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Risk Assessment for Periodontal Disease - A Review

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Abstract: ***Periodontitis is a multifactorial inflammatory disease that affects the supporting structures of the teeth, primarily caused by specific microorganisms. This review article synthesizes the comprehensive information presented in the provided document, covering the definitions, risk factors, clinical assessments, and treatment implications associated with periodontitis. The article emphasizes the importance of understanding the various determinants influencing the initiation and progression of periodontal disease, including microbial, systemic, genetic, and environmental factors. The different contributing factors of periodontitis underscore the necessity for dental professionals to adopt a holistic approach in evaluating and treating patients.***

Keywords: Periodontitis, risk factors, determinants, markers, indicators, attachment loss, pocket

1. Introduction

Periodontitis is an inflammatory disease caused by specific microorganisms, leading to progressive destruction of the periodontal ligament and bone, with pocket formation or recession. The key distinction from gingivitis is detectable attachment loss. Clinical signs like color changes and bleeding may not always indicate attachment loss, but persistent bleeding on probing is a reliable sign of inflammation and potential loss. Periodontitis is multi-factorial, initiated by microbial plaque and influenced by genetics, behavior, systemic health, and tooth-related factors. (1) Periodontal diseases are caused by bacteria like *Porphyromonas gingivalis* and *Actinobacillus actinomycetemcomitans*. Local risk factors include pre-existing disease, deep probing depths, and plaque retention from faulty restorations. Systemic factors, such as poorly controlled diabetes, smoking, and neutrophil dysfunction, play a major role in progression. Managing these factors is key to prevention and treatment. Stress, osteopenia, and factors like gender, age, and heredity also contribute. (2) A thorough case history is essential for identifying risk factors, which then guide the creation of an appropriate treatment plan. This review article explores the role of these risk factors in chronic periodontitis.

2. Risk Factors

A risk factor is a characteristic linked to an increased likelihood of a disease, though it doesn't necessarily cause it. Risk factors can be modifiable, like environmental or behavioral factors, or non-modifiable, such as those intrinsic to the individual, also called determinants.(3)

1) Microorganisms as Risk Factors: The specific plaque hypothesis suggests that only certain plaque is pathogenic, depending on the presence or increase of specific microorganisms. These bacteria produce substances that contribute to the destruction of host tissues, leading to periodontal disease.(4) In gingival health, gram-positive bacteria like *Actinomyces* and *Streptococci* dominate the plaque. With poor oral hygiene over time, plaque matures, leading to a shift toward gram-negative anaerobes and motile organisms. Common bacteria linked to periodontal diseases include *Porphyromonas gingivalis*, *Prevotella intermedia*, *Bacteroides forsythus*, *Campylobacter rectus*, *Actinobacillus actinomycetemcomitans*, and *Treponema*

denticola. The mechanism states that these bacteria release leukotixin that kill human primate polymorphonuclear leukocytes and pheripheral blood monocytes, thereby evading the innate immune response. Leukotoxin mediated killing is related to the induction of apoptosis. [5] The fimbriae of *P. gingivalis* enable adherence to host cell receptors and induce bacterial internalization by activating the epithelial cell cytoskeleton. P.gingivalis fimbriae also modulate the production of proinflammatory cytokines such as IL-1b, IL-6 and TNFα.[6]

2) Tobacco Smoking as Risk Factor for Periodontitis: Smoking significantly impacts periodontal tissues, leading to less gingival bleeding, greater alveolar bone and attachment loss, and impaired wound healing. It reduces the effectiveness of both non-surgical and surgical treatments, though its effect on grafting and connective tissue healing remains unclear. While smoking's influence on bacterial populations in periodontal pockets and the gingival inflammatory response is uncertain, it may alter pro-inflammatory factors. Nicotine and smoke can also affect cells involved in periodontal repair, and the role of genetic polymorphisms in smokers' periodontal health is not yet fully understood. [7]

