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From Your Dentistry for Diabetics (DFD) Professional
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Informed

The truth about the diabetic & oral care

Glycemic Control and Periodontal Destruction

Much has been written about the destructive relationship between poorly-controlled diabetes and periodontal disease. It has been well documented that diabetics displaying marginal glucose levels contract periodontal disease up to 3.4 times more often than non-diabetics or diabetic subjects with good control.

We also know that the severity of periodontal destruction is often matched by HbA1c levels. As glucose levels worsen, so does periodontal health. Additionally, the 2007

report from the American Diabetes Association that stated that only **7.3 percent** of diabetic subjects achieved all three **treatment goals at once**.¹ If both are true, then collaboration between physicians and dentists treating diabetics may be an imperative in the ethical care of diabetic patients.

But what are the mechanisms behind glycemic control and periodontal destruction? This month's newsletter will attempt to answer that question.



Did You Know?

Periodontal disease and oral candidiasis (thrush) are markers for poorly controlled diabetes.

Etiology Behind Glycemic Levels and Periodontal Disease Rates

It would be simple (and also correct) to say that the high incidence and increased severity of periodontitis in diabetic patients is due to many of the same mechanisms and defects that drive other “classic” complications of diabetes. However, by understanding the etiology of periodontitis when combined with diabetes mellitus, we are better able to prevent onset of oral health complications and treat their effects.

According to the American Academy of Periodontology (1999), there are few (if any) differences between the subgingival microflora of non-diabetic subjects versus diabetic subjects, who have periodontal disease. That finding is supported by multiple studies.²⁻⁴ However, this lack of pathogenic differentiation suggests that immune response may be a causal factor for diabetic patients.

As you know, cell function for neutrophils, monocytes and macrophages can be inhibited by progression of diabetes. When that occurs, the result is often inhibited destruction of bacterial pathogens in perio tissue (as well as other vulnerable tissue). Propagation of such pathogens ultimately expedites soft tissue destruction. In the case of the periodontium, it carves out larger and larger pockets in which bacterium continue to grow, infect and inflame host tissue.

In addition, during phagocytosis, monocyte-macrophages become hyper-responsive due to bacterial antigens and may trigger increases in pro-inflammatory cytokines and mediators.

What may be most telling are the findings by Salvi et al that examined monocytic secretion patterns in diabetic patients (1997).⁵ In it, diabetic subjects were tested against non-diabetic subjects to measure TNF-

alpha levels. Researchers found that, when given antigens from *Porphyromonas gingivalis*, subjects with DM had elevated TNF-alpha levels compared with non-diabetic subjects. *P. gingivalis* has been found to lead to prolonged inflammatory response – unrelated to pathogens, suggesting that inflammation may be directly related to TNF stimulation.

HbA1c levels greater than 8 percent displayed crevicular fluid levels of interleukin -1 beta that were ~2X that of test subjects whose HbA1c levels were less than 8 percent.

This inflammatory response can be traced via elevated serum levels in gingival crevicular fluid, which is also directly related to glycemic control. In fact, a study published in the 2004 J. Periodontics found that subjects with HbA1c levels greater than 8 percent displayed crevicular fluid levels of interleukin -1 beta that were ~2X that of test subjects whose HbA1c levels were less than 8 percent.

These findings suggest that inflammation is strongly influenced by blood glucose levels, specifically in subgingival tissue.

1. Standards of Medical Care in Diabetes—2007; Diabetes Care, American Diabetes Assoc. 2007. Vol. 30 (Sup. 1); S4-S33.
2. Sastrowijoto S, Hillemans P, van Steenberg T, Abraham-Inpijn L, de Graaaf J. J. Periodontal condition, microbiology of healthy & diseased periodontal pockets in type 1 diabetes mellitus patients. J Clin Periodontol 1989;16:316-322.

Check it out:

In a cross-sectional study of patients with type 1 diabetes for a mean duration of more than 16 years, subjects with poor glycemic control had more interproximal attachment loss and alveolar bone loss than well-controlled subjects .

Safkin-Seppala B., Ainamo J. Periodontal conditions in insulin-dependent diabetes mellitus
J Clin Periodontol 1992; 19:24-29

3. Zambon JJ, Reynolds H, Fisher JG, Shlossman M, Dunford R, Genco RJ. Microbiological and immunological studies of adult periodontitis in patients with non-insulin dependent diabetes mellitus. J Periodontol 1997;24:8-16.
4. Sbordone L, Ramaglia L, Barone A, Ciaglia RN, Iacono VJ. Periodontal status and subgingival microbiota of insulin-dependent

juvenile diabetics: A 3-year longitudinal study. J Periodontol 1998;69:120-128.

5. Salvi GE, Collins JG, Yalda B, Arnold RR, Lang NP, Offenbacher S. Monocytic TNF- α secretion patterns in IDDM patients with periodontal diseases. J Clin Periodontol 1988;59:23-31.

Mechanisms Behind Periodontal Destruction

Altered immune response may explain increased incidence of periodontal disease and long-term inflammation of the periodontium. But what are the mechanisms that lead to advanced destruction of periodontal tissue, alveolar bone loss, attachment loss and endentulous?

In addition to altered immuno-inflammatory response, studies have found that changes in metabolism impact connective tissue resorptive and formative function. Similarly, glycemic state affects osseous healing by inhibiting osteoblastic cell growth.¹⁻³ The result is reduced alveolar bone, and diminished mechanical properties of newly formed bone.

