

## Chapter 23

# Oral Complications in Diabetes

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### SUMMARY

**D**ata regarding oral complications in diabetes prior to the insulin era are scarce, possibly due to the limited scope of oral health care of that time and the short life span of the insulin-requiring diabetic patient. During the past 40 years, much data have been generated emphasizing the frequent occurrence of oral afflictions in patients with insulin-dependent diabetes mellitus (IDDM) and non-insulin-dependent diabetes mellitus (NIDDM). Perhaps the most important finding is that periodontal disease is more severe and occurs with higher frequency in diabetic patients (both NIDDM and IDDM), especially if the diabetes is not well controlled and there are other complications, such as retinopathy. The reason for the greater occurrence of periodontal destruction in diabetes is not clear. However, studies of the periodontal flora find similar microorganisms in diabetic and nondiabetic individuals, suggesting that alteration in host responses to periodontal pathogens account for these differences in periodontal destruction. For example, increased susceptibility to infection by periodontal bacteria associ-

ated with altered phagocyte functions and reduced healing capacity associated with altered collagen metabolism may explain, in part, the increased levels of periodontal disease in diabetes.

Caries in the crowns of teeth appear to be greater in adults with poor control of IDDM. However, the prevalence of root caries requires further studies. Oral infections aside from dental caries and periodontal disease are often more severe. Life-threatening deep neck infections and palatal ulcers exemplify the severity of these conditions. Mucosal abnormalities and oral bacterial and fungal infections may reflect undiagnosed diabetes or identify poorly controlled diabetes. Successful management of oral infections, including periodontal diseases, seems to depend on establishing metabolic control in diabetic patients. Knowledge of oral co-morbidity among people with diabetes is generally poor and suggests the need for appropriate health education and health promotion to improve the oral health of diabetic patients.

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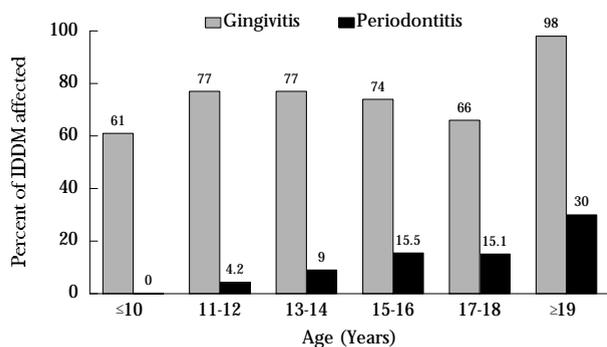
### CARIES (TOOTH DECAY)

Children with IDDM have been reported to have caries incidence that is higher<sup>1</sup>, lower<sup>2-4</sup>, or similar to that of nondiabetic children. This contradiction may possibly be explained by cohort characteristics, degree of diabetes control, and degree of adherence to dietary prescriptions. Adult patients with poor control of their IDDM seem to have more coronal caries<sup>5-7</sup>. In the general population, the frequency of root caries increases with age and is three times more prevalent in those age  $\geq 65$  years compared with young adults<sup>8</sup>. However, very few studies have reported on the incidence of root-surface caries as a significant problem in older patients with IDDM or NIDDM<sup>9</sup>.

### PERIODONTAL DISEASE

Periodontal disease is the most prevalent oral complication in IDDM and NIDDM patients and has been labeled the "sixth complication of diabetes mellitus"<sup>10</sup>. Numerous studies have shown both increased prevalence and severity of periodontal disease in patients with IDDM. Diabetic children and adults with less than optimal metabolic control show a tendency towards higher gingivitis scores<sup>11-17</sup>. Early case reports suggested that diabetic adolescents and teenagers may suffer from periodontitis<sup>18</sup>. In a more recent study, the prevalence of periodontal disease was 9.8% in 263 patients with IDDM, compared with 1.7% in people without diabetes<sup>19</sup>. Most of the periodontal disease was found in those age 11-18 years (Figure 23.1).

