The Relationship Between Periodontics and Cardiovascular Disease

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The Relationship Between Periodontics and Cardiovascular Disease

Effective Date: 01/01/2016 Expiration Date: 01/01/2019

Learning Objectives: After reading this article, the individual will learn: (1) what is periodontal medicine, and (2) the current state of the medical and dental literature regarding the association between periodontal disease and hypertension, atherosclerotic diseases, metabolic syndrome, and obesity.

About the Authors

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Disclosure: Dr. Brown reports no disclosures.

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Disclosure: Dr. Makhene reports no disclosures.

The mouth has been called “the window to your body’s health.” The body undergoes many biological processes that interact with one another, and as such, oral health is inextricably linked to general systemic health. Health is defined not only as the absence of disease or infirmity but a state of complete physical, mental, and social well-being. An unhealthy mouth can result in an unhealthy body. In 1900, William Hunter, MD, introduced the concept of oral sepsis in a paper entitled, “Oral Sepsis as a Cause of Disease,” in which he discussed the association between oral infection and systemic disease.

Today, it is known that maintaining optimal oral health is a crucial part of achieving and maintaining optimal general health. Tissues of the oral cavity receive their blood supply, innervation, and lymphatics from branches of blood vessels, nerves, and components of the immune system that serve other parts of the body. The mouth is the first portion of the digestive system of the human body; it receives food and nourishment. Other functions of the mouth include mastication, drinking, breathing, swallowing, speaking, tasting, conveying expressions and emotions through our facial expressions, and nursing/sucking for infants. In addition to these basic functions, the mouth can also be used for erotic purposes, as in kissing.

Not only can a complete oral examination detect diseases, lesions, and abnormalities of the oral cavity, but it can detect certain immune system disorders, systemic diseases and disorders, cancers, and nutritional deficiencies. Williams and Offenbacher coined the term “Periodontal Medicine” in 1996, which focuses on periodontal health or disease and their relationships to systemic health or disease. In 1945 Miller, a periodontist, initiated the American Academy of Oral Medicine, and in 1956 he initiated the American Board of Oral Medicine.

**PATHOGENS ASSOCIATED WITH PERIODONTAL DISEASE**

It is believed that there are more than 500 species of microbial species that are the primary etiologic agents for periodontal disease. In addition to microbial pathogens, viruses and fungi are also associated as putative pathogens for periodontal disease. In disease, the putative periodontal pathogens reside in and colonize the gum tissue collar that surrounds the tooth, forming a niche called the periodontal pocket. Up to 109 bacteria may be harbored in the deeper periodontal pockets. Despite the potential for many pathogens to be associated with periodontal disease, there are a small number most frequently associated with active periodontal disease.

Socransky et al divided the pathogens into 2 main clusters of microorganisms and deemed them the “red” and “orange” complexes. The red complex includes the following Gram-negative, anaerobic pathogens: *Porphyromonas gingivalis, Treponema denticola, Tannerella forsythia*. The orange complex...
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Pathogens include *Fusobacterium nucleatum*, *Prevotella intermedia*, *Prevotella nigrescens*, *Peptostreptococcus micros*, *Campylobacter rectus*, *Centroburoides gracilis*, *Campylobacter showae*, *Eubacterium nodatum*, and *Streptococcus constellatus*. Other microbial pathogens highly associated with periodontal disease are *Aggregatibacter actinomycetemcomitans* and *Eikenella corrodens*.

There are 3 pathways by which oral/periodontal microbial pathogens may contribute to nonoral diseases. The inflamed, ulcerated tissues of the periodontal pocket serve as a point of entry for periodontal microbial pathogens into the bloodstream. Once in the bloodstream, the pathogens produce a bacteremia, which may then be disseminated throughout the body. This pathway of infection is called metastatic transient bacteremia. The periodontal pathogens may have unique structural components such as capsules, lipopolysaccharide (LPS), or active secretion of substances such as toxins that damage the host tissues directly or allow the microbial pathogens to evade the host’s defensive mechanisms. This pathway is called metastatic toxic injury. Finally, the bacteremia that is caused by the periodontal pathogens may react with antibodies to form complexes that cause inflammatory reactions in the host, resulting in host injury. This pathway is called metastatic immunological injury.