3) Diabetes as Risk Factor for Periodontitis: In diabetes, immune cell function is impaired, with neutrophil defects hindering bacterial killing in the periodontal pocket, while monocyte/macrophage hyper-responsiveness leads to increased proinflammatory cytokine production, contributing to periodontal destruction. Hyperglycemia inhibits osteoblastic proliferation and collagen production, leading to reduced bone formation and increased cell death, which contributes to more severe periodontal attachment loss in diabetic patients.[8] Elevated plasma and gingival crevicular fluid glucose levels in diabetics impair fibroblast attachment and spreading, hindering wound healing and tissue turnover in the periodontal pocket. In sustained hyperglycemia, proteins form Advanced Glycation End Products (AGEs), which accumulate in the periodontium, particularly on collagen, increasing cross-linking and stability, and impairing tissue turnover. AGEs activate the RAGE (Receptor for Advanced Glycation Endproducts) receptor, increasing proinflammatory cytokine production and oxidative stress in monocytes, leading to vascular permeability, thrombus formation, and chronic inflammation, contributing to atheromatous lesions in diabetic individuals.[9]

4) Puberty, Pregnancy, Menopause & Hormonal Contraceptives: Hormonal fluctuations in women, whether physiological or non-physiological, significantly impact periodontitis, especially in the presence of pre-existing plaque-induced gingival inflammation, with the gingiva being the primary target of steroid hormones. Estrogen and progesterone affect periodontitis by altering salivary peroxidases, stimulating collagen metabolism and angiogenesis, and modulating vascular responses and connective tissue turnover. These hormones also interact with inflammatory mediators, increasing inflammation during hormonal fluctuations and promoting cytokine release. Gestational hormones act as growth factors during pregnancy gingivitis, increasing *Porphyromonas gingivalis* (Pi) in subgingival plaque by satisfying its naphthoquinone requirement. Increased progesterone levels raise vascular permeability, enhancing the gingival response to bacterial pathogens and increasing exudate, while also affecting gingival keratinization by increasing epithelial glycogen and reducing the epithelial barrier. Hormonal contraceptives, which use synthetic gestational hormones, are linked to increased periodontitis, with higher BOP (Bleeding on Probing), PPD (Pocket probing depth), and clinical attachment loss in users; progesterone and estrogen raise vascular permeability and inflammation, highlighting the need to minimize plaque levels when starting oral contraception.[10]

5) Blood Disorders as Risk Factor for Periodontitis: Blood disorders can indeed be a risk factor for periodontitis, a multifactorial infection of the tooth-supporting tissues. Conditions such as anemia, sickle cell disease, hemochromatosis, and leukemias can manifest with oral and periodontal symptoms, increasing susceptibility to infections including periodontal disease. Blood disorders often involve chronic inflammation, exacerbating periodontal conditions like anemia of inflammation, which results in reduced erythrocyte survival and increased iron trapping in macrophages. Additionally, disorders affecting white blood cells, such as cyclic neutropenia, leukemias, neutrophil defects, neutropenia, Chédiak-Higashi syndrome, lazy leukocyte syndrome, agranulocytosis, and leukocyte adhesion deficiency, can compromise the immune response, making individuals more prone to severe periodontal destruction. Furthermore, periodontal infection can modulate the activity of bone marrow and affect the fate of hematopoietic stem cells, leading to systemic consequences[11]. Therefore, managing these systemic conditions is crucial for maintaining periodontal health.

