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Prevalence and Severity of Periodontitis and its Correlation with Risk Factors among a Convenient Sample of Egyptians: A Cross-Sectional Study: A Mini Review

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Abstract

Periodontal diseases have proven to be one of the most prevalent oral diseases globally, in both developed countries and poor countries, especially periodontitis, which is ranked sixth among the most common human diseases, and more than 10% of the population. This makes this disease the focus of attention among specialists and researchers. Furthermore, periodontal diseases are linked to an increased risk of systemic conditions, including cardiovascular diseases, metabolic disorders, adverse pregnancy outcomes, rheumatoid arthritis, respiratory illnesses, and kidney diseases. Associations have also been drawn with oral and systemic malignancies. Understanding the epidemiology and characteristics of periodontal diseases in adults is crucial for devising effective prevention and control strategies. There is a lack of sufficient data in the periodontal literature regarding the prevalence of periodontal diseases among Arab and African adults. Furthermore, limited data exist on the prevalence of periodontitis in Egyptian population. This cross-sectional study aimed to investigate the prevalence of periodontitis in a sample of Egyptian population along with staging and grading of the disease, and its correlation with different risk factors, specifically smoking and diabetes mellitus.

Keywords: Periodontitis; Gingivitis; Smoking; Diabetes Mellitus; Stage; Grade; Localized; Generalized

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1. Introduction

Periodontal diseases arise from complex interactions between supportive structures of periodontium and diverse microbial communities residing in the oral cavity, creating a dynamic environment in which homeostasis can be disrupted [1-3]. These conditions span a spectrum that begins with mild gingival inflammation and can progress to severe destruction of the periodontal ligament and alveolar bone, often resulting in tooth loss if not managed appropriately [4-6]. The development and progression of periodontal pathology are driven by an interplay between an exaggerated host immune-inflammatory response and shifts in microbial ecosystem, commonly referred to as dysbiosis [7-8]. Advances in diagnostic criteria and refined classification systems have greatly enhanced our ability to assess disease severity predict treatment outcomes [9-11].

2. Periodontium and Periodontal Diseases

Periodontium is composed of gingiva, periodontal ligament, cementum, and alveolar bone, each of which plays a critical role in maintaining tooth support and function [1]. A detailed understanding of normal histo-anatomical features of these tissues establishes baseline against which pathological changes can be compared, highlighting significance of tissue integrity and vascular supply in maintaining periodontal health [1-5]. Disruptions in this finely balanced system, often initiated by microbial plaque accumulation, can lead to a cascade of inflammatory events that ultimately result in tissue breakdown and loss of support [3]. Transition from health to disease in periodontium marked by specific alterations in cellular architecture and extracellular matrix composition, which serve as foundation for subsequent diagnostic and therapeutic interventions.

3. Clinical Manifestations and Diagnostic Criteria

The initial clinical manifestation of periodontal disease is often gingival inflammation, characterized by erythema, edema, and bleeding upon gentle probing, which indicates an early immune response to bacterial biofilms [4]. As the condition progresses, deeper periodontal pockets form and clinical attachment loss becomes evident, leading to tooth mobility and, in advanced cases, eventual tooth loss [6-10]. Clinical assessments are typically performed through a combination of visual examination and periodontal probing, which help quantify probing pocket depth (PPD) and clinical attachment loss (CAL), two critical parameters for staging the disease. Digital intra-oral radiography further enhances diagnostic accuracy by providing clear images of alveolar bone levels and identifying bony defects that may not be detected through clinical examination alone [12-13] Together, these diagnostic modalities enable clinicians to determine the extent and severity of periodontal destruction, thereby guiding individualized treatment planning.

4. Histopathological Alterations in Periodontal Tissues

At the microscopic level, the onset of periodontal disease is marked by infiltration of inflammatory cells, such as neutrophils, lymphocytes, and plasma cells, into gingival connective tissue, which initiates tissue-destructive process [14-15]. Early histopathological changes include localized collagen degradation and subtle alterations in extracellular matrix, which precede visible clinical symptoms [15]. With persistent plaque accumulation, these early lesions evolve into chronic inflammatory states characterized by an increased density of immune cells and progressive destruction of connective tissue fibers [14]. In more advanced stages, histological examination reveals extensive breakdown of periodontal ligament, apical migration of epithelial attachment, and significant resorption of alveolar bone, correlating with clinical signs of deep periodontal pockets and tooth mobility [16-17]. These cellular and molecular alterations underscore importance of early intervention to prevent irreversible periodontal damage.

