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Potential Role of Parasitic Infection in Periodontal Disease

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Abstract

Dental plaque formation is linked to periodontal disease, a chronic, multifaceted inflammatory disorder. Microbial an imbalance has been connected to the pathophysiology and fundamental the cause of periodontal disease. Research is still being done to determine the causes of dysbiosis of microbes in the oral cavity. The structure of bacteria and how they create biofilms or microbial films on a variety of substrates, including the oral cavity, have been the subject of research. Protozoa are among the microbes found in human gingival bio films, according to recent studies. The article attempted to describe the possible role of parasite infection in the etiopathogenesis of periodontitis.

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Introduction

Periodontal disease is a multifactorial inflammatory condition that results from the complex interplay between microbial dysbiosis, host immune responses, and environmental risk factors. Microbial infection of the gum sulcus causes periodontitis. Bacteria are the main cause of periodontal disease, and the mouth cavity is home to a diverse microbiota composed of pathogenic fungi, viruses, parasites, and bacteria [1]. The infiltration of leukocytes to inflammatory tissue in the gingival region and their transit to the periodontal pocket canal are thought to induce both the breakdown of tissue and the growth of the bacteria found within the cavity. Inflammation is always present in periodontitis. It is challenging to determine each bacterial species' impact on gum disease, and the involvement of parasites in the development of the illness has been overlooked [2].

The pathophysiology of gum disease may be significantly influenced by parasites. The protozoa *Entamoeba gingivalis* (*E. gingivalis*) and *Trichomonas tenax* (*T. tenax*) are commonly found in periodontal pockets, according to research gathered across years. The possible involvement of these protozoa in oral infectious illnesses is unknown, even though they are significantly enriched in individuals with periodontitis [3]. Therefore, this review's goal is to describe how parasite infection may contribute to the etiopathogenesis of periodontitis.

Periodontal Disease

In addition to providing teeth with protection and nourishment, the periodontium is an interconnected system of tissues that holds teeth firmly to the alveolar area. The root cementum, periodontal ligaments, alveolar bone, and gingival complex make up the periodontium, according to Zalewska et al. [4]. Continuous inflammation of the gingiva and other periodontal tissues leads to periodontal disease [5]. The periodontal a ligament, cementum, and alveolar bone are among the oral tissue surrounds that are gradually destroyed by periodontal disease, a state of inflammation brought on by a dysbiotic the microbes [6]. Numerous risk factors, such as familial susceptibility, diabetes, tobacco use, tension, inadequate dental care, and particular drugs, can contribute to gum recession. Identifying those who are at elevated risk and putting therapeutic and preventive strategies in place to control the illness depend on an understanding of these contributing variables [7]. Dental patients

as well as professionals can collaborate to avoid and treat periodontal disease, thereby lowering the likelihood of missing teeth and related general wellness implications, by understanding how such variables interact. To decrease the effects of hazards and maintain dental wellness, an integrated strategy to periodontal care is necessary, including routine screenings, customized treatment planning, and patient instruction [8].

Etiopathogenesis of Periodontal Disease

The host-induced inflammatory process of the periodontal tissue, which is linked to imbalances in bacterial biofilms, is the hallmark of periodontitis, a chronic multifaceted condition that eventually destroys the tooth-supporting structure and causes a breakdown in periodontal connection (9, 10). Dental inflammation is caused by the formation of bacterial biofilms, but the development and course of periodontitis rely on dysbiotic alterations in the microbes in response to nutrients from gingival inflammatory and tissue breakdown products as well as antibacterial processes that attempt to manage the microbial difficulty within the gum sulcus area once infection has started. This triggers a number of important molecular processes, which in turn trigger host- produced enzymes that facilitate the apical emigration of the junctional epithelium, the disappearance of marginal ligament fibres, and the apical dispersion of the bacterial biofilm along the root surface [9]. Thus, dental pockets, gum bleeding, and the loss of periodontal tissue assistance, as seen by clinical loss of attachment and imaging-measured alveolar bone loss, are the main characteristics of periodontitis [10,11].

