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Diabetes and dental disease: An insight

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Abstract---This study's purpose is to provide up-to-date knowledge on the connection between periodontal disease and diabetes from the standpoint of oral health, and its goal is to accomplish this through presenting the material. Both diseases are the consequence of a range of different factors that can either cause the disease to begin or modify the disease's progression. The likelihood that a person may develop either sickness varies from person to person, and both diseases are caused by a variety of different conditions. Chronic periodontal disease has been hypothesized to influence how diabetes develops over time, while diabetes has been hypothesized to increase the prevalence of periodontitis. Both hypotheses have been tested. Because atherosclerosis, poor sugar metabolism, and oral infections

all appear to be related to one another, there is a possibility that there is a connection between metabolic syndrome and periodontal disease. Those with type 2 diabetes who are successful in treating their periodontal disease may have an improvement in their ability to control their blood sugar. If glucose regulation is improved, there is a possibility that periodontal disease can be managed more effectively.

Keywords---diabetes, dental disease, chronic periodontal disease, hypothesized.

Introduction

The physiognomies of diabetes and periodontal disorder are analyzed and addressed in this research, in addition to their known interactions and the supporting scientific data, with the primary emphasis being placed on clinical investigations. There has been a significant amount of investigation into the link between diabetes and periodontitis over the course of the last few decades (Ponts et al., 2007; Bascones Martinez et al., 2011, 2014). On the other hand, the impact that periodontal therapy has on the way diabetics control their blood sugar levels is not yet well understood. The influence that periodontal treatments have on glycemic control has been the focus of a significant number of studies conducted in the scientific community; however, the influence that glycemic control has on the periodontal state is not as well recognized (Taylor et al., 2008; Mealey, 2006). This review not only provides information on the current state of our understanding of these two disorders, but it also highlights the need for future research to explain the connection between the two conditions. The purpose of the research was to provide up-to-date knowledge on the topics from the perspective of oral health.

Diabetes

Diabetes mellitus, a metabolic condition that is frequently characterized by the traditional triad of polydipsia, polyuria, and polyphagia (Goldberg, 2019). These signs and symptoms are a result of poor glucose homeostasis. The true prevalence in the general population, which has been reported to range from 1% to 6% depending on the diagnostic criteria used, is difficult to determine (Dubey and Mishra, 2017). Ninety percent of cases of type 2 diabetes are in people who do not need to take insulin (Zaccardi et al., 2016). Retinopathy, nephropathy, neuropathy, wound healing, and cardiovascular issues are the main complications of diabetes (Lotfy et al., 2017). These complications are all linked to changes in macro- and microvascular circulation that are brought on by the disease's ineffective metabolism of glucose, lipids, and proteins (Filla and Edwards, 2016). One of the most typical consequences of diabetes is retinopathy. Given how frequently people experience both diabetes and periodontal disease at the same time, it has been hypothesized that periodontal disease is the sixth effect of diabetes. There are still some questions about some parts of this relationship, though.

Periodontal Disease

The localized infections that are known as periodontal disease can affect both the structures that make up the periodontium and the tissues that support teeth. Periodontal disease is also known as gingivitis (i.e., gingiva, periodontal ligament, root cementum, and alveolar bone). Periodontal disease is an umbrella term that encompasses both reversible (gingivitis) and irreversible (periodontitis) disorders that can affect the teeth and gums. Periodontitis is characterized by the destruction of the connective tissue of the tooth attachment apparatus, which in turn leads to the migration of the apical apparatus and, ultimately, tooth loss (Nibali, 2016). The earliest clinical symptom of periodontal disease is the presence of periodontal pockets, which are characterized by the presence of an environment that is favorable for the colonization of bacteria (Mombelli, 2018). It can be identified using a combination of an X-ray examination and a clinical examination that uses a periodontal probe to determine the depth of the pockets between the teeth (Mombelli, 2018). After that, methods from the field of microbiology are applied to conduct an accurate analysis of the infectious agents. The extremely high prevalence of periodontal disease is one factor that adds to the clinical relevance of the condition. Although there is some variation in the numbers, most of the research carried out in countries such as the United States indicated that 14% of the population suffers from periodontal disease and that 50% of the population has had gingivitis at some point in their lives (Mealey and Oates, 2006). The numbers for periodontal disease may possibly be an understatement of its true prevalence considering that earlier studies had showed that 25–36% of the population was affected. According to reports, the diagnostic approach that was selected influences estimates of the disease's prevalence.

