THE SILENT KILLER:
Understanding and Addressing the Inflammatory Pathway

Why another course on the oral-systemic link?

FACT: The information has been redefining our understanding of how inflammation is at the very core of today’s complex, prevalent and deadly diseases

FACT: Moderate to severe chronic periodontitis has significant systemic implications

FACT: The AAP has redefined periodontal disease as an inflammatory disease with far reaching effects.

FACT: We need to reconsider our therapeutic endpoints to ensure that the impact of oral disease does not continue to threaten overall health

FACT: We need to meet the needs of today’s population

SCIENCE MEETS THE DEMOGRAPHIC

We OWN This:
Defining of a healthcare professional:
“An occupation whose core element is work based upon the mastery of a complex body of knowledge and skills....to be used in the service of others. Professions and their members are accountable to those served and to society. Society rewards health professionals...this status, however, comes with professional obligations.”

Self-Evaluation: Rate Your Present Periodontal Therapy Program
1. How satisfied are you with your present periodontal therapy program?
2. Are you receiving predictable outcomes?
3. What do you feel would elevate your periodontal program to the next level?
4. How are you addressing the inflammatory component of periodontal disease?
5. Do you have an evidence based risk assessment program in place?
6. Do you feel your medical history update is uncovering sufficient information to fully address the needs of your dental hygiene patient?
7. Do you have adequate resources to educate your patient about the oral-systemic link?
8. What treatment modalities have you incorporated into your periodontal therapy program in order to reduce the bacterial burden?
9. What treatment modalities have you incorporated into your periodontal therapy program in order to address the host response?
10. What are your determinants and criteria for referring to a periodontist?

Presented by: Jo-Anne Jones, RDH
President, RDH Connection Inc.
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Synopsis:
Are we still treating periodontal disease as an infection when leading authorities have redefined periodontitis as an inflammatory disease? Is our practice compliant with the most recent JADA guidelines?

The link between periodontal disease and systemic health is becoming increasingly clear and is the benchmark of innovative healthcare delivery. Long running, ongoing chronic disease such as periodontal disease tips the body’s balance towards chronic inflammation.

Inflammation often being referred to as the ‘silent killer’ is one of the hottest topics of research. The latest research surrounding the oral systemic link is both startling and compelling. What if we now could slow down the destruction caused by chronic inflammation and impact not only oral health but systemic health? We have the ability to change lives through the power of our message and the science of our treatment.

Learning Outcomes:
1. Recognize the role of the inflammatory pathway in initiating disease states within the body
2. Elevate understanding and treatment of periodontal disease as an inflammatory rather than an infectious disease
3. Incorporate new therapeutic modalities and communication strategies to enhance treatment outcomes

References:
References & Resources:


http://www.cdho.org/Advisories/CDHO_Advisory_Hy pertension.pdf

(p. 21, 22)

Is it mandatory to take my patient’s blood pressure?
Definition of Hypertension:
A condition where blood pressure persistently exceed specified limits
One of the leading health problems preceding stroke, heart attack, kidney failure, dementia and sexual dysfunction
About 75 million American adults (29%) have high blood pressure... 1 out of every 3 adults
Only about half (54%) have their condition under control
Often asymptomatic; referred to ‘silent killer’
Responsibility of Today’s Dental Hygienist:
- Important to have a baseline as part of initial assessment
- Requirement of blood pressure to be taken when medical history indicates a need
- Ensure patient is not being placed at risk before initiating dental hygiene treatment
- If patient’s history is clear, a registrant is encouraged to take a baseline assessment; prudent and proactive to periodically monitor as often asymptomatic

The Facts on Diabetes:
FACT: Nearly 30 million Americans have diabetes and face its devastating consequences
FACT: Periodontal disease is listed as the 6th complication
FACT: 82% of diabetic patients with severe periodontitis experienced the onset of one or more major cardiovascular, cerebrovascular or peripheral vascular events compared to only 21% of diabetics without periodontitis.
Understanding the Oral-Systemic Link: Diabetes
Research supports that infectious and inflammatory processes increase insulin resistance resulting in hyperglycemia.
Hyperglycemia (elevated blood glucose) diminishes the ability of WBC, neutrophils in particular to track, adhere and kill bacteria
Diabetes increases risk through an amplified inflammatory response and depressed wound healing; elevated blood glucose leads to elevated glucose levels in GCF hindering wound healing capacity of fibroblasts.
GCF contains elevated concentrations of cytokines producing higher levels of MMPs that promote tissue destruction and disease severity

