Aggressive Periodontitis and its Multidisciplinary Focus: Review of the Literature

Periodontitis agresiva y su enfoque multidisciplinario: Revisión de literatura

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ABSTRACT

The purpose of this review is to have a current prospect of periodontal diseases and, in particular, aggressive periodontitis. To know its classification and clinical characteristics, such as the extent and age group affected, as well as its distribution in the population, etiology, genetic variations, among other factors that could affect the development of this disease. Also, reference is made to different diagnostic options and, likewise, the current treatment options.

KEYWORDS

Aggressive periodontitis; Periodontal diseases; Diagnosis.

RESUMEN

El propósito de esta revisión es tener un panorama actual de las enfermedades periodontales y, en particular, de la periodontitis agresiva. Conocer su clasificación y características clínicas, como la extensión y grupo etario afectado, así como su distribución en la población, etiología, variaciones genéticas, entre otros factores que pudiesen afectar el desarrollo de dicha enfermedad. Así mismo, se hace referencia a distintas opciones de diagnóstico y, de igual forma, las opciones de tratamiento actuales.

PALABRAS CLAVE

Periodontis agresiva; Enfermedad periodontal; Diagnóstico.

INTRODUCTION

Periodontal disease is an endogenous microbial disease that damages the dental structure and 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).

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).

ETIOLOGY

This is a complex disease that possesses four risk factors: subgingival microbiota; individual genetic variations; lifestyle, and systemic factors.

SUBGINGIVAL MICROBIOTA

AP is associated with Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, and abnormal neutrophil function. Both bacteria, due to the action of their virulence factors, act on the host, activating its mechanism of proliferation and destruction, damaging the defense cells and bone resorption, creating periodontal bursae, loss of insertion, mobility, and loss of dental organs (7).

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. AP 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 AP, 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, Immunoglobin G (IgG), and Immunoglobin 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).

LIFESTYLE

A person's lifestyle plays a fundamental role in the appearance or inhibition of AP. With respect to the appearance of AP, local and systemic factors are present, such as age, gender, stress, and socioeconomic level. Disease severity increases with age, there is a greater prevalence in women linked with hormonal changes at the pubertal stage, periods of stress diminish the immune response of the organism, and the disease has been associated with low socioeconomic level, which is characterized by deficient habits of hygiene and diet (11).

Stress comprises a risk factor for the development of AP. The effects of the organism's response are anxiety, depression, cognitive alliteration, and an alteration in self-esteem, which give rise to alteration of health behaviors and include negligent oral-hygiene practices and the installation of bruxism. Depression is considered an indicator of stress and is related with the consumption of tobacco, alcohol, and the ingestion of an unhealthy diet, increasing the patient's susceptibility to develop AP. There is individual variability in the immunological response to stress; in some cases, IL-1B increases and regulation of the immune system diminishes. The defense of the immune system against antigens is due to the interaction of the behavior between the innate Central Nervous System (CNS) and the immune system cells. The defense of the immune system against antigens is due to the interaction of the behavior between the CBS and the immune-system cells (12).

Smoking is the most significant risk factor in the prevalence and progression of AP, whose severity depends on the dose consumed (13). The effects of smoking are related with the formation of dentobacterial plaque and the inflammatory response. The physiopathological effects are generated by the nicotine, smoke, and the carbon monoxide resulting from incomplete combustion, which favor a series of molecular events causing the diminution of osteoprotegerin, supporting the interaction of the RANK- RANKL pathway, intervening in the differentiation and activation of osteoclasts, in turn giving rise to resorption (14).

Tobacco smoke increases the proliferation of bacteria in dentobacterial plaque and subgingival microbiota and diminishes the proliferative capacity of the lymphocytes, of Immunoglobin G2 (IgG2) in the saliva and the synthesis of betadefensin 2. It generates vasoconstriction and increases platelet

adhesion, augmenting the risk of microvascular occlusion and tissue ischemia reinforced by the CO2 released, forming carboxyihemoglobin, reducing the contribution of oxygen to the tissue, cellular respiration, and formation of Free Radicals (FR). Nicotine produces an increase in bacterial adhesion to epithelial cells, of the activity of the fibroblastic collagenase enzyme, with a diminution of collagen favoring osteoclastic activity due to the increase of PGE2. It increases the release of inflammatory cytokines such as IL-6, IL-8, and NTF-α alfa, generating bone loss, destruction of connective tissue, and diminishing repair (15). In addition, it causes peripheral vasoconstriction of the blood vessels and in appearance reduces the clinical signs of gingivitis (16,17).

Ingestion of drugs for the control of systemic diseases such as high blood pressure, diabetes mellitus, and radiotherapy can exert secondary oral effects such as xerostomia, black tongue, recurrent aphthous ulcers, and gingival enlargement, which generate a predisposition for AP. Other drugs, in particular those in liquid or chewable form that contain added sugar, alter the pH and the composition of the plaque. Additionally, drugs such as calcium blockers, Cyclosporine, and anticonvulsants produce gingival hyperplasia (18). These drugs are structurally different, but have in common the action of inhibiting cell calcium uptake, a mechanism implied in the pathogeny of gingival enlargement. It has been demonstrated that young patients exhibit an exaggerated response to drugs, due to the higher level of androgens at the blood level, generating an active metabolite that acts directly on the subpopulations of fibroblasts, this response with greater frequency in the masculine gender (19).

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 (20).

The prevalence of LAP in European population varies among adolescents and young adults between 0.1 and 0.2% (21). 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% (22,23).

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.

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.

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. Immunoglobin 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 (24).

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 (25).

TREATMENT

In AP, 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 (26).

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 diana 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 (27), 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, Porphyromonas intermedia, and A. actinomycetemcomitans (28).

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