**Figure 23.1**  
Prevalence of Periodontal Disease in IDDM Patients, by Age

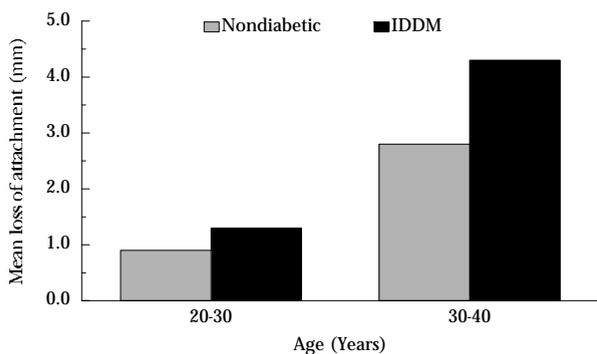


Source: Reference 19

However, earlier rapid periodontal destruction was not found in adolescent patients with IDDM in Finland<sup>15</sup>. This difference may be related to different levels of metabolic control in participants of the two studies. For example, case reports suggest a strong relationship between rapid periodontal breakdown and elevated blood glucose levels<sup>20</sup>.

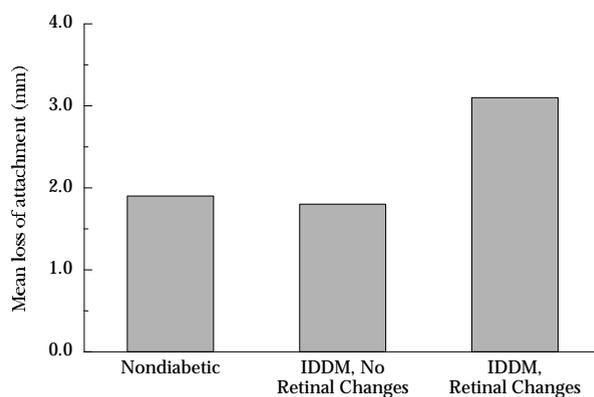
Patients with IDDM of >10 years duration had greater loss of periodontal attachment compared with those of <10 years duration<sup>21</sup>. This was found to be particularly true for patients age ≥35 years (Figure 23.2). More recently, it was reported that IDDM patients age 40-50 years with long IDDM duration had significantly more sites with advanced periodontal destruction and alveolar bone loss than people without diabetes<sup>17</sup>. It has also been demonstrated<sup>21</sup> and confirmed<sup>16</sup> that in IDDM patients with retinal changes

**Figure 23.2**  
Periodontal Attachment Loss in IDDM Patients and Nondiabetic Persons



Source: Reference 21

**Figure 23.3**  
Periodontal Attachment Loss According to Retinal Changes in IDDM Patients and Nondiabetic Persons



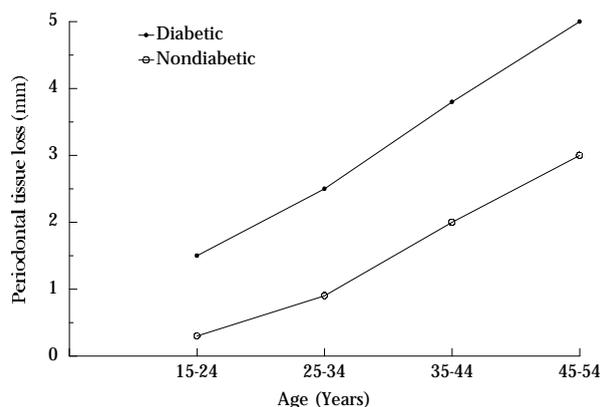
Source: Reference 21

the loss of periodontal attachment is significantly larger than in IDDM patients without retinal changes (Figure 23.3).

Several studies have clearly demonstrated that IDDM patients with poor long-term control of diabetes have increased extent and severity of periodontal disease, whereas those who maintain good metabolic control have minimal periodontal problems. Patients with IDDM of long duration who have retinopathy tend to exhibit more loss of periodontal attachment as they reach age 40-50 years. Good oral home care and frequent professional check-ups and care are important for these patients<sup>22</sup>.