References & Resources:
Last edited August, 2015

Diabetes: Clinical Considerations for Dental Hygiene Practice
NEW REPORTS CONFIRM PERIO-SYSTEMIC CONNECTION AND OUTLINE CLINICAL RECOMMENDATIONS
A diabetes management program should involve on-going comprehensive periodontal assessments
Independent association between moderate to severe periodontitis and increased risk for development or progression of diabetes
AAP and EFP Consensus Report... “periodontal interventions may provide beneficial effects on diabetes outcomes in some patients, so regular comprehensive periodontal evaluations should be part of an ongoing diabetes management program”
Solicit feedback regarding diabetic status;
• Type 1, 2 or pre-diabetic or familial history? What is their blood glucose target? What were the results of their last A1C test?

<table>
<thead>
<tr>
<th>Recommended blood glucose targets for people with diabetes*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Target for most patients with diabetes</strong></td>
</tr>
<tr>
<td>Fast. gluco. before meals (mmol/L)</td>
</tr>
<tr>
<td>≤7.0</td>
</tr>
<tr>
<td>≤5.0</td>
</tr>
<tr>
<td>≥4.0</td>
</tr>
<tr>
<td>≥3.0</td>
</tr>
<tr>
<td><strong>Blood glucose 2 hours after eating (mmol/L)</strong></td>
</tr>
<tr>
<td>5.0 to 10</td>
</tr>
<tr>
<td>4.0 to 6.0</td>
</tr>
</tbody>
</table>

- What was their blood glucose level the morning of the appointment? What times of the day are best for scheduled appointments?

Understanding the Oral-Systemic Link between Obesity and Periodontal Disease:
- A pro-inflammatory state exists in obesity as a result of the release of several cytokines and hormones from adipose tissue into systemic circulation; similar cytokines are released into circulation in periodontal disease
- A person with a BMI of 30 or more is generally considered obese; research is debating whether BMI, waist circumference (WC) or both should be used to determine disease risk
- Obesity is a major risk factor for a number of chronic diseases including type 2 diabetes, hypertension, cardiovascular disease, metabolic syndrome, liver disease, musculoskeletal disease, reproductive abnormalities and cancer. Recent studies have reported an association between obesity and periodontitis.
- Studies prove that a high prevalence of PD can be expected among obese adults

Obesity Rates in North America:
Between 1985 and 2011, the prevalence of overweight adults increased by 21% to 33.6% and obesity increased 200% from 6.1% to 18.3%.

Respiratory Disease

Biological Link between Periodontal Disease and Respiratory Disease:
Possible mechanisms for presence of oral bacteria in pathogenesis of respiratory invasions include;
- Dental plaque may serve as a reservoir for pulmonary pathogens responsible for aspiration pneumonia in high risk patients
- Enzymes associated with periodontal disease may facilitate adherence of respiratory pathogens to the mucosal tissues in the oral cavity and ultimate in the airways
- Hydrolytic enzymes associated with periodontal disease pathogens may destroy salivary pellicles and reduce their host defense capabilities
- Cytokines and other inflammatory mediators originating from the periodontal tissues may alter respiratory epithelium resulting in pathogen adherence and colonization.
Rheumatoid Arthritis (RA)
Understanding the Oral-Systemic Link with Rheumatoid Arthritis:
• Periodontal disease (PD) is an infection characterized by chronic inflammation, and may ultimately lead to tooth loss
• Rheumatoid arthritis (RA) is a chronic disease, characterized by inflammation of the synovium of the joints, and may ultimately lead to destruction of the joint
  o RA begins with inflammation of the synovial membrane...lymphocytes, neutrophils and other inflammatory cells migrate into the joint and release inflammatory chemicals that destroy body tissues
• Chronic inflammatory mediators are shared by both these diseases, and this has prompted researchers to investigate the possibility of a relationship between RA and PD