Subjects with type 2 diabetes also displayed greater alveolar bone loss associated with increased glucose intolerance or poor metabolic control.

What is interesting, according to a study published in Endocrinology (2003), is that when insulin treatment was given, reduced expression of Cbfa1 and Dlx5 was reversed.⁴⁻⁷

Studies have found that infection of *P. gingivalis* can trigger the hyperglycemic state itself, which in turn increases apoptosis. What follows is diminished fibroblastic and osteoblastic capability (those cells responsible for matrix production). The associated

cell death, and weakened growth and differentiation, may explain (with strong evidence) why patients with periodontal disease AND diabetes suffer attachment loss and bone destruction at a rate as high as 11 times greater than non-diabetics.⁸

Immune response, inflammation, alteration in connective tissue metabolism, decreased matrix-producing cell activity are just some of the mechanisms by which diabetes contributes to elevated periodontal destruction. All are related to glycemic control. All can be treated, prevented or delayed with proper medical and dental care.

According to research, a treatment program that combines mechanical and chemical debridement may reduce circulating TNF- α and serum levels of glycosylated hemoglobin – ultimately helping to regulate metabolic levels.⁹

The treatments must be used in tandem to defer transient bacteremia or lipopolysaccharidemia caused by mechanical debridement by itself. When the following were incorporated into treatment:

- Ultrasonic bactericidal curettage
- Topic antimicrobial
- Systemic doxycycline

...Researchers found that test subjects had improved metabolic control three months after the initial treatment.⁹

Did you know?

Type 1 diabetic subjects with poor metabolic control over the preceding 2-5 years had a significantly greater prevalence of deep probing depths and advanced attachment loss than subjects with good glycemic control.

IN SUM

In sum, the impact of glycemic control on periodontal health for the diabetic patient is complex and multifaceted. As with all systemic diseases that lead to morbidity, prevention is key. Management, oral and systemic treatment, patient education, properly administered medications and more contribute to oral health as well as overall health. Collaborative relationships between treating physicians and dentists, who understand the special circumstances surrounding the diabetic state, is the first step in proper, ethical care.

Next month's newsletter will examine micro-vascular change, and its effect on oral health.

1. Inaba M, Nishizawa Y, Mita K, et al. Poor glycemic control impairs the response of biochemical parameters of bone formation and resorption to exogenous 1,25-dihydroxyvitamin D3 in patients with type 2 diabetes. *Osteoporos Int* 1999;9:525-531.
2. Tisdell CL, Marcus RE, Heiple KG. Triple arthrodesis for diabetic

peritalar neuroarthropathy. *Foot Ankle Int* 1995;16:332-338.

3. Beam HA, Parsons JR, Lin SS. The effects of blood glucose control upon fracture healing in the BB Wistar rat with diabetes mellitus. *J Orthop Res* 2002;20:1210-1216.
4. Beam HA, Parsons JR, Lin SS. The effects of blood glucose control upon fracture healing in the BB Wistar rat with diabetes mellitus. *J Orthop Res* 2002;20:1210-1216.
5. Gooch HL, Hale JE, Fujioka H, Balian G, Hurwitz SR. Alterations of cartilage and collagen expression during fracture healing in experimental diabetes. *Connect Tissue Res* 2000;41:81-85.
6. Lu H, Kraut D, Gerstenfeld LC, Graves DT. Diabetes interferes with bone formation by affecting the expression of transcription factors that regulate osteoblast differentiation. *Endocrinology* 2003;144: 346-352.
7. Amir G, Rosenmann E, Sherman Y, Greenfeld Z, Ne'eman Z, Cohen AM. Osteoporosis in the Cohen diabetic rat: Correlation between histomorphometric changes in bone and microangiopathy. *Lab Invest* 2002;82:1399-1405.
8. Taylor GW, Burt BA, Becker MP, et al. Non-insulin dependent diabetes mellitus and alveolar bone loss progression over 2 years. *J Periodontol* 1998;69: 76-83.
9. Vernillo, A.T., DDS, PhD, Dental considerations for the treatment of patients with diabetes mellitus. *JADA*, 2003; Vol. 134:24S-32S

What's Clicking? Dentistry For Diabetics

The job of this newsletter is to provide in-depth information about the many connections between oral health and diabetes. We have examined research findings focused on the proclivity by diabetic patients to suffer advanced oral diseases. We have discussed the reasons why diabetic patients are unwilling or unable to get the vigorous oral care needed to prevent and treat oral diseases. We have reviewed risk factors and lifestyle choices that contribute to systemic and oral complications.

Today, we introduce **DentistryForDiabeticssm**, an organization made up of dentists dedicated to the care of diabetic patients. Their mission is to support physician-directed management programs while aggressively treating oral health diseases related to diabetes. To that end, they are trained in the following:

- **General diabetes management practices** — Dietary, oral health and lifestyle counseling. DFD dentists may assist the physician in monitor and counsel of patients. They may also assist in smoking cessation programs.

- **Glycemic assessment** — periodontal disease and candidiasis are markers for poorly controlled diabetes; patients presenting with these and other risk factors, may be tested for A1C levels and referred to physician
- **Drug interaction** — Common medications used in oral care such as steroidal treatment for Lichen planus can lead to hyperglycemia, if not managed carefully. Cyclosporine (used following organ transplants) may cause gingival overgrowth. More than **400 medications** in use today produce dry mouth, which can be damaging to gum tissue and trigger periodontal disease and dental carries.

To learn more of about **DentistryForDiabeticssm**, contact the dentist who sent this newsletter.

From:

To: