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Introductory Chapter: Periodontitis - A Useful Reference

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1. Etiology

The oral cavity is a unique part of the human body and is considered the window of general health. Gingivitis is primarily caused by bacterial colonization called dental plaque, and it is one of the common oral diseases that are preventable [1]. Gingivitis can be resolved with proper oral hygiene and professional intervention. Periodontitis occurs if inflammation of the gingiva is not intervened and progresses, which leads to an active destruction of the periodontal supporting tissues including the alveolar bone. Periodontitis can occur either as a generalized or localized form. It can be chronic or aggressive in nature. The World Health Organization (WHO) estimates nearly 15–20% of the adult population aged between 35 and 44 years suffers from an advanced form of periodontitis [2]. The Center for Disease Control (CDC) reported that approximately 50% of the general population in the USA is affected with some types of periodontitis [2]. Periodontal inflammation establishes as a sequela of a complex interaction of mixed oral pathogens (*Porphyromonas gingivalis, Tannerella forsythia, Treponema denticola, Fusobacterium nucleatum, and Streptococcus gordonii*) in the endogenous plaque or the biofilm, and their toxic by-products exert pathogenicity on host tissues. This disruption develops a shift in the bacterial ecosystem [3] from good health to disease. The ultimate result is bone deterioration and tooth loss in vulnerable patients.

There is an extensive list of local and systemic risk factors associated with the periodontal disease [4]. These risk factors include smoking, diabetes, medications, hormonal imbalance in women, immunocompromised status, etc. Most commonly, periodontal disease occurs when plaque [5] is allowed to build-up along tooth and under the gingiva. Genetic and environmental factors also contribute to the progression of periodontitis. In addition to the exacerbated inflammatory responses and other risk factors, another important consideration is adverse

effects from undesirable tooth movement. Improper application of forces on the healthy teeth such as parafunctional habits like bruxism (teeth grinding), malocclusion, or even during orthodontic therapies itself induces and provokes gingival recession, clinical attachment loss, and extensive bone resorption. Periodontal disease most often occurs without overt symptoms and is rather painless. As far as periodontal disease is concerned, frequent professional maintenance and excellent home care are the best prevention before initiation of the disease.

2. Oral and general health

The mouth serves as a diagnostic mirror for many systemic diseases. The dentist should be aware of all the associated systemic conditions that manifests in the oral cavity [6]. This assists both the clinicians and patients in early diagnosis, prevention, and better treatment results. In recent developments, a study on the pathological link between periodontal health and systemic ailments [7] invited more interest and follow-up among researchers and public health organizations. There are significant findings supporting the association between periodontal disease and systemic conditions [8] namely cardiovascular disease [9], cerebrovascular diseases, peripheral arterial disease, respiratory diseases, mental disorders, type 2 diabetes mellitus [10], digestive diseases, endocrine disorders, obesity, adverse pregnancy outcomes [11], renal disorders, and rheumatoid arthritis (RA). The risk associated with a bacteremia from a dental treatment prior to any invasive cardiac surgeries, organ transplants, and prosthesis implantations is being investigated. On the other hand, the optimization of dental health before high-risk surgeries and in patients with pre-existing systemic disorders like cardiovascular defects should be recognized and reinforced among patients and dental professionals. In recent days, there are numerous emerging clinical studies substantiating the links of chronic inflammation and predisposition to various cancers. The effect of chronic periodontitis on head and neck squamous cell carcinoma (OSCC) is of utmost priority in the world of research [12]. OSCC lesions have been reported to harbor very high levels of oral microbial colonies relative to contiguous healthy mucosa [13]. Hence, the importance of primary oral hygiene measures should be emphasized soon after the primary teeth erupts.

3. Role of immune system

Many of the latest studies enlighten a possible bidirectional link between the mechanism of periodontitis and metabolic diseases where both conditions could exacerbate each other. Various clinical studies determined any derangements in the equilibrium of immune system because of chronic inflammation of the periodontium that has a greater impact on the general body health of patients with this disease relative to those who are free of gum disease. It has also been postulated that periodontal inflammation can modify the host susceptibility to acquire other diseases. Experimental animal studies have shown elevated levels of inflammatory markers in periodontitis. A proper knowledge of immune pathogenesis during chronic periodontitis can be a reliable source for clinicians to apply and modify new therapeutic approaches. The

innate immune response is the first line of defense against invasion of pathogens. The blood leukocytes, neutrophils, and monocytes possess arsenal of receptors that enable the detection of invading pathogens and production of reactive oxygen species, cytokines, and chemokines. There occurs a disorder in the immune homeostasis, which leads to further increase in bacterial population and release of additional inflammatory mediators [14]. These inflammatory mediators elicit exaggerated immune response in the body, which subsequently favors the pathogens like *P. gingivalis.*

4. Host response to periodontitis

The interaction between host response and the dysbiotic microbial community is significant for causing periodontitis. The keystone pathogen, especially *P. gingivalis,* is known to influence the host directly by tissue damage and indirectly by stimulating and modifying the immune system to favor their survival and multiplication. In normal healthy status, the gingival epithelium [15] acts as the physical barrier against mechanical stress, exogenous substances, and pathogenic bacteria. When inflammation of the periodontium exists, *P. gingivalis* employs unique strategies to survive, sustain, and persist in the oral cavity, particularly in the antigen-presenting dendritic cells [16–18]. Hajishengallis discovered *P. gingivalis* surviving in the macrophages [19]. *P. gingivalis* causes inhibition of IL-8 production by the epithelial cells and impairs the protective functions of polymorphonuclear neutrophils (PMNs). During periodontal inflammation, the epithelial surface becomes ulcerated by proteolytic activity from the by-products of bacterial colonies, resulting in exposure of the connective tissues and blood capillaries to the bacterial biofilm, which allows further periodontal destruction and progression of the overall disease process. More studies are needed to specify how these pathogenic bacteria induced by biofilm affect the host immune homeostasis. Investigations should also focus on how bacterial dissemination to distant sites in the human system causes different conditions such as cardiovascular diseases, diabetes, rheumatoid arthritis (RA), macular degeneration [20], Parkinson, and Alzheimer's disease [21].

5. Treatment planning, outcome, and impact

A comprehensive periodontal diagnosis allows opportunity for appropriate treatment. A thorough assessment of the local cause (plaque), systemic factors, functional (presence of caries, occlusal status), and esthetic need (orthodontic therapy) induces an effective treatment plan. The chief goal of periodontal therapies is to control the prevailing infection and prevent further damage to the periodontium. Therapeutic control is accomplished with the least invasive methods and surgical treatments as needed. Nonsurgical strategies range from improvements in basic oral hygiene practices to professional scaling and root planning with antimicrobial medications. When treating severe periodontal destruction, surgical and regenerative therapies such as reconstructive tissue therapy guided tissue regeneration (GTR) with the aid of bioactive substitutes and bone grafts are indicated. A combined interdisciplinary

approach with orthodontic management and periodontal therapy is most effective in adults with severe periodontitis to restore the disordered function, compromised esthetics, and overall well-being. On the other hand, it is vital to identify, eradicate, or modify the causal factors and risk factors associated with periodontitis.

It is appropriate to advance our knowledge of oral pathogens, their impact on host immune response, and the cellular level modifications, which occur during normal health, disease, and periodontal management. The collection of chapters in this book will be a valuable guide in this regard.

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