Arthritis Prevalence in U.S.
• >50 million adults have doctor-diagnosed arthritis; 1 in 5 over age 18
• >300,000 babies and children have arthritis or a rheumatic condition; 1 in 250 children
• Most common form is osteoarthritis (OA) which affects an estimated 31 million Americans
• More than 78 million people expected to have doctor-diagnosed arthritis by the year 2040
• Arthritis is the nation’s No. 1 cause of disability

Pregnancy (PLBW)
Understanding the Oral Systemic Link with Pregnancy (PLBW):
• 1 in 10 infants born are considered to be preterm; improvements in neonatal intensive care medicine have improved the survival rate however rate of premature delivery has steadily climbed since the 1950’s
• Other risk factors include race, smoking, alcohol and drug use, lower socioeconomic status and lower education; more than ¼ of all complicated pregnancies occur for no apparent reason
Periodontal disease may contribute by presenting an infectious, inflammatory ongoing challenge to the fetus

References & Resources:
Scannapieco FA. Role of oral bacteria in respiratory infection. J Periodontol. 1999; 70:793-802
Feb 2015 Statement:

During normal pregnancy, the placenta invades the surrounding uterine tissue and provides an exchange of nutrients and waste between mother and fetus via the umbilical cord. As pregnancy progresses, amniotic fluid levels containing prostaglandin E2 (PGE2) and inflammatory cytokines—tumor necrosis factor (TNF-α) and interleukin 1 (IL-1β)—steadily rise to reach the threshold that induces labor. Thus, normal labor and delivery are induced by inflammatory signaling. One theory for the association between periodontal diseases and preterm birth is that women with periodontitis, a bacterial infection, exhibit an increase in fluid mediator levels and inflammatory cytokines, which can trigger labor prematurely. Furthermore, an increase in other markers of inflammation such as C-reactive protein (CRP) has been associated with an elevated risk for preeclampsia and intrauterine growth restriction.

Osteoporosis

**Biological Link between Periodontal Disease and Osteoporosis:**
- In periodontal disease, chronic oral inflammation results in destruction of oral bone and periodontal ligament
- Increased production of cytokines, IL-6 stimulate osteoclast activity and promote bone resorption
- Similar mechanism may contribute to osteoporosis
- Evidence indicates there is an association between the two diseases
- Common risk factors; age, genetics, estrogen deficiency, calcium and Vitamin D deficiency, alcohol intake and smoking

Alzheimer’s disease

**Understanding the Systemic Link between Alzheimer’s and Periodontal Disease:**
- Alzheimer’s disease (AD) is a degenerative disease of the brain characterized by neurofibrillary tangles and the accumulation of beta amyloid plaques
- A strong positive correlation was found between midlife C-reactive protein levels, a marker of inflammation and the risk of developing AD. The chronic nature of oral infections, such as periodontitis, may further amplify the mechanisms that lead to the onset or progression of AD.

References & Resources:


Notes:

References & Resources:

- It is possible that periodontal pathogens may directly invade the central nervous system via systemic circulation; oral Treponema may have reached the brain via the trigeminal nerve.

**Recognize the Role of the Inflammatory Pathway in Initiating Disease States within the Body**

**FACT: The Common Link – Inflammation:**
- Today’s diseases of influence are linked by the inflammatory pathway
- Periodontal disease is the most common chronic inflammatory disease known to mankind
- Living longer, consequences of Western lifestyle adding to today’s inflamed body

**Elevate Understanding and Treatment of Periodontal Disease as an Inflammatory Disease**

**We’ve Lost the Battle...** when we focus on reducing the bacterial component only, we do not achieve the reduction of the host response. **Inflammation** and destruction continues placing healing, repair and systemic health in jeopardy.

**FACT: American Academy of Periodontology Statement:**
- «Research has shown that periodontal disease is associated with several other diseases. For a long time it was thought that bacteria was the factor that linked periodontal disease to other disease in the body; however, more recent research demonstrates that inflammation may be responsible for the association. Therefore, treating inflammation may not only help manage periodontal diseases but may also help with the management of other chronic inflammatory conditions.»

