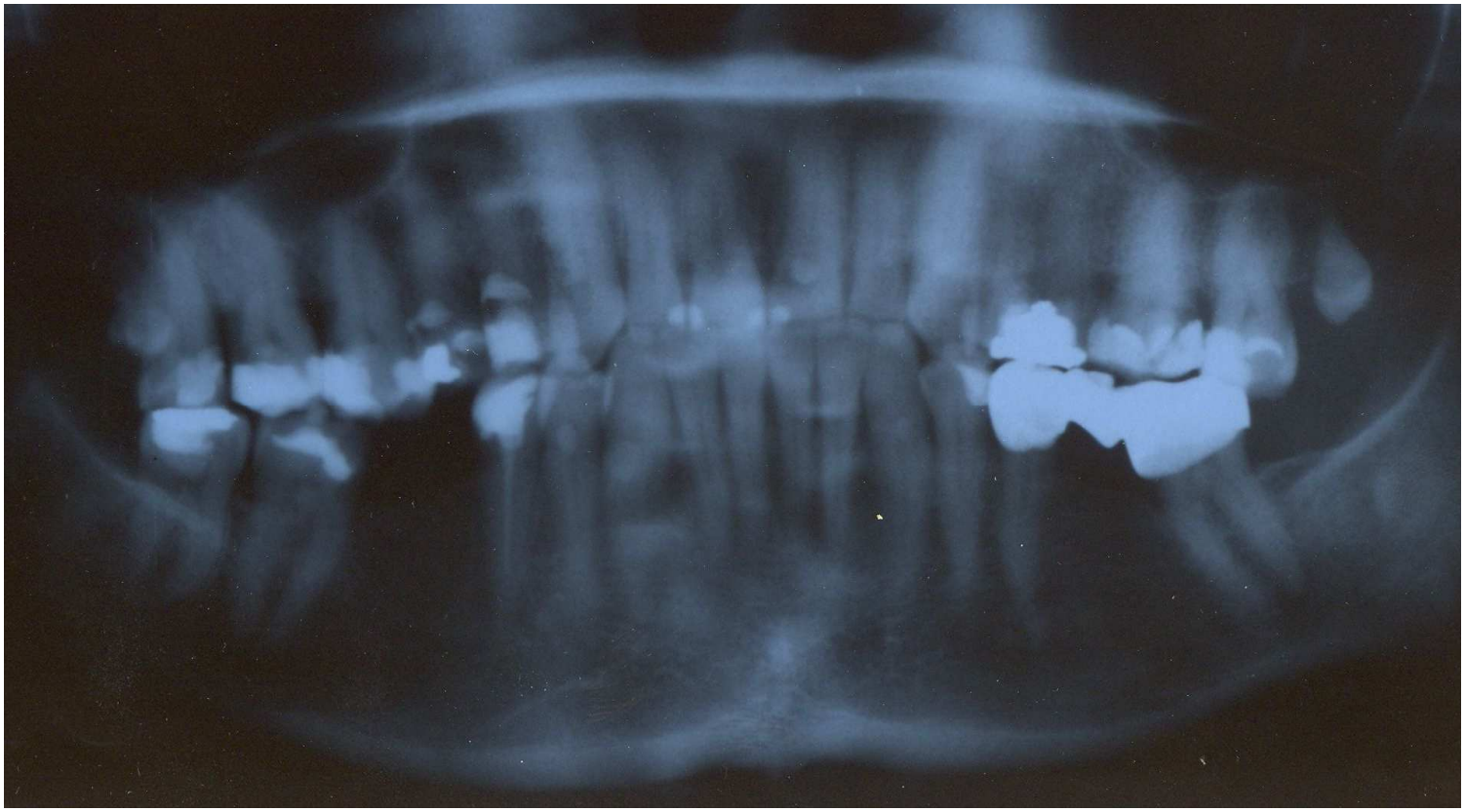
HEALTH AND DISEASES OF ORAL CAVITY



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Lifestyle and relationship with periodontal disease

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ABSTRACT

Lifestyle is composed of behaviors and habits that refer to the customs of individuals, some behaviors may be protective factors for health, while others can be harmful facilitating the development of some disease such as periodontitis. A risk factor is defined as that circumstance that increases individual changes of contracting an illness or any health problem. The prevalence and severity of periodontal disease varies according to social, environmental, systemic and oral diseases, particularly the situation of individual oral hygiene. Among the determinants for the presence of this pathology in young people are age, gender, stress, socioeconomic level and academic instruction. The severity of the disease increases with age, there is a higher prevalence in women linked to hormonal changes in the

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pubertal stage, stress period and has been associated with the low socioeconomic level characterized by poor hygienic and deficient dietary habits.

