

Progression of age-related periodontitis: Literature review

Gabriela Michelle Alvarez, Katusca Anabell Rodriguez * and Katherine Andrea Romero

Dentistry School, University of Cuenca, Cuenca, Ecuador.

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Abstract

Background: Periodontitis is currently defined as a multifactorial chronic inflammatory disease associated with deregulation of the biofilm with the susceptible host, which can lead to damage the periodontal tissue due to an inappropriate immune response, characterized by a neutrophilic inflammation with subsequent destruction of proteolytic connective tissue. Nowadays, there are arguments that associate the progression of this disease in elderly people, for this reason the objective of this article is to carry out a literature review that links the progression of periodontitis associated with age, in addition to determining if at an older age there is higher prevalence of periodontitis.

Methodology: A systematic bibliographic search was carried out in 5 scientific databases: PubMed, Cochrane, Scielo, Science Direct, EBSCO of 45 articles published in English and Spanish in the last 5 years (2018-2022).

Results: Evidence suggests that elderly patients (60 years and older) are more likely to develop periodontitis due to a deficient immune response that keep them off from having a correct inflammatory response to various factors, there is also postulated a prolonged exposure to the etiological factor.

Conclusions: Evidence reports that older patients have a higher prevalence of periodontitis because aging decreases immune response, which is responsible for protecting the body against different factors. Finally, as patient's age, nutritional status is altered thereby the digestive process and nutrient absorption from food, which interact with other well-defined risk factors to increase susceptibility to periodontal disease.

Keywords: Periodontitis; Inflammation Chronic; Immunity; Aging; Periodontitis by age; Periodontitis in the elderly; Periodontitis; Inflamación; Inmunidad; Envejecimiento; Periodontitis por edad y Periodontitis en ancianos.

1. Introduction

Periodontitis is a multifactorial chronic inflammatory disease, associated with the accumulation of biofilm that progressively affects the supporting structures of the teeth. Patients with periodontitis are characterized by probing depth ≥ 4 mm, bleeding on probing, clinical attachment loss, radiographic evidence of alveolar bone loss, pathologic mobility, and pathologic migration of teeth [1].

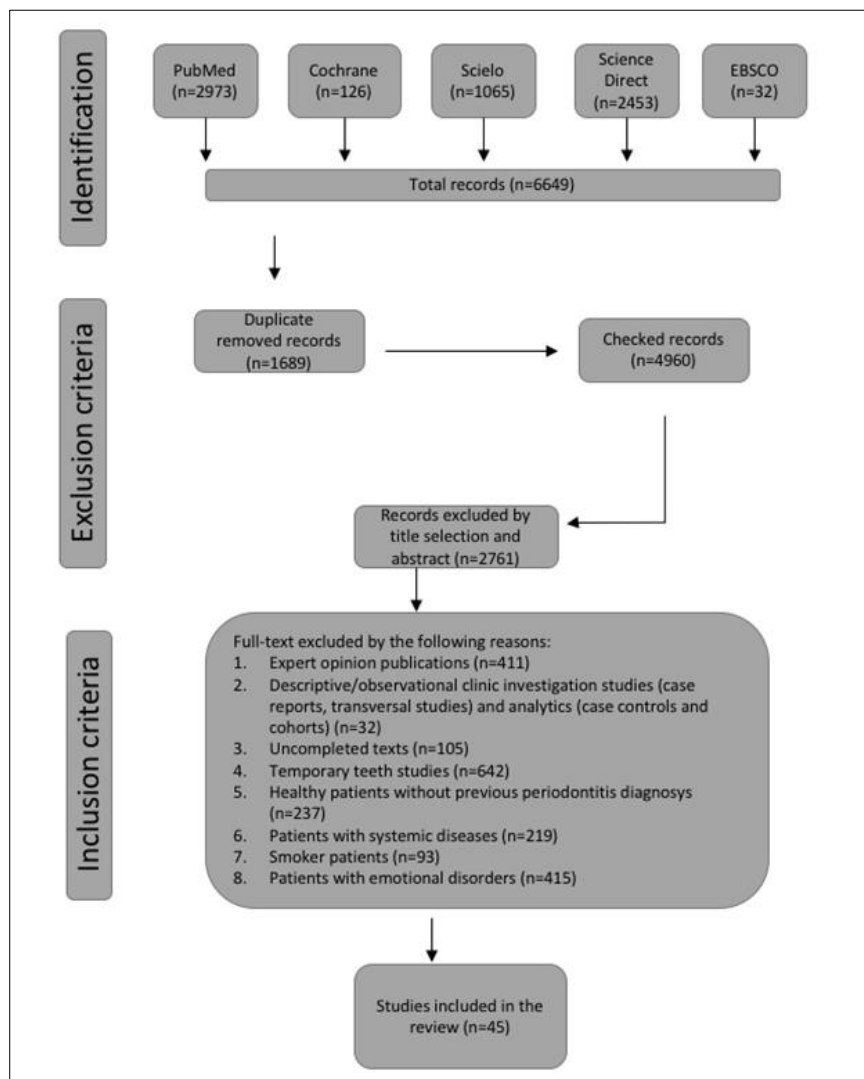
Inflammatory/infectious diseases are considered to be the result of the imbalance between the immunological interaction of the susceptible host and the flora of the biofilm, frequently associated with a group of specific anaerobic gram-negative species, defined by Socransky as red complex (*Porphyromonas gingivalis*, *Tannerella forsythia* and *Treponema denticola*), found mainly in periodontal pockets, capable of leading a chronic inflammation by stimulating macrophages and other inflammatory cells, resulting in the production of pro-inflammatory substances such as: tumor necrosis factor alpha (TNF)- α , interleukin (IL)-1, prostaglandin E2 (PGE2), among others [1, 2, 3, 4].

* Corresponding author: Katusca Anabell Rodriguez
Dentistry School, University of Cuenca, Ecuador.

The presence of these proinflammatory cytokines induces expression of receptor activator of nuclear factor-kappa B ligand (RANK-L) in osteoblasts and helper T cells, which interact with receptor activator of nuclear factor kB (RANK) in osteoclast progenitor cells, generating the formation and maturation of osteoclasts, which mediate the destruction of alveolar bone. On the other hand, virulence factors produced by pathogenic periodontal bacteria, stimulate the production of matrix metalloproteinases (MMPs) in macrophages, fibroblasts, neutrophils, and junctional epithelial cells, which are capable of mediating the rupture of collagen fibers in periodontal tissue especially in the periodontal ligament [1].