**Today’s Periodontal Therapy Program Objective:**
- Traditional clinical periodontal examination includes assessment of already existing damage to periodontal tissues as well as measure of periodontal inflammation
- Cost effective, simple method in determining the location and severity of diseased periodontal tissues
- However, in predicting future periodontal breakdown or even just quantifying current disease activity...especially inflammation, these methods are far from ideal
- Oral inflammatory load (OIL) not pocket depths or periodontal pathogenic bacteria explains the linkages with systemic disease

**FACT: Call to Action for Dental Hygienists**
- “If we, in dentistry, are indeed healers, it is imperative for us to take a different approach... the goal is to help patients become and remain inflammation-free.”

Dr. Tim Donley
Understand and Apply the 2015 JADA Guidelines into Clinical Practice and Treatment Delivery

1. Adequate Management of Risk Factors:
   - Philips Oral Healthcare CARE tool (Customized Assessment and Risk Evaluator) web-based patient interview and integration of risk management program into dental hygiene clinical practice: https://www.philipcare.com

2. Adequate Bacterial Reduction:
   - Biofilm leads to bacteremia
   - Onset of bacteremia initiates inflammatory response
   - Systemic involvement

Solutions for Effective Patient Self-Care
www.curaprox.com
www.philipsoralhealthcare.com
www.3m.com/3M/en_US/oral-care-us/

3. Address Host Response

Periodontal Inflammation and Destruction:
   - Cytokines are an intermediate mechanism between bacterial stimulation and tissue destruction; may also be produced by fibroblasts and osteoblasts
   - The host response is the major contributing factor for chronic maladaptive periodontal disease. A deficient host response initiates the chronic condition and response that leads to further tissue breakdown
   - Bacteria initiate periodontitis. They are essential but insufficient. What is required is a susceptible host.
   - Primary etiologic basis for periodontal disease is bacterial however the excessive host inflammatory response or inadequate resolution of inflammation is critical to the pathogenesis of periodontitis.

Host Modulation: Low-dose doxycycline (LDD)
Medical and Dental Benefits
About 30 years ago, Golub et al discovered that doxycycline had the unexpected ability to inhibit host-derived tissue-destructive enzymes known as MMPs by mechanisms unrelated to the antibacterial/antibiotic properties of these drugs
These enzymes when present in pathologically-excessive levels are largely responsible for degrading collagen fibers and mediating bone resorption related to various medical and dental diseases
Over the past decade this novel non-antimicrobial LDD has been tested in patients with medical disorders which excessive MMPs and inflammatory mediators play a role

References & Resources:
http://www.dentalproductsreport.com/dental/article/5-things-consider-regarding-connection-between-stroke-inflammation
T Van Dyke, C Serhan, A Novel Approach to Resolving Inflammation, Oral and the Whole Body Health; 2006;42-45
D Graves, Cytokines That Promote Periodontal Tissue Destruction, J Periodontol (Suppl.), 2008; 1585-1591
TIME Magazine article: http://www.inflammationresearchfoundation.org/inflammation-science/inflammation-details/time-cellular-inflammation-article/
Therapeutic Benefit of Inhibiting Collagen Breakdown:
- Inhibit breakdown of collagen in diseased joint (synovial) tissues reducing severity of symptoms in ARTHRITIS
- Inhibit breakdown of collagen in connective tissues around CANCER cells: reduced local invasiveness and metastasis
- Protect collagen “cap” stabilizing cholesterol-rich arterial plaques: reduced risk for MYOCARDIAL INFARCTION & STROKE
- Reduce diagnostic biomarkers of skeletal bone resorption for POST MENOPAUSAL OSTEOPOROSIS with no effect on biomarkers of bone formation
- Reduce blood levels of Hemoglobin A1C after SDD + SRP for DIABETICS

STATEMENT ON PERIOSTAT® AS AN ADJUNCT TO SCALING AND ROOT PLANNING

Reduces the elevated collagenase activity in the gingival crevicular fluid of patients with adult periodontitis. Periostatin® is a systemically delivered collagenase inhibitor consisting of 3-10 mg capsule of dicyclic hydrochloride for oral administration. This is the first FDA approved systemic drug for host modulation as an adjunct to scaling and root planing in the treatment of periodontitis. Periostatin® administered bid reduced the elevated collagenase activity in the gingival fluid of patients with adult periodontitis.