Dental Plaque

Dental plaque is a microbial biofilm that accumulates on the teeth and gingiva, which is the first stage of gingivitis. In the absence of treatment, gingivitis can advance to periodontitis, a condition characterized by deep periodontal pockets that reflect the illness and may result in the loss of teeth [12]. Interestingly, studies estimate that a human host may have up to 150 different types of microbes in their biofilm. In addition, human dental plaque contains over a thousand species. Research on the healthy residential microbiome using current DNA methods reveals an overabundance of Gram-positive microbes, despite the existence of periodontal pathogens [13]. This makes them symbionts, or obligate inhabitants of the subgingival and submarginal regions of the dental biofilm formation. Further spirochetes, viruses, protozoa and Gram-negative anaerobic bacteria are all possible causative pathogens [14]. Particularly, *Treponema denticola*, *Porphyromonas gingivalis* (*P. gingivalis*), and *Tannerella forsythia* are members of the red complicated collection of anaerobic microorganisms thought to be the effective bacterial species affecting the onset and progression of periodontal disease within the many different and extensive range of bacteria residing in subgingival biofilm formation. It is possible that microbial biofilm presence alone is insufficient for periodontal disease development. The disease develops when a microbial biofilm and its host have lost their equilibrium due to dysbiosis brought by the presence of "keystone" bacteria or an excessive response of the immune system in the host to the existence of microbes [15]. Moreover, inflammation is triggered when the host reacts to the typical residing plaque as it builds, which alters the microbial ecology and dysbiosis. An "ecological catastrophe" can be brought by a pathological malfunctioning of the immune response. An upward cycle of escalating dysbiosis feeds on it- self. In particular, the immune cell remnants (after apoptosis), the complete spectrum of host immune elements encompasses antibodies, component of complement, proteins of blood and inflammatory cytokines. Chemokines as well collagen breakdown products are elevated during inflammation. Furthermore, plasma discharge are present in inflamamatory area due to enhanced capillary permeability. There is an increase in anaerobic bacteria because an anoxic environment occurs in inflammatory situations. Therefore, given the new environmental circumstances, certain symbionts become pathobionts, i.e., commensals become potentially pathogenic due to an aberration in homeostasis. Pathobionts' proliferation such as *P. g.* might intensify provoke and exacerbate reaction to inflammation. The transcriptome investigation of subgingival microbes in periodontal disease demonstrated increase the transcription of genes encoding proteolytic agents, iron acquisition, and lipopolysaccharide synthesis; this strongly suggests that numerous saccharolytic, anaerobic, and Gram-negative bacteria in periodontal inflammation take advantage of ecological alterations for their nourishing and enormous requirements [16,17]. As periodontal inflammation worsens, the bacterial biomass of biofilms is associated with human periodontitis development. Thus, the dysbiosis and inflammation encourage one another, creating a vicious cycle that could continue periodontal inflammation and the disease's development [18]. The altered ecology then promotes the outgrowth of pathobionts, which might exacerbate inflammatory reactions and prolong the disease's course. In addition to the sub-gingival biofilm of microbes on the tooth root surface and and epithelium covering (a), genetic susceptibility and epigenetic variations (b), lifestyle-associated hazards (c), systemic illnesses (d), and other factors (e) have been identified as periodontitis-causing indicators [19]. Regarding the degradation of bone and connective tissue in periodontitis, the host response is thought to play a crucial role. Antigens of microbes and virulent elements trigger inflammation and immunological responses. These reactions include both innate and adaptive immune responses. Furthermore, there is individual diversity in response due to differences in cytokine and other antimicrobial responses and the individual's environmental circumstances and genetic predispositions. The host's reaction to bacterial invasion entails activating and stimulating various inflammatory cell types and resident tissue cells [20].

Parasitic Infection

Parasitic disorders are illnesses triggered by helminths and protozoa that affect human and/or animals. The human body can hold as many as 300 different types of parasites. Not all parasitic diseases have manifestations; some, like HIV/AIDS, might just come up when the immune system is compromised. Humans can serve as either a transitional or final host, where a parasite develops into an adult and engages in sexual reproduction. They can appear in several body areas, including the digestive, muscular, neurological, and defense systems, as well as exterior signs, such as emotions. Ingestion is the most typical method of transfer. Parasite disease is encouraged by eating uncooked meat, eating contaminated fruits and veggies, drinking contaminated water, and neglecting private sanitation guidelines. Numerous types of bacteria, fungus, and protozoa naturally reside in the mouth cavity, which frequently serves as an indicator of the health of the body. Lesion development occurs when the biological equilibrium in the mouth is disrupted [21]. Certain protozoa may communicate with host cells and cause inflammation in the oral tissue by encouraging the expression of inflammatory substances and the attraction of neutrophils, which helps to the development of periodontal disease, according to evidence collected over the years [22].

