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Assessment of the Risk Factors for Periodontal Diseases

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ABSTRACT

With the advances in the understanding of the etiology and pathogenesis of the periodontal diseases, the role of different risk factors in the diseases process is also gaining importance. Though microbial dental plaque is the primary etiologic factor for the Chronic periodontal disease, the role of other secondary or modifying risk factors cannot be undermined. There is enough evidence to suggest the possible role of diabetes, smoking and stress in the periodontal destruction. This paper highlights the role of such factors for the dental practitioners to understand the possible role of the systemic factors. Dentists should be encouraged to communicate with physicians about the health of their patients, and physicians should be alerted to the possible risks of severe periodontitis.

Key words: Risk Factors, Periodontal disease, Risk Assessment

INTRODUCTION

Periodontics like many other specialized areas of dentistry is undergoing yet another change. The current era of change in periodontics is focusing on host factors. This evolution is being stimulated by new evidence that suggests a link between systemic factors and the severity of periodontal disease. Assessment of risk remains an integral part of diagnosing and treating periodontal disease. It was reported that evidence to support risk assessment in clinical decisions is substantial[2]. Evidence emerging from clinical research has shown that patients with systemic diseases (e.g., diabetes) as well as patients with other systemic factors (e.g., smoking) have an increased risk and severity of periodontal disease[4,5]. As a result, these systemic "host" factors are now being recognized as significant contributors to development and progression of periodontal disease[4].

Local Risk Factors for Periodontal diseases

There is ample evidence to demonstrate a relationship between bacterial plaque and gingival inflammation. While periodontal pathogens are essential for periodontal disease progression, these pathogenic microorganisms

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alone are not sufficient to explain the differences observed in periodontal disease severity. Factors in addition to bacterial plaque may also contribute to periodontal disease destruction; Periodontitis is now seen as resulting from complex interplay of bacterial infection and host response, often modified by behavioral factors. Perhaps the most fundamental change in our understanding of periodontal diseases is that not all individuals are equally susceptible to severe disease. Some individuals are more at risk for periodontitis than others. One of the best predictors of future disease progression appears to be past disease. An individual who has suffered from periodontitis previously is more likely to experience future disease destruction than an individual who has not had previous disease. However, the other possibility is that systemic or environmental factors that originally predisposed that individual to periodontitis may continue to predispose him or her to future disease. Hence, the existence of previous disease is a positive indicator for that individual's "risk" of future disease. This latter view suggests a significant role of the host in the development and progression of periodontitis. Host factors that may have more influence on disease progression than periodontal pathogens include diabetes, smoking, stress, and genetic predisposition. Thus, it has become essential to identify systemic "host" factors that increase the risk of periodontal disease destruction[12].

Systemic Host Risk Factors

Diabetes Mellitus

Diabetes is a systemic condition that has long been associated with an increased risk and severity of periodontal disease[5.7.12]. The reasons for this relationship are many and relate to the pathogenesis and control of diabetes. The mechanism responsible for increasing the risk in diabetics is likely to be related to an increased susceptibility to infections, an impaired immune response, poor wound healing, or a combination of these factors. An altered periodontal microflora has also been suggested as a possible cause for the increased periodontal disease seen in diabetics.

The accumulation of advanced glycosylated end products in the tissues of diabetics alters the integrity and function of the affected tissues. This may be mechanism responsible for some of the complications observed in diabetics[14,15].

Well-controlled diabetics with good oral hygiene do not show an increased risk of developing severe periodontitis. In fact, well-controlled diabetics have fewer systemic complications than poorly controlled diabetics and have been shown to respond well to periodontal therapy[14,15].

Smoking and Tobacco

Smoking has long been associated with adverse systemic health effects such as respiratory disease arid cancer. Analysts of data from the 1971-75 National Health and Nutritional Examination Survey in the United States showed a clear relationship between smoking and periodontitis[8]. Many studies since then have provided strong evidence to support an increased risk relationship between smoking and periodontal disease severity. Proposed mechanisms include ail altered immune response, decreased vascularity, impaired polymorphonuclear neutrophil leukocyte chemotaxis and phagocytosis, and decreased antibody production. Smoking also appears to decrease local oxygen levels. It has been suggested that the resulting decreased oxygen tension may encourage growth of anaerobic pathogens[3,9].

