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Undergraduate

Importance of Improving Communication Between Health Professionals:

Implications of Avoiding Precautionary Measures for Periodontal and Systemic Diseases

By Macayla Kinney

Abstract

Although most patients believe maintaining oral hygiene only affects the mouth, many risk factors from untreated gingivitis involve developing serious medical complications at distant sites within the body because the bacteria can spread into the bloodstream. As a result dentists need to better coordinate their treatment of periodontal disease with medical practitioners because oral disease may indicate both an undiagnosed medical problem and cause a medical problem. Dentists, doctors and patients need to work together to take precautionary measures to help reduce the risks of developing periodontal disease or systemic diseases. Therefore, more efficient collaboration between medical professionals treating dental health as a part of general health will aid in reducing the risks for certain medical complications such as diabetes mellitus and coronary heart disease.

Oral disease may indicate an undiagnosed medical problem. For example, periodontal disease increases the risk of developing cardiovascular diseases and diabetes mellitus [4]. The inflammation caused by disrupted bacteria within tissues and organs play a role in the development of specific diseases. Diabetes mellitus, a common endocrine disorder, causes the body to process glucose poorly [2]. This disorder decreases insulin production and elevates the patient's blood glucose level [2]. When glucose levels raise a patient's risk for developing gingivitis increases as a result. They provided an example of a patient's poorly maintained gums (Figure 1 and 2), which detected diabetes mellitus from excessive inflammation.



Fig 1. Unusually intense gingival response to dental plaque in a 38-year-old female. The gingivae are bright red, have a glossy surface and bleed profusely on probing. The inflammation extends throughout the attached gingiva of the affected areas. Diabetes mellitus was diagnosed on subsequent referral to the patient's general medical practitioner.



Fig 2. Localized gingival swelling and inflammation extending through the attached gingiva of teeth 23 and 24 in a 32-year-old uncontrolled diabetic. The bluish tinge of the gingival tissues is a common, although not an exclusive sign of diabetes-conditioned gingival inflammation. Another feature is that the severe gingival inflammation does not necessarily affect all gingival areas.

The results of several studies suggest oral inflammation causes elevated levels of antibodies and proteins [1,3,4,5]. Keiko Watanabe, suggested a bi-directional relationship between diabetes mellitus type 1 and 2 and periodontal disease [1]. Data shows an increase in gingival inflammation in children with T1DM by ten percent compared with adults and children without T1DM with less than two percent of inflammation [1]. Subjects with periodontitis show a greater amount of ketoacidosis, retinopathy, and neuropathy including micro vascular complications from severe inflammation [1]. Watanabe mentions, “the death rate from diabetic nephropathy in subjects with severe periodontal disease is 8.5 times as high” than individuals with healthy periodontal conditions, while “subjects with severe periodontal disease have a 3.2 times greater risk of cardio-renal mortality compared with patients with healthy periodontal tissues” [1]. Patients with T2DM show “three to four times greater risk of developing destructive periodontitis than non-diabetic subjects” [1].

A study induced periodontitis on pre-diabetic rats showed impaired glucose tolerance leading to the onset of T2DM in rats fed a high fat diet, which portrays a relationship between periodontitis and DM. Although mechanisms are not certain, data suggests hyperglycemia modifies advanced glycated end products (AGEs), which worsen patients and animals DM might influence a periodontal condition [1]. AGEs tend to

activate monocytes, macrophages and endothelial cells which release proinflammatory cytokines

and proteases that destruct bone supporting the tooth and gingival tissues. Data shows increased AGE receptor (RAGE) in patients with T2DM and periodontitis, which helps support a potential mechanism of the relationship between DM and periodontitis [1]. The results suggest treatment of periodontitis may improve glycemic control and help decrease organ damage over time. The activation of proinflammatory cytokines such as TNF α , IL1 β and IL-6 stem from bacteria and their by products such as lipopolysaccharides (LPS). A pattern recognition receptor that recognizes products of viruses and bacteria called TLR4 initiates immune and inflammatory responses to infection [1].

Every patient's case differs in severity because of length of oral care provided or how well insulin levels have been maintained. Some treatment suggested by Bjelland and colleagues for type one diabetes include insulin therapy, diet control, exercise as well as oral hypoglycemic agents. Possible reduction of inflammation may result from periodontal treatment, which improves glycemic control aiding in decreasing organ damage [1]. When periodontal lesions occur, high levels of cytokines are released or LPS in gingiva, which may enter the systemic circulation and influence organs at distant sites.