6) Tooth Anatomic Factors as Risk Factor for Periodontitis: Tooth-related factors, such as tooth position, anatomy, and treatment, can predispose the periodontium to disease by facilitating bacterial accumulation or entry. These factors include root shape abnormalities, furcations, root concavities, developmental grooves, cervical enamel projections, bifurcation ridges, and occlusal discrepancies. Issues like root proximity, open contacts, tooth restorations, pulpal involvement, fractures, and external root resorption can lead to localized problems, contributing to gingival inflammation, attachment loss, and bone loss. These factors often require targeted treatment in otherwise healthy periodontium. [12]

7) Other Predisposing Factors as Risk Factor for Periodontitis: Other predisposing factors for periodontitis include margins of restorations, microbial tooth deposits, materia alba, food debris, calculus, improper contours [13] and contacts [14], restoration materials, the design of removable partial dentures, restorative dentistry procedures, malocclusion [15, 16], plaque retention, gingival trauma, toothbrush trauma, chemical irritation by drugs, and radiation therapy. Therefore, managing these systemic conditions and predisposing factors is crucial for maintaining periodontal health.

3. Risk Determinants

Risk determinants encompass a broad range of elements, including genetic predispositions, environmental exposures, lifestyle choices, and demographic factors. These determinants can either increase or decrease the likelihood of developing a disease. Understanding risk determinants is crucial in epidemiology for identifying vulnerable populations and formulating effective prevention strategies.

1) **Aging as Risk Determinant for Periodontal Disease:** Age is no longer considered a direct risk factor for attachment loss; instead, it reflects cumulative periodontal breakdown over time rather than an intrinsic age-related susceptibility to disease. As age increases, the periodontal ligament shows reduced cell density, irregular structure, and decreased mitotic activity.[17] With aging, fibroblasts in the periodontal ligament may shift to synthesizing type I collagen, along with increased elastic fibers and fewer epithelial rests. As age advances, the host response to bacterial plaque changes, with shifts in oral cavity conditions affecting microorganism attachment, growth, and metabolism. The plaque-driven classification of periodontal diseases is largely based on biological assumptions, lacking sufficient epidemiologic evidence, and doesn't fully account for factors like smoking, diabetes, socioeconomic status, and age.[17] Epidemiological studies show periodontitis is more prevalent and severe in older age groups compared to younger ones. With age, clinical changes in the oral cavity include loss of gingival stippling, increased gingival recession, more severe periodontal disease, and loss of alveolar bone and teeth. While gingival recession, epithelial attachment loss, and tooth loss may result from prolonged exposure to etiologic factors, intrinsic host changes with age likely also affect the host response, though this is not well-documented.
2) **Gender as Risk Determinant for Periodontal Disease**- Gender is a significant risk determinant for periodontal disease, with studies showing that men generally have a higher prevalence and severity of periodontitis compared to women. This difference is attributed to both biological and behavioral factors. Biologically, sex hormones like estrogen in women have immune-protective and anti-inflammatory effects, which can result in better periodontal health in premenopausal women. However, after menopause, the decline in estrogen levels can lead to an increased risk of periodontal disease in women, sometimes surpassing that of men. Behaviorally, men are often less likely to seek regular dental care and maintain good oral hygiene practices compared to women, contributing to the higher prevalence of periodontal disease.[18]
3) **Socio Economic Status as Risk Determinant for Periodontal Disease**- Socioeconomic status (SES) plays a crucial role as a risk determinant for periodontal disease, as it reflects various factors such as income, education, and occupation. These factors significantly impact an individual's access to healthcare, their health literacy, and overall well-being. People with lower SES often face barriers to dental care services, including limited access to preventive treatments and oral health education. This lack of resources and knowledge can result in poor oral hygiene practices, a higher likelihood of developing periodontal disease, and a greater severity of its progression compared to those with higher SES.[19,20,21]
4) **Genetic Factors as Risk Determinant for Periodontal Disease**- Genetic factors play a significant role as risk determinants for periodontal disease. According to research, variations in genes related to the immune response, such as interleukin-1 (IL-1) and tumor necrosis factor-alpha (TNF-α), can lead to heightened inflammatory responses to bacterial plaque, resulting in more severe periodontal issues.[22, 23] Additionally, polymorphisms in collagen genes (COLIA1 (Collagen type I alpha 1 chain) and COLIA2 (Collagen type I alpha 2 chain) can affect the structural integrity of periodontal tissues, making them more susceptible to bacterial damage. Variations in matrix metalloproteinase (MMP) genes can increase enzyme activity, leading to rapid deterioration of periodontal tissues. Studies have shown that individuals with a family history of periodontal disease are more likely to develop the condition, regardless of environmental factors. Understanding these genetic factors can help in developing personalized treatment strategies and early interventions for those at higher risk.[24, 25,)
5) **Stress as Risk Determinant for Periodontal Disease:** Psychological stress disrupts the bi-directional communication between the nervous, endocrine, and immune systems, downregulating immune function and impairing homeostasis. The link between stress and periodontal conditions was first noted with Acute Necrotizing Ulcerative Gingivitis (ANUG) in soldiers during World War I, suggesting that stress-induced lowered resistance could trigger inflammatory periodontal disease. Murayama[26] found that periodontal disease in 149 males aged 35-44 was linked to higher work stress and poor marital quality, while other studies observed gingival inflammation in military recruits with extreme personality traits or high physical stress levels.
6) **Race as Risk Determinant for Periodontal Disease:** The role of race as a risk factor for periodontal disease is complex. A study by Beck[27] and colleagues found that black patients had three times more advanced periodontitis than white patients in the same age group (above 65years). Further analysis showed that socio-economic status played a larger role for blacks, and P. intermedia was a risk indicator for blacks but not whites. However, when matched for socio-economic status, racial differences often disappeared in other studies.