Psychosocial Stress

Psychosocial stress has been associated with periodontal disease. In World War II soldiers on the battlefield presented with trench mouth, a condition also known as Acute Necrotizing Ulcerative Gingivitis. This form of periodontal disease is known to be stress-related. The mechanism of stress-induced periodontal disease destruction is not well defined. However, it has been known for many years that increases in corticosteroids, whether exogenous or endogenous, decrease the immune response. Psychosocial stress may predispose a susceptible host by decreasing the immune response to pathogenic bacteria and altering

wound healing.

The role of stress in aggravating systemic conditions (e.g., cardiovascular disease) has been well-documented. However, the evidence to support a relationship between psychosocial stress and periodontal disease is limited. In a case-controlled analysis of psychosocial factors and adult periodontitis, Moss and colleagues showed that individuals testing positive for antibody to the periodontal pathogen B. forsythus and rating high on the depression scale were 5.3 times more likely to have periodontitis than individuals testing negative for B, forsythus antibody and depression. Although these findings are suggestive, it is too early to make conclusive statements about the relationship of stress and periodontal disease destruction[11].

Genetic Predisposition

Genetics or familial inheritance of periodontal disease has long been suspected. With their study of adult twins and periodontal disease, Michalowicz and colleagues were the first to demonstrate that, indeed, periodontal disease was linked to genetics. Several studies have subsequently shown a similar relationship between juvenile periodontitis and genetic predisposition[10]. The first evidence of a specific genetic marker for susceptibility to severe chronic adult periodontitis was the discovery of a relationship between specific polymorphisms of the IL-1 genotype and the expressed phenotype of severe adult periodontitis. Subsequently, the first commercial genetic test for susceptibility to periodontitis became available (PST, Medical Science Systems, Flagstaff Ariz.). The degree of increased risk of severe periodontitis for genotype-positive patients is estimated to be about 6.8 times greater as compared to genotype negative individuals. It is estimated that approximately 30 percent of the population may be positive for this genetic marker[8].

Assessment of Risk Factors

The accurate assessment of risk factors for any condition depends on the technical or scientific reproducibility and validity of the diagnostic test. Diagnostic tests have properties that can be quantitatively determined, and these properties are described regularly in literature. They often include sensitivity, specificity and predictive value. The value of diagnostic information obtained irons a "good" test tor the purpose of clinical decision-making can be quantitatively determined for each patient using decision analyzing methodology.

In assessing risk for disease periodontitis can be thought to be more like some of our common medical conditions. Risk assessment efforts applied to periodontitis have only recently received attention, primarily because our prior paradigm regarding the etiology and progression of disease involved a ubiquitous condition, gingivitis, inevitably leading to periodontitis.

In an attempt to clarify the process of identifying high-risk individuals, the research group at the University of North Carolina has delineated a 4-step process.

The **first step** is identification of risk factors that are associated with the disease. The **second step** is development of a risk assessment model entails putting together the relevant risk factors into a multivariate model that identifies the combination of factors that will most efficient distinguish between those who are at high ride or low risk of developing the disease. The **third step** involves screening population groups for the factors included in the risk assessment model and using the model to predict each individual's risk of developing disease, this step is called assessment. The **fourth step** is targeting, involves the application of some health promotion/ disease prevention regimen or treatment procedure to the individuals at increased risk with evaluation of the effectiveness of the intervention[8].

CONCLUSION

The approach to the diagnosis and treatment of periodontal disease is changing. The disease has not changed, but dentistry's understanding of the pathogenesis and appreciation for the influence of host factors has improved As a result, the approach to the management of the disease is evolving. There is a rapidly growing body of data that supports a periodontal medicine interrelationship. Current

evidence suggests that systemic factors contribute to the severity of periodontitis, and periodontitis may be a risk factor for systemic diseases. As part of a comprehensive examination of patients for periodontal disease, dental practitioners must act more like physicians to evaluate systemic illnesses and other conditions that may contribute to the risk and severity of periodontal disease. Conversely, physicians must understand the role of periodontitis in the health of their patients and become aware of the signs of severe periodontitis. This is the beginning of a new era in periodontics that provides the opportunity for dentists to develop new relationships with physician colleagues.

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