Protozoan Infection in Periodontal disease

The connection among protozoa and gum disease has been investigated in several nations. Recent research points to a complex

interaction between both protozoa and bacteria in periodontal disorders. The dysbiotic state caused by pathogenic bacteria could be associated with the existence of protozoa [23-26].

E. gingivalis Infection in Periodontal Disease

After that, the investigator looked at their virulence ability and infection techniques. According to the study, E. gingivalis entered the gingival tissue once the epithelial barrier broke, migrated around, and nourished on the host cells. E. gingivalis infected significantly raised the pro-inflammatory chemokine IL-8 in isolated gingival cell types. It has been discovered that the protozoan increases the production of collagenase enzyme in periodontal fibroblasts and inhibits cell growth. This implies that the hitherto underappreciated involvement of E. gingivalis in the growth of lethal forms of gum disease may be substantial. Research indicates that this parasite could be an indicator for oral disorders because it is particularly common in people with periodontal disease. Additionally, the incidence was also noted in the research, which is first report of *E. gingivalis* identification and genetic variants. To fully comprehend this amoebae's unique involvement in the pathophysiology of periodontal conditions, more research is necessary [27,28]. Another study [29] found that patients with diabetes had a higher percentage of this protozoal infection and that there was a clear correlation between the prevalence of parasites that cause gingivitis and periodontics and diabetes, hypertension, cigarettes, and cardiovascular disease.

Trichomonas tenax Infection in Periodontal Disease

Periodontal illness has been linked to the oral protozoa *T. tenax*. According to [30], individuals who had periodontal disorders showed a greater incidence of *T. tenax* than that healthy control. This outcome demonstrated a favorable association with periodontitis. This parasitic could have a role in the inflammatory process of gum disease, according to researchers. Trichomonads may play a role in the onset of periodontal disease, according to some preliminary research. However, more investigation is required to completely comprehend their precise role. Furthermore, no trichomonas was discovered in healthy locations; only diseased areas with significant bone loss had them. The protozoan was associated with conditions such severe attachment loss, thick calculus, and tooth movement. These findings imply a correlation among the activity of periodontal condition and the presence of this parasite [31,32]. Moreover, a study investigated the association between *T. tenax* and the severity of periodontal disease and diverse strains of *T. tenax,* with specific strains linked to more severe periodontal disease [31,33,34]. In turn, Bracamonte-Wolf et al.'s [35] study investigated the prevalence of this protozoan in patients with gingivitis and periodontal disease. There is a substantial correlation between the presence of *T. tenax* in normal and sick oral tissues, according to several research [33,34] that investigated the relationship between the microorganism and periodontal condition. Trichomonads were found to be more common in oral locations that were sick. Additionally, the study showed that parasite can create enzymes that degrade oral tissues, indicating a direct involvement in the onset and course of periodontal illness. Additionally, a study examined the relationship among different species of *T. tenax* and the severity of disease; certain strains were associated with aggressive severe periodontatitis [31]. The incidence of this protozoan in an individual with gum disease was also examined [35]. Over fifty percent of the study subjects had this protozoan, and the incidence was much greater in individuals with periodontitis than in gingivitis. The more severe the periodontal disease, the more *T. tenax* is present. Another intriguing aspect is that this parasite is not found in infants as well in individuals who are entirely edentulous, suggesting that it is linked to the existence of teeth [31,35].

