Interrelation of Cardiovascular Diseases and Periodontal Pathologies - A Literature Review

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Abstract:
Oral infections are a source of bacteremic organisms that can infect damaged heart valves causing bacterial endocarditis and other forms of cardiovascular disease including stroke. This review discusses the relationship of oral infections, bacterial endocarditis and coronary artery disease, especially periodontal disease, while suggesting guidelines for best practice clinical management.

Key words: Periodontal disease, coronary heart disease, endocarditis prophylaxis.

Introduction:
Cardiovascular diseases are a leading cause of death, with a marked increase among the geriatric population. Coupled with recent evidence linking severe periodontitis with coronary artery disease this suggests that the role of periodontists extends beyond treating local pathology and they must be prepared to provide safe and effective therapy to patients with various types of heart conditions.

Patient at risk of infective endocarditis:
Patient management requires the taking of a thorough medical history and physical examination with evaluation of vital signs and medical management when indicated. The two most common microorganisms associated with infective endocarditis are Streptococcus viridans and Staphylococcus aureus that may be present as commensals in the oral cavity and the biological load of these organisms increases in the presence of chronic periodontitis. Individuals with a cardiac valve prosthesis are especially susceptible to infective endocarditis although those with native valvular damage and even those with undamaged heart valves may also develop endocarditis. Preventive measures include methods to reduce the potential for significant bacteremia from the oral cavity, the skin, the upper respiratory tract and gastrointestinal and urinary tracts.

Valvular heart disease:
Rheumatic fever, aortic stenosis, senile valvular calcification, congenital anomalies, ischemic heart disease, Kawasaki disease, systemic lupus erythematosus are all associated with valvular heart disease. Congenital heart anomalies may induce cardiac blood turbulence and permanent valve damage therefore should be considered as a lifetime risk for infective endocarditis although the risk may be low.

Dental Consideration:
The patient with valvular heart disease faces the risk of congestive heart failure, hemodynamically significant arrhythmias and infective endocarditis. Blood borne bacteria may lodge on damaged and abnormal heart valves in the endocardium or in the endothelium near congenital anatomic defects resulting in infective endocarditis or endarteritis.

Dental procedures that involve manipulation of soft tissue and result in bleeding can produce a transient bacteremia; 43% of patients with periodontitis experienced transient bacteremia following routine periodontal probing. The incidence and severity of odontogenic bacteremias increase markedly in the presence of periodontitis or focal oral infections with or without manipulation of oral tissues. The American Association of Periodontology recognized that one of every four cases of infective endocarditis was caused by periodontal organisms such as Actinobacillus spp, Eikenella corrodens, etc.

Transient bacteremias may be induced by some surgical or non-surgical periodontal treatments such as oral subgingival irrigators and air polishing devices although these bacteremias rarely persist longer than 15 minutes and most dissipate within 3-5 minutes.

The American Heart Association (AHA) recommends the following specific prophylactic antibiotic regimens for dental procedures: for adults, oral administration of 2 gm of amoxicillin one hour before the dental procedure. In patients allergic to the penicillins the American Heart Association recommends clindamycin 600mg or cephalaxin/cefadroxil or azithromycin/clarithromycin one hour before the dental procedure.

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losporins should not be used due to the potential for microbi
cross-resistance between cephalosporins and penicillin derivatives (9) nor are tetracyclines recommended for prophylactic cardiovascular antibiotic coverage (7, 8, 9, 13) although patients with periodontal disease associated with tetracycline-sensitive organisms may be best treated by administering tetracycline two to three weeks prior to the periodontal treatment followed by a one week delay and then performance of periodontal therapy using AHA recommended prophylactic regimens (15).

The relationship between infective endocarditis and periodontal treatment incorporating local delivery of antibiotics or antimicrobials into gingival pocket is not known although the AHA recommends systemic prophylaxis anticipating potential traumatic injury and bleeding during these procedures (8).

The patient on anticoagulants:

The INR for patients with normal prothrombin time (PT) is approximately 1.0 and patients on anticoagulant therapy are maintained at an INR ranging between 2 and 3.5 (16). Infiltration anesthesia, scaling and root planing can be done safely with an INR <3.0; block anesthesia, minor periodontal surgery, simple extraction can be done with an INR <2.0; and complex surgery and multiple extractions need an INR <1.5.

Local hemostatic measures to be taken include atraumatic surgery, adequate wound closure using sutures, application of post-surgical pressure, use of topical clotting agents like foamed gelatin, thrombin, oxidized cellulose or synthetic collagen. Oral rinses with tranexamic acid reduce the risk of hemorrhage markedly without altering the INR level (14, 17). On occasion pharmacologic manipulation becomes necessary for the anticoagulated patient and vitamin K administration reverses the effect of coumarin. More urgent situations may require blood transfusion or infusion of fresh frozen plasma or packed platelets (14). Aspirin is often used as an antithrombotic agent in small dosages and at those levels the medication will not significantly alter bleeding time (13, 18). Patients on higher aspirin levels are at a slight risk of prolonged post-operative hemorrhage following periodontal therapy and so for such individuals the medication should be discontinued four to seven days prior to the procedure, with the approval of the cardiologist (2, 13).

