellitus is a group of metabolic diseases that are characterized by chronic hyperglycaemia resulting from defects in insulin secretion, insulin action, or both¹. The majority of diagnosed cases of diabetes can be differentiated into 2 main etiopathogenetic categories: type 1 diabetes mellitus and type 2 diabetes mellitus¹. Type 1 diabetes mellitus (also known as juvenile diabetes or insulin dependent diabetes mellitus) is caused by an absolute deficiency of insulin secretion, mostly resulting from auto-immune destruction of the pancreatic β -cells. Approximately 5-10% of diabetes patients are related to this category¹. More prevalent is type 2 diabetes mellitus (90-95% of diabetes patients). Type 2 diabetes mellitus (also known as adult-onset diabetes or non-insulin-dependent diabetes mellitus) is often caused by a combination of resistance to insulin action and an inadequate compensatory insulin secretory response, leading to a relative deficiency of insulin secretion¹. The risk of developing type 2 diabetes mellitus increases with age, weight and lack of physical activity. Chronic hyperglycaemia in diabetes patients is associated with a wide range of secondary diseases, including retinopathies, nephropathies, peripheral neuropathies and cardiovascular diseases¹.

Periodontal disease is a destructive inflammation of the tooth supporting tissues resulting from a complex multi-

factorial disorder, which involves various microorganisms organized in a dental plaque biofilm and interactions of host cells. Furthermore, genetic predispositions², systemic diseases such as diabetes mellitus³, and personal behaviour such as smoking⁴ and oral hygiene^{5,6} play an important role in the etiopathogenesis of periodontitis, which may lead to loss of attachment, destruction of alveolar bone, and to periodontal pocket formation, ultimately causing tooth loss.

Diabetes mellitus and periodontal disease are both multifactorial diseases with a high prevalence worldwide. The International Diabetes federation estimates that from 2011 to 2033 the global number of people with diabetes mellitus will grow from 366 million to 552 people⁷. Numerous cross-sectional and longitudinal prospective clinical studies provide evidence for a bidirectional relationship between both diseases and intense efforts have been devoted to elucidate the underlying mechanisms^{3,8}. Recent evidence indicates that diabetes mellitus promotes the occurrence, the progression and the severity of periodontitis, whereby the glycaemic control of the patients seems to be the strongest influencing factor. *Vice versa*, periodontal inflammation complicates the glycaemic control of diabetes and seems to have an impact on the risk and onset of diabetes associated complications. The presented case shows how interdisciplinary collaboration between attending physician and dentist can significantly improve oral conditions and metabolic control.

Case Report

A 50-year-old female patient was referred from her dentist to university hospital complaining of pain in the left maxilla and the right mandibular region. The patient reported that tooth #36 was extracted by her dentist 2 month ago because of swelling and strong pain. Furthermore, she reported about a stroke 1 year ago and a myocardial infarction 3 years ago. She was smoker (10 cig/d for 30 years, 30 pack years) and suffered from fibromyalgia, polyneuropathy, cardiac arrhythmia, hypertension, obesity (body-mass-index of 39) and diabetes mellitus type 2 (HbA1c of 7.7%). The diabetes was diagnosed 4 years ago and controlled with insulin (Insuman Comb 25® 24-0-24; Humalog® according to requirements) and oral anti-diabetic medication (Metformin 1000mg 1-0-1). In addition, the patient took a plenty of other medications for her other diseases (Aggrenox, Ramipril, Torasemid, Bisoprolol, Pantoprazol, Amineurin, Simvastatin, Novalgin, Gabapentin, Amlodipin) and an allergy against penicillin was known.

Intraoral examination revealed dental plaque on all teeth (plaque control record- PCR- was 100%). The gingiva showed generalized signs of inflammation and in region #24 to #27 there was a swelling and suppuration with a fistula in the buccal region of #24. A mild gingival overgrowth in the mandibular and maxillary front area was observed. The patient had lost several teeth and had no prosthodontic treatment. Teeth #24 to #27 were flared out because of missing antagonists (Fig. 1). Caries was diagnosed on following teeth: #45, #46, and #36. All teeth, except #46, were vital. Periodontal probing depth (PPD) ≥ 4 mm was measured on all teeth. Teeth #16 to #23 and #24 to #27 showed a PPD ≥ 6 mm. Furcation involvements were diagnosed on teeth #16, #26, #27 and #46. Tooth 46 had a mobility degree of III. Bleeding on probing (BOP) was diagnosed in 24% of all sites examined (Fig. 2). Radiographic examination revealed generalized horizontal bone loss up to the middle of the dental roots, calculus and localized severe vertical bone loss. Tooth 46 showed a periapical and intra-radicular radiolucency and in region #36 the X-ray revealed a fresh extraction socket (Fig. 3).



Figure 1. Clinical conditions before systematic periodontal treatment

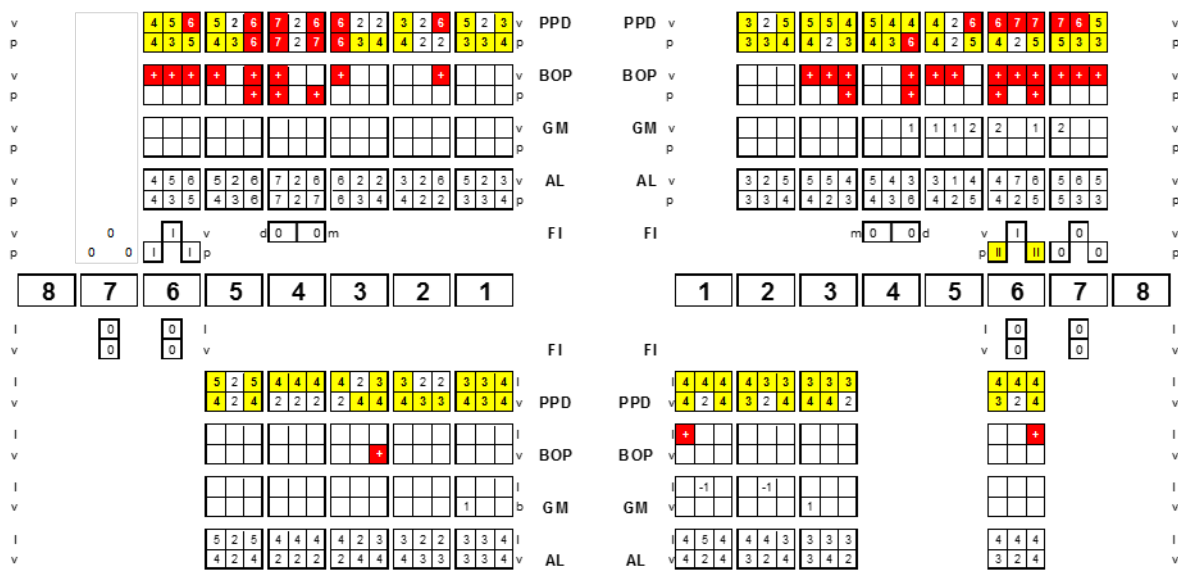


Figure 2. Periodontal status before systematic periodontal therapy. PPD: periodontal probing depth. BOP: bleeding on probing. GM: gingival margin. AL: attachment level



Figure 3. Panoramic radiograph of the patient

Following diagnoses⁹ were made: 1. severe generalized chronic periodontitis modified by diabetes mellitus type 2; 2. periodontal abscess region #24 to #27; 3. gingival overgrowth associated with amlodipine; 4. tooth #46- profound caries and symptomatic apical periodontitis. Prognosis¹⁰ of tooth #46 was set to be hopeless, while prognosis of teeth #24, #25, #26, and #27 was set to be unfavourable. Prognosis of all other teeth was expected to be favourable.

A collaborative dental-medical treatment plan was devised together with the attending physician. First of all, the abscess was drained and tooth #46 was extracted. An interim prosthesis was fabricated and inserted. The

patient revealed oral hygiene training and professional tooth cleaning, as well as restorative therapy of the carious lesions (3 appointments within 7 weeks). Full-mouth disinfection (FMD) was performed under adjuvant antibiotic therapy in agreement with the attending physician (300 mg Clindasaar® 4 times per day for 7 days, beginning 1 day before FMD). Chlorhexidine 0.2% mouth rinse was prescribed for the following 2 weeks. Meanwhile, the attending physician reinstructed and motivated the patient again for diabetic therapy and optimized anti-diabetic medication (in addition: Victoza® 1,2 mg/ml 0-0-1). Re-evaluation of the periodontal status was performed 3 months after FMD. No signs of gingival inflammation and gingival overgrowth were observed in this visit. BOP was decreased to 2.4% of sites. All teeth showed PPD ≤ 4 mm without BOP except teeth #26 and #27 (5 and 6 mm). Subgingival scaling was repeated at teeth #26 and #27. Individual risk assessment¹¹ recommended supportive periodontal therapy (SPT) in a 3-month interval. The patient showed good compliance and missed no SPT appointment and no appointment with her physician. One year after FMD, periodontal examination showed no site with PPD > 4 mm and continuously low oral hygiene indices (PCR: 35%; GBI: 6%) as well as clinically healthy conditions (Figs. 4 and 5). Radiographic re-evaluation of the region #24 to #27 revealed osseous regeneration (Fig. 6). HbA1c-value dropped to 7.3%.



Figure 4. Clinical condition 1 year after full-mouth disinfection

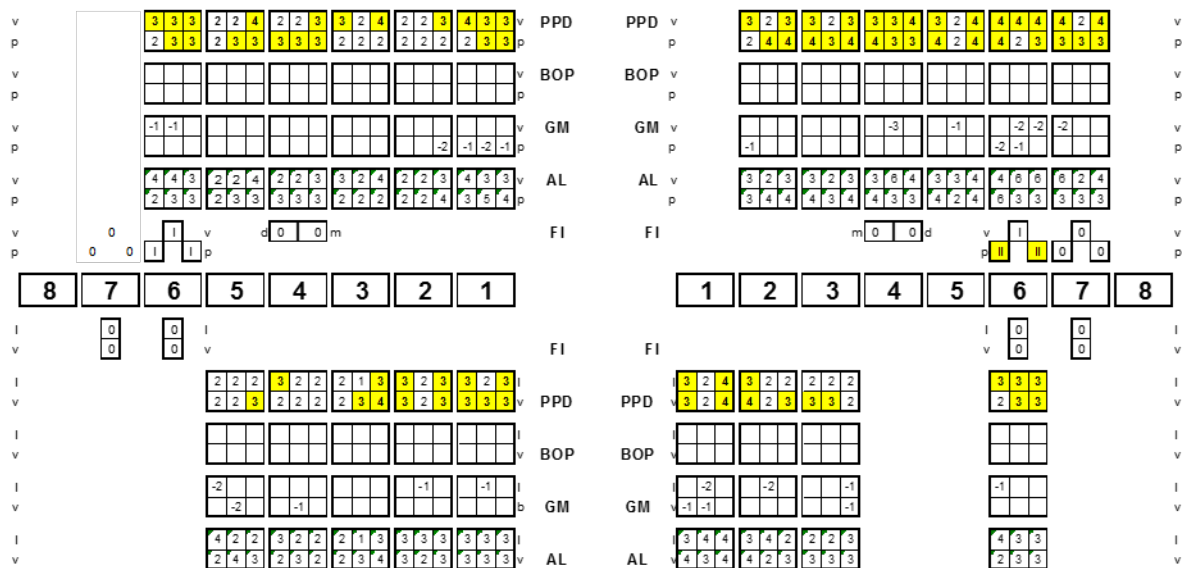


Figure 5. Periodontal status after 1 year of supportive periodontal therapy. PPD: periodontal probing depth. BOP: bleeding on probing. GM: gingival margin. AL: attachment level