Keywords: Lifestyle; Periodontitis.

1. INTRODUCTION

Lifestyle and other factor can contribute in periodontal disease (PD). This is an endogenous microbial disease that damages the structure of the periodontium [1]. The disease derives from the cellular and humoral response of the host, altering the homeostasis of the periodontal tissues and causing inflammation and destruction by means of bacterial enzymes and virulence factors [2].

PD is divided in aggressive and localized Periodontitis. Aggressive periodontitis (AP) is a complex disease that promotes microbial alteration and cellular dysfunction in systemically healthy patients. It begins at any age and prevails in adolescents and young adults. It is characterized by rapid loss of adherence and bone destruction, inconsistent with the amount of microbial deposits present on dental surfaces in local or generalized form [3].

Localized Aggressive Periodontitis (LAP) begins at peri-pubertal age, with interproximal periodontal destruction in primary molar and in no more than two additional affected teeth [4]. The presence of dental calcifications on dental surfaces is not frequent; the tissues inflammation and bone-loss patterns are vertical and “U” in form [5]. Generalized Aggressive Periodontitis (GAP) affects more than three teeth in addition to the primary molars and incisors, and it presents loss of interproximal insertion in persons aged <30 years and episodic destruction of alveolar bone [6].

2. ETIOLOGY

This is a complex oral disease that possesses four principal risk factors: subgingival microbiota, individual genetic variations, lifestyle and systemic factors. All factors are play an important role in development of periodontitis.

2.1. Subgingival microbiota

The composition of the oral microbiota is influenced by factors how: temperature, pH, atmosphere, host defense and host genetics. Bacteria are responsible for stimulating the host response which defines tissue changes caused periodontal lesions. When bacteria work in association within a glycocalyx forming a biofilm, which allow microorganism to join and multiply on different surfaces. The biofilm protects microorganisms from toxic substances in the environment, it's also facilities the uptake of nutrients, the cross-feed, the elimination of metabolic products and the development of an appropriate environment with suitable physicochemical condition for their growth. In the periodontal surface it's not an exception because is associated with multiple oral microbiota in gingivitis and periodontitis phases. In periodontal disease in particular exist three bacteria with participation active in initiation and progression of this damage: *Aggregatibacter* (=*Actinobacillus*) *actinomycetemcomitans (Aa)* and *Porphyromonas gingivalis (Pg)* and *Tannerella forsythia (Tf)*. These bacteria, due to the action of their virulence factors: immunosuppression factors (inhibit blastogenesis, antibody production and activate t-suppressor cells, lipopolysaccharides, antimicrobial resistance, leukotoxin, killed PMN and monocytes, resistant to complement-mediating killing, gingipain (collected nutrients for the *Pg* to survive), evasion of the host defenses and immune response, fimbriae, apoptosis-inducing activity, production of methylglyoxal and trypsin-lyke protease. These bacteria were called major pathogens or complex red in periodontal area [7].

2.2. Genetic variables

These are associated with biological or endophenotypic intermediaries, which have the potential to modify the host barrier function, inflammatory responses, and microbial colonization patterns. PD comprises a group of distinct conditions, with similar clinical and superimposed presentations in which each of these is influenced by human genetic variation. A non-protective inflammatory response presents that interacts with the biofilm of the dental surface, generating dysbiotic microbial changes and the establishment of the clinical disease [8].

