Review Article

THE EFFECT OF PERIODONTAL DISEASE ON QUALITY OF LIFE: LITERATURE REVIEW

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ABSTRACT

Periodontitis and gingivitis poorly impact patients' function and appearance, directly affecting their Oral Health-Related Quality of Life (OHRQoL). Systemic problems, including poor pregnancy outcomes, cardiovascular diseases, type 2 Diabetes Mellitus (DM), respiratory disorders, deadly pneumonia in hemodialysis patients, chronic renal disease, and metabolic syndrome, have all been linked to periodontitis. The Medline, Pubmed, Embase, NCBI, and Cochrane databases were searched for studies of patients with non-alcoholic fatty liver disease. Incidence, etiology, and management options were analyzed. At this stage, it is clear that dental plaque (a microbial biofilm) causes gingival inflammation, and the extent and severity of the inflammation are influenced by various systemic and oral factors. Furthermore, plaque accumulates faster in inflamed gingival sites than in non-inflamed sites, resulting in a complex dynamic between the dental plaque biofilm and the host's immune-inflammatory response. However, it should be noted that not all inflammatory sites will progress to periodontitis. treating gingivitis with appropriate local therapeutic intervention is still necessary to prevent attachment loss and destruction of periodontal tissue. Gingival conditions may be diagnosed in the future using objective analytic approaches such as transcriptome characterization or epigenetic change categorization.

Key words: Periodontics, Periodontal debridement, Quality of life, Cardiovascular diseases, Diabetes mellitus

Introduction

A bacterial plaque buildup at and below the gingival margin causes plaque-induced gingivitis, an inflammatory response of the gingival tissues [1]. Although it doesn't directly cause tooth loss, controlling gingivitis is a key periodontitis prevention measure [2]. Plaque-induced gingivitis, the most prevalent kind of periodontal disease, has been demonstrated by epidemiologic data to be commonplace in dentate populations of all ages [3, 4]. The initial shift from healthy to plaque-induced gingivitis cannot be seen clinically [5], bringing up significant discussions over the clinical limits for differentiating between pathologic and normal inflammation. However, clinical signs and symptoms become visible as plaque-induced gingivitis develops into more advanced types of this disease. Plaque-induced gingivitis starts at the gingival margin and can spread to the remaining gingival unit. In the case of established forms, patients may have symptoms such as bleeding when brushing their teeth, blood in their saliva, gingival swelling and redness, and halitosis [5-7]. Clinical symptoms and indicators will differ in severity across people [3] and between sites within a dentition. Aside from bleeding, pain, and enlargement, erythema, edema, and bleeding are typical clinical indicators of plaque-induced gingivitis [2]. The structure of the tooth and root, restorative and endodontic considerations and other tooth-related issues affect the severity of plaque-induced gingivitis. In most cases, losing supporting structures in people with plaque-induced gingivitis cannot be detected by radiographic analysis or by probing attachment levels. The rete ridges' extension into the gingival connective tissue, blood vessel vasculitis next to the junctional epithelium, progressive destruction of the collagen fiber network with altered collagen types, cytopathologic changes in local fibroblasts, and a developing inflammatory/immune cellular infiltrates are all examples of histopathologic changes [4, 8]. Although current research suggests that the bacterial phylotypes linked to gingivitis and periodontitis differ, untreated periodontal disease will eventually affect the quality of life, according to OHIP-14 (oral health impact quality of life). The fourteen OHIP items—functional limitation, physical discomfort, psychological discomfort, physical disability, psychological

disability, social impairment, and handicaps—are broken down into seven categories [5].

Epidemiology

Epidemiology is the study of population health and disease and how biology, heredity, the physical environment, the social environment, and individual behavior affect these states. Our understanding of periodontal diseases has fundamentally transformed due to recent research advancements. The following tenets were part of the prevalent model for the epidemiology of periodontal diseases as recently as the middle of the 1960s: [3] All people were thought to be roughly equally susceptible to severe periodontitis; gingivitis often advanced to periodontitis, causing a loss of bone support and ultimately, tooth loss; periodontitis susceptibility increased with age and was the primary cause of tooth loss beyond age 35–55. Since the creation of this paradigm, improvements in our understanding of periodontal diseases have prompted a reevaluation of this disease model [3]. According to current knowledge, periodontitis does not demonstrate a linear progression and is not age-dependent.

Additionally, host vulnerability and risk factors significantly impact the disease's distribution and severity. Numerous epidemiological investigations of destructive periodontal diseases have been looking for correlations in the never-ending search for risk factors for these conditions. Identif ying risk factors for diseases, measuring the strength of those connections, and estimating the causality of those associations are the goals of analytical epidemiology [2]. Understanding risk variables can help physicians develop hypotheses of causation, which can subsequently be used to develop treatment procedures.

