The association between diabetes mellitus and periodontitis has been well established. Diabetic condition may impair periodontal health in many ways, and compromise the host’s response to opportunistic bacteria that cause periodontal disease. A case of a severe periodontal condition as a result of long-term diabetes mellitus combined with difficult life conditions, smoking and poor oral hygiene is presented.

INTRODUCTION

There is an increasing evidence that a number of complex diseases are associated with opportunistic infections in periodontal tissues. New interest has arisen in oral microbial etiology, mucosal immunity, and associations with systemic conditions, of which diabetes mellitus (DM) is one of the most frequent and dangerous diseases. The remarkable consistency in finding either the prevalence, severity or extent of at least one manifestation of periodontal disease (PD) in the overwhelming majority of the studies shows that there should be further rigorous, controlled studies of treatment of PD in diabetics in order to confirm the extent to which treatment not only resolves periodontal infection but enhances glycemic control.

There is also a need for further investigations and assessment of the extent to which control of the DM status is related to control of PD status. Case reports as well as clinical experience support this contention.

CASE REPORT

A male patient (born in 1963) was admitted to the Department of Periodontology, Zagreb School of Dental Medicine, at the beginning of 2000 for initial therapy. The patient was born in Vukovar, and during the war in 1991 he had to leave his hometown. Not only did he lose his parents, but after coming to Zagreb he lived in the appalling conditions, without running water and electricity. In 1995 he got married but his wife left him soon and he has been living alone since then. His medical history revealed a long-term DM type I, with the onset at age 14, which had never been properly controlled. In 1992, the first signs of arterial hypertension with oscillatory blood pressure occurred, which required antihypertensive therapy.

First hospitalization was in 1996, when DM type I, diabetic retinopathy, diabetic sensorimotor polyneuropathy and diabetic nephropathy were diagnosed. In February 1998, he was admitted for hemodialysis and has since been on a regimen of 4-hour sessions three-times a week. The patient is a smoker, >20 cigarettes per day, but does not consume alcoholic beverages.
Table 1. Mean ALOSS values before (ALOSS 1) and after one year (ALOSS 2) of initial treatment, and difference (1-year ALOSS) between two measurements (mm)

<table>
<thead>
<tr>
<th>Tooth</th>
<th>16</th>
<th>14</th>
<th>13</th>
<th>23</th>
<th>24</th>
<th>25</th>
<th>27</th>
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<th>34</th>
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<th>36</th>
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</thead>
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<td>ALOSS 1</td>
<td>4</td>
<td>1.75</td>
<td>0.5</td>
<td>0.25</td>
<td>2.25</td>
<td>3</td>
<td>2.5</td>
<td>1.5</td>
<td>1.5</td>
<td>2.75</td>
<td>3</td>
<td>2.5</td>
</tr>
<tr>
<td>ALOSS 2</td>
<td>5.75</td>
<td>4.75</td>
<td>2.75</td>
<td>3.5</td>
<td>4.5</td>
<td>5</td>
<td>6.25</td>
<td>3</td>
<td>4.25</td>
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<tr>
<td>1-year</td>
<td>1.75</td>
<td>3</td>
<td>2.25</td>
<td>3.25</td>
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<td>2</td>
<td>3.75</td>
<td>1.5</td>
<td>2.75</td>
<td>2.25</td>
<td>3.25</td>
<td>1</td>
</tr>
</tbody>
</table>

ALOSS = attachment loss

He is constantly on the following medication: ACT 12, 12, 12 IU; IT 14 IU at 10 p.m.; verapamil (Tonicardin) 3 x 80 mg tbl; doxazosine (Tsonocardin) 2 x 4 mg tbl; Gopten 2 mg 1 tbl in the evening; isosorbide-3-mononitrate (Olicard) a 60 mg 1 tbl in the evening; furosemide (Edemid) F 2 x 1 tbl; CaCO₃ 3 x 1 tbl; nifedipine (Nifedipin) 1, 1, 0 tbl; nifedipine (Cordipin XL) a 40 mg 1 tbl in the evening; Cosar a 50 mg 1 tbl at noon.

His blood pressure measures 150-210/75-100 mm Hg, with extremely high blood pressure in the morning, when he experiences tinnitus. Blood glucose level is never stable, varying between 250 and 310 mg/dl.

Periodontal examination revealed missing teeth, pocketing, plaque and calculus (supragingival and subgingival) as well as halitosis. Initial periodontal charting is shown in Fig. 1. Figure 2 presents an orthopantomograph at the first visit. After initial examination 7 teeth were extracted because of their hopeless status.