Hereditary autosomal recessive mechanisms have been related to the appearance and progression of periodontal disease. In PD, there are interindividual differences in the degree of production of inflammatory cytokines, such as interleukin (IL)-1, tumor necrosis factor alpha (TNF-α), and prostaglandin E2 (PGE2), following a stimulation due to a leukocytic endotoxin. In addition to the presence of the genetic polymorphism associated with the

differences in the interindividual production of IL-1 and TNF-α, there are variants associated with AP, such as the *IL-4* gene [9]; this cytokine stimulates the production of B lymphocytes, Immunoglobulin G (IgG), and Immunoglobulin E (IgE) antibodies, and differentiation into T cells inhibits the inflammatory response of the macrophages and the production of IL-1. The polymorphism present in the receptor gene of vitamin D is related with bone density; thus, it is associated with LAP [10].

2.3. Immune response in periodontal disease

The physiopathology of PD is poorly understood, oral inflammation is relatively rare, usually is present in the tissue around the teeth, because of the supra and subgingival plaque stimulus; resident Dendritic Cells maintaining the immunological homeostasis instead bacterial presence. With the purpose to stablish an immune response, a group of molecules called Toll-Like Receptor (TLR) which are transmembrane proteins expressed in macrophages and dendritic cells and also in mucosal cells; those receptors are associated to damage/danger molecular patterns, they recognize molecules in intra and extracellular pathogens. It was demonstrated that TLR2 is expressed in Periapical granulomas and Periapical cyst and TLR4 is overexpressed leads to NF-β expression and translocation, as consequence, inflammatory and adaptive immune response against oral microbial are induced [11].

On other hand, Langerhans cells (LC) in the mucosa, are considered the first cells to sample antigens (Ag) from the biofilm and elicit regulatory or proinflammatory response [12]. The continuous stimulation of plaque and Antigen presenting cells activation, promote the specific repertory of immune cells, in this way the role of CD4+ Th cells, Th1, Th2, Th17 or iTregs in soft tissue and bone destruction is should be related to cytokine expression as IL-10, IL-17, IFNγ and RANKL, that influence Th1 phenotype, osteoclast activation and damped iTregs [13]. Considering this cytokines network and the role of LC, *P. gingivalis* antigens recognized and processed by LC, it seems that polarized the immune response to Th17 influenced by LC [14]. Also the expression of IL-1α and IL-1β induced in gingival epithelial cells by *A. actinomicetemcomitans* extracts, triggers inflammatory mediators and expression of IL-18 [15]; Both cytokines IL-1α and IL-18 improve the inflammations associated with the immunomodulatory effects of β-glucans and its antimicrobial activity of the immune cells and mediators [16, 17]. The expression of immunological receptors in gingival fibroblasts because

the interaction with bacterial components in the dental plaque, and the constant mechanical stimulation, maintains the secretion of IL-6, IL-8 and IL-1 producing the chronic inflammatory response [18].

In chronic periodontitis, some others factors related with the immune regulation are involved in periodontal disease, some of them is the stress hormone called cortisol, this hormone is important, because of the regulation through the hypothalamic pituitary adrenal axis, cortisol participate in the recruitment of immune cells, promotes the inflammatory response and induce an imbalance between Th1/Th2 with the subsequent polarizations to Th2 cells [19].

Besides the cell mediate immune response, higher levels of serum IgG to *P. gingivalis, A. actinomycetemcomitans and Prevotella intermedia,* favors an adequate immune response, in contrast to lower levels of IgG to *T. forsythia, Treponema denticola* and *Fusobacterium nucleatum*, indicating that these microorganism are poorly immunogenic, suggesting increased risk for periodontal disease progression as compared to the first group of periodontal pathogens [20]. Finally it will be considered, that antimicrobial peptides expressed in response to oral bacteria or bacterial components should be used as a biomarker for the diagnosis, progression and risk development of periodontal disease, matrix metalloproteases 8, 9 and 13, are related to gingival and alveolar bone degradation, receptor activator for nuclear factor κB (RANKL) and decreased expression of osteoprotegerin induce osteoclastogenesis activation, IL-1β, IL-6 and TNFα are considered in periodontitis, as a promotors of inflammatory, rather than bacterial presences, and should be determinant to periodontal destruction, in accordance with this findings, used as a tool, all of this biomarker it will help for the diagnosis and prognosis in periodontal diseased patient [21].