Watanabe suggested a variance within results because patients have differing diabetic control and severity of periodontitis. However, "it cannot be ruled out that poorly controlled diabetic subjects or subjects with T1DM of long duration have poorer oral hygiene, poorer compliance with dental appointments predisposing patients to more severe gingival inflammation and periodontitis" [1]. Although the relationship between diabetes mellitus and periodontitis requires further research to better understand the mechanism of the effect on organ damage [1]. However, data suggests a close correlation between DM and periodontitis using animal models indicate periodontitis does influence the progression of diabetes and treatment helps glycemic control to reduce organ damage. Therefore, Watanabe states communication between health professionals becomes critical for optimal care in DM patients. Although the bi-directional relationship may not always increase risk it will still benefit patients to take precautionary measures.

Oral disease may cause a medical problem. Procedures that may disrupt bacteria include brushing, extractions or surgery allowing bacteria such as *P. gingivalis* to use a pathway like the endocytic pathway causing complications [3]. Patients with cardiovascular disease test positive for secretions of inflammatory mediators, VCAM1, E-selectin, and ICAM-1 within the arteries [3]. Another contributing bacterium includes *C. pneumoniae* increasing patient's chances of developing coronary heart disease [3]. The bacterium does not normally show within normal arteries, which leads to the hypothesis that periodontal pathogens do have the ability to invade vascular cells and increase inflammation [3]. Myocardial ischemic events also occur from specific dental plaque bacteria [3]. Chui studied thirty-three different cases of carotid atherectomy specimen, which detected an infectious agent within atherosclerotic plaque [3]. The atherosclerotic plaque then interacts with vascular cells increasing the infectious agents present as well as neurovascularity. [3].

Some infectious agents within the bloodstream include bacterium named *porphyromonas gingivalis*, which invades gingival tissues and advance to deeper epithelial tissues [3]. *P. gingivalis* causes complications by secreting inflammatory mediators and spreading into the bloodstream. *C. pneumoniae* correlates to increasing patient's risk of coronary heart disease

because normal arteries do not contain *C. pneumoniae* [3]. The risk for developing cardiovascular disease increases when bacterium reaches the epithelial tissues. Periodontal pathogens have to ability to invade vascular cells through the bloodstream [3]. These microorganisms contain LPS like mentioned for DM patients that evoke an antibody response to infections [3]. Specific dental plaque bacteria induce platelets that aggravate the bacterial surface platelet aggregation-associated protein, triggering an acute myocardial ischemic event [3]. Chui mentions data from thirty-three human carotid atherectomy specimens were studied for the presence of multiple infectious agents in human carotid atherosclerotic plaques and both *C. pneumoniae* and *P. gingivalis* were present within carotid plaques. The microorganisms interact with host vascular cells and begin the stages of developing atherosclerosis and trigger acute coronary syndrome or ischemic stroke.

Similar to patients developing atherosclerosis Deshpande and colleagues claim periodontitis, “a bacterially induced chronic inflammatory disease that is the major cause of tooth loss in the adult population,” associates with cardiovascular disease. Destructive inflammatory response caused by the periodontal pathogen *P. gingivalis* interacts with the immune system. *P. gingivalis* may be found in gingival tissues, which suggests the bacteria’s interaction with the epithelial barrier plays a role in a systemic spread of the organism [4]. This occurs because the connective tissues in the periodontium are highly vascularized allowing microorganisms to easily enter the bloodstream [4]. The study consisted of transmission electron micrographs data showing *P. gingivalis* ability to invade endothelial cells (see figure two from [4]). Further data includes scanning electron microscope images of *P. gingivalis* infecting fetal bovine heart endothelial cells, bovine aortic endothelial cells, and human umbilical vein endothelial cells (see figure 3 from [4]). Although the pathway by which *P. gingivalis* uses to invade endothelial cells remains uncertain, “the invasion . . . by electron microscopy supports the endocytic pathway of engulfment” [4]. Treatment of epithelial cells with monodansylcadaverine and ouabain inhibit coated pits from forming therefore reducing the amount of *P. gingivalis* invasions. Overall, coated pits and engulfment may be a possible mechanism *P. gingivalis* uses to spread into distant sites.

The concept of *P. gingivalis* causing entering gingivae lesions may be caused from tooth scaling, periodontal surgery, dental extraction, and tooth brushing allowing bacteria to circulate the bloodstream putting patients at risk for cardiovascular disease. Human arteries in atherosclerotic areas contain secretions of inflammatory mediators, E-selectin, ICAM-1, and VCAM-1 which all contribute to the pathology of cardiovascular disease [4]. A study performed by Roivainen and colleagues found an “association between high levels of antibodies to enteroviruses, measured by use of a group-specific antigen, and the risk of myocardial infarction” [5]. Viruses found in coronary heart disease and atherosclerosis includes cytomegalovirus (CMV), herpes simplex 1 (HSV-1) and HSV-2 within lesions. Participants within the study had controls “who completed a follow-up without coronary heart disease” and “matched for treatment group (gemfibrozil/placebo) and place of residence” [5]. Researchers measured enterovirus group specific antibodies via enzyme immunoassay (EIA). Enterovirus infections were found by the peptide KEVPALTAVETGATC act as a group antigen for diagnosis [5]. Adenovirus specific antibodies, HSV-1 and CMV antibodies, *C. pneumoniae* IgA serum antibodies, specific immune complex bound IgG antibodies were also measured. Results showed that HSV-1 (IgG) and *C. pneumoniae* and immunocomplex bound antibodies (IC) were all higher in coronary heart disease patients rather than controls [5]. Levels of *H. pylori*,

enteroviruses, and adenovirus were the same for CHD cases and controls. The most significant risk of CHD includes elevated levels of Cpn and HSV-1 antibodies [5].

While no association was confirmed between high levels of adenovirus, cytomegalovirus, enterovirus and *H. pylori* to increased risk of CHD. Data showed 103 out of 241 cardiac events occurred while *C. pneumoniae* were present in atherosclerotic lesions. Overall, HSV increased with low levels of CRP while high *C. pneumoniae* required elevated CRP levels, which also increases with smoking [5]. Either of these two infectious agents increases the risk of CHD, Roivainen and colleagues state “results thus support the hypothesis that inflammatory reaction can be one of the major factors in the pathophysiology of atherosclerosis and suggest that at least 2 different infections are capable of triggering this reaction” [5]. These high antibody levels show that an active and chronic infection leads to an increased risk of coronary heart disease [5]. Lamster and colleagues suggest medical professionals and dentists should “change perceptions regarding oral health and disease so that oral health becomes an accepted component of general health” in an efficient manner to improve oral health [6]. The first study of the relationship between oral inflammation and systemic diseases including DM and CHD began in 1989. They claim periodontitis has been recognized as the sixth clinical complication from diabetes mellitus [6]. Health professionals need to work together to find specific treatment recommendations for patients including but not limited to treatment of periodontal disease to improve glycemic control. More evidence continues to emerge explaining the benefits of periodontal therapy and its relationship between systemic diseases and oral inflammation [6]. With further collaboration between medical and dental professions, dental schools may modify the programs to include general health care in order to change the future of the profession. Curriculum and conferences may be included to improve communication about dental information to medical professionals and even patients. The new knowledge may increase awareness of the connection between diseases to improve overall health outcomes. Lamster and colleagues mention continuing education events, outreach visits, workshops and conferences to change professional practice by implementing new techniques. Professionals within the dental field and dental schools should take responsibility to promote education about periodontal disease to nurse practitioners, midwives and physician assistants to reduce patients risk of developing systemic diseases from periodontitis [6]. If health professionals can work together to improve disease prevention then infection rates will decrease as a result. Although it is easy to argue that dentists have limited time each day so collaboration may be limited sufficient evidence urges the importance of improving interpersonal collaboration.

Therefore dentists and doctors must consider oral health as an “accepted component of general health,” and work harder to educate other professional practices outside of dentistry as well as increase patient’s knowledge about risk factors involved. Improved collaboration such as opening joint practices to share patients may be a great solution will reduce the risks of further complications because dental checks may detect undiagnosed diseases and reduce the chances of developing a systemic disease. If dentists and doctors have low priority with collaboration then what alternatives may be implemented in order to increase prevention via treatment?

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Biography

Macayla Kinney is currently a fifth year at the University of California, Merced and graduated with her Bachelor's degree in Human Biology and minor in Psychology with the Class of 2015. She has been involved in many extracurricular activities on campus including holding an officer position within Project Smile the Pre-Dental organization, volunteering in a research lab studying Hepatitis C virus and is currently an alumni member of the Fraternity, Delta Delta Delta that works with fundraising for St. Jude's Children Research Hospital. She is an aspiring Dentist and is applying to Dental school in summer of 2016. This paper was inspired by her interest in the connections between dentistry and systemic diseases. Macayla plans to incorporate these aspects of interpersonal collaboration with other medical professionals during her career. She is also interested in further research to incorporate within her practice to aid in systemic and periodontal disease prevention.