According to several studies, the prevalence of adults diagnosed with periodontitis is approximately 8.9% in people aged 21 to 30 years, 10.2% between 31 to 63 years, 20.6% of adults between 64 to 74 years and 45.7% among the population between 75 and 84 years old. On the other hand, a prevalence of 14.3% was found among the population aged 90 to 95 years old. These data suggest some degree of association with advancing age, which could be explained by the degenerative changes typical of this stage, especially since the expression of Del-1 (developmental endothelium locus 1) decreases with aging, which results in a greater production of IL-17 increasing inflammation and as a consequence periodontal bone loss. However, prolonged exposure to risk and etiological factors throughout a person's life can also lead to loss of periodontal support [2, 5, 6, 7].

2. Material and methods



Source: Gabriela Michelle Alvarez and Katusca Anabell Rodriguez.

Figure 1 Flowchart summarizing the bibliographic search and the selection of articles

A literature review was carried out to identify the articles that meet the requirements established in the inclusion and exclusion criteria, both in Spanish and English literature, in order to obtain information about the progression of age-related periodontitis.

An electronic literature search was performed in bibliographic databases such as: PubMed, Cochrane, Scielo, Science Direct, EBSCO using the keywords: “*Periodontitis OR Inflammation Chronic AND Immunity and Aging AND periodontitis by age AND periodontitis in the elderly, Periodontitis, Inflamación, Inmunidad, Envejecimiento, Periodontitis por edad y Periodontitis en ancianos*”.

The inclusion criteria used for the selection of articles were the following: systematic reviews, full-text meta-analyses published in Spanish and English, scientific articles published in the last 5 years, studies conducted in permanent dentition and patients with a previous diagnosis of periodontitis. There were excluded publications with expert opinions, descriptive/observational (case reports; cross-sectional studies) and analytical (case controls and cohorts) clinical research studies, incomplete texts, studies conducted in deciduous dentition, healthy patients without previous diagnosis of periodontitis, patients with systemic diseases, smoking patients and patients with syndromes or emotional disorders (Figure 1).

3. Results and discussion

3.1. Periodontitis

Periodontitis is a multifactorial chronic inflammatory disease associated with the deregulation of the bacterial biofilm in periodontal pockets, which causes damage to the attached units due to an inappropriate immune response. Older people present a dysregulated immune response characterized by neutrophilic inflammation with subsequent proteolytic destruction of connective tissue [8, 9, 10].

Patients with periodontitis are characterized by probing depth ≥ 4 mm, bleeding on probing, clinical attachment loss, and radiographic evidence of alveolar bone loss, pathologic tooth mobility and migration (Table 1) [1].

Table 1 Periodontitis diagnostic criteria

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|--|
| Diagnostic criteria. |
| Gum inflammation. |
| Bleeding on probing. |
| Clinical attachment loss. |
| Radiographic evidence of alveolar bone loss. |
| Probing depth ≥ 4 mm. |
| Pathological mobility. |
| Pathological migration of teeth. |

Source: Taken from Keene and Ribeiro work [1, 8].

Another systematic review found that periodontitis is considered a "silent" disease with few or no symptoms, which may explain the results of studies that show no association between periodontitis and quality of life. Although periodontitis may not directly cause impaired quality of life, it can eventually lead to tooth loss, which can negatively affect quality of life in older adults [11, 12].

3.2. Etiopathogenesis

Inflammatory/infectious diseases are considered to be the result of the imbalance between the immunological interaction of the susceptible host and the flora of the biofilm, frequently associated with a group of specific anaerobic gram-negative species, defined by Socransky as red complex (*Porphyromonas gingivalis*, *Tannerella forsythia* and *Treponema denticola*), found mainly in periodontal pockets, capable of leading to chronic inflammation by stimulating

macrophages and other inflammatory cells, resulting in the production of pro-inflammatory substances such as: tumor necrosis factor alpha (TNF) - α , interleukin (IL)-1, prostaglandin E2 (PGE2), among others [1, 2, 3, 4].

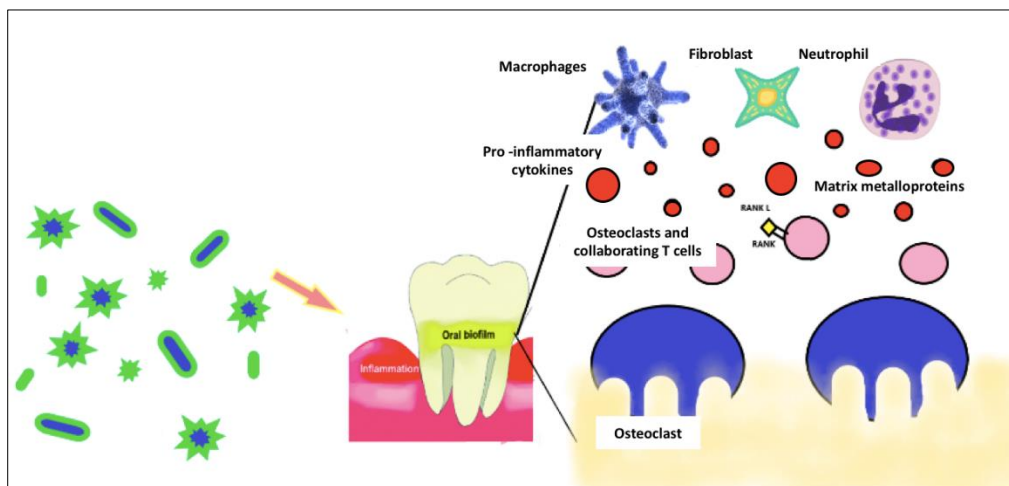
The presence of these proinflammatory cytokines induces expression of receptor activator of nuclear factor-kappa B ligand (RANK-L) in osteoblasts and helper T cells, which interact with receptor activator of nuclear factor kB (RANK) in osteoclast progenitor cells, generating the formation and maturation of osteoclasts, which mediate the destruction of alveolar bone (Figure 2) [1].