4. Risk Indicators

1) HIV as a Risk Indicator for Periodontal Disease: HIV-positive patients and those with AIDS often develop a severe, painful, and rapidly progressing form of periodontitis called Necrotizing Ulcerative Periodontitis (NUP). The occurrence of NUP is linked to

immunosuppression, with a CD4 count below 200 cells/mm3, suggesting that altered cellular responses may increase periodontal disease by modifying normal regulatory mechanisms. In a 20-month study by Barr C et al. (1992), HIV-positive men with a CD4 count <200/mm3 had a 6-fold higher risk of 3mm or more attachment loss compared to those with CD4 >200/mm3.[28] Reviews suggest that periodontitis in HIV-infected individuals has a more common clinical and radiographic appearance than NUP, despite some uncommon organisms in the periodontal microflora. HIV infection may be a risk indicator for periodontitis, often manifesting as NUP, with variations depending on HIV stage, lifestyle, care, antiretroviral use, and other factors. Immune suppression from conditions like blood dyscrasias, hormones, autoimmune disorders, AIDS, and drugs can increase periodontitis risk, with RA showing a correlation due to shared inflammatory responses, though no direct cause-effect link is established. AIDS, caused by HIV, profoundly impairs the immune system, affecting CD4+ T-helper cells, monocytes, macrophages, and Langerhans cells, with oral lesions being common in both HIV-positive individuals and those with AIDS, though geographic factors may vary. Common oral lesions in HIV/AIDS include oral hairy leukoplakia, Kaposi's sarcoma, candidiasis, and lymphoma, while periodontal conditions may progress to linear gingival erythema, NUG, cancrum oris, and NUP.[29, 30,31]