2.4. Lifestyle and periodontal disease

Life styles can be understood to be a sum of actions and behaviors that an individual develops towards a form of good health or bad health, including aspects relating to drug use, physical exercise, nutrition, sexuality, leisure activities and stress control [22]. Life styles are determined by the presence of risk factors and/or factors that protect and lead towards wellbeing. This is why they should be seen like a dynamic process that is not only formed by an individual's actions and behaviors because they are also actions based on a social nature. A

risk factor is defined as those circumstances that increase the individuals' possibility of obtaining a disease or any type of health issue [23].

Life style is not a vague concept that can be modified voluntarily it is closely interrelated with life conditions, therefore it is not just a simple decision of the individual. Since limits exist for open options for the individual because of environmental causes, social media and economic media, as well [24]. This is how some actions can be protection factors towards health, while others can be harmful facilitating the attainment of many diseases, like what could be of a dental type or specifically a periodontal disease [25].

The prevalence and severity of periodontal disease varies in function of the risk factors, where you can find actions, life styles, systemic, amicrobic, philological, psychosocial, family, sociodemographic and those related with the individuals' dental hygiene [26]. Among the predetermining factors that lead to the presence of this pathology specifically in young populations, are the modifiable and the non-modifiable [27]. The modifiable can be intervened with or controlled to reduce the risk of initiation or progression of the periodontal diseases. For example: the action factor and life styles that include tobacco alcohol or drug use, stress management, obesity, low social economic level, and the level of educational orientation [28].

On the other hand, the non-modifiable factors or are generally intrinsic to the individual, which is why there are non-controllable. For example: genetic characteristics, family aggregation, among others [28]. To that affect, it has been observed that the severity of the disease is increased with ageing, and in the case of gender there exists more prevalence in women related with hormone change in the puberty stage [29, 30]. It should be noted that each and every one of the factors included continuously was reviewed and analyzed thoroughly. Here provided below are some of the risk factors of actions and life styles:

2.4.1. Smoking

The use of tobacco is considered the principal risk factor for the affection of the prevalence and progression of periodontitis, in which the severity depends on the doses of consumption [31]. The effects that provoke tobacco use are represented by the formation of dental bacterial plaque and the inflammatory response of the diseases progress. The physical pathological effects are due to the harmful actions of the nicotine, the smoke and carbon monoxide that result from the incomplete combustions favor a serious of molecular events [32].

The number of cigarettes and years of use increase the severity of periodontitis, less response exists to periodontal therapeutic treatment and its use increases the loss of teeth settings and it promotes osteoporosis that of the alveolar crestal height supports [33]. There exists enough evidence that demonstrates the very close relationship that exists between tobacco use in periodontal disease [34]. Such is the case of authors Zini, Sgan and Marcenes [35] who found that smoking exerts a substantial destructive effect on periodontal tissue an increases the rate of progression of the periodontal disease. It has also been found that the hosts' response can be modified and provoke a proliferation of bacteria of the dental plaque [36].

Smokers with periodontal disease apparently seem to show less signs of clinical inflammation and gum bleeding in comparison with nonsmokers. This could be explained by the fact that nicotine exerts local constriction of blood vessels, reducing blood flow, the edema and clinical signals of inflammation. Nicotine acetylcholine receptor has been found to play an important role in the development of nicotine related periodontitis [37].

In Table 1 the signs symptoms and changes of the periodontal tissue attributed to tobacco use are summarized.

Table 1. Findings from smoker patients with periodontal disease

Parameter	Smoker patients
Gum bleeding	Less gum bleeding and higher amount of small blood vessels
Bone height	Greater bone lose
Level of Insertion (NI)	Greater lose of (NI)
Sondeable depth	Greater sondeable depth
Number of lost teeth	Number of lost teeth

Source: Adapted from Rivera-Hidalgo [38]

On the other hand, it has also been compared that when the smoking habit is suspended favorable changes can occur on the immune system against germ attack. It has been reported that when the habit is suspended it can holt the periodontitis progression and better the results of the treatment and the periodontal prognostic. The periodontal state of the patients that were smokers and that currently are not is intermediate among those that have never smoked and current active smokers; in other words adopting a healthy life style like leaving the smoking habit has shown to positively affect the periodontal state [39] .