Under normal health conditions, inflammation enters in a programmed resolution cycle in which a limit of the damage area is established until the pathogen is eliminated locally [13].

The inflammation process is characterized by dilation of blood vessels, increased permeability of capillaries, increased blood flow, and recruitment of leukocytes. The first leukocytes that accumulate at the site of inflammation are polymorphonuclear neutrophils as the first line defense of the innate immune system, since they have phagocytic and microbicidal functions. Pathogens are recognized by pattern recognition receptors such as Toll-type receptors, activation that generates the production of inflammatory cytokines, chemokines and proinflammatory lipid mediators such as prostaglandins. These mediators are essential to generate an effective inflammatory response and eliminate bacteria. Proinflammatory mediators such as interleukin-1 beta, interleukin-6, tumor necrosis factor alpha, and prostaglandins are produced in the inflamed gingiva [14].

To detect the inflammatory response, the innate immune cells and the resident cells trigger the production of mediators that will regulate the fate of inflammation. In addition, the presence of chemokines, with chemoattractant functions, induce the migration of defense cells to the site of infection. Regarding genetic regulation, the pathways regulated by nuclear factor kappaB are activated by pattern recognition receptors, which are lipopolysaccharides, through a Toll-like receptor pathway [14].

Periodontitis is considered to be the result of the imbalance between the immunological interaction of the susceptible host due to an increase in the basal activity of NF- κ B (nuclear factor enhancer of kappa light chains of activated B cells) of DCs (mature dendritic cells), in addition to the production by macrophages of PGE2; related to increasing age, which could contribute to inflammatory bone loss. The innate immune response can be affected with old age, presenting an inefficient communication since they express lower levels of adhesion molecules and show a reduced response to growth factors [7].



Source: Gabriela Michelle Alvarez and Katusca Anabell Rodriguez.

Figure 2 Inflammatory response mechanism in periodontitis

This suggests that the frequency of apoptotic events could be reduced with aging. One such endogenous inhibitor, which is produced in periodontal tissue, is a 52-kDa protein secreted by endothelial cells called developmental endothelial locus-1 (Del-1), which competes with intercellular adhesion molecule-1 (ICAM-1) by binding to integrin LFA-1 on neutrophils, thereby inhibiting its firm adhesion to the endothelium and subsequent transmigration associated with chronic neutrophil recruitment. Del-1 expression decreases in old age, which correlates with the development of periodontitis (Figure 3) [7].

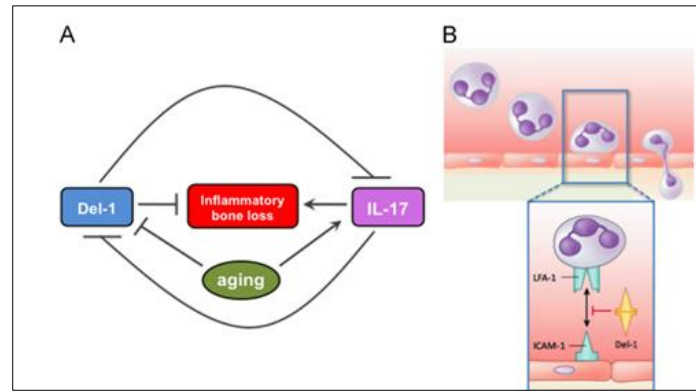
Table 2 Effects of aging on phagocytes and antigen presenting cells

| Effects of aging on phagocytes and antigen presenting cells. | Cell type | | |
|--|---|--|---|
| | Neutrophils | Monocytes/macrophages | Dendritic cells |
| Reduced | Signal transduction (GM-CSFR, TLR2, TLR4, CD14, CD11b), phosphorylation of ERK, p38, Akt, PLC- γ) Receptors recruitment to lipid rafts (TLR4, TREM-1) Chemotaxis (fMLP, GM-CSF) CD16 expression Phagocytosis Microbicidal activity Reactive oxygen species (LPS, fMLP, GM-CSF, opsonized bacteria). | Signal transduction (e.g., total levels and/or activation of STAT-1 α , p38 and JNK MAPKs, MyD88, NF- κ B) Cytokine production (IL-12, IL-6, TNF, MIP-1 α , MIP-1 β , MIP-2) Chemotaxis Phagocytosis Reactive oxygen species Reactive nitrogen species Intracellular killing Expression of costimulatory molecules (CD80 and CD86), MHC Class II. Expression of CD14, TLR1, and TLR3. | Antigen presentation Chemotaxis Endocytosis Production of IFN type I and III (pDC) IL-12 PI3K activity and Akt phosphorylation Expression of TLR-1, -3, -5, -7, and -8. |
| Maintained | Total number of circulating neutrophils Basal levels of receptor expression Expression of adhesion molecules and adhesion to endothelial cells. Apoptosis (spontaneous). | Expression of IFN- γ receptor and TLR2. Expression of TLR negative regulators (e.g. SOCS-1, IRAK-M, A20, PPAR- γ) b. | Expression of TLR2 (mDC) and TLR9 (pDC). |
| Increased | Apoptosis (under priming conditions; e.g. impaired anti-apoptotic signals after exposure to GM-CSF) Activity of cytokine-signaling inhibitory molecules (SHP-1, SOCS). | PGE2 production. TLR5 expression. | TNF, IL-6, IL-23 Basal expression of CD80, CD83, CD86 Basal NF- κ B activity. |

Source: G. Hajishengallis [7].

Bodineau, Agnes et al. evaluated the proportions of the inflammatory cell subset in gingival connective tissue, including mature dendritic cells (DCs), in elderly and young patients with chronic generalized periodontitis to better understand the effect of aging on the gingival inflammatory phenomenon. The CD45RB+ leukocyte re-count in the upper connective tissue revealed that both groups presented a similar inflammatory state. Therefore, the observed changes in the proportions of the inflammatory cell subsets could be related to age and could allow further comparison of the effect of aging on the gingival inflammatory phenomenon [15].

According to Agnes Bodineau et al., Langerhans cells (LC) (dendritic cells located in the epithelium of the oral mucosa) may play an important role in the initiation and maintenance of periodontitis. LCs can capture and process foreign antigens to travel to regional lymph nodes and initiate proliferation of antigen-specific T cells.



