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Trichosoma tenax and *Entamoeba gingivalis*: pathogenic role of protozoic species in chronic periodontal disease development

Abstract

Periodontal disease is a complex inflammation/immune-mediated compromising of connective and epithelial tissues in dental periodontal ligament. Serving as a stabilizing and mechanical absorption system, periodontal ligament consist in a complex and organized structure presenting a really delicate balance with oral microbioma and immunomediated alterations. A large number of microbiological assays have been developed to understand, prevent and even stabilize an advanced disease form. Specific protozoic organism, usually not triggered in conventional microbiological assays, could not be evaluated and underestimated by the clinician. Their role, pathogenetic mechanism and agonist activity is far to be completely known. As a matter of fact, protozoic organism is still possibly involved in determination of chronical periodontitis and their knowledge is essential for a comprehensive overview in microbioma-mediated oral and gingival alteration. *E. gingivalis* and *T. tenax* are strongly associated with non responsive chronic periodontal disease. These pathogen organisms must be clearly and carefully identified and evaluated for a possible antagonistic spontaneous conversion. These conditions could be largely observed in unbalanced oral microbiome and patient with poor oral hygiene. Understanding prevalence, epidemiological aspects, pathological mechanism, therapies and role of hygiene therapy must be a fundamental knowledge of modern dental clinicians.

Keywords: *Trichosoma tenax, Entamoeba gingivalis*, periodontal

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Introduction

Approximately 5 to 20 percent of the world's population suffers from severe generalized periodontitis. Immuno-modulation, alteration in oral bacterial population and its related inflammation damage are clearly involved in the etiology. Despite this fundamental knowledge, a large amount of real pathways of this multi-factorial disease are still unclear. Periodontal lesions contain numerous neutrophils, motile bacteria, spirillae, spinning rods, and protozoa, multicellular highly pathogenic organisms. Nevertheless, there is accumulated evidence that periodontal diseases mainly develop in permissive hosts, who have local dysimmunity. Despite the fact that more experiments are needed, it can be hypothesized that one or several events may upset the balance of host– microbiome homoeostasis. A local dysimmunity may favour the development, selection and pathogenicity expression of periopathogens (including bacteria and protists), leading to a shift from 'eubiosis' to 'dysbiosis'. Recent literature findings estimate and subsequently strongly confirmed an evaluable linkage between oral periodontal lesions and systemic inflammation. This largely accepted relationship provides a remarkable knowledge necessity of every aspect in etio pathogenesis. Periodontal alteration was been often considered a microbial mediated disease. A possible parasites pathogenetical pathway must be discussed and investigated due to their habitual oral commensal finding.

Methods

A comprehensive review was made using NCBI and PUB MED data library. Keywords used for article selection were: Parasites, and/or *Trichosoma tenax* and/or *Entamoeba gingivalis* and Protozoa Periodontal disease. Inclusive selection criteria enclosed every paper made from year 2000 until 2018, included historical milestone paper and/or posters and/or grey literature findings. No country or language limit was applied.

Current status of knowledge

Protozoa: pathogenetic mechanism and body response

The possible role of parasites in the development of periodontitis and oral tissue damages has been poorly studied. The impact of parasites in periodontium pathophysiology is still debated and little information are available regarding the conditions favourable for their occurrence.[1–6] Reported clinical cases include a large number of lesion mediated by commensal parasites organism usually located in oral and oro-pharyngeal areas.[2–5] In a general view, parasites present a complex and sophisticated pathogenetic mechanism.[3] Parasitic infections present hard diagnosis, high chronicity, typical inflammation symptoms (fever, asthenia, weakness, chronical fatigue, abscessual and purulent lesions, tumor, dolor, rubor, calor and function lease localized in specific areas). Despite this, parasitic organism present highly complex hiding mechanism and classical systemical immunomediated recognizing and antigens elimination systems are often unsuccessful.[4,5] Global parasitic pathogenetic mechanism is mediated by various aspect: Target system/organ damages: Parasitic organisms are largely known for their often expressed organ-related pathogenicity (trophism). Direct damages are usually mediated by progressive tissue direct elimination, blood vessels damages, direct hemorragia, nervous alteration and organ specific damages. Tissue

degradation is often silent and could be underestimated by patient and clinician. Immunomodulated damage: parasites surface antigens leads to a inflammation bacterial derived response model. A large amount of immune modulator is functionally called to attend in antigen presentation. Interleukin and Cytokines are often reported to take a role, emphasizing the inflammation process and consequential chronicity. Supplies decreasing: Parasitic organisms often challenge with host for nutrition and life supplies. Thus compromising ideal and regular systemical and local functions.[1–7]

Analysis of protozoa mediated periodontal diseases, prevalence and clinical aspects

It has been shown that protozoa can both induce tissue damage and benefit from modified micro-environment.