Few studies have dealt with NIDDM subjects. In a

**Figure 23.4**  
Severity of Periodontal Disease Among Diabetic and Nondiabetic Pima Indians



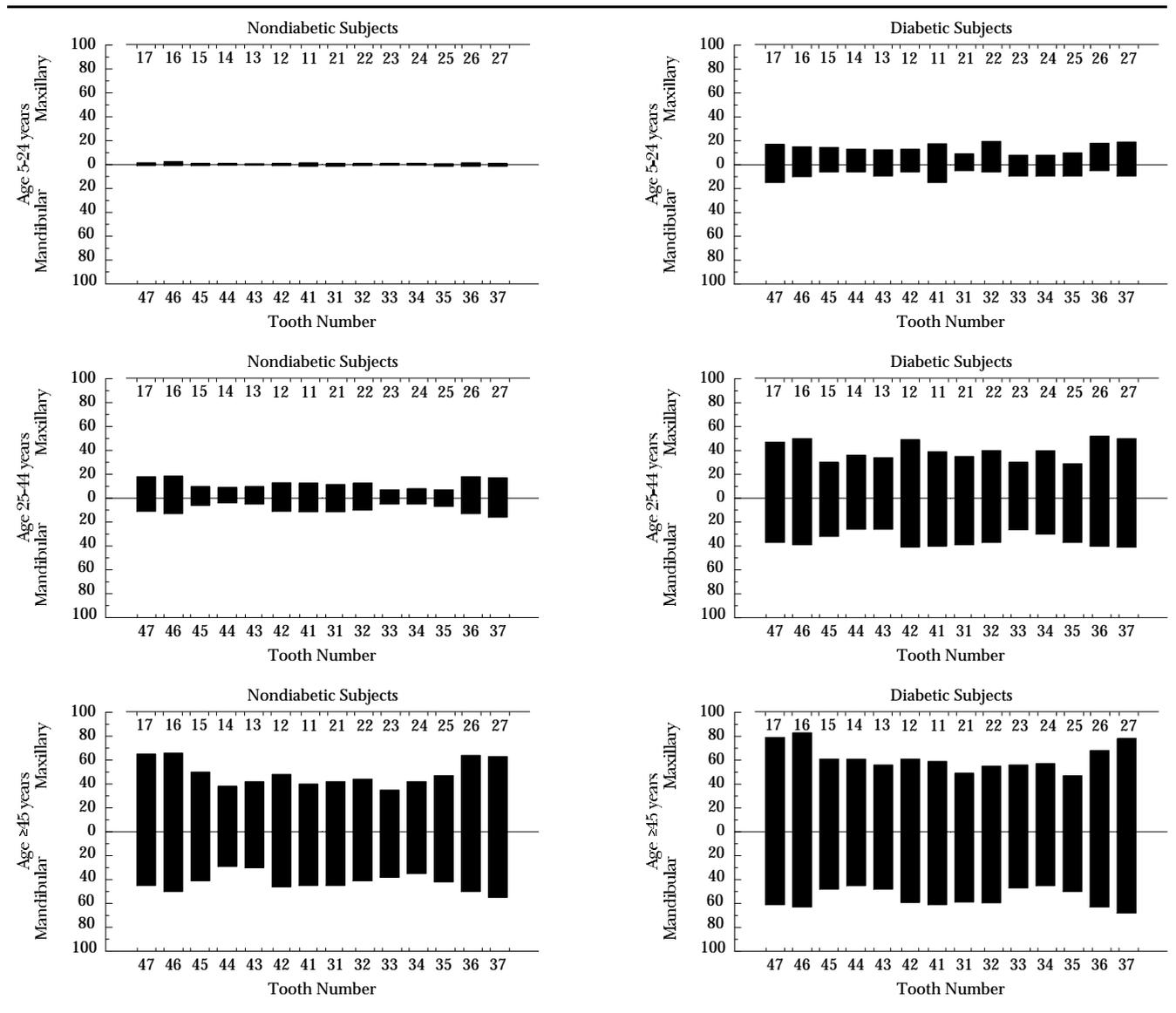
Source: Reference 23

study of Pima Indians, 40% of whom have NIDDM, diabetic patients age <40 years had increased attachment loss, and alveolar bone loss was associated with increased glucose intolerance<sup>23</sup>. Periodontal tissue loss increased with age and was higher in people with diabetes compared with people without diabetes in all age groups (Figure 23.4). Alveolar bone loss also increased with age and was substantially more frequent in patients with NIDDM compared with nondiabetic people age 5-44 years (Figure 23.5). Toothlessness was 15 times higher in the diabetic than in the nondiabetic group. Indeed, 30% of these young adults with

NIDDM had no teeth. The odds ratio for subjects with NIDDM for increased risk of periodontal destruction was 3.43 (95% confidence interval (CI) 2.28-5.16)<sup>24</sup>. In this population, the age- and sex-adjusted incidence of periodontal disease in subjects with NIDDM was 75 cases per 1,000 person-years, which was substantially higher than the rate of 29 cases per 1,000 person-years in subjects without diabetes<sup>25</sup> (Table 23.1).