2) Osteoporosis as Risk Indicator for Periodontal Disease: Osteopenia and osteoporosis are systemic disorders marked by low bone mass and microarchitectural deterioration, with osteoporosis defined by a BMD (Bone mineral density) 2.5 SD (Standard deviation) below normal and osteopenia by BMD between 1 and 2.5 SD below normal. Osteoporosis risk factors include non-modifiable factors like age, sex, and early menopause, as well as modifiable factors such as lack of calcium, exercise, smoking, and alcohol, which overlap with risk factors for advanced periodontal disease. A study of 41 dentate Caucasian women aged 20-78 found that maxillary alveolar process density was significantly related to mandibular density and systemic bone densities, with maxillary density declining with age. Studies on the relationship between tooth loss and BMD show mixed results, with some linking tooth loss to reduced systemic BMD, but the underlying causes of tooth loss are often unclear. A 2-year study found that osteoporotic women experienced more alveolar bone height and density loss than women with normal BMD, with estrogen deficiency linked to increased bone density loss, suggesting osteoporosis as a risk factor for alveolar bone loss in post-menopausal women with periodontitis. Studies suggest that osteoporosis, particularly postmenopausal osteoporosis, increases the risk of alveolar bone loss, especially in patients with pre-existing periodontitis, indicating low systemic BMD as a risk factor for periodontal disease progression. A direct link between skeletal osteopenia and mandibular bone loss in postmenopausal women suggests that systemic bone abnormalities may exacerbate bone resorption from periodontal infection, with periodontitis and osteopenia potentially sharing common etiological factors, warranting further study in larger populations with controlled variables. The relationship between osteoporosis and periodontal bone loss remains uncertain, with studies showing correlations between skeletal and mandibular bone density, but prospective studies are needed to confirm a causal link between osteoporosis and periodontitis.[32, 33]

3) Infrequent Dental Visits as Risk Indicator for Periodontal Disease- The role of infrequent dental visits as a risk factor for periodontitis remains controversial. One study found an increased risk of severe periodontitis in patients who hadn't visited the dentist for three or more years, while another study showed no significant difference in attachment or bone loss between individuals who did or did not seek dental care over a 6-year period. These differing results may be due to variations in the age of the subjects. Further longitudinal and interventional studies are needed to conclusively determine whether infrequent dental visits contribute to periodontal disease progression.[34, 35]

4) Drugs as Risk Indicator for Periodontal Disease- Calcium channel blockers, particularly Nifedipine, have been linked to gingival overgrowth, with prevalence varying by drug and influenced by plaque control and genetic factors, but no direct association with periodontal disease has been established. Phenytoin and Cyclosporin are known to induce gingival overgrowth, while steroid therapy, though linked to osteoporosis, shows conflicting results regarding its impact on alveolar bone loss. Evidence suggests an association between osteoporosis and alveolar bone loss, with treatments like calcium, vitamin D, estrogen replacement, and osteoporosis drugs potentially reducing alveolar bone and tooth loss.[36, 37]

5. Risk Markers

A risk factor that can be used to predict the future course of the disease which may include previous history of periodontal disease and bleeding on probing.

1) **Previous History of Dental Visit as Risk Marker for Periodontal Disease:** A history of periodontal disease is a strong predictor of future risk, with patients having severe attachment loss at higher risk for further progression, while those without periodontitis are at lower risk.[34]
2) **Bleeding on Probing as Risk Marker for Periodontal Disease:** Bleeding on probing is a key indicator of gingival inflammation, and when combined with increasing pocket depth, it becomes an excellent predictor of attachment loss, while the absence of bleeding indicates periodontal health. [34]
3) **Nutrition as Risk Marker for Periodontal Disease**: Vitamins are coenzymes required for metabolism and health. They have been classified as fat soluble (A, D, E and K) and water soluble (B & C). Vitamin A deficiency can cause dermatologic, mucosal, and ocular changes, with hypervitaminosis A leading to gingival erosions, ulcerations, bleeding, swelling, and loss of keratinization, as reported by De'menezes. Vitamin B complex deficiency can lead to oral changes such as glossitis, angular cheilitis, atrophy of lingual papillae, and oral ulcerations, although it doesn't cause specific oral changes. Vitamin C deficiency, leading to scurvy,

adversely affects collagen synthesis, resulting in periodontal changes like gingivitis, increased gingival sulcus permeability, and altered capillary permeability. Vitamin D deficiency causes osteomalacia, leading to periodontal ligament destruction, alveolar bone resorption, and fibrous dysplasia, while Vitamin K deficiency results in gingival bleeding and post-extraction hemorrhage. Deficiencies in trace metals like zinc, manganese, and copper impair immune function and wound healing, while severe protein deficiency affects host defenses, leading to gingival inflammation and periodontal bone loss, though no evidence supports altered resistance to periodontal disease.[38, 39, 40]