Prevalence of protozoan mediated periodontal disease is still under debate. From 1992 a large number of authors discussed statistical changes and analysis in community and cohort studies, particularly in low healthcare status community and developing countries. *T. tenax* is currently considered as a member of the oral biofilm.[8–10] Its prevalence in the oral cavity ranges from 4 to 53% worldwide;[11] however, in patients with periodontitis; it is 3 to 4 times more than healthy individuals. *Trichomonas tenax* (*T. tenax*) is an anaerobic commensal of the human oral cavity. Several studies describe presence of *T. tenax* in patients with chronic periodontitis.[11–15] Transmission can be performed through saliva, air spray, and use of contaminated dishes and drinking water.[16] Worldwide, its prevalence in the mouth ranges from 4 to 53%.[16,17] *Trichomonas* spp. can be found in oral mouth and their pathogenic activities can present peculiar trophism with broncopulmonary tract, leading to a global increased attention level l.[1–17] Wantland examined 700 patients with periodontitis and found a prevalence of 26.5%.[18] Feki et al.,[15] in France reported a prevalence of 28% of *T. tenax* and 50.7% of *E. gingivalis* among 300 patients. The mean age was 30-32 years old for carriers of *E. gingivalis* and 45-54 old for those of *T. tenax*. Mahdi in Iraq examined the saliva of 143 patients with poor oral hygiene and reported a prevalence of 8.4% for *T. tenax* but later investigations showed that saliva was not a suitable media for detection of parasite.[15] Cambon et al.,[19] showed that neither the sex, maxillofacial anomalies nor smoking had any influence on parasite incidence and *E. gingivalis* was encountered in 85% of subjects free from paradonthopathy.[18,19] Age, social status, alcohol consumption, dental condition, and gingival pathology influenced the presence of *T. tenax*.[19] Suleyman et al.,[20] shown a peculiar dynamic agohonistic duo for Trichomonas and Entamoeba spp. In oral cavity. Cultivation of sampled *T. tenax* and *E. gingivalis* obtained from 38 periodontitis patients shown that *T. tenax* was present in samples from only 3 periodontitis patients. Both *E. gingivalis* and *T. tenax* were found together in the samples from 2 periodontitis patients. In total, 22 and 2 gingivitis patients were found to be infected with *E. gingivalis* and with *T. tenax*, respectively. Only 1 gingivitis patient was found to be infected with both *E. gingivalis* and *T. tenax*.[20] *T. tenax* is commensal. Its harmful conversion is opportunistic and may be relevant for immuno-suppressed patient or with immunal complications due to its peculiar oral/pneumo-elective trophism. Undercover prevalence undergoing over new studies can be of fundamental importance. Parasite mediated PD can be easily undiagnosed and they can be a possible cause in non responsive periodontal diseases. Parasites may evolve to grow and exacerbate the injuries strategies by genotype changes in oral cavity. A parasitic periodontal disease often occurs in tandem with bacterial alterations caused by an imbalance of the bacterial flora leading to a real opportunistic colonization. Recent studies have emphasized the ability of parasites to induce changes in some features of microbial communities. Opportunistic parasitic infections involves either man or woman aged in 40-50 by range with, debilitation, compromised systemical health, compromised immune system or immunodeficiency. Along with inflammation and the deepening of the periodontal pockets, the periodontal environment becomes more anaerobic, resulting in a bacterial shift from a Gram+ positive to a Gram-negative flora. This decrease in the partial pressure in oxygen may explain why the depth of periodontal pocket may be a critical factor for the anaerobic parasites colonization and growth.

3) Trichosoma tenax and *Entamoeba gingivalis*: role in periodontal pathogenetic pathways

Trichomonas tenax and *Entamoeba gingivalis* are largely reported as oral commensal. These pathogenic organisms were reported to be often related to periodontal disease and gum alterations and their identification in host tissues is often related to oral hygiene, global and immunal patient health, geographical factors and QOL (quality of life) status. This comprising water consumption, environmental pollution and water filtering systems.