5. Etiopathogenesis: Microbial Dysbiosis and Host Immune Response

The pathogenesis of periodontal diseases is fundamentally linked to changes in the composition of the oral microbiota, where a shift toward a dysbiotic microbial community triggers an inappropriate host immune response [18-19]. In this context, even low-abundance periodontal pathogens can act as keystone species, significantly altering the host-microbe equilibrium and promoting a sustained inflammatory response [20-41]. The host immune system, in attempting to eliminate these pathogens, releases pro-inflammatory cytokines and enzymes such as matrix metalloproteinases (MMPs) which, although intended to control infection, also contribute to the degradation of periodontal tissues [4-21]. This vicious cycle of microbial challenge and host response results in chronic inflammation that underpins the progression of periodontal disease, highlighting the need for therapeutic strategies that address both microbial control and modulation of the host response.

6. Risk Factors and Systemic Influences

Periodontal disease is multifactorial, with genetic predisposition, systemic health conditions, and lifestyle behaviors all playing significant roles in disease susceptibility and progression [22-23]. Systemic conditions, particularly diabetes mellitus, exacerbate periodontal destruction by altering the host's immune-inflammatory responses and impairing tissue repair mechanisms, thereby accelerating the progression of periodontal breakdown [24-26]. Moreover, behavioral factors such as poor oral hygiene, stress, and suboptimal nutrition further contribute to the development and severity of periodontal disease [27-28]. Understanding these risk factors is essential for the development of preventive strategies and personalized treatment plans that target both local and systemic contributors to periodontal pathology.

7. Effects of Smoking and Alcohol on Periodontal Health

Tobacco smoking is a well-documented risk factor that significantly increases the incidence and severity of periodontal disease by impairing local blood flow, altering immune cell function, and enhancing the virulence of periodontal pathogens [29-31]. The toxic compounds in cigarette smoke induce vasoconstriction and reduce the oxygen supply to periodontal tissues, which not only compromises healing but also predisposes the tissue to infection and destruction [32-33]. Similarly, chronic alcohol consumption negatively impacts periodontal health by impairing neutrophil function, reducing the regenerative capacity of bone, and contributing to malnutrition, which in turn further deteriorates the periodontal environment [34-40]. These lifestyle factors, particularly when present in combination, can synergistically accelerate the progression of periodontal lesions, emphasizing the critical need for behavioral modifications in affected individuals [35-36].

8. Advances in Classification Systems and Diagnostic Refinements

Historically, periodontal diseases were classified primarily based on clinical parameters, with distinctions made between chronic and aggressive forms; however, this approach often oversimplified the heterogeneous nature of the disease [9]. Recent advances have led to the development of more comprehensive staging and grading systems that integrate clinical findings, radiographic evidence, and prognostic factors, thereby providing a more nuanced understanding of disease severity and progression [10-11]. These refined systems enable clinicians to categorize periodontal disease not only by the extent of tissue destruction but also by the complexity of management and the individual's risk profile, thus supporting a more personalized approach to treatment [10-37]. The continuous evolution of these classification frameworks reflects the dynamic nature of periodontology as a discipline that adapts to emerging scientific evidence.

9. Epidemiological Trends and Prevalence of Periodontal Diseases

Epidemiological studies consistently reveal that periodontal diseases are highly prevalent on a global scale, affecting diverse populations ranging from adolescents to older adults [2-38]. Variations in disease prevalence are observed across different geographic regions and socio-economic groups, which are influenced by factors such as access to dental care, oral hygiene practices, and cultural differences in lifestyle behaviors [39-42]. The public health

burden of periodontal disease is considerable, as these conditions are linked to functional impairments, increased risk of systemic diseases, and reduced quality of life due to tooth loss and compromised masticatory function [1-11]. Robust epidemiological data are therefore essential for informing prevention strategies and guiding public health policies aimed at mitigating the impact of periodontal diseases.

10. Conclusions

An integrated understanding of periodontal diseases spanning the anatomical, clinical, histopathological, etiopathogenic, and epidemiological dimensions provides a robust framework for improving diagnosis, prevention, and treatment strategies. The interplay between microbial dysbiosis and an overactive host immune response is central to the progressive tissue destruction observed in these conditions, while systemic factors and lifestyle behaviors further influence disease outcomes. Advances in diagnostic methodologies and the refinement of classification systems have enhanced our ability to predict disease progression and tailor treatment approaches, ultimately aiming to reduce the public health burden of periodontal pathology. Continued research and clinical vigilance remain essential for further advancing our understanding and management of these complex diseases. There is a lack of evidence regarding the prevalence of periodontal disease in Egypt through cross-sectional epidemiological studies. Additionally, there are only few studies in the periodontal literature that discuss the new classification of periodontal diseases and its prevalence among the Egyptian population. Given this, the present study was conducted as a hospital-based cross-sectional study with the objective of determining the prevalence of periodontitis disease, based on the new classification, among a sample of Egyptian adult population.

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