2.4.2. Alcohol

Alcoholism is related to the harmful effects over the hosts' response. The consumption of alcohol could have significant impact over hemostasis on the periodontal bacteria and the hosts' response [40]. Abusing the use of alcoholic beverages during a long period of time is related to the origin, severity and evolution of gum and periodontal disease with an even higher probability of attainment, in relation with non-alcoholic individuals. The production of periodontal pathologies in the alcoholic patient is based on criteria over the effect of alcohol on the tissue. For example, alcoholic patients show an altered immune response, alcohol has a toxic effect over the liver causing alterations of the coagulation mechanisms. Those individuals that are classified as conspicuous smokers' frequently present nutritional disorders, and with that resulting in protein and vitamin deficiencies [41].

Other risk factors for periodontitis pathologies in alcoholic patients is a deficiency in oral hygiene due to an overall lack of personal hygiene and from the low saliva flow or xerostomia as a consequence of the morphological and functional alteration of the glandules due to an ethanol effect. Alcohol produces epithelial atrophy in the oral mucosae, it increases permeability of mucosae increases the solubility of the toxic substances like those derived from smoking [42].

2.4.3. Drug-induced disorders

Another important factor in the appearance of periodontal diseases there are found disorders caused by the consumption of drugs which produce a decrease salivary flow among which antihypertensive, narcotic analgesics, some tranquilizers and sedatives, antihistamines, and antimetabolites [41]. Other drugs in particular those in liquid or chewable forms that contain added sugar. They alter the Ph. and the plaque composition. Also documented is that drugs such as anticonvulsants, calcium channel blocking agents, and cyclosporine may induce gingival overgrowth. It has been demonstrated that young patients have an excessive response to drugs due to the higher level of androgens in blood levels [43].

2.4.4. Stress

Regarding stress, it has been found that it is a risk indicator for the development of the periodontal disease; the effects to the response of the organism to anxiety, depression,

cognitive alteration and self-esteem alteration are what causes distortion in health conducts therefore the incorporation of negligent practices of oral hygiene and formation of bruxism. Also depression is a stress indicator and is related to tobacco use, alcohol and intake of an insufficient diet, hence forth provoking the increase in the susceptibility of the patient to infection due to bacterial development [44].

Patients with inadequate behavioral strategies on stress (defensive adaptation) are in greater risk of severe periodontal disease [45]. Stress is associated with an increased risk of glucocorticoid secretion that can depress immune function, increased insulin resistance and potentially, periodontitis. Studies have found some periodontal disease indicators such as tooth loss and gingival bleeding to be associated with work stress and financial strains [46].

2.4.5. Obesity

Obesity is multifactorial chronic disease considered the most commune nutritional disorder in America, therefore a risk factor for many systemic diseases. Chronic inflammation has a multidirectional relationship with obesity and chronic periodontitis, among other diseases. Furthermore several explanations for the association between obesity and periodontal disease in younger adults have been provided. Younger people may have different dietary patterns than older study participants [47].

Research in dietary trends in adolescent's ages from 11 to 18 reveals a significant decrease in raw fruit and non-potato vegetables, which are sources of vitamin C. In addition, adolescents have decreased their calcium intake and increased their intake of soft drinks and non-citrus juices. This is important to oral health because low dietary intake of calcium and vitamin C has been associated with periodontal disease. People who consume less than the recommended dietary allowance (RDA) for calcium and vitamin C have slightly higher rates of periodontal disease [48].

3. EPIDEMIOLOGY

There are reports in the literature that AP affects 47.2% of the U.S population; the prevalence of AP in adolescents has been estimated at between 0.1 and 2%. Other studies whose objective was to determine periodontal disease in young population found a prevalence in persons aged between 13 and 20 years of <1%, while in adolescents between the ages of

15 and 17 years, the prevalence was estimated at 0.2% for Caucasians and at 2.6% for Blacks. Similarly, greater prevalence was found in women than in men, and <1% of the population aged under the age of 30 years had AP [49].

The prevalence of LAP in European population varies among adolescents and young adults between 0.1 and 0.2% [50]. In industrialized countries, it was found that LAP affects primary dentition in children aged between 5 and 11 years, with a frequency ranging between 0.9 and 4.5% [51, 52].