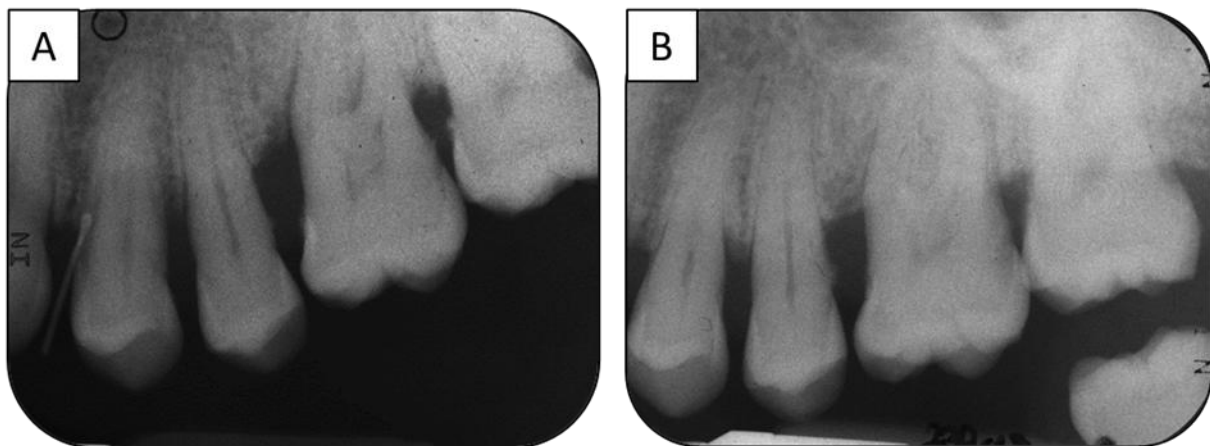


Figure 6. Radiography of region #24 to #26. A: Before periodontal therapy. Fistula tract region #24. B: 1 year after full mouth disinfection. Calculus at root surface #25 was removed afterwards

Discussion

The presented case report demonstrates the successful periodontal treatment of a female diabetes patient with a periodontal abscess, chronic periodontitis, and medical induced gingival overgrowth. Moreover, the patient's glycaemic control could be improved from HbA1c 7.7% to 7.3% after periodontal treatment and additional diabetic medication. The therapeutic results agree with recent evidence regarding response of diabetes patients to periodontal therapy.

Controlled studies have shown, that mechanical periodontal therapy of diabetes patients with periodontitis can improve HbA1c-value approximately 0.4%-points⁸. On the other hand, there is a consistent and robust evidence that severe periodontitis affects glycaemic control. Also, there is evidence for a direct dose-dependent relationship between severity of periodontal inflammation and diabetes complications, as well as growing evidence for an increased risk for diabetes onset in patients with severe periodontics¹². The current understanding of biological mechanisms behind the bidirectional relationship is described as follows: type 2 diabetes is preceded by systemic inflammation, leading to insulin resistance and reduced pancreatic beta-cell function and apoptosis of these cells. Periodontal inflammation increases systemic inflammation by entrance of periodontal pathogens and their virulence factors into circulation¹². Recent evidence suggests that local changes in the periodontal tissues are characterized by enhanced interactions between leukocytes and endothelial cells and by altered leukocyte functions (resulting in increased levels of reactive oxygen species and of pro-inflammatory cytokines- interleukin-1 β , interleukin-6 and tumour necrosis factor- α). These local changes are amplified by the enhanced accumulation

of advanced glycation end-products (AGEs) and their interaction with receptors for advanced glycation end-products (RAGEs). Furthermore, the increased levels of pro-inflammatory cytokines lead to an up-regulation of RANKL in periodontal tissues, stimulating further periodontal tissue breakdown. These complex changes resulting from diabetes conditions modify the local inflammatory reaction in the periodontium of diabetes patients, leading to a pro-inflammatory state in the gingival tissue and microcirculation¹³. In the presented case, the improvement of the HbA1c of 0.4%-points probably resulted from a combination of optimized medication and periodontal therapy. Dental interventions were planned in cooperation with the attending physician and treatment time was monitored closely and restricted between meals and medicine uptake, so that the patient was able to cope with her systemic challenge.

In addition to periodontitis, the patient also suffered from slight gingival enlargement induced by amlodipine. Recent evidence suggests that approximately 30% of patients with an intake of amlodipine develop gingival overgrowth¹⁴. Pathogenesis of calcium channel blocker induced gingival overgrowth remains unclear, but it is assumed that the secretory function of fibroblasts or collagenase synthesis is affected, resulting in the increased fibroblastic proliferation and collagen synthesis that may be enhanced by inflammatory changes within the gingival tissue¹⁵. Case series have shown that FMD is an adequate treatment concept for drug-induced gingival overgrowth reducing the need for further surgical interventions even in severe cases¹⁶. In the presented case, we diagnosed only a mild form of gingival overgrowth and adequate supra- and sub-gingival plaque control resulted in complete remission. It should be mentioned that the smoking habit of the patient also negatively influenced the periodontal status; however, all efforts to help the patient to quit smoking were not successful.

Conclusion

This case demonstrates that systematic periodontal treatment and better control of diabetes can result in remarkable improvements of periodontal and systemic conditions. To avoid risks that might interfere with treatment or its outcomes, the interdisciplinary coordination between dentist and attending physician plays a fundamental role in the treatment of periodontitis and diabetes.

Note: The results of this paper were presented as a part of an invited lecture at the 21st BaSS Congress.

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