Early studies of the pathogenesis of periodontal disease in diabetic patients centered on the general feature of "basement membrane thickening"<sup>26</sup> and possi-

**Figure 23.5**  
**Distribution of Interproximal Alveolar Bone Loss in Diabetic and Nondiabetic Persons**



Bone loss is expressed as percent of individuals who have teeth affected by  $\geq 25\%$  bone loss as determined by measurements from panoramic radiographs. The 2-digit tooth numbers are: first digit refers to the quadrant (1, upper right; 2, upper left; 3, lower left; 4, lower right); second digit refers to tooth type (7 and 6 are molars; 5 and 4 are premolars; 3 is a canine; 2 and 1 are incisors).

Source: Reference 23

Table 23.1

**Incidence of Periodontal Disease in Pima Indians by Diabetes Status**

Diabetes status	Age- and sex-adjusted incidence (new cases/1,000 person-years)
Nondiabetic	28.9
NIDDM	75.5
Relative risk	2.6

Source: Reference 25

ble changes in the vasculature<sup>27-29</sup>. More recent studies have focused on the role of the periodontal infection<sup>15</sup>, the microflora of dental plaque<sup>30</sup>, collagen metabolism<sup>31,32</sup>, leukocyte function<sup>33</sup>, and other aspects of the host response<sup>34,35</sup>. All of these factors may individually or synergistically contribute to periodontal disease.

The reason for the greater occurrence of periodontal destruction in diabetics is not clear. However, studies of the periodontal flora find similar microorganisms in diabetic and nondiabetic people<sup>30,36</sup>, suggesting that alteration in host responses to periodontal pathogens account for these differences in periodontal destruction. For example, increased susceptibility to infection by periodontal bacteria associated with altered phagocyte functions and reduced healing capacity associated with altered collagen metabolism may explain, in part, the increased levels of periodontal disease in diabetic patients.

The response to treatment suggests that the periodontal lesions are eminently treatable<sup>37,38</sup> and that eradication of the infection and the inflammatory foci may reduce insulin requirements<sup>39</sup>. The knowledge among people with diabetes of oral co-morbidity is generally poor<sup>40</sup> and suggests the need for appropriate health education and health promotion to improve the oral health of diabetic patients.

**SALIVA**

Reduced salivary secretion has been a frequent finding in experimental diabetes in animals<sup>41</sup> as well as in IDDM patients. A non-inflammatory, non-neoplastic enlargement of the parotid gland is believed to occur in 25% of patients with moderate to severe diabetes and especially in IDDM patients with poor metabolic control<sup>42</sup>. The etiology of this condition is unknown, but it is speculated that the enlargement occurs in response to decreased insulin production or that the Sjögren's syndrome may underlie this symptom<sup>43</sup>. Also, the possibility that in some cases these enlarge-

ments may be due to a low degree of mumps infection has been mentioned<sup>44</sup>. Increased concentration of Ca<sup>++</sup> in both parotid and submandibular saliva of IDDM subjects<sup>45</sup> might explain the frequently reported increase in calculus formation in such patients. However, in well-controlled individuals with altered glucose metabolism, salivary gland function does not seem to be significantly impaired<sup>46</sup>.

**OTHER PATHOLOGICAL FEATURES OF THE ORAL CAVITY**

Other pathology associated with diabetes includes oral infections other than those responsible for dental caries and periodontal destruction. Case reports on life-threatening deep neck infection from a periodontal abscess<sup>47</sup> and fatal palatal ulcers<sup>48</sup> exemplify the severity of these conditions. To what extent such incidents are part of the broader issue of increased occurrence of infection in people with diabetes, or may have strictly a local etiology, is open to question. In addition to these infections, other localized or regional infections such as mucormycosis, "malignant otitis media," necrotizing cellulitis, urinary tract infections, skin infections, and pneumonia have also been found more often in poorly controlled diabetic patients than in others. There are also indications that patients with elevated salivary glucose levels carry candida intraorally more often than those with lower glucose levels<sup>49</sup>. Moreover, a study of 40 patients with lichen planus found that 11 patients (28%) had overt or latent diabetes, compared with none of the control group<sup>50</sup>, the implication being that diabetes may be related to the pathogenesis of lichen planus. The evidence for an immunological defect<sup>51</sup> and deficient leukocyte functions superimposed on the metabolic abnormality of diabetes seems increasingly convincing.

Finally, it should be mentioned that diabetes may initially manifest with oral symptoms other than thirst<sup>52</sup>. Mucosal abnormalities, such as erosive lichen planus, burning tongue, and gingival bleeding, as well as sialorrhoea and sialosis, have been found in undiagnosed NIDDM, most of which resolved on treatment directed at improving glycemic control<sup>52</sup>.

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