Source: G. Hajishengallis [7].

Figure 3 Del-1 vs. IL-17 in aging and periodontitis

In aging, the proportion of intraepithelial LC decreased and LC showed a rounded profile with a decrease in the number and length of dendritic processes. All samples were located in labial/buccal sites and most gingival tissue samples (12 of 16) were from mandibular gingiva; In addition, the CD45RB+ leukocyte count in the upper connective tissue confirmed that both groups present a similar inflammatory state. Therefore, the observed changes in the number and morphology of Langerhans cells could be related to age [15].

With respect to the periodontium, the biofilm microbiota of healthy individuals in their mid-60s consists predominantly of aerobic gram-positive bacteria and less anaerobic gram-negative ones. Actinomyces Spp., lactobacilli and yeasts are among the microorganisms housed at the supragingival level of individuals older than 60 years [16].

According to Christoph A. Ramseier, relying on probing depth as an exposure variable for periodontitis in older age groups is not ideal, since apical junctional migration is mainly associated with normal gingival recession of the aging process, which is minimal, therefore it can be the result of repetitive and localized trauma caused during tooth brushing or the result of the fragility of the gingival tissues as a consequence of biological processes [17, 9, 2, 18, 19].

3.3. Risk factor's

There are risk factors that affect the appearance and progression of periodontitis, which can be divided into 2 categories: non-modifiable factors (age, sex, race and genotype) and modifiable factors (poor oral hygiene, peripheral pathogens in the oral biota, complex symptoms of periodontitis, nicotine). Additionally, there are other circumstances that influence the development of this disease such as: systemic conditions, low socioeconomic level, level of education, poor diet and stress [8, 20, 21, 21, 22].

Some studies show that people with a low level of education and training have a higher incidence of periodontitis since they do not have the possibility of preventive activities, easy access to elements and techniques that effectively help to eliminate the biofilm [23, 22, 24].

The main factor associated with the destruction of tissue as a physiological result of the aging process is the production of free radicals, which means that as people age, the risk of chronic diseases will increase [25].

The relationship between age and HRQoL (Health-Related Quality of Life) was analyzed in a group of women between 70 and 92 years old using a OHIP descriptive analysis (an instrument that contributes to the evaluation of the patient's own perception regarding the oral health and quality of life), which showed that that at an older age the impact of tooth loss was less [26]. The report projected that more than 20% of older people have periodontitis, that men are more affected than women, and that low-income older people are at higher risk [27].

3.4. Consequences of periodontitis

Periodontitis causes considerable damage to the periodontal support and can cause significant tooth loss, and this translates into a loss of masticatory function, affecting the patient's quality of life. In the absence of adequate control of periodontitis, the dentition is at risk of being lost. This stage is characterized by the presence of deep periodontal lesions that extend to the apical portion of the root and/or a history of multiple tooth loss. It is frequently complicated by dental

hyperlaxity due to secondary occlusal trauma and the sequelae of tooth loss: posterior bite collapse and deviation. Case management often requires stabilization/restoration of masticatory function [28, 29].

3.5. Prevalence

According to the World Health Organization (WHO), older adults are those who are over 60 years old and are divided into the following categories: Third age: 60-74. Fourth age: 75-89. Longevity: 90-99. Centenarians: 100 years old and further. When analyzing, these data are consistent with the theory that the prevalence and severity of periodontitis increases with age.

Gingivitis is the main disease in late adolescence (17-25 years). Patients with stage IV periodontitis are mostly middle-aged (46-55 years). Compared with other periodontal diseases, the incidence of periodontitis is 15% in the young group, 38% in the adult group, and 47% in the elderly group [2, 29].

The rationale for classification according to severity encompasses at least two important dimensions: complexity of treatment and extent of disease. It is also worth discussing the important limitations of severity definitions in the context of recent therapeutic improvements that have enabled the successful management of progressively severe periodontitis [12]. Conventional definitions of severe periodontitis should be checked over to better discriminate the most severe forms of periodontitis [29, 30].

Table 3 Studies related to the progression of periodontitis according to age carried out in different countries

| Country | Authors | Title-Publication date | Results | Conclusions |
|---------------------------|---|---|---|--|
| Costa Rica | William Lao Gallardo Huberth Araya Rodriguez [2]. | Periodontal disease in Costa Rica year 2017. | Tooth loss due to periodontitis is reported to be approximately 0.0004% for patients between 13-19 years old, gradually rising from that age to 2.60% in adults between 20 to 45 years old, and then increasing disproportionately to 37.83% in the elderly. | The dental loss caused by periodontitis is directly proportional to the increase in age and inversely proportional to the behavior of the hygienic condition. |
| Colombia | Rubiel Marin Jaramillo Andres Duque-Duque [31]. | Modifying conditions for the risk of periodontal disease: a narrative review of the evidence in Latin America 2021. | Periodontitis increased in severity with increasing age; periodontitis prevalence was mild in central Americans from 18 to 44 years old and moderate in South Americans between 65 and 74 years old. However, severe periodontitis decreases its prevalence in patients between 45 and 54 years old. | Periodontitis increased in severity with increasing age. Various conditions can negatively affect periodontal health. |
| United States and Germany | Monisha Billings, Birte Holtfreter, Panos N. Papapanou, Gabriela Lopez Mitnik, Tomas Kocher, Bruce A. Dye [32]. | Age-dependent distribution of periodontitis in two countries: Findings from NHANES 2009 to 2014 and SHIP-TREND2008 to 2012. 2018. | There were made divisions by age, in which 95% of people aged 30 to 34 years had a mean CAL (clinical attachment loss) of ≤ 2.5 mm and a mean clinical recession of ≤ 0.3 mm, while the top 5% achieved a mean CAL of 6.0 mm and a mean clinical recession of 2.5 mm. In contrast, the oldest 95% of participants had a mean CAL ≤ 4.5 mm and a mean clinical recession of < 2.7 mm, whereas the top 5% had a mean CAL | Despite the substantial differences in the overall severity of attachment loss between the two samples, common patterns of CAL and relative contribution in the relationship between R (recession) and PD (pocket depth) |