Among the parasites found in dental plaque, *Trichomonas tenax*, an anaerobic motile-flagellated protozoan, may play a role in the pathophysiology of periodontal diseases. This 12–20 μm long and 5–6 μm wide organism is either ellipsoidal or ovoid in shape and has four anterior flagella of unequal lengths. *Trichomonas tenax* is a protozoan classified in the same genus as Trichomonas vaginalis.[19–23] *Entamoeba gingivalis* (*E. gingivalis*) is found in the oropharynx, but rarely in the head and neck lesions. This microorganism is commonly found in patients with poor dental health and oral hygiene, periodontal disease and immune suppression. It was the first commensal found in the human oral cavity. E. gingivalis can be usually observed on the surface of the teeth and gum tissues. Inflammatory process produces a propitious anaerobic environment for parasites growth. Pathogenic bacteria involved in periodontal host colonization and immune subversion use complement and toll-like receptor (TLR) bacteria –like signalling pathways. Parasites are recognized by TLR. Stimulation of the innate immune system via TLR4 by *T. vaginalis* has been reported. Similar mechanism within the periodontium was also hypothesized for *T. tenax*. Massive neutrophils recruitment found in human periodontitis can be explained by observation of patients infected by *T. vaginalis* in which a similar development of a predominant tissue recruitment of neutrophils can be identificated.[1–10] Moreover; these pathogens produce fibronectin-like proteins, responsible for tissue adhesion. Given this pathogenic property, host tissue disruption and lysis may be induced by *T. tenax* secretion of peptidases such as cathepsin B-like proteinases for matricial type 1 collagen and gelatine hydrolyses or haemolysins for erythrolysis. Recently Ribeiro confirmed the ability of the flagella to adhere to periodontal epithelial cells. After only 6 h, *T. tenax* in co-culture caused significant direct damage, disrupting some of the cells in the tissue, inducing membrane damage and cell apoptosis. In contrast, *T. vaginalis* has been found in the oral cavity but failed to elicit in vitro damage on periodontal cells.[10–20]

Clinical overview

Clinical evaluation and diagnosis of parasitic periodontitis may result difficult. A large number per year of parasites mediated periodontal diseases remain undiagnosed due to a real lack of

recognizable distinction and pathognomonic signs. These types of periodontal disease can show evaluable signs of a common aggressive periodontitis, a fast and unstoppable progressive damage of eptihelial tissues and connective structure of periodontal ligament. Suppuration, oedema, hyperaemia, erythema and lack of pain can possibly associate to these general signs.[23] A fast progression of damages, apical migration of gingival free margin and adherent mucosa as well as the delection of primary gingival cervical groove, can possibly be signs of parasitic progression, in absence of any primary systemical illness. Absence in specific pathogenic bacteria complex after microbiological assays can be possibly associated. This interpretation must be conducted carefully due to parasites' natural agohonistic activity that raises specific pathogens presence. Due to its fast immune mediated modulation and fast flogosis progression, a bone resorbtion can be observed in vertical dimension and may be consensual to a dramatic tissue apical migration. In patient with low oral hygiene adherence, presence of parasites can be globalized and substain, exacerbate or amplificate flogosis, diapedesis of Interleukin, TNF and immune modulation factors. As it starts as an aggressive disease, a parasitic periodontal disease could progress and become chronic. As they can't be processed as bacterial pathogens, parasites migrate in various sectors, rising inflammation and bacterial opportunistic proliferation. With these conditions, real causes of chronical periodontitis could be undiscovered and every clinical management may result uneffective or harmful. Furthermore *T.Tenax* and *E. gingivalis* can show peculiar and specific pathogenic activities with direct effect on connective and epithelial tissues in periodontal ligament.[1–23]