The patient was included in the initial therapy program performed by last year periodontology students. The cause related therapy included extraction of non-retainable teeth, instructions in oral hygiene, plaque and supragingival calculus removal, filling polishing, and deep scaling and root planing. In July 2000, the patient was scheduled for re-evaluation.

The compliance of the patient was not satisfactory, so he was recalled to the Department only one year after initial treatment. Repeat periodontal probing at that time revealed a worsened situation, although the clinical signs of inflammation were not present. The patient was scheduled for prosthetic rehabilitation with two partial dentures.

Figure 3 shows probing depths at the end of initial therapy, Fig. 4 periodontal status in January 2001, and Fig. 5 an orthopantomograph taken at the same time. Table 1 illustrates changes in the mean attachment loss (ALOSS) values between two measurements.

It should be stressed that the patient was not motivated, and smoked more than 20 cigarettes per day. He is to be scheduled for kidney transplantation.
The overall prognosis of the patient is poor, considering his low motivation and other factors that have exerted impact on his life so far. It can be expected that in less than a year he will lose all of his remaining teeth.

DISCUSSION

The effect of diabetes on the periodontium has been thoroughly studied, but it is difficult to make any conclusions from many of the performed studies since they are of a heterogeneous design, they differ in study populations, there have been some changes in the classification of periodontal diseases (PD) and diabetes mellitus (DM), and due to other factors studied and reported in the articles. Modern epidemiologic methods used in large populations have clearly established that DM is a risk factor for periodontal disease.

The relationship between DM and PD is well documented (1-3). There is a number of possible mechanisms proposed by which DM may affect the periodontal tissues. Primarily, these are related to changes in the subgingival microbiota, gingival crevicular fluid (GCF) glucose levels, periodontal vasculature, host response, and collagen metabolism (4). Although early studies showed possible differences in subgingival bacterial colonization between diabetic and nondiabetic patients, more recent research reports have not demonstrated significant differences, so it may be acclaimed that periodontally diseased sites harbor similar species as comparable sites in nondiabetic individuals (5-7).

Changes affecting the renal, retinal, and perineural vasculature in diabetes also occur in the periodontal tissues. Increased thickness of the gingival capillary endothelial cell basement membranes and the walls of small blood vessels may be seen in diabetic individuals (8-10).

The formation of advanced glycosylation endproducts (AGEs), according to Schmidt et al. (11), show a twofold accumulation in diabetic gingiva as compared with the gingiva from nondiabetic patients. The underlying mechanism responsible for the widespread vascular injury associated with diabetes may be the enhanced oxidant stress. The formation of AGEs stimulates arterial smooth muscle cell proliferation, increasing thickness of vessel walls. In the capillaries, enhanced cross-linking of AGE-modified collagen in the basement membrane inhibits the normal degradation of these proteins, increasing the thickness of the basement membrane. The AGE-modified arterial collagen in gingival blood vessel walls can bind circulating low density lipoproteins (LDL), resulting in atheroma formation and further narrowing of the vessel lumen. All these events may play a role in altering the tissue response to periodontopathic bacteria, resulting in increased severity and progression of periodontitis.

A rather important question for the dental practitioner is, will the DM patients with PD respond favorably to periodontal treatment. Christgau et al. (5)
found similar responses to scaling and root planing when compared with nondiabetic subjects four months after treatment. Conversely, patients with poorly controlled DM often have a less favorable response to treatment than those with well-controlled DM (12).

DM patients must be examined individually to assess their potential response to periodontal therapy. The mere presence of DM does not condemn the person to a less favorable periodontal outcome. A diabetic patient with good glycemic control can be expected to respond in a fashion similar to the nondiabetic subject. The presence of poor glycemic control may place the patient at risk of less favorable response. In addition, other factors, such as smoking or poor plaque control may adversely affect the response to periodontal treatment in DM individuals.

It has been established that diabetic adults having gingivitis or mild periodontal disease have a significantly lower prevalence of cardiovascular and kidney complications during the 1- to 11-year follow-up, when compared with patients with severe periodontal disease (13). This was despite the fact that hemoglobin A1c levels were similar in both groups, indicating a similar level of long-term glycemic control. Thus, the classic complications of DM may be closely associated with periodontal disease in these individuals, lending further credence to the concept that periodontal disease may be the “sixth complication of diabetes” (14).

REFERENCES