A randomized, multi-center, double blind study was performed to compare the efficacy of scaling and root planing (SRP) plus placebo to scaling and root planing plus Periostatin® administered bid. That study revealed statistically significant pocket depth reduction with adjunctive use of Periostatin® at 3, 6, and 9 months post initial therapy (for initial depth > 7 mm, 120 vs. 66.8 mm, at depths 4.6 mm, 0.69 vs. 0.95 mm) and gain of clinical attachment (for initial depths > 7 mm, 1.17 mm vs. 1.35 mm, at depths 4.6 mm, 0.86 vs. 1.03 mm). Mean changes in pocket depth and attachment level across large numbers of patients and both sites were small, and may not reflect the magnitude of change that may occur in an individual patient or tooth site. For example, if SRP + Periostatin® was compared with SRP + placebo, more sites initially demonstrating 5 to 8 mm probing depth exhibited >2 mm reduction in probing depth (41% vs. 30%, 886 vs. 640 sites).

In a 3-month follow-up study, where patients received no additional therapy, pocket depth reductions and clinical attachment level gains observed following 9 months adjunctive Periostatin® were maintained. The Academy is not aware of any data regarding treatment outcomes for periods longer than 12 months.

Periostat: Mechanism of Action
Periostat will help to reduce the over-production of collagenase (enzymes responsible for the destruction of collagen) and osteoclasts (bone cell responsible for the resorption of bone) that are present in overabundance during a chronic, prolonged & destructive inflammatory response.

This exaggerated inflammatory response is common among inflammatory diseases such as periodontitis, cardiovascular disease and rheumatoid arthritis.

Therefore, Periostat, when used (BID) for 6 to 9 months, will help to modulate the chronic, prolonged & destructive inflammatory response into a normal & healthy inflammatory response process.

References & Resources:
Sub-antimicrobial Dose Doxycycline (SDD) Study Listing at end of handout
“My patient doesn’t want to take any more medications”
KEY TALKING POINTS:

“I’m concerned with my patient experiencing side effects from taking an antibiotic for so long”
KEY TALKING POINTS:

“My patient doesn’t have any real medical concerns at this time”
KEY TALKING POINTS:

“My patient comes in regularly, has effective self-care measures. Insurance covers a 3 month maintenance interval. There is still inflammation present however I don’t feel he needs Periostat.”
KEY TALKING POINTS:
Non-antibacterial tetracycline formulations: clinical applications in dentistry and medicine.

Gu Y, Walker C, Ryan ME, Parne JR, Golub LM

Abstract
In 1963, it was first reported that tetracyclines (TCs) can modulate the host response, including (but not limited to) inhibition of pathologic matrix metalloproteinase (MMP) activity, and by mechanisms unrelated to the antibacterial properties of these drugs. Soon thereafter, strategies were developed to generate non-antibacterial formulations (subantimicrobial-dose doxycycline: SDD) and compositions (chemically modified tetracyclines: CMTs) of TCs as host-modulating drugs to treat periodontal and other inflammatory diseases. This review focuses on the history and rationale for the development of (a) SDD which led to two government-approved medications, one for periodontitis and the other for acne/rosacea and (b) CMTs, which led to the identification of the active site of the drugs responsible for MMP inhibition and to studies demonstrating evidence of efficacy of the most potent of these, CMT-3, as an anti-angiogenesis agent in patients with the cancer. Kaposi’s sarcoma, and as a potential treatment for a fatal lung disease (acute respiratory distress syndrome, ARDS). In addition, this review discusses a number of clinical studies, some up to 2 years’ duration, demonstrating evidence of safety and efficacy of SDD formulations in humans with oral inflammatory diseases (periodontitis, pemphigoid) as well as medical diseases, including rheumatoid arthritis, post-menopausal osteopenia, type II diabetes, cardiovascular diseases, and a rare and fatal lung disease, lymphangioleiomyomatosis.