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| | | | of 11.1 mm and a mean clinical recession of 6.7 mm. When comparing across lifespans by age group, the contribution of recession to CAL appeared to increase substantially between ages 35 and 54. | and CAL were identified with increasing age. |
| United States of America | Leslie R. Halpern [33]. | Geriatric Syndrome and Oral Health 2020. | Epidemiological studies have shown that plaque accumulation and consequent gingivitis/periodontitis become more severe as the patient ages (1.8% to 3.3% after 65 years old). Atherosclerosis develops in the alveolar bone and the ligamentous vasculature becomes dysfunctional. The result is gingival recession and an increase in the length of the crown of the teeth with the risk of caries formation in the cementum exposed to the oral cavity. With aging, the gingival epithelium thins and cell density increases. | The decrease in salivary flow in the elderly contributes to the progression of periodontitis. |
| New York | Manresa C, Sanz-Miralles EC, Twigg J, Bravo M [34]. | Supportive periodontal therapy (SPT) for maintaining the dentition in adults treated for periodontitis (Review) 2018. | Reports on NHANES data showed that the prevalence of periodontitis was 68% in adults ≥65 years old and 46% in adults ≥30 years old. The prevalence of severe periodontitis was also higher in older adults, with 11% of adults ≥65 years old and 8.9% of adults ≥30 years old diagnosed with severe periodontitis. | They have shown that periodontitis may be associated with a poor quality of life related to oral health. |
| Brazil | Andrew Tawse-Smith [35]. | Age and oral health: current considerations 2017. | The age groups 35-44 and 65-74 presented high prevalence values of chronic periodontitis, 92% and 100% respectively. Several studies have shown that at early ages, there is a minimal loss of localized initial attachment; at the age of 30 years the cumulative attachment loss reaches 1 mm; and as participants approach 40 years, the average loss reaches 1.5 mm. | Regardless of the age group, it is essential to understand which risk factors are involved in the onset and progression of caries and periodontitis. |
| United States of America | Persson RE, Persson GR [10]. | The elderly at risk for periodontitis and systemic diseases 2018. | Three groups of older subjects are identified based on chronological age: (1) young (65 to 74 years old), (2) elderly (75 to 84 years old), and (3) elderly (>85 years old). The report projected that more than 20% of older people have periodontitis, that men are more affected than women, and that low-income older people are at higher risk. | To manage the senior's health care needs, coordination between medical and dental care providers will be necessary. Such risk assessment of older people should take a holistic approach and focus on reducing the |

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| | | | | infectious burden and improving self-efficacy. |
| Holland | Van de Rijt LJM, Stoop CC, Weijnenberg RAF, de Vries R, Feast AR, Sampson EL, et al [11]. | The Influence of Oral Health Factors on the Quality of Life in Older People: A Systematic Review. The Gerontologist. July, 15th 2020. | OHQoL in people 65 years and older is positively associated with a greater number of teeth, a greater number of occlusive pairs, over implant-retained dentures, and the concept of shortened dental arch, and is negatively associated with xerostomia, orofacial pain, and poor chewing ability. | Having a functional dentition (whether natural or prosthetic) is important for good OHQoL, while painful or functional complaints are associated with impaired OHQoL. |
| China | Wong FMF, Ng YTY, Leung WK [20]. | Oral Health and Its Associated Factors Among Older Institutionalized Residents. 26 October, 2019. | The reported oral hygiene and health of the surveyed institutionalized elderly was poor (>50% of residents had calculus; denture hygiene index >80%). Problems of the gums (approximately 30% of dentate residents had moderate to severe periodontitis), teeth (more than 20 decayed, missing, or filled teeth), mucous membrane (>10% had mucosal lesions), and dentures (up to 40%) were common and associated with poor OHRQoL. | The relationship between oral health, OHRQoL, and nutrition in this population is at risk, as people with poor OHRQoL may show signs of poor nutrition. |
| United Kingdom | Yaacob M, Han TM, Wahab SMA, M Sham S' Atiqah, Abllah Z [12]. | Chronic periodontitis patients: their knowledge and its correlation with oral health related quality of life. Mater Today. January, 2019. | More than 90% of periodontitis patients reported at least one experienced oral impact occasionally, quite frequently, or very often (OFOVO) compared with 53.8% of periodontally healthy controls (P < 0.001). | Patients with periodontitis have a worse quality of life than periodontally healthy individuals, being the differences clinically significant. |
| Sweden | Kato T, Abrahamsson I, Wide U, Hakeberg M [26]. | Peri Periodontal disease among older people and its impact on oral health-related quality of life. Gerodontology. 2018. | Among men and women aged 70 years, generalized periodontitis was associated with worse HRQoL. However, a multivariate analysis failed to demonstrate this association (OR = 1.02, 95% CI: 0.72-1.44). In participants with 1-10 remaining teeth, the OHIP-14 score was significantly increased, indicating poor OHRQL, compared to participants with ≥21 remaining teeth (OR = 1.57, 95% CI: 1.13 -2.19). Similar findings were observed among women aged 70 to 92 years. | Periodontitis did not show an association with poor HRQoL; however, a significant association was found between the number of teeth and poor HRQoL. |
| Egypt | Omar Khaled Gamila,b, Dina FahimahmedC, Khaled Mohamed | Frequency and Risk Indicators of Periodontal Diseases in a | The frequency of stage I periodontitis is (38%), stage II (20.4%), stage III (1.6%) and stage IV (0.4%). | Stages III and IV periodontitis showed the lowest frequency in a sample of adult |