In inflamed tissues, as noted with periodontal disease, there is an increase in the presence of periodontal microbial pathogens. This can result in a pathological and destructive inflammatory process or cycle, through the activation of the body’s host defense cells. Examples of the body’s host defense cells include macrophages and their precursors, monocytes, lymphocytes, and polymorphonuclear leukocytes or PMNs (such as neutrophils). Microbial components, such as LPS, found in the cell wall of Gram-negative anaerobic microbes, activate macrophages to produce and secrete proinflammatory cytokines such as interleukin-1 beta (IL-1β) and tumor necrosis factor-alpha (TNF-α). IL-1β promotes bone resorption. It also stimulates the release of prostaglandin E2 by fibroblasts. Fibroblasts are the primary and most abundant cell type found in connective tissue in humans. Fibroblasts synthesize collagen, which is the primary structural protein of connective tissue and the main fiber component of gingival tissue and bone.

Fibroblasts and inflammatory cells (neutrophils and macrophages) synthesize matrix metalloproteinases (MMPs), which are enzymes or proteins that facilitate biologic reactions. For example, MMP1 and MMP8 are enzymes that degrade collagen, which weakens periodontal support. Activation of the body’s host defense or immune system is the body’s way to protect itself against a pathogenic challenge; however, the result is often damaging to the host through destruction of tissue. This is due to the release of cytokines, proinflammatory mediators, and MMPs. These agents not only act locally, affecting the periodontal tissues, but once in the systemic circulation, they induce and perpetuate systemic effects.

The primary etiological factor for developing periodontal disease is dental plaque biofilm; however, a number of risk factors may also play a role. These include age, certain medications, genetics, poor nutrition, obesity, certain systemic diseases, clenching or grinding teeth, smoking, and stress. Smoking may be one of the most significant risk factors because of its negative impact on periodontal health, which can lead to the development as well as progression of periodontal disease.

Psychological stress may also increase the risk of developing periodontal disease, perhaps because in this population optimal oral hygiene is less likely to be performed.

Periodontal disease is the result of a variety of complex interactions between pathogenic microbes, a susceptible host, and risk factors, which work together to elicit a host immune-inflammatory response. This response has an impact on connective tissue and bone metabolism, ultimately resulting in the clinical expression and progression of periodontal disease. Periodontal plaque biofilm has access to the gingival circulation through ulceration of the periodontal pocket, thus providing a pathway for spreading systemically. The spread of plaque biofilm systemically can activate an inflammatory response, leading to the production of mediators with systemic effects. Due to these various interactions, periodontal disease may affect a person’s susceptibility to systemic diseases and conditions.

**PERIODONTAL DISEASE AS A RISK FACTOR**

Periodontal disease is thought to be a risk factor associated with several systemic diseases and conditions. They include cardiovascular diseases (CVDs) such as hypertension, atherosclerotic disease, coronary heart disease (CHD) and cerebrovascular disease (ie, stroke); diabetes; female reproductive issues such as pregnancy complications, fetal mortality, preterm and low-birthweight deliveries, and preeclampsia; male reproductive issues such as prostatitis and erectile dysfunction; autoimmune diseases such as rheumatoid arthritis, Sjögren’s syndrome, ankylosing spondylitis, and inflammatory bowel disease.
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disease; osteoporosis; cognitive impairment such as Alzheimer’s disease; respiratory disorders such as pneumonia and chronic obstructive pulmonary disease; chronic kidney disease; and cancer.16-35 Periodontal disease and several of the previously mentioned systemic diseases share common risk factors such as aging, gender, ethnicity, race, genetic factors, smoking, alcohol abuse, and low socio-economic status.