4. DIAGNOSIS

Periodontal clinical parameters are as follows: Probing Depth (Pd); Level of Clinical Insertion (LCI), and Bleeding on Probing (BOP). Pd is the space than can measure between 1 and 3 mm in the absence of clinical inflammation; a periodontal pocket is defined as the pathological depth of the depth of the periodontal groove, rendered by bone loss and periodontal insertion. For practical clinical effects, a periodontal pocket represents one of the cardinal signs of periodontitis, given that it is produced by the loss of insertion, and can be considered as such from 4mm. The National Informatics Centre (NIC) makes reference to the fibers of the connective gingival tissue that are inserted into the radicular/root cement through the Sharpey fibers. In the clinical ambit, NIC is utilized to refer to the magnitude of support loss, but it depends on the particular radicular length of each tooth.

BOP is the main predictor of periodontal disease and is induced by penetration of the periodontal probe. It should be interpreted in a global manner, because its presence is not absolutely indicative of disease, while its absence is indeed a reliable indicator of periodontal health.

4.1. Radiographic bone loss

Radiographically, periodontal bone pathology presents loss in the continuity of bone and cortical crests, loss of bone height, formation of bone defects, and periodontal ligament enlargement and furcation. Bone loss severity is classified according to the distance from the Cemento-Enamel Junction (CEJ) to the tooth apex: cervical or mild; medium or moderate, or apical or severe.

4.2. Diagnostic alternatives

Different complementary diagnostic alternatives to the clinical diagnosis: the use of immunoproteomic approaches implied in the immune response. There is a wide variety of potential proteomic periodontal markers that are included within the immnunoproteome: from immunoglobins to bone remodelation proteins. Immunoglobulin M (IgM) is a natural antibody that can bind specific antigens to those to which the host has never been exposed, and it presents traits that allow it to bind to antigens to the degree of invasion, resulting in the activation of the complement as a mechanism of first-line defense, participating in early recognition of bacteria in periodontal disease [53].

C-reactive protein (CRP) is a plasma protein that reflects a measurement of the acute-phase response to inflammation, and is one of the markers-of-choice in the follow-up of this response. It is a recognition molecule of patterns that bind specific molecules that are produced during cell death or that are found on the surfaces of diverse bacterial pathogens. The rapid increase of CRP synthesis during the first hours of the progression of an infection suggests its contribution to defense of the host as part of the innate immune response. CRP is produced in response to many types of distinct lesions of periodontitis, which is found regulated by diverse cytokines. The changes in the cellular and molecular compartments of peripheral blood can be found in patients with periodontitis due to inflammatory changes in the periodontal tissues [54].

5. TREATMENT

In PD, conventional mechanical therapy and oral hygiene is not sufficient to control the disease. The use of broad-spectrum antibiotics, such as amoxicillin/clavulanic acid, metronidazole, clindamycin, ciprofloxacin, tetracycline, and azithromycin are efficient in pharmacological treatment, in addition to treatments such as surgery, laser therapy, and photodynamic therapy [55].

Photodynamic Therapy (PDT) is a non-invasive tool that functions from the generation of free radicals (FR) and oxygen molecules through a photosensitizer placed in an inactive target tissue, inactivating microorganisms or molecules that react with the light activator. The cytoplasmic membrane of the bacterium is damaged, leading to the inactivation of the membrane transport system, inhibition of the plasma membrane, enzymatic activities,

and lipid peroxidation, [56] destruction of proteins and ion channels, elimination of critical metabolic enzymes, cell agglutination, and direct inhibition of exogenous virulence factors such as lipopolysaccharides, collagenase, and protease. PDT acts in microorganisms such as fungi, viruses, and protozoans, infections due to simple herpes virus, *P. gingivalis*, *P. intermedia*, and *A. actinomycetemcomitans*. In the other hand plants natural products (*Camellia sinesis*, *Quercus rubra*, *Caria illinoinensis*, *Smilax glyciphylla*) and phytomedicine were proposed how new alternatives in treatment of periodontal disease, present inhibit of biofilm formation, antibacterial activity and inhibition of cariogenic potential [57].

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