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| | Keraad, Noha Ayman Ghallaba, Weam Elbattawya [36]. | Sample of Adult Egyptian Patients: A Hospital-Based Cross-Sectional Study 2021. | | Egyptian patients compared to stages I and II. |
| Norway | Gro Eirin Holde, nils oscarson, Tordis A. Trovik, Anders Tillberg, Birgitta Jonsson [37]. | Prevalence and severity of periodontitis in adults: a cross-sectional study in Norwegian circumpolar communities 2017. | The prevalence of periodontitis increased with age; in the older group, it was five times higher than in the youngest group. Periodontitis was more prevalent among men (56.7%) than among women (42.6%). The prevalence of radiographic bone loss (BL) skyrocketed from the age of 35, reaching almost 100% in the age group of 65-79 years. The extent of the BL also increased rapidly with age. | In conclusion, this study reveals a high periodontitis burden in circumpolar communities of Norway, with half the adult population affected. |
| Poland | Paśnik-Chwalik B, Konopka T [8]. | Impact of periodontitis on the Oral Health Impact Profile 2020. | All studies indicated a significant influence of periodontitis on the deterioration of OHRP-14 values. It was shown that this relationship is directly modified in proportion to the degree of advancement of periodontal disease and the extent of periodontal tissue damage. | The impact of periodontitis on the deterioration of OHRQoL is clearly explained by the clinical symptoms of periodontitis. According to patients, the most important problems that periodontitis can cause include psychological discomfort, stress, problems in interpersonal relationships or even difficulties in daily activities. |
| China | Florence M F Wong , Yannies T Y Ng , W Keung Leung [20]. | Oral Health and Its Associated Factors Among Older Institutionalized Residents-A Systematic Review 2020. | Oral hygiene and health of the elderly surveyed were poor (>50% of residents had calculus; denture hygiene index >80%). Periodontal problems (approximately 30% of dentate residents had moderate to severe periodontitis), teeth (more than 20 decayed, missing, or filled teeth), mucosa (>10% had mucosal lesions), and dentures (up to 40%) were common and associated with poor OHRQoL, especially in women, socially disadvantaged residents, or those with mild or higher cognitive impairment. | The awareness of health policy makers and health promotion teams regarding oral health were increased. |

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| Sweden | Takahiro Kato, Ingemar Abrahamsson, Ulla Wide, Magnus Hakeberg [26]. | Periodontal disease among older people and its impact on oral health-related quality of life 2018. | The relationship between age and health-related quality of life (HRQoL) was analyzed in a group of women aged 70 to 92 through a descriptive analysis, which showed that age was not associated with the sum of OHIP-14 scores (instrument that contributes to the evaluation of the patient's own perception regarding oral health and quality of life). | Periodontitis did not show an association with poor HRQoL, however a significant association was found between the number of teeth and poor HRQoL. |
| England | Dur Durham J, Fraser HM, McCracken GI, Stone KM, John MT, Preshaw PM [21]. | Imp Impact of periodontitis on oral health-related quality of life. J Dent. Abr 2018. | The mean age of the participants was 47 ± 9 years, and patients with periodontitis had, on average, 33 ± 23 sites showing probing depths ≥ 5 mm. | People with periodontitis report substantial impacts on functional, physical, psychological, and social OHRQoL. |
| Argentina and Israel | Catunda,Raisa Queiroz,Levin Liran,Kornerup,Idal Gibson,Monica Prasad [6]. | Prevalence of periodontitis in young populations: A systematic review 2019. | The average reported prevalence of periodontitis in young populations was 1.7% (range 0.66% in Argentina and 5.9% in Israel). The prevalence was higher for the localized form of this disease. Permanent teeth were the most commonly affected dentition (114 of 115 affected patients). Regarding age, the prevalence was 0.6%, 0.8% and 1.6% for the age groups 2-12, 20-25 and 13-20, respectively. | The prevalence of periodontitis in young patients varies widely, which could indicate population predispositions, under diagnosis, or lack of diagnostic standardization. |
| Italy | Fari Farina R, Simonelli A, Baraldi A, Pramstraller M, Minenna L, Toselli L, et al [41]. | Tooth loss in complying and non-complying periodontitis patients with different periodontal risk levels during supportive periodontal care. Clin Oral Investig. October, 1st 2021. | In both compliant and non-compliant, TLR was significantly lower or similar to the positive baseline at PerioRisk level 3 (0.08 and 0.03 teeth/year, respectively) and PerioRisk level 4 (0.12 and 0.18 teeth/year, respectively). Although marked and clinically relevant in the non-compliant, the difference between the TLR of the compliant (0.32 tooth/year) and the non-compliant (0.52 tooth/year) with PerioRisk level 5 and the negative baseline was not significant. | An SPC protocol based on a 3- to 6-month review interval can effectively limit long-term tooth loss in periodontitis patients with PerioRisk levels 3 and 4. A fully adhered 3-month SPC protocol appears ineffective when applied to PerioRisk level 5 patients. |
| Dublin | Winning L, Polyzois I, Sanmartin Berglund J, Renvert S [9]. | Peri Periodontitis and airflow limitation in older Swedish individuals 2020. | The proportion of participants with periodontitis in the airflow limitation group was 65.1% compared to 41.5% with normal respiratory function ($p < 0.001$). Multiple logistic regression analysis showed that periodontitis was independently associated with airflow limitation with an odds ratio of 2.31. | In this group of older dentate people, periodontitis was significantly associated with airflow limitation, independent of other known risk factors. |

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| Brazil | Susilena Arouche Costa, Cecilia Claudia Costa Ribeiro, Kheops Renoir de Oliveira, Cláudia Maria Coelho Alves, Erika Barbara Abreu Fonseca Thomaz, Renato Corrêa Viana Casarin, Soraia de Fátima Carvalho Souza [39]. | Low bone mineral density is associated with severe periodontitis at the end of the second decade of life: A population study 2020. | Statistically significant correlation between age and periodontitis severity in which it was shown that of 78 people analyzed who were in the pre-elderly classification, 24 people had Stage III periodontitis progression and 27 were located in stage IV, and in the elderly classification, 4 people were located in Stage III and 23 were located in Stage IV. It should be noted that the age range of pre-elderly is 45-59 years and the elderly 60 years. Immune system, genetic factors have been shown to play a role in periodontal changes in the elderly, although the mechanism is still unclear. | Low BMD (bone mineral density) was found to be associated with the severity and extent of periodontitis in adolescents. Adolescents in the peak age of bone mass who have a low BMD are more likely to have severe periodontitis. |
| Argentina | Cat Catunda RQ, Levin L, Kornerup I, Gibson MP [6]. | Prevalence of Periodontitis in Young Populations 2019. | The average reported prevalence of periodontitis in young populations was 1.7% (range 0.66% in Argentina and 5.9% in Israel). | The prevalence of periodontitis in young patients varies widely, which could indicate population predispositions, under diagnosis, or lack of diagnostic standardization. |

Source: Various authors detailed in references

3.6. Prevention

In first place, the prevention of gingivitis is the first step to prevent periodontitis, therefore, self-oral care and the professional's performance in the control, treatment and maintenance of dental support tissues, must be considered [42].