Association between periodontal disease, atherosclerosis and coronary heart disease:

Both periodontal disease and arteriosclerosis increase with age and it has been hypothesized that the circulatory impairment induced by vascular changes may increase the susceptibility to periodontal disease (17). Conversely there is recent evidence to suggest that individuals with periodontal disease may be at greater risk for heart disease as a result of chronic periodon-
tal infections and inflammation. The immuno-inflammatory response to bacteria and their products, systemic challenge with their agents also induce a major vascular response which may offer explanatory mechanisms for interactions between periodontal infection and many systemic diseases including coronary heart disease /atherosclerosis.

Various studies have been published on the interrelation of dental health and ischemic heart disease. In a cross-sectional study (19, 20) on patients with acute myocardial infarction (MI) or confirmed coronary heart disease compared with age and gender matched control patients. Myocardial infarction patients had significantly worse dental health than did controls. This association between poor dental health and myocardial infarction was independent of known risk factors for heart disease such as age, serum cholesterol level, hypertension, diabetes and smoking.

As atherosclerosis is a major determinant of coronary heart disease related events, coronary disease has also been related to coronary atheromatosis. Mattila and colleagues correlated the oral radiographic examination and coronary angiographic examination in patients with ischemic heart disease (21) and found a significant correlation between severity of dental disease and the degree of coronary atheromatosis. Cross sectional studies suggest a link between the two diseases although unable to determine the causality in this relationship as dental disease may be an indication of general health status. A seven year follow up study of 182 males and 32 females with fatal and non-fatal coronary events found that dental disease was significantly related to the incidence of new fatal and nonfatal coronary events as well as the overall mortality (22).

In a prospective study a natural sample of 9,760 adults was followed for 14 years after dental and medical evaluation (23). Subjects with periodontal disease had a 25% increase in the risk of coronary heart disease compared with the minimum or no periodontal condition after adjusting for the known risk factors. Among males aged 25-49 years periodontal disease increased the risk of coronary events by 70%. Patients with poor oral hygiene had a two-fold increased risk for coronary heart disease.

In 1996 Beck and colleagues (24) published a study of 1147 males with baseline radiographic measurements of alveolar crest height and mean alveolar bone loss divided into high and low categories and found that the cumulative incidence of coronary heart disease increased with greater levels of age-adjusted alveolar bone loss at baseline suggesting the more periodontal disease at baseline the greater was the cumulative incidence of coronary heart disease over time. They also identified a common genetically determined hyperinflammatory macrophage phenotype in periodontal disease which they believed contri-
buted to atherosclerosis although Destefano and colleagues (23) considered that periodontal disease and poor oral hygiene were stronger indicators for an association between the two diseases.

These studies and others (21, 22, 23, 24) in which the periodontal conditions is known to have preceded the coronary event support the concept that periodontal disease is a risk factor for coronary heart disease, independent of other classic risk factors. Although the underlying mechanisms linking oral or periodontal disease and cardiovascular diseases remain largely unknown, new data have indicated that periodontitis can elicit a systemic inflammatory response by activating the hepatic acute phase response (25).

Both diseases have complex causation, genetic and gender predisposition and potentially share many risk factors, age, race, male gender and stress, the most significant which may be smoking, and so when considering oral health status as a possible factor for systemic conditions, it is important to recognize that other known risk factors such as host susceptibility factors may also play a part in the risk of cardiovascular disease; the true association may actually be between risk factors rather

Table 1 Cardiac conditions requiring prophylaxis for dental treatment.

<table>
<thead>
<tr>
<th>High Risk</th>
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<td>Prosthetic cardiac valves.</td>
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<td>Previous infective endocarditis.</td>
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<td>Complex congenital cardiac malformations.</td>
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<td>Systemic pulmonary shunts (surgically constructed)</td>
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<th>Moderate Risk</th>
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<tr>
<td>Rheumatic heart disease, Kawasaki disease, connective tissue disorders and conditions with valvular dysfunctions, Hypertrophic cardiomyopathy, Mitral valve prolapse with regurgitation, Most other congenital cardiac malformation.</td>
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Table 2 Cardiac conditions not requiring endocarditis prophylaxis

<table>
<thead>
<tr>
<th>Isolated secundum atrial septal defect</th>
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<tr>
<td>Surgical repair of secundum atrial septal defects, ventricular septal defects, or patent ductus arteriosus after 6 months and without residua.</td>
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<tr>
<td>Previous coronary artery bypass graft.</td>
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<td>Mitral valve prolapse without regurgitation.</td>
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<tr>
<td>Physiologic, functional, or innocent heart murmurs.</td>
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<tr>
<td>Previous rheumatic fever, Kawasaki disease or connective tissue disorders without valvular dysfunction.</td>
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<td>Cardiac pacemakers and implanted defibrillators.</td>
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Table 3. Dental procedures likely to induce significant bacteremia

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<th>Dental extraction</th>
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<tr>
<td>Implant placement and tooth reimplantation.</td>
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<td>Surgical and non surgical periodontal procedures.</td>
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<td>Endodontic instrumentation beyond root apex or endodontic surgery.</td>
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<td>Initial placement of orthodontic bands.</td>
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<td>Intraligamentary injection.</td>
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<td>Prophylaxis when bleeding is expected.</td>
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<td>Subgingival placement of antibiotic fibres or strips.</td>
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From Dajani AS, Taubert KA, Wilson W. Prevention of bacterial endocarditis - Recommendations by the American Heart Association JAMA 1997; 277:1794 – 1801.
References:


