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Review Article

A Double Threat to Oral health: The Inflammatory Processes of Diabetes and Tobacco Use

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Abstract

The incidence debilitating conditions such as diabetes and risk factors such as the use of tobacco products continue to climb, so does the need for better understanding of these complex conditions. Long-term presence of diabetes and tobacco use may result in deterioration. Additionally, diabetics who use tobacco products have many adverse oral conditions. Of these factors, inflammatory responses represent a complex interplay and offers a unique challenge. Strong medical consultation team approaches contribute to improving outcomes of diabetes and its comorbid disorders.

Keywords: Oral Health; Diabetes; Inflammation; Tobacco Use; Multi-System Decline

Introduction

Diabetes continues to grow as a national health priority. Recent estimates suggest that diabetes affects more than 11% of the US population, with approximately 90% to 95% of cases representing Type 2 diabetes. Risk factors such as tobacco use in diabetic patients, adversely affects the course of many co-morbidities [1]. Tobacco kills more than 480,000 people annually – more than AIDS, alcohol, car accidents, illegal drugs, murders and suicides combined [2]. If current trends continue, 5.6 million kids under age 18 alive today will eventually die from smoking-related diseases. Smoking causes over 80 percent of all cases of lung cancer deaths and about 30 percent of all cancer deaths. In addition, smoking causes nearly 80 percent of all COPD deaths and 32 percent of all coronary heart disease deaths . This paper aims to raise awareness about the impact of declining oral health in Type 2 diabetics who use tobacco products [3].

Diabetic patients who use tobacco generally undergo metabolic decline in multiple systems, including changes in the oral cavity. Heightened activation of immune systems and local adipose inflammation and the secretion of a plethora of pro-inflammatory adipokines from visceral adipose tissues is indicated in metabolic disease [4].

Effects of Inflammatory Processes and Oral Tissue Alterations

Oral mucosal lesions that may result from tobacco use include leukoplakia, erythroplakia, cancers of the oral cavity and pharynx, oral diseases of the gingival and periodontal tissues may involve inflammatory processes [5,6]. Smokeless tobacco use is again popular in the forms of snuff and snus. After long-term use, snuff-dippers lesions, and other inflammatory-related lesions such as sores may occur. Snuff submucosal deposits which may lead to dental caries [7].

Oral tissue alterations occur due to tobacco use, especially along the oral mucosal areas where the tobacco is placed. Zhang et al. showed that inflammatory responses and osteoclastogenesis are commonly involved in the development of periodontitis [8]. Animal studies revealed that the periodontium is aggravated by nicotine, causing ischemia and inflammation. Nicotine, the addictive component in tobacco products is associated with inflammatory mediators which aggravate oral mucosal tissues [8].

The association of periodontitis was found to be statistically significant with tobacco smoking, diabetes, hypertension and age. Higher age predicted greater incidence of periodontal disease. The

most common risk factors attributed to periodontal diseases are tobacco use, diabetes, pathogenic bacteria and tooth deposits [9,10]. Fouad et al. investigated treatment outcomes in root canal therapy and found that patients with diabetes have a reduced likelihood of successful treatment in cases with peri-radicular lesions [9,11]. High prevalence of periodontitis occurs after long-term, heavy use of tobacco products [12].

Similar mechanisms are induced in the diabetic patient who uses tobacco products [12]. These factors enhance periodontal and carious process. Diabetic patients who use tobacco products are more likely to experience extreme tooth loss [13,14].

Tobacco use and inflammatory processes

Smokeless tobacco products deliver substantial doses of nicotine along with powerful cancer-causing chemicals [15]. Local irritation from salt may increase the absorption of smokeless tobacco carcinogens in the oral cavity, and may lead to chronic inflammation. Absorption of nicotine across biological membranes is highly pH dependent [7]. Free nicotine (unionized and pH above 6.5) is readily absorbed into biological tissues, and well absorbed through the mouth and buccal membranes [14]. Smokeless tobacco users and those who smoke cigarettes have comparable levels of nicotine in the blood [16]. Nicotine found in smokeless tobacco and cigarettes, is absorbed through the oral cavity tissues directly continues into the blood, where it goes to the brain [15,16]. Even after the tobacco is removed from the mouth, nicotine continues to be absorbed into the bloodstream [14]. Orally absorbed nicotine stays in the blood longer for users of smokeless tobacco, than for cigarette smokers and is easily absorbed through the oral mucosal lining [17]. Approximately twice as much as nicotine is absorbed per dose of smokeless tobacco than cigarettes (4mg vs. 2mg).

Cigarette smoke (CS) causes considerable morbidity and mortality by inducing cancer, chronic lung and vascular diseases, and oral disease. Despite the well-recognized risks associated with smoking. CS also contains trace amounts of microbial cell components, including bacterial lipopolysaccharide [18]. These and other CS constituents induce chronic inflammation at mucosal surfaces and modify host responses to exogenous antigens CS also contains trace amounts of microbial cell components, including bacterial lipopolysaccharide. The net effect of CS on immunity depends on many variables, CS also impairs immunity in the oral cavity and promotes gingival and periodontal disease and oral cancer [18,19].

Inflammatory processes are a common thread in patients with diabetes and in tobacco users, and these inflammatory changes affect the oral cavity [16]. There is growing evidence for the relationship between exposure to mainstream and side-stream smoke and diseases resulting from reactive oxidant challenge and inflammation directly as a consequence of the combined activity of neutrophils, macrophages, dendritic cells, eosinophils, basophils, as a humoral immunological consequence of sensitization, and that the metal components of the particulate play a role in adjuvant effects [20].

When cigarette smoke is sustained, a chronic inflammatory process ensues that has the potential to promote enhanced microbial colonization and infection [18]. Gaseous and particulate cigarette smoke (CS) constituents interface with the immune system at the mucosal surfaces lining the oral cavity, sinuses and airways. T-cells may be induced to proliferate cytokines that mediate important inflammatory functions [18,21].

Inflammation and diabetes

Insulin resistance has been suggested as the metabolic link for periodontal disease contributing to poorer glycemic control in diabetes. Rigorous management of glycemic control reduces the risk of coronary heart disease and chronic kidney disease in people with diabetes [12], and there is evidence that non-surgical periodontal therapy significantly contributes to improvement in glycemic control [9]. Patients with Type 2 diabetes have a high prevalence of periodontal disease, xerostomia and increased caries [12]. Bi-directional relationships further perpetuate the cycle of diabetes, periodontal disease and other chronic diseases [22] (Figure 1).

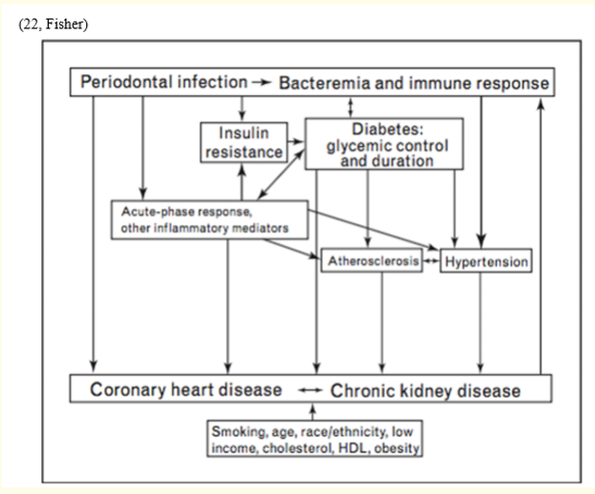

Figure 1

Independent of the pathogenetic mechanism among the different types of diabetes, the common pathway in TD2 seems to be the inflammation in the pancreatic Langerhans beta cell islets (insulitis), in the concept of an auto-inflammatory process, which results in reduction in both beta cell number and function [23]. It has been suggested that in people with a genetic predisposition, the 'stressed' beta cell may stimulate local inflammation and modify the balance between beta cell mass and function in the islets of Langerhans [23].

Several experimental models as well as observational studies in humans have demonstrated that macrophages play a key role in the

islet inflammation seen in T2D [18]. It is likely that other immune cell types are involved in islet inflammation in T2D, while islet autoimmunity has also been suggested to contribute to beta cell functional decline during the course of T2D2 [23].

Ueta et al. found that patients with diabetes had a high number of pulpal and periodontal infections and that diabetes was a predisposing condition for endodontic infections [24]. Animal studies in diabetic rats given a sucrose solution resulted in very large periradicular lesions [25].

Chronic hyperglycemia is one of the most frequent effects of diabetes and the major underlying cause for associated complications. Diabetes patients may experience severe lesions many affecting eye function, peripheral nerves and blood vessels [26]. Significant health risks to major organ systems may occur within the human body, including atherosclerotic vascular disease, neuropathy or peripheral artery disease, and in severe situations amputations, loss of kidney function and progression of multi-organ complications. Diabetes affects several functions of the immune system making the diabetic patient more vulnerable to chronic inflammation, progressive tissue damage and reduction of tissue repair [26]. Diabetic glucooxidative stress impairs the healing response and disrupts the flow of overlapping healing phases. In addition to healing impairment, other factors include non-healing wounds which are a major predisposing factor or entry point for infections [27]. A microbiota-biofilm comprises symbiotic bacteria, yeast and fungal loads and can silently spread, amplifying the underlying healing deficit [27].

Chronic and generalized inflammation occur in patients with diabetes, and in those who use tobacco. Both of these high risk conditions impact oral health. Patients with diabetes are impacted by chronic and generalized inflammation. Chronicity of inflammation presents the strongest plausibility for detrimental effects of inflammatory events that could also link periodontal disease and other oral diseases to diabetes. Diabetes and tobacco users are double threats to systemic health and oral health [12].

Conclusion

Continued efforts are needed to assess the public health impact and dangers associated with the long-term Type 2 diabetes, use of tobacco products, and pro-inflammatory oral conditions. There is a continued need to evaluate cytological and morphologic alterations of oral mucosal tissues. Numerous epithelial layer cells and oral mucosal membranes are altered by chemicals and constituents in cigarette smoking and in the use of smokeless tobacco . Additionally, there are significant inflammatory processes and health risks to major organ systems within the human body, including atherosclerotic vascular disease, neuropathy or peripheral artery disease, non-healing wounds, and in severe situations amputations, loss of kidney function and progression of multi-organ complications. The breakdown of many cellular systems may concurrently result in a complex process of diminishing oral conditions, organ damage and organ failure.

Patient-centered care in diabetic patients who use tobacco can result in long-term communication and interactions between the patient management team to provide the best overall care for the patient. Collaboration and coordination of all of the medical specialties is critical in patients with complex chronic diseases.

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