PERIODONTAL DISEASE AND CARDIOVASCULAR DISEASE
CVD refers to a group of disorders associated with the heart and vasculature (ie, blood vessels) and includes high blood pressure, CHD, congestive heart failure, heart valve diseases, stroke, and myocardial infarction (ie, heart attack).34 CVD is the number one cause of death around the world. It accounts for approximately 17.5 million deaths per year and approximately 3 in every 10 of all deaths worldwide.34-36 Atherosclerotic disease is usually the underlying etiology in the majority of these deaths.34

Following is a discussion of certain forms of CVD and their relationship with periodontal disease.

Perhaps then, periodontal disease is contributing to systemic inflammation.

Hypertension
Hypertension is a medical condition in which there is an elevation in the force of the blood against the arterial walls as it courses through the vessels.37 In most people, there is no known cause for hypertension. Hypertension tends to develop gradually over time, which is known as essential or primary hypertension.37 Secondary hypertension is the other form. It is a result of a known cause, usually associated with kidney disease, thyroid disorders, and tumors of the adrenal gland, alcohol abuse, and illicit drug usage.37 Hypertension afflicts 67 million, or one in 3 American adults.38 It is estimated that hypertension costs the United States approximately $47.5 billion each year in healthcare services, medications, and days missed from work.38

In general, inflammation is not usually associated with hypertension; however, research is being done to assess the role that vascular inflammation may play in the progression of hypertension.39,41 Periodontal disease is a local chronic inflammatory disease with systemic effects. It has been proposed that the elevation in C-reactive protein levels associated with periodontal disease leads to endothelial dysfunction.41 Throughout time, endothelial dysfunction increases the risk for hypertension. Researchers have found an association between periodontitis and endothelial dysfunction.43,44 Likewise, they have found that periodontal therapy may result in a reduction not only in systemic inflammation but also in endothelial dysfunction, which may be useful in the treatment of hypertension.45

Atherosclerotic Disease
Atherosclerotic disease, or hardening of the arteries, can affect different vessels in the body. When it affects the arteries of the heart, it is called CHD and is manifested by heart attack and angina pectoris. When it affects the vessels of the brain, it is called cerebrovascular disease and is manifested by stroke. When atherosclerosis affects the arteries of the legs, arms, kidneys, and stomach, it is called peripheral arterial disease. CHD is the leading cause of death in the United States; most are due to heart attack.46 More than 81 million Americans have some form of CVD, and the numbers are increasing annually.47 Cerebrovascular disease is the fourth leading cause of death in the United States.48 There is a tremendous economic burden on individuals, families, and the country as a result of atherosclerotic disease due to illness, injury, and death.

Atherosclerotic disease is a specific type of atherosclerosis, where the walls of a vessel thicken as a result of invasion and accumulation of white blood cells, cellular waste products, cholesterol, and triglycerides to form plaques or atheromas.49 Additional contributing factors to vessel thickening and vascular injury include mechanical stress, smoke exposure, hyperhomocysteinemia, and chronic infection.50 Eventually over time, advanced lesions are encased in calcium and other crystalized materials, as well as fibrin (a clotting material in the blood). This is a chronic disease that may begin in the childhood years but usually in the 20s and 30s, and may lay dormant and asymptomatic for years.51 Atherosclerosis typically becomes dangerous when a person reaches their 50s to 60s.51

The body identifies these plaques as foreign entities and mounts an inflammatory immune response in an effort to wall off and rid the body of the intruder. This response is injurious to the vessel wall. As atherosclerosis advances, 2 things may happen: a piece of the plaque or lesion may break off and rupture, causing injury to the wall of the artery, or a blood clot may form on the surface of the plaque.49 Either situation results in a complete or partial blockage of the vessel lumen. When the blockage occurs in a blood vessel as a result of a fibrin clot and stays lodged in the vessel, the blockage is called a thrombus. A small piece of a blood clot that breaks off, travels through the bloodstream, and lodges in a vessel and blocks it is called an embolus. If the thrombus or embolus completely occludes or blocks the lumen of the vessel, then blood flow to the organ that the artery supplies is blocked. As a result of the blockage, tissue death occurs.49 This event is
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called an infarction. An infarction that occurs in the coronary artery is called a myocardial infarction.\textsuperscript{49} If there is an infarction in an artery in the brain it is called a cerebrovascular event.\textsuperscript{49} If the thrombus is in an artery and restricts blood flow (meaning some blood is able to get downstream to supply and nourish the tissue) but the flow is diminished, the result is ischemia.\textsuperscript{49} If an ischemic event occurs in a coronary artery, one may experience angina pectoris. If an ischemic event occurs in an artery of the brain, one may experience short periods of numbness or tingling, also known as a transient ischemic attack.