Risk factors

Identification of independent risk factors has been made possible by risk factor analysis and the statistical rescaling and stratification of populations to remove the impact of confounding variables. These different but changeable periodontal disease risk factors include behavioral factors, including smoking and alcohol use [8, 9]. In addition, they comprise illnesses and harmful circumstances such as osteoporosis, osteopenia, metabolic syndrome, diabetes mellitus, and low dietary calcium and vitamin D. These risk factors can be changed, and managing them is an integral part of the care for many patients with periodontal disease receive today. Genetic variables also influence periodontal disease, which makes it possible to target specific people for early identification and prevention [10]. It is obvious how hereditary factors play a part in aggressive periodontitis. Although it is strongly hypothesized that genetic variables (i.e., specific genes) may be linked to chronic adult periodontitis, there is currently no conclusive data in the general population to support this. Because genetic variables may help identify people susceptible to developing chronic periodontitis, it is crucial to continue studies to determine the genetic markers linked to this condition. Most individuals with periodontal disease treated in clinics and dental offices are likely to be affected by several of the systemic risk factors for the condition, including smoking, diabetes, obesity, and osteoporosis in postmenopausal women. As a result, identifying and managing risk factors has become crucial in treating patients with periodontal disease [11]. Periodontitis is associated with illnesses that produce inflammation in the body, including arthritis, COVID-19, and cardiovascular disease. Several systemic disorders, such as poor pregnancy outcomes6, type 2 diabetes, respiratory illnesses, pneumonia mortality in hemodialysis patients, chronic renal disease9, and metabolic syndrome, have all been linked to periodontitis. As a result of endothelial and microvascular dysfunctions, severe chronic periodontitis may be linked to the early stages of atherosclerosis. The low systemic inflammatory burden linked to periodontitis is the main factor supporting the biological plausibility of these associations.

Symptoms and signs

According to the disease's stage, the symptoms can vary but typically include bleeding gums when you brush or floss, bad breath, shifting or loose teeth, receding gums, red, tender, or swollen gums, plaque or tartar buildup on the teeth, pain when chewing, tooth loss, an unpleasant taste in the mouth, and an inflammatory response throughout the patient's body.

Treatments

In terms of infection management, lowering probing pocket depth (PPD) and increasing clinical attachment level (CAL), non-surgical (NSPT), and surgical periodontal treatment (SPT) are predictable procedures [12]. Data relating to tooth brushing behavior supported the idea that effective plaque control is essential for maintaining periodontal health [13]. According to an 11-year study, brushing your teeth reduces the number of teeth with periodontal pockets. The frequency of cleaning your teeth and the change in the number of teeth with PPD under 4 mm were correlated [14]. Daily interdental brushing or flossing was the least expensive preventive therapy and the most effective method for lowering plaque and gingivitis scores. The clinical effectiveness of scaling and root planning (SRP), which is regarded as the gold standard non-surgical treatment for periodontitis, has been thoroughly established by numerous systematic reviews [15]. Even severely damaged teeth can still be kept and given treatment. In about 45% of sites, plaque reduction and SRP may minimize gingival bleeding on probing. The type of tooth, the extent of periodontal disease, regional circumstances, patient age, and medical history could all affect how well SRP works. After SRP, non-molar teeth showed a greater PPD reduction than molars [6]. However, in patients with severe periodontitis, NSPT may not be enough to restore periodontal health [6, 7]. For SRP effectiveness enhancement, additional therapies such as lasers, antiseptics, systemic antibiotics, host modulators, and probiotics have been proposed [16]. Deep intrabony flaws were the principal indication for

regenerative procedures. Similar PPD reduction and CA gains were seen in guided tissue regeneration and enamel matrix derivatives. Additionally, the possibility of tooth loss may be increased by furcation abnormalities.

Conclusion

Gingival inflammation is caused by dental plaque (a microbial biofilm), and the extent and severity of the inflammation are influenced by various systemic conditions and oral factors at this stage. Furthermore, plaque accumulates faster at inflamed gingival sites than at non-inflamed sites, resulting in a complicated dynamic between the dental plaque biofilm and the host's immune-inflammatory response [17]. However, it should be noted that not all inflammatory sites will progress to periodontitis. However, no scientific evidence exists to date that allows us to determine which gingivitis sites are susceptible to progression to periodontitis. Thus, treating gingivitis with appropriate local therapeutic intervention is still necessary to prevent attachment loss and destruction of periodontal tissue. Gingival conditions may be diagnosed in the future using objective analytic approaches such as transcriptome characterization or epigenetic change categorization.

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