Treatment of early seropositive rheumatoid arthritis: doxycycline plus methotrexate versus methotrexate alone.

Abstract
OBJECTIVE: To compare the efficacy of doxycycline plus methotrexate (MTX) versus MTX alone in the treatment of early seropositive rheumatoid arthritis (RA), and to attempt to differentiate the antibacterial and antimatrix metalloproteinase effects of doxycycline.

METHODS: Sixty-six patients with seropositive RA of <1 year’s duration who had not been previously treated with disease-modifying antirheumatic drugs were randomized to receive 100 mg of doxycycline twice daily with MTX (high-dose doxycycline group), 20 mg of doxycycline twice daily with MTX (low-dose doxycycline group), or placebo with MTX (placebo group), in a 2-year double-blind study. Treatment was started with an MTX dosage of 7.5 mg/week, which was titrated every 3 months until remission was reached (maximum dosage of 17.5 mg/week). The primary end point was an American College of Rheumatology 50% improvement (ACR50) response at 2 years.

RESULTS: ACR50 responses were observed in 41.6% of patients in the high-dose doxycycline group, 38.9% of those in the low-dose doxycycline group, and 12.5% of patients in the placebo group. Results of chi-square analysis of the ACR50 response in the high-dose doxycycline group versus that in the placebo group were significantly different (P = 0.02). Trend analysis revealed that the ACR20 response and the ACR50 response were significantly different between groups (P = 0.04 and P = 0.03, respectively). MTX doses at 2 years were not different among groups. Four patients in the high-dose doxycycline group, 2 patients in the low-dose doxycycline group, and 2 patients in the placebo group were withdrawn because of toxic reactions.

CONCLUSION: In patients with early seropositive RA, initial therapy with MTX plus doxycycline was superior (based on an ACR50 response) to treatment with MTX alone. The therapeutic responses to low-dose and high-dose doxycycline were similar, suggesting that the antimatrix metalloproteinase effects were more important than the antibacterial effects. Further studies to evaluate the mechanism of action of tetracyclines in RA are indicated.


Engelbrecht SP, Hey-Hadavi J

Abstract
In vitro and animal studies suggest a possible role for the tetracycline class of drugs in the inhibition of non-enzymatic protein glycation. We conducted a 3-month, randomized placebo-controlled pilot clinical trial of conventional sub-gingival debridement (periodontal therapy), combined with either a three month regimen of sub-antimicrobial-dose doxycycline (SDD), a two week regimen of antimicrobial-dose doxycycline (ADD), or placebo in 45 patients with long-standing type 2 diabetes (mean duration 9 years) and untreated chronic periodontitis. Subjects were taking stable doses of oral hypoglycemic medications and/or insulin. Treatment response was assessed by measuring hemoglobin A1c (HbA1c), plasma glucose, and clinical periodontal disease measures. At one-month and three-month follow-up, clinical measures of periodontitis were decreased in all groups (data to be presented elsewhere). At three months, mean HbA1c levels in the SDD group were reduced 0.9% units from 7.2% units±2.2 (±SD), to 6.3% units±1.1, which represents a 12.5% improvement. In contrast, there was no significant change in HbA1c in the ADD (7.5%±2.0 to 7.8%±2.1) or placebo (8.5%±2.0 to 8.5%±2.6) groups. Mean HbA1c change from baseline was significantly greater in the SDD group compared with the ADD group (p=0.04) but not placebo (p=0.22). Moreover, a larger proportion of subjects in the SDD group experienced improvement (p<0.05) compared to the ADD or placebo groups. Mean plasma glucose levels were not significantly different between or within the groups. The results of this pilot study suggest that the treatment of periodontitis with sub-gingival debridement and 3-months of daily sub-antimicrobial-dose doxycycline may decrease HbA1c in patients with type 2 diabetes taking normally prescribed hypoglycemic agents.
The effect of nonsurgical periodontal therapy on hemoglobin A1c levels in persons with type 2 diabetes and chronic periodontitis: a randomized clinical trial. 