Triggers of inflammation associated with atherosclerosis include smoking, diabetes mellitus, and obesity.\textsuperscript{52} Several studies have shown an association between periodontal disease and atherosclerotic CVD.\textsuperscript{53} Poor periodontal health has been noted as a risk factor for the development of CHD along with smoking, age, hypertension, and race, among others.

Periodontal microbial pathogens such as \textit{P. gingivalis}, \textit{T. denticola}, \textit{C. rectus}, and \textit{A. actinomycetemcomitans} have been isolated from atherosclerotic plaques and in arterial walls of coronary arteries.\textsuperscript{34} \textit{P. gingivalis} has also been isolated in higher concentrations in patients with more severe periodontitis compared to those with milder forms of periodontitis.\textsuperscript{54} It has been suggested that the LPS found on the cell wall of Gram-negative periodontal pathogens such as \textit{P. gingivalis} stimulates an inflammatory response through the release of inflammatory cytokines such as interleukin and TNF-\textalpha in the walls of coronary blood vessels.\textsuperscript{55} IL-6 has been found to be an important prognostic factor for a future occurrence of a major cardiovascular event.\textsuperscript{55} IL-6 induces expression of C-reactive protein, among other hepatic acute phase proteins.\textsuperscript{57} C-reactive protein levels are associated with an increased atherosclerotic risk and endothelial dysfunction.\textsuperscript{58} Researchers have suggested that the microbial burden of a patient from several chronic sources such as \textit{Helicobacter pylori}-associated gastric ulcers, \textit{Chlamydia pneumoniae}-associated bronchitis, and periodontitis are associated with an increased risk for CHD.\textsuperscript{59}

This inflammatory response contributes to plaque instability, resulting in a cascade of atherosclerotic events.\textsuperscript{60} Others have suggested the possibility that \textit{P. gingivalis} may induce platelet aggregation, thus increasing the likelihood of the development of blood clots.\textsuperscript{61} Periodontal microbial pathogens, such as \textit{P. gingivalis}, have also been associated with endothelial dysfunction and in the development of atherosomas.\textsuperscript{65} All of these occurrences may be associated with an increase in a cascade of atherosclerotic events.

Just as studies have suggested that periodontal disease may be a risk factor for atherosclerotic disease, there are those who have reported a potential bidirectional influence of systemic inflammation, especially as it relates to cardiac health, on periodontal health. C-reactive protein is considered a golden marker for systemic inflammation. Its concentration increases in the presence of infection and inflammation.\textsuperscript{63} Periodontal disease is associated with an increase in the level of C-reactive protein.\textsuperscript{64} Increases in C-reactive protein are also associated with an increased risk of atherosclerosis.\textsuperscript{64} Researches have found elevated levels of C-reactive protein in patients with both periodontal disease and atherosclerosis.\textsuperscript{64,65} These levels were elevated above what is seen with only one disease or the other.\textsuperscript{64,65} Perhaps then, periodontal disease is contributing to systemic inflammation.