To achieve optimal control of the biofilm, brushing should be complemented with interdental cleaning, the modified Bass technique can be used, which is recommended to use with a soft bristle brush. In order to perform the technique, the head of the brush is placed parallel to the occlusal plane, with the tip directed distally, the bristles are placed on the gingival margin, an angle of 45 degrees is established towards apical, vertical vibratory pressure is exerted on the bristles, so they are introduced into the groove and interdental niches making approximately 20 short movements and finally moving the head of the brush towards the free faces of the tooth. In addition, the auxiliary elements available are: dental floss and interdental cleaners such as wooden or plastic toothpicks and interdental brushes [42].

3.7. Treatment

According to Roberto Fariña, Elisa Maietti et al., patients with periodontitis have a residual risk and there may be recurrence or progression of the disease, so they were treated with supportive periodontal care (SPC in which was based on preventive and therapeutic treatment). The SPC sessions incorporate the evaluation of periodontal and general health, the motivation for adequate oral hygiene, the control of risk factors, the professional mechanical plaque removal (PMPR) and subgingival instrumentation of residual pockets [43].

Currently, patients who are treated periodontally have a better chance of preserving these teeth. According to Yong Zhang, 2022 mobile mandibular anterior teeth after splinting decreased probing depth (PD) from 4.31mm to 2.93mm and clinical attachment loss (CAL) from 5.02 mm to 4.58 mm keeping the alveolar bone stable [44].

Another treatment that can be performed is ultrasonic subgingival scaling combined with manual root planning, producing a better therapeutic effect for treatment of chronic periodontitis in elderly patients [44, 45].

Regarding periodontitis treatment, the following care protocol is postulated:

- Carry out emergency treatment if considered.
- Instruction to the patient about the course of the disease, factors that contribute, perpetuate and trigger the disease.
- Instruction on oral hygiene, evaluation and reinforcement of biofilm control measures.
- Study, diagnosis and treatment of occlusal disharmonies and temporomandibular disorders.
- Bacterial sampling from selected bags, cultures and antibiograms, may be considered.
- Previous dental treatments if necessary
- Supra and subgingival scaling plus root planing.
- Atraumatic extraction of non-viable teeth and ridge preservation.
- Local and systemic antibiotic treatment. The application of antibiotics through both routes presents advantages and limitations [39].

4. Conclusion

In conclusion, there is conflicting evidence regarding the progression of periodontitis associated with age, since some studies consider age as one of the risk factors for alveolar bone loss and loss of clinical attachment, while other authors discuss the association.

The older there is a higher prevalence of periodontitis because with aging the immune response decreases, in the same way the expression of Del-1 (locus-1 of the developmental endothelium) is reduced, which competes with the intercellular adhesion molecule-1 (ICAM-1) by binding to the integrin LFA-1 in neutrophils, inhibiting adhesion to the endothelium and transmigration, which are responsible for locally autoregulating persistent inflammation associated with chronic neutrophil recruitment.

Currently, there are several alternatives for the prevention and treatment of periodontitis, however, some elderly patients present minimal attachment loss even with a lifetime maintenance plan.

Finally, the incidence of periodontal disease increases with age because as patients age, they are more likely to suffer from a great number of diseases that force patients to use medications that reduce saliva flow, causing various complications in the mouth, in addition to reduced immune function, impaired nutritional status and thus impaired digestive process and nutrient absorption from food, vocalization is severely affected, which interact with other well-defined risk factors to increase susceptibility to periodontal disease.

Recommendations

After having carried out an exhaustive literature review, we can confirm that the implementation of preventive and corrective measures is the best treatment to maintain good oral health from a young age, preserving a good quality of life in a long term. It is important to strengthen the communication process with the patient and reinforce self-care practices, which, although simple, are effective measures to prevent the disease. Undoubtedly, it is necessary to carry out studies that broaden the knowledge about the progression of periodontal disease in association with age, since it would promote prevention policies established according to age.