Engebretson SE1, Hyman LG2, Michalowicz BS1, Schenkein DE2, Gallo MC2, Hou W, Seagust ER4, Reddy MS5, Lewis CE6, Gates TW, Tripathy D, Katnir A, Orlandi PR10, Paguette DW11, Hanson NO12, Tsai MY12.  

Author information

Abstract

IMPORTANCE: Chronic periodontitis, a destructive inflammatory disorder of the supporting structures of the teeth, is prevalent in patients with diabetes. Limited evidence suggests that periodontal therapy may improve glycemic control.

OBJECTIVE: To determine if nonsurgical periodontal treatment reduces levels of glycated hemoglobin (HbA1c) in persons with type 2 diabetes and moderate to advanced chronic periodontitis.

DESIGN, SETTING, AND PARTICIPANTS: The Diabetes and Periodontal Therapy Trial (DPPTT), a 6-month, single-masked, multicenter, randomized clinical trial. Participants had type 2 diabetes, were taking stable doses of medications, had HbA1c levels between 7% and less than 9%, and untreated chronic periodontitis. Five hundred fourteen participants were enrolled between November 2009 and March 2012 from diabetes and dental clinics and communities affiliated with 5 academic medical centers.

INTERVENTIONS: The treatment group (n = 257) received scaling and root planing plus chlorhexidine oral rinse at baseline and supportive periodontal therapy at 3 and 6 months. The control group (n = 257) received no treatment for 6 months.

MAIN OUTCOMES AND MEASURES: Difference in change in HbA1c level from baseline to each 6-month visit for groups with and without intervention. Secondary outcomes included changes in probing pocket depths, clinical attachment loss, bleeding on probing, gingival index, fasting glucose level, and Homeostasis Model Assessment (HOMA2) score.

RESULTS: Enrollment was stopped early because of futility. At 6 months, mean HbA1c levels in the periodontal therapy group increased 0.17% (SD, 1.0), compared with 0.11% (SD, 1.0) in the control group, with no significant difference between groups based on a linear regression model adjusting for clinical site (mean difference, -0.05% [95% CI, -0.23% to 0.12%]; P = 0.56). Periodontal measures improved in the treatment group compared with the control group at 6 months, with adjusted between-group differences of 0.28 mm (95% CI, 0.18 to 0.37) for probing depth, 0.25 mm (95% CI, 0.14 to 0.36) for clinical attachment loss, 13.1% (95% CI, 8.1% to 18.1%) for bleeding on probing, and 0.27 (95% CI, 0.17 to 0.37) for gingival index (P < 0.01 for all).

CONCLUSIONS AND RELEVANCE: Nonsurgical periodontal therapy did not improve glycemic control in patients with type 2 diabetes and moderate to advanced chronic periodontitis. These findings do not support the use of nonsurgical periodontal treatment in patients with diabetes for the purpose of lowering levels of HbA1c.


Clinical and biochemical results of the metalloproteinase inhibition with subantimicrobial doses of doxycycline to prevent acute coronary syndromes (MIDAS) pilot trial.

Brown DL1, Detzi K, Yavah BA, Nounah C, Lee HM, Golub LM.  

Author information

Abstract

BACKGROUND: Vulnerable plaque demonstrates intense inflammation in which macrophages secrete matrix metalloproteinases (MMPs) that degrade the fibrous cap, ultimately leading to rupture, in situ thrombosis, and an associated clinical event. Thus, inhibition of MMP activity or more general suppression of vascular inflammation are attractive targets for interventions intended to reduce plaque rupture. We hypothesized that subantimicrobial doses of doxycycline (SDD) (20 mg twice daily) would benefit patients with coronary artery disease by reducing inflammation and MMP activity and thus possibly prevent coronary plaque rupture events.