When atherosclerotic disease results in an infarction in an artery in the brain, similar to an infarction in an artery of the heart, it is called a cerebrovascular event or an ischemic stroke. There are 2 types of stroke: ischemic and hemorrhagic. An ischemic stroke is the most common type, accounting for approximately 80\% of all strokes.\textsuperscript{66} An ischemic stroke occurs due to a blood clot that blocks a blood vessel in the brain. There are 2 types of ischemic stroke: thrombotic and embolic. A thrombotic stroke is as a result of a blood clot or thrombus that forms inside an artery that supplies the brain with blood. An embolic stroke is as a result of a piece of a blood clot or a piece of plaque that breaks off and travels through the bloodstream (an embolus). When an embolus lodges in an artery that supplies blood to the brain, a stroke occurs.\textsuperscript{67} A hemorrhagic stroke happens if an artery in the brain leaks or ruptures. The increased blood volume due to the leakage or rupture causes damage to brain cells.\textsuperscript{57}

Poor oral health, and periodontal disease in particular, has been found to be a risk factor for cerebrovascular disease, especially in ischemic stroke.\textsuperscript{35,68,69} Poor oral health, especially periodontal diseases that increase the level of C-reactive protein, a marker for systemic inflammation, has been found to be a risk factor for stroke.\textsuperscript{70} \textit{P. gingivalis} has been found in atherosclerotic plaques from ischemic stroke.\textsuperscript{71} Studies have suggested that periodontal pathogens such as \textit{P. gingivalis} induce an inflammatory response by eliciting macrophages to produce and secrete proinflammatory cytokines, such as IL-1\beta, IL-6, and TNF-\textalpha. These cytokines stimulate the production of acute phase proteins, like fibrinogen, which plays a role in clot formation in arteries, contributing to atherosclerotic events.\textsuperscript{72} Such events may result in ischemic stroke or cerebral vascular accident (CVA).

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\textbf{Dentists and physicians alike should educate patients about the importance of both oral and systemic health.}
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Metabolic Syndrome

Metabolic syndrome is a term used for a group of risk factors that raise one's risk for developing CHD, stroke, and diabetes. It is defined by the presence of obesity plus any 2 of the following risk factors: hypertension, high atherogenic lipid profile, insulin resistance, and elevated C-reactive protein and fibrinogen levels. Additional risk factors include physical inactivity, hormonal imbalance, smoking, and aging. When an individual is diagnosed with having at least 3 of these metabolic risk factors, then the diagnosis of metabolic syndrome can be made. Approximately 34% of adults age 20 years or older meet the criteria for metabolic syndrome in the United States. Metabolic syndrome contributes to the development of atherosclerotic disease. There are studies suggesting that perhaps periodontal disease should be considered a risk factor for metabolic syndrome and that both periodontitis and metabolic syndrome are associated with systemic inflammation, insulin resistance, and endothelial dysfunction. Severe periodontitis has been associated with metabolic syndrome. Moreover, there are common pathophysiologic pathways between periodontal disease and metabolic syndrome that increase the risk for CVD in patients with periodontitis. Others have found that a reduction in periodontal inflammation has occurred in those treated for periodontitis who have metabolic syndrome.

Obesity

Obesity, one of the risk factors associated with metabolic syndrome, is defined as an excessive accumulation and storage of fat in the body. One of the causes of obesity is taking in more calories than what are being used through physical activity. The World Health Organization has classified obesity as a calculation of the ratio of body weight (kg) by the square of height (m). This calculation is also known as the Body Mass Index (BMI). A person is considered obese when having a BMI ≥ 30 kg/m2. According to the National Health and Nutrition Examination 2007 to 2008, it is estimated that 154.7 million Americans (approximately 32.2% of the adult male and approximately 35.5% of the adult female population) are obese.

Obesity has been associated not only with metabolic syndrome but with hypertension, diabetes, CVD, osteoarthritis, respiratory disease, and periodontal disease. There is a clear relationship between obesity and periodontitis. It has been suggested that LPS endotoxin found on the surface of Gram-negative periodontal microbial pathogens triggers adipose or fat tissue to produce cytokines, specifically proinflammatory cytokines such as TNF-α and IL-6. These proinflammatory cytokines induce the production of acute-phase liver proteins such as fibrinogen (a clotting factor) and C-reactive protein, which may increase one's risk for atherosclerotic disease. Studies have also shown that normal-weight people who are physically active show a decreased incidence in periodontal disease and proinflammatory markers.