Clinical management

Due to its predominantly silent progression, therapeutically aspect of parasitic periodontal disease may be complex and very different. Most cases may remain undiagnosed or not clearly identified. A resistance to chemo antimicrobial therapy was often considered as a common aspect between many kind of parasitic induced periodontal disease.[15–23] Immunal response, systemical health and pharmacotherapy should be considered in every therapy. Clinical management begins with parasitic evaluation. A concern in literature reports was the method of parasite identification. The method most frequently used was direct microscopy, with or without staining (e.g. Giemsa). This method is time consuming and insufficient to discriminate parasite species, because *T. tenax* is morphologically close to T. vaginalis. Molecular methods are also been reported to be effective methods for *Entamoeba gingivalis* and *Trichomonas tenax* identification but more studies are needed to clearly understand incidence and prevalence of false positive/negative. In vitro culture is still largely used to really declarate a valid diagnosis. Mechanical causal therapy has been largely described in literature reviews and RCT. Non-surgical periodontal therapy (NSPT) was successfully described by May body in 2016. A Primary Goal in NSPT could be a quantitative decreasing of parasitic species, facilitating their non aggressive/commensal re-conversion.[23] After clinical diagnosis of moderate and severe non responsive periodontitis, patient in both sex were enrolled and underwent to plaque and saliva sample collection. Giemsa coloration was performed for *T. Tenax* and *E. gingivalis* identification. An ultrasonic scaler treatment was performed after CAL and PPD detection including charting. The frequency of *Entamoeba gingivalis* was reduced in saliva (p=0.007) and plaque (p=0.027) three weeks after the treatment. Likewise, the frequency of *Trichomonas tenax* reduced in saliva (p=0.030); however, the decrease was not significant in plaque (p=0.913). Results shown moderate statistical evidence supporting the efficacy of *standalone NSPT* in performing complete *T. tenax* and *E. gingivalis* reduction. Oral Trichomonas are sensitive to anti-parasitic treatment. Zvetkova et al.,[24] in 1988 investigated the clinical improvement and the evaluation of antibodies after anti-parasitic treatment with tinidazole in patients infected by oral *Trichomonas*.[24] After 3 days of treatment, oral clinical signs had disappeared, and titration of antibodies was normalized by months 6–12 after treatment. Tinidazole is also efficient against anaerobic bacteria involved in periodontal disease as described by Alou et al.[25] Evidence shown that both pharmacological and NSPT stand alone performed therapies were not considered effective. A dinamic-aghonistic duo should be considered in *T.tenax*, *E. gingivalis* related periodontal infection treatment. However, additional studies are needed to support this critical relationship.[24,25] A possible efficacy of probiotic enzyme linked therapy, should be considered due to its microbiome balancing capabilities. This could lead in a non-pathogenical devolution of parasitic species after repopulation of antagonist bacterial species. An adequate number of patients should be enrolled in RCT designed studies. Patient with diagnosed parasitic oral infections should be adequately monitorized. A punctual and precise recall plan must be always designed. First control is reported to be effective and usually hypnotizable within 30 days after a primarily mechanical treatment. In a practical therapeutically point of view, Diagnosis and therapy of any form of periodontal disease should always respect largely used and approved protocol, regardless to the inborn activity or the specie of a single pathogen. Oral cavity must be considered as a balanced environment and pathogen population as a single unit entity with dynamic linkage to every other system.

Conclusion

Compression of parasites pathogenic mechanism is essential for comprehensive understanding of periodontal disease. Aggressive initial form of PD and subsequent chronic manifestation are common signs of microbiomal changes in oral flora but a non responsive disease to common used therapy should alert clinicians. Hypothesis of parasitic infection should be adequately analyzed with common first level diagnosis assays. Use of probiotic, anti-parasitic therapy associated with mechanical hygiene therapy is reported to be effective and an eligible therapy for parasites mediated periodontal disease. Oral parasites represent a real issue and their comprehensive understanding is a fundamental topic for clinicians. Protocols for parasites eradication may be evaluated in hospital and clinical structures for hospitalized patients undergoing in long terms mechanical ventilation assisted therapies. Due to their common presence in oral microbiome, parasites should be known by the modern dental clinical and their role in periodontitis pathogenic mechanism should be adequately analyzed in future RCTs.

What is already known on this topic?

I. Parasites such *T. tenax* and *E. gingivalis* are frequently encountered human oral commensal.

II. Parasites can alternate oral microbiome leading to periodontal chronic flogosis.

III. *T. tenax* and *E. gingivalis* could possibly exacerbate a non responsive periodontal disease leading to a difficult diagnosis and hard treatment.

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What this study adds

I. A large number of authors agreed to encourage SRP and causal therapy for parasites mediated PD treatment.

II. A large number of authors agreed to encourage anti parasitic pharmacological therapy for parasites mediated PD treatment XXXX.

III. According to recent findings a comprehensive integrated both pharmacological and mechanical treatments have to be considered the eligible therapy for parasites mediated PD.

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Conflicts of interest

The author declares there is no conflicts of interest.

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