The inconsistent diagnosis of the illness may be partially attributed to the lack of a proper classification. According to the genesis and clinical signs of periodontal disease, there have been numerous attempts to categorize the condition (Marchetti et al., 2012). According to these categories, periodontal disease exists independently of the patient and can manifest symptoms. The six categories recognized by the American Association of Periodontology (AAP) classification, which is currently the most popular classification, are gingival disease, chronic periodontitis, aggressive periodontitis, periodontitis as a manifestation of systemic disease, necrotizing periodontal disease, and periodontal abscess (Germen et al., 2021). Gum disease is under one of the categories. The inability to classify periodontal disease according to its origin and the lack of clearly defined clinical criteria for its diagnosis are both disadvantages. As a result, it is up to the medical practitioner to determine the patient's diagnosis.

Diabetes and periodontal disease

Diabetes is a systemic condition that can have an impact on both the life expectancy and quality of life of a diabetic patient. It is possible for this to influence the neurological, cardiovascular, and renal systems, in addition to the healing of wounds (Edelman et al., 2002). Additionally, diabetes either brings on periodontal disease or makes the condition more severe (Casanova et al., 2014). Findings from a study conducted on the Pima Indians of Arizona revealed that diabetics of all ages experienced more periodontal attachment and bone loss than

non-diabetics (Casanova et al., 2014). There is a correlation between diabetes and periodontal disease, although the origins of this connection are unknown currently. Others feel that the AGEs that are produced by diabetes are what cause the hyper-inflammatory cells, while others believe that the two diseases share a shared ancestry (Garcia et al., 2001).

We will give a quick overview of the relationship between diabetes and gum disease. There is a strong correlation between periodontal disease and diabetes in families, which accounts for many cases of each illness. Both conditions are considered polygenic because there is no single genetic mutation or issue that can be related to either of them. There is a connection between genetics and susceptibility to sickness. The intricate interplay between host response mechanisms and dangerous microbes in periodontal disease makes it difficult to clarify genetic variables (Pranckeviciene et al., 2014). A common HLA genotype is responsible for both diseases. Changes in the way antigen is presented to T cells and the immune response of the patient can be brought about by HLA anomalies, which can put a person at risk for diabetes and periodontitis (Sanz et al., 2018). The molecule known as HLA is located on chromosome 6. It is necessary to conduct further research before passing judgement. Both diabetes and periodontal disease can be traced back to problems in the immune system. It is essential to exercise control over cytokines as well as soluble factors to lessen the destructive impact of both diseases.

It appears that the disorders are linked by their hyperinflammatory characteristics. People who have type 1 or type 2 diabetes or periodontal disease are more likely to experience an imbalance in their soluble cytokine levels, as well as an increased rate of cytokine release (Mesia et al., 2016). Diabetes is caused by both infectious agents and environmental pollutants, whereas smoking and stress are risk factors for periodontal disease. Patients suffering from periodontal disease and diabetes have cells that produce higher levels of the cytokines prostaglandin E2 (PGE2), interleukin 1 (IL-1), and tumor necrosis factor alpha (TNF-alpha). This up-regulation of immune response mediators has been seen in vitro as well as in comparisons between sick animals and controls, demonstrating that there is a relationship between the two conditions. To begin, we will combine the many hypotheses regarding diabetes and periodontal disease. A flawed gene combination on chromosome 6 makes the host more vulnerable to the effects of environmental pressures. If the host's immune system and other defense mechanisms are impaired because of this vulnerability, diabetes and/or periodontal diseases may develop.