4) **Obesity as Risk Marker for Periodontal Disease:** Obesity is considered to be important risk factor for Type II diabetes, hyperlipidemia, and hypertension. Obesity disrupts metabolic and endocrine functions of adipose tissue, increasing production of hormones and cytokines like TNF-α and IL-6, which contribute to insulin resistance, inflammation, and altered immune function. Maulani C et al[41] found significant associations between obesity (BMI (Body mass Index) ≥25) and increased severity of periodontitis and attachment loss, with obesity being linked to higher odds of periodontal disease. Nishimura & Murayama[42] suggested that the pro-inflammatory cytokine TNF-α from adipose tissue in obesity may contribute to chronic inflammation, linking obesity to an increased risk of periodontal disease.
5) **Alcohol as Risk Marker for Periodontal Disease:** Excessive alcohol consumption has detrimental effects on oral health, increasing the risk and severity of periodontal disease through mechanisms such as impaired host response, liver toxicity, interference with protein metabolism, stimulation of bone resorption, and direct toxic effects on tissues. Tezal[43] et al. found that alcohol consumption >5 drinks per week increased the odds of gingival bleeding and clinical attachment loss, with the strongest effect on gingiva, though no significant link was found to alveolar bone loss or subgingival microorganisms.[44]
6) **Psychological Factors as Risk Marker for Periodontal Disease-** Selye's General Adaptation Syndrome explains that while the initial HPA axis response to stress is beneficial, prolonged stress can exhaust the body's ability to cope, leading to detrimental effects. Poor coping skills, such as anxiety or depression, can lead to neglecting oral hygiene, increased smoking, alcohol use, altered food intake, bruxism, and reduced salivary flow, all of which contribute to plaque accumulation and oral health issues. Prolonged physical or emotional stress has been linked to increased susceptibility to systemic diseases, delayed wound healing, periodontitis, and behavioral changes, though its significance is difficult to assess due to the multifactorial nature of these conditions.[45] Genco[46] et al. found that financial strain and poor coping skills were associated with greater attachment and alveolar bone loss, suggesting that effective coping may be more crucial for periodontal health than the stress itself. NUG, linked to psychosocial stress, anxiety, and depression, involves neuroendocrine changes and immune dysfunction, but its treatment relies more on controlling microflora than on addressing stress, suggesting stress has a limited role in periodontal disease.

Risk Assessment in Periodontal Disease

1) Periodontal risk calculator (PRC)[47],
2) Health information status (OHS), the Den Plan Excel/ Previsor Patient Assessment (DEPPA) [48]
3) The hexagonal risk diagram for periodontal risk assessment (PRA), model developed by Chandra [49]
4) The simplified method (UniFe) (Union of European railway industries) for periodontal risk assessment.[50]
5) Health Improvement in Dental Practice Model (HIDEP)[51]
6) Risk Assessment-Based Individualized Treatment (RABIT) [52]

6. Conclusion

Risk assessment is crucial in clinical dentistry, particularly for diagnosing and treating periodontitis. Evidence-based approaches guide clinicians in evaluating risk factors like bacterial pathogens, smoking, diabetes, and genetic factors, all of which contribute to periodontal disease. However, due to the multifactorial nature of periodontitis, determining definitive risk factors can be complex, as studies often yield conflicting results. A thorough patient history, including lifestyle factors and co-morbid conditions, along with the clinician's experience, helps in identifying risk factors and predicting disease progression. With modern risk assessment tools, including microbiologic, histologic, and biochemical markers, clinicians can better diagnose, prevent, and manage periodontal disease, ensuring more effective treatments and reducing recurrences.

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