METHODS AND RESULTS: We conducted a prospective, randomized, double-blind, placebo-controlled pilot study of 6 months of SDD or placebo treatment to reduce inflammation and prevent plaque rupture events. A total of 50 patients were enrolled, of whom 24 were randomized to placebo and 26 to SDD. At 6 months, there was no difference in the composite endpoint of sudden death, fatal myocardial infarction (MI), non-fatal MI, or troponin-positive unstable angina in SDD compared with placebo-treated patients (8.4% versus 0%, P = 0.491). Biochemical markers of inflammation were assessed in plasma at study entry and after 6 months of therapy in 30 patients. In SDD-treated patients, high-sensitivity C-reactive protein (CRP) was reduced by 46% from 4.8±0.6 microg/mL to 2.6±0.4 microg/mL (P = 0.007), whereas CRP was not significantly reduced in placebo patients. Interleukin (IL)-6 decreased from 22.1±13.7 pg/mL at baseline to 14.7±18.8 pg/mL at 6 months in SDD-treated patients (P = 0.025) but did not decrease significantly in placebo-treated patients. On zymography, pro-MMP-9 activity was reduced 50% by SDD therapy (P = 0.011), whereas it was unchanged by placebo treatment.

CONCLUSIONS: SDD appears to exert potentially beneficial effects on inflammation that could promote plaque stability. These findings should be investigated in a larger study.
Empower the Patient through the Provision of Resources to Understand the Oral Systemic Connection

Colgate Professional;  
www.colgateprofessional.com/professional-education/oral-systemic-health  

CDHO Knowledge Network;  
http://www.cdho.org/knowledge-network.asp  
American Academy of Periodontology Consumer Site;  
http://www.perio.org/consumer/other-diseases  
What’s Your Real Age?  
www.realage.com

Oral Systemic Link Professional and Public Information;  
www.oralsystemiclink.pro  
www.oralsystemiclink.net

Product References:  
Curaprox brushes, interdental brushes www.curaprox.com  

Thank you for your time and participation. If there is anything further that I may assist you with in regards to today’s presentation please do not hesitate to contact me.

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Medical History Update

Patient Name: __________________________________________ Date: __________________

Recent research indicates a strong relationship between the mouth and the body. Since we now know how closely they are related, we are going to be asking you some questions about your family history and your overall health that we may not have asked you about before. This additional information will assist us in providing the best possible care to maintain your oral health and overall wellness.

Any changes in your health since your last dental visit? □ Yes □ No  If yes, please list:
__________________________________________________________________________________________________

What medications are you taking? _____________________________________________________________________
______________________________________________
____________________________________________________

Any changes in medication dosage or medications? □ Yes □ No  If yes, please list:
________________________________________________________________________________________
__________________________________________________________________________________________________

What over the counter or ‘herbal/natural’ supplements are you taking on a regular basis? Please list:
________________________________________________________________________________________

Are you taking any bisphosphonates in the past or presently? □ Yes □ No  If yes, please provide details:
________________________________________________________________________________________

Do you have a persistent sore throat, hoarseness, ear ache or feeling of something being caught in your throat? □ Yes □ No  If yes, please provide details:

Have you had any surgery or been hospitalized since your last visit? □ Yes □ No
If yes, please explain: __________________________________________

Are you being treated for any medical problem presently? □ Yes □ No
If yes, please explain: __________________________________________

Have you ever taken antibiotics prior to having your teeth cleaned or before dental work? □ Yes □ No
If yes, please explain: __________________________________________

Any allergies to drugs, food, metal or latex? □ Yes □ No
If yes, please list: __________________________________________

History of illness or disease in family?
If yes, please explain: __________________________________________

Have you been diagnosed with diabetes? □ Type I □ Type II □ Pre-diabetes
□ Diet-controlled □ Medication controlled Under control: □ Yes □ No

Have you had any heart problems or a knee, hip or prosthetic joint replacement? □ Yes □ No
If yes, provide details: __________________________________________

Have you had a bone mineral density test? □ Yes □ No Results: __________________________________________

Female patients; Are you pregnant? □ Yes □ No

On a scale of 1 to 10 (10 being highest), how would you rate your general health at this time? __________________

How would you rate your level of stress presently? □ Low □ Moderate □ High

On a scale of 1 to 10 (10 being highest), how closely related is the health of your mouth to your overall health in your opinion? __________________
References: Subantimicrobial Dose Doxycycline (SDD) *


*Note: This does not comprise a complete listing of studies related to SDD.