Discussion

Many studies and researchers have established an association between periodontal disease and atherosclerosis, heart disease, C-reactive proteins, and CVAs; however, a cause and effect relationship has yet to be established. Kalburgi et al reported increased C-reactive proteins serum levels and neutrophils in chronic periodontitis, but noted that the specificity of these markers was insufficient to document a role in patients' systemic health. Furthermore, Holtfreter et al reported that further clarification was necessary regarding an analysis of CRP and endothelial function with regard to a periodontal-systemic health connection.

There is no current evidence that life expectancy can be increased by successfully treating periodontal disease and thus decreasing atherosclerosis, CVD, and CVAs. It is possible that there may be an increased genetic risk for atherosclerosis in individuals with genetic precursors for periodontal disease. If so, treating periodontal disease would not increase clinically successful outcomes for people who have atherosclerotic disease. However, the successful treatment of periodontal disease is important for achieving optimal oral health and a lower risk of systemic chronic inflammation and bacteremias. Therefore, it is possible that such treatment could reduce the risk for the development of atherosclerotic disease.

Many of the associations linking periodontal disease to systemic conditions and diseases are associated with age and biologically complex conditions such as atherosclerotic disease. These associations provide the opportunity for interprofessional communication among general dentists, periodontists and other dental specialists, hygienists, primary care physicians and specialists, nurses, therapists, and other healthcare providers. Many people may see their dentist more often then their primary care physician, or vice versa. Dentists and physicians alike should educate patients about the importance of both oral and systemic health.

Conclusion

The oral cavity is an integral part of the body, and as such it has important systemic interactions. It is important for dental and medical professionals to educate patients about their oral health as well as the potential increased risk for systemic diseases secondarily associated with poor oral health. In addition, interprofessional communication in the area of oral/systemic health is vital, including appropriate referrals among all involved disciplines.
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**POST EXAMINATION QUESTIONS**

1. Which of the following Gram-negative anaerobic pathogens is a member of the “red complex”?
   a. Streptococcus mutans.
   b. Actinomyces viscosus.
   c. Porphyromonas gingivalis.
   d. Candida Albicans.

2. What is the name of the pathway of periodontal infection that contributes to nonoral disease by producing an infection in the blood that becomes disseminated throughout the body?
   a. Metastatic transient bacteremia.
   b. Metastatic toxic injury.
   c. Metastatic immunological injury.
   d. None of the above.

3. What is the primary and most abundant cell type found in human connective tissue?
   a. Melanocyte.
   b. Fibroblast.
   c. Mast cell.
   d. Erythrocyte.

4. Which of the following is not a component of the attachment apparatus?
   a. Cementum.
   b. Periodontal ligament.
   c. Alveolar bone.
   d. Gingiva.

5. What is the leading cause of death in the United States?
   a. Cancer.
   b. Coronary heart disease.
   c. Diabetes.
   d. Suicide.

6. Thickening of arterial walls from the accumulation of white blood cells, cellular waste products, cholesterol and triglycerides produces a/an ___?
   a. Embolus.
   b. Stent.
   c. Arthroma.
   d. Cyst.
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7. Complete occlusion or blockage of a blood vessel resulting in tissue death is known as?
   a. Infarction.
   b. Syncope.
   c. Ischemia.
   d. Asphyxiation.

8. Which periodontal pathogen has been isolated from atherosclerotic plaques and in arterial walls of coronary arteries?
   a. Escherichia coli.
   b. Staphylococcus aureus.
   c. Capnocytophaga spp.
   d. Treponema denticola.

9. What is the most common chronic disease that affects children?
   a. Foot and mouth disease.
   b. Herpes simplex virus type 1.
   c. Dental caries.
   d. Herpangina.

10. The presence of what is considered the golden marker for systemic inflammation?
    a. Interleukin-25.
    b. C-reactive protein.
    c. Vitamin C.
    d. Cox-2.
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