Other possibilities for the connection between the two diseases involve the binding of AGEs to their cell receptors (RAGEs), which are found in the endothelium or phagocytic cells of some diabetes patients. It is possible for either mononuclear or polymorphonuclear cells to produce AGEs, which reduces the cells' capacity to chemo tact and phagocytose, hence facilitating the growth of gram-negative anaerobic bacteria. This helps to explain why patients with diabetes have more severe forms of periodontal disease. This hypothesis postulates that AGE-stimulated macrophages and polymorphonuclear cells hyper-react to bacterial biofilm, resulting in greater connective tissue degeneration in these patients. This is because the release of more cytokines and

soluble mediators (Fig. 1). The binding of AGE receptors in fibroblasts may influence collagen, which is a component of periodontal tissue. Interfering with the turnover of collagen would make it more difficult for diabetics to heal wounds and would raise the risk of developing periodontal infections. The deterioration of connective tissue is caused by AGEs, which work by stimulating collagenase and other enzymes in the tissue (Reynolds, 1996).

MMPs are found in larger concentrations in gingival pockets of patients with advanced periodontal disease, and this correlation is also shown between advanced periodontal disease and higher MMP levels in the saliva. Patients suffering from periodontitis have higher levels of serum MMP, which may be associated to an infection caused by periodontal bacteria. It is possible that MMP-9 is the link between cardio pathogenesis and periodontal disease (Isola et al., 2021). There is a scientifically feasible mechanism by which diabetes weakens the host's defenses. The presence of the same bacterial species in diabetic and non-diabetic periodontium demonstrates that an immune issue that produces AGE is the root cause of the increased risk of periodontal disease seen by diabetic patients (Preshaw et al., 2012). Periodontal infections have the potential to upregulate cytokines and enzymes that degrade tissue, which can have systemic repercussions.

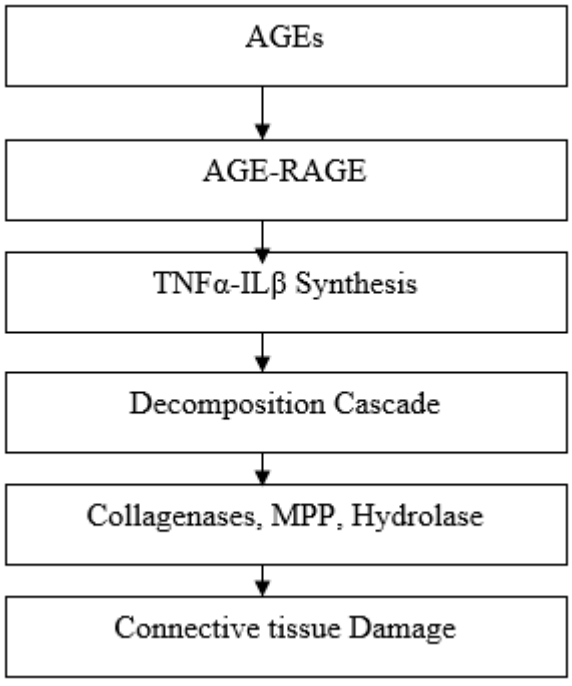

Fig 1. Diabetic periodontal disease model

Periodontal disease and diabetes

We now have a much better understanding of the pathogenesis of periodontal disease because of the revelation that subgingival plaque is a microbial biofilm

(Berezow and Darveau, 2011). Microbial biofilms are made up of bacteria that have embedded themselves in an extracellular matrix as well as bacteria that have adhered to one another or to a surface. The development of the subgingival biofilm requires bacterial adhesion, and the pathogenic potential of the biofilm depends on the growth and maturation of the relevant bacteria. A component of these bacteria's cell structures is constantly ejected into the spaces they have dug for themselves. Gram-negative bacteria's cell walls play a significant role in the etiology of periodontal disease.

These structures, which contain lipopolysaccharides and vesicles with protein content and can induce an innate host response, are a normal part of the bacteria's cell wall turnover process and have the power to do so. Even though the host's immune system is unaffected, the structure of the biofilm produces conditions that are exceptionally favorable for the survival of the bacteria that make it. These microbes' virulence factors are what trigger the series of pathogenic occurrences that lead to periodontal disease (Dahlen et al., 2019). Tannerella forsythia (T.f.) and Porphyromonas gingivalis (P.g.), periodontal bacteria, have been demonstrated to increase MMP-9 levels in gingival crevicular fluid and serum (Seo and Hwang, 2022). The pathogenic model outlined in suggests that periodontal disease may worsen the high cytokine levels already present in diabetes individuals and, thus, lead to the emergence of systemic inflammation (Fig. 2). The most frequent cause of diabetes-related problems is the overproduction and buildup of AGEs in tissues (Cade, 2008). These molecules attach to neutrophils and trigger a series of events that worsen the inflammatory response and boost cytokine output (Panagakos and Scannapieco, 2011). The LPS of gram-negative bacteria (like P. g.) in the subgingival biofilm is more reactive to these previously activated neutrophils, hastening the destruction of periodontal connective tissue and worsening diabetes (Sheets, 2006).