E. gingivalis and *T. tenax* Co-infection in Periodontal Disease

The widespread presence of both protozoa in the mouth of patients with and without periodontitis was found in a prior study [36] that reevaluated the co-infection of both protozoa in periodontitis. *T. tenax* was discovered in two percent of patients with periodontitis and was linked to *E. gingivalis*, while *E. gingivalis* was detected in ten percent of patients with periodontitis but not in healthy controls. The authors noted that the overall incidence of these parasites may have been undervalued by direct examination, implying that the adoption of alternative methods could result in a larger prevalence rate. The results point to a potential link between *E. gingivalis* and periodontitis despite the study's limitations. Other research that found that those with periodontal conditions had a greater incidence of *E. gingivalis* than normal controls lend credence to this. The strongest proof for a connection between *E. gingivalis* and *T. tenax* and periodontitis was presented by [37]. Individuals with this disease had a substantially greater prevalence of both types of protozoa than normal individuals. This means that there may be a connection among these protozoa and the onset or advancement of periodontal illness. These results support the function of protozoa in oral oral hygiene and add to the increasing amount of evidence that indicates protozoa, in addition to bacteria, are involved in periodontitis. *E. gingivalis* and *T. tenax* have a significant association with periodontal disease, according to many reports. Research indicates that these protozoa are far more common in people with gum disease than in healthy subjects. Some research shows a greater connection with periodontitis, whereas other studies show a higher prevalence of gingivitis. Furthermore, various areas may have varying prevalence rates, indicating possible affecting factors like habits, socioeconomic level, and surrounding conditions. Nonetheless, a number of theories have been put forth even though the precise processes by which E. gingivalis and T. tenax cause periodontal disease are not entirely understood: (a) Because of their digestive enzymes and incurable characteristics, these protozoa may directly harm dental tissues; (b) suppression of the immune response may trigger a reaction of inflammation that aids in tissue damage; (c) *E. gingivalis* could contribute to the built-up of biofilms, which provide an environment that is conducive to the growth of other pathogenic bacteria; and (d) infection together involving both parasites may increase their potential for pathogenicity [38–40].

Protozoa and bacteria's relationship in periodontal disease

Numerous investigations assessed the intricate interaction among both protozoa and bacteria in periodontitis [41,42]. Scientists investigated *E. gingivalis's* distribution, frequency, and association to the subgingival microbial community in individuals with the disease. After evaluating more than 100 sites from sixty subjects, it was discovered that *E. gingivalis* was linked with *Porphyromonas, Treponema, and Tannerella*, and that its frequency and distribution raised considerably in locations with periodontal disease. The authors came to the belief that *E. gingivalis* can be considered an important agent in periodontitis since it is directly linked to microbial dysbiosis and cytokines in the GCF [41]. Further intriguing investigation [42], who found that individuals with periodontitis had protozoa and bacterial infections along with related medical features.

The disease was linked to the *T. tenax and E. gingivalis* and the periodontopathogenic bacteria. Furthermore, some of these bacteria were linked to *T. tenax* alone. Gingival oedema and elevated overall bacterial count in subgingival biofilm specimens were strongly correlated with *E. gingivalis*. This is explained by the reality that amoebae require bacteria that constantly divide to create an environment that is conducive to their growth. Hence, more research remains necessary to fully comprehend the relationship between bacteria and protozoa in this illness. There is substantial proof that both protozoa and bacteria interact intricately in the pathophysiology of gum disease. Bacteria may be shielded from host defenses by this internal habitat, which could allow them to survive and possibly become more virulent. The simultaneous presence of protozoa and bacterial species, such as pathogens from periodontal tissue containing *E. gingivalis*, indicates an interconnected related that drives the advancement of illness. *P. gingivalis*, *Treponema denticola*, and *Tannerella forsythia* are among the bacteria found in *E. gingivalis* in patients with periodontal disease, according to a study. According to this study, relationships among bacteria and parasites might change the gingival microbiota and result in serious periodontal conditions [43]. These discoveries have important ramifications for the comprehension and management of periodontal disease. Effective therapy may require the development of techniques that target both bacteria and protozoa, and evaluating the function of protozoa in sheltering and defending harmful bacteria can reveal information about the course and severity of disease.

Conclusion

There is a strong correlation with the incidence of periodontal disease and the existence of the two protozoa, according to various studies. Due to their invasion into periodontal cells that stimulation of inflammatory conditions, restricting proliferation of cells, and modulation of proteins involved in the breakdown of periodontal tissue, these parasites have a high potential for pathogenicity. There is compelling proof suggesting *E. gingivalis* and *T. tenax* are involved in periodontal disease. Developing successful preventative and therapeutic approaches will require a deeper comprehension of their interactions with microorganisms.

Conflict of Interest

Every author affirms that they have no conflicting interests

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