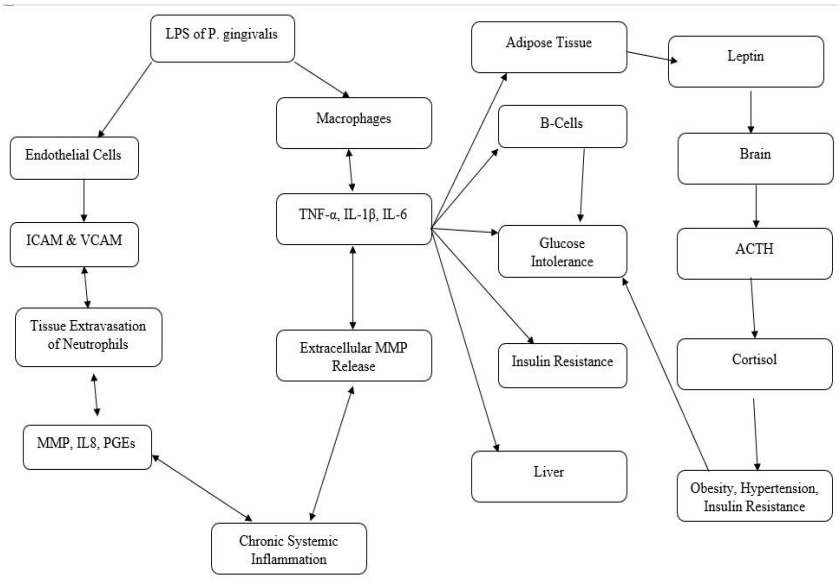

Figure 2. Model showing how periodontal infection contribute to systemic inflammation, sugar imbalance, and diabetes

Diabetes and periodontal care

Although the association between diabetes and periodontal disease has been established beyond a shadow of a doubt, there is still a degree of controversy surrounding the impact that the metabolic control of diabetes has on periodontal disease as well as the impact that periodontal therapy has on the ability of diabetic patients to maintain metabolic control. People who have diabetes are one of the groups of patients who, in general, are given higher priority for periodontal therapy than other patients (Llambes et al., 2008). This is because periodontal disease may pose a risk to the overall health of diabetic patients. Publications have been made including protocols for the treatment and prevention of infections in diabetic patients, with an emphasis on managing acute infections (Albridge et al., 1995; Kiran et al., 2005; Wang et al., 2014). The treatment of chronic infections appears to be much less effective than the treatment of acute infections.

Conclusion

Based on published studies on people with diabetes type 1, it is not feasible to say for sure how periodontal therapy affects glucose management. The different causes of type 1 and type 2 diabetes may be better understood with the use of insulin therapy. The researchers treated type 1 diabetics who had periodontal issues but found no difference in their HbA1c levels after treatment. The main area of investigation into the relationship between periodontal disease and type 2 diabetes should be this problem. The sole effect of doxycycline gel that was supported by meta-analysis was its impact on metabolic regulation. Before drawing firm conclusions, more research on this topic is necessary. It's crucial for diabetic patients to keep in regular contact with their dentist because periodontal disease and diabetes are related. Despite the relationship between these disorders, more investigation is necessary to completely understand the clinical ramifications. It is essential to consider the possibility of simultaneous occurrence of both illnesses to establish an early diagnosis. Medical professionals should urge diabetic patients to visit their dentists frequently to better control potentially harmful factors, such as the accumulation of bacterial plaque in the periodontal pocket, given the high prevalence of these diseases and the potentially serious consequences associated with them. Dentists need to watch out for diabetes and issues with sugar metabolism since these can exacerbate periodontal disease.

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