Compliance with ethical standards

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References

- [1] Kwon T, Lamster IB, Levin L. Current Concepts in the Management of Periodontitis. *Int Dent J*. December 1st 2021;71(6):462-76.
- [2] Lao Gallardo W, Araya Rodríguez H, Lao Gallardo W, Araya Rodríguez H. Periodontal disease in Costa Rica 2017. *Odontol Vital*. Decemeber, 2018;(29):59-68.
- [3] Nath SG, Raveendran R. Microbial dysbiosis in periodontitis. *J Indian Soc Periodontol*. 2013;17(4):543-5.
- [4] Díaz Caballero AJ, Vivas Reyes R, Puerta L, Ahumedo Monterrosa M, Arévalo Tovar L, Cabrales Salgado R, et al. Biipels as an expression of the Quorum Sensing mechanism: a review. *Av En Periodoncia E Implantol Oral*. diciembre de 2011;23(3):195-201.
- [5] Botero Z L, Vélez L ME, Alvear E FS. Prognosis factors in periodontics. *Rev Fac Odontol Univ Antioquia*. June, 2008;19(2):69-79.
- [6] Catunda RQ, Levin L, Kornerup I, Gibson MP. Prevalence of Periodontitis in Young Populations: A Systematic Review. *Oral Health Prev Dent*. 2019;17(3):195-202.
- [7] Hajishengallis G. Aging and its Impact on Innate Immunity and Inflammation: Implications for Periodontitis. *J Oral Biosci JAOB Jpn Assoc Oral Biol*. 1 de febrero de 2014;56(1):30-7.
- [8] Paśnik-Chwalik B, Konopka T. Impact of periodontitis on the Oral Health Impact Profile: A systematic review and meta-analysis. *Dent Med Probl*. 2020;57(4):423-31.
- [9] Winning L, Polyzois I, Sanmartin Berglund J, Renvert S. Periodontitis and airflow limitation in older Swedish individuals. *J Clin Periodontol*. junio de 2020;47(6):715-25.
- [10] Persson RE, Persson GR. The elderly at risk for periodontitis and systemic diseases. *Dent Clin North Am*. abril de 2005;49(2):279-92.
- [11] van de Rijt LJM, Stoop CC, Weijenberg RAF, de Vries R, Feast AR, Sampson EL, et al. The Influence of Oral Health Factors on the Quality of Life in Older People: A Systematic Review. *The Gerontologist*. 15 de julio de 2020;60(5):e378-94.
- [12] Yaacob M, Han TM, Wahab SMA, M Sham S 'Atiqah, Abllah Z. Chronic periodontitis patients: their knowledge and its correlation with oral health related quality of life. *Mater Today Proc*. 1 de enero de 2019;16:2302-8.
- [13] Suárez DLS, Martínez IOP, Hernández ALG. Inflammatory mechanisms in periodontal destruction. *Rev Odontológica Mex* [Internet]. 2019 [January, 5th 2023];23(3). Disponible en: <https://www.revistas.unam.mx/index.php/rom/article/view/75626>
- [14] Freire MO, Van Dyke TE. Natural resolution of inflammation. *Periodontol 2000*. octubre de 2013;63(1):149-64.
- [15] Bodineau A, Coulomb B, Folliguet M, Igondjo-Tchen S, Godeau G, Brousse N, et al. Do Langerhans cells behave similarly in elderly and younger patients with chronic periodontitis? *Arch Oral Biol*. febrero de 2007;52(2):189-94.
- [16] Belibasakis GN. Microbiological changes of the ageing oral cavity. *Arch Oral Biol*. diciembre de 2018;96:230-2.
- [17] Costa MR, Silvério KG, Rossa CJ, Cirelli JA. Periodontal conditions of teeth presenting pathologic migration. *Braz Oral Res*. 2004;18(4):301-5.
- [18] Papapanou PN, Kornman KS. Periodontitis Classification. *J Am Dent Assoc* 1939. marzo de 2020;151(3):159.
- [19] Burt BA. Periodontitis and aging: reviewing recent evidence. *J Am Dent Assoc* 1939. marzo de 1994;125(3):273-9.
- [20] Wong FMF, Ng YTY, Leung WK. Oral Health and Its Associated Factors Among Older Institutionalized Residents- A Systematic Review. *Int J Environ Res Public Health*. 26 de octubre de 2019;16(21):4132.
- [21] Durham J, Fraser HM, McCracken GI, Stone KM, John MT, Preshaw PM. Impact of periodontitis on oral health-related quality of life. *J Dent*. abril de 2013;41(4):370-6.
- [22] Otomo-Corgel J, Pucher JJ, Rethman MP, Reynolds MA. State of the Science: Chronic Periodontitis and Systemic Health. *J Evid Based Dent Pract*. September, 1st 2012;12(3, Supplement):20-8.
- [23] Fernández PCV, Fernández GLH, Rodríguez JS, Villalón SA. Autocuidado de las encías y salud periodontal. *Rev Inf Científica*. October, 23th 2018;97(4):868-79.

- [24] Schaudinn C, Gorur A, Keller D, Sedghizadeh PP, Costerton JW. Periodontitis: An Archetypical Biofilm Disease. *J Am Dent Assoc.* 1 de agosto de 2009;140(8):978-86.
- [25] Escudero-Castaño N, Perea-García MA, Bascones-Martínez A. Chronic periodontitis review: evolution and its clinical application. *Av En Periodoncia E Implantol Oral.* abril de 2008;20(1):27-37.
- [26] Kato T, Abrahamsson I, Wide U, Hakeberg M. Periodontal disease among older people and its impact on oral health-related quality of life. *Gerodontology.* 2018;35(4):382-90.
- [27] Belibasakis GN. Microbiological changes of the ageing oral cavity. *Arch Oral Biol.* December, 1st 2018;96:230-2.
- [28] Costa MR, Silvério KG, Rossa CJ, Cirelli JA. Periodontal conditions of teeth presenting pathologic migration. *Braz Oral Res.* 2004;18(4):301-5.
- [29] Tonetti MS, Greenwell H, Kornman KS. Staging and grading of periodontitis: Framework and proposal of a new classification and case definition. *J Clin Periodontol.* 2018;45(S20):S149-61.
- [30] Prevalencia de periodontitis crónica en Iberoamérica [January, 6th2023]. https://www.scielo.cl/scielo.php?script=sci_arttext&pid=S0719-01072016000200020
- [31] Jaramillo RM, Duque AD. Condiciones Modificas del Riesgo de Enfermedad Periodontal: una revisión Narrativa Sobre la Evidencia en América Latina. *CES Odontol.* 15 de junio de 2021;34(1):82-99.
- [32] Billings M, Holtfreter B, Papapanou PN, Mitnik GL, Kocher T, Dye BA. Age-dependent distribution of periodontitis in two countries: Findings from NHANES 2009 to 2014 and SHIP-TREND 2008 to 2012. *J Periodontol.* 2018;89(S1):S140-58.
- [33] Halpern LR. The Geriatric Syndrome and Oral Health: Navigating Oral Disease Treatment Strategies in the Elderly. *Dent Clin North Am.* enero de 2020;64(1):209-28.
- [34] Manresa C, Sanz-Miralles EC, Twigg J, Bravo M. Supportive periodontal therapy (SPT) for maintaining the dentition in adults treated for periodontitis. *Cochrane Database Syst Rev.* 1 de enero de 2018;2018(1):CD009376.
- [35] Tawse-Smith A. Age and oral health: current considerations. *Braz Oral Res.* 2007;21:29-33.
- [36] Gamil O, Ahmed D, Keraa K, Ghallab N, Elbattawy W. Frequency and Risk Indicators of Periodontal Diseases in a Sample of Adult Egyptian Patients: A Hospital-Based Cross-Sectional Study. 22 de diciembre de 2021;
- [37] Holde GE, Oscarson N, Trovik TA, Tillberg A, Jönsson B. Periodontitis Prevalence and Severity in Adults: A Cross-Sectional Study in Norwegian Circumpolar Communities. *J Periodontol.* 2017;88(10):1012-22.
- [38] E LM, Vm MN, E SO. Mesenchymal Stem Cells for Periodontal Tissue Regeneration in Elderly Patients. *J Gerontol A Biol Sci Med Sci [Internet].* 16 de agosto de 2019 [citado 4 de enero de 2023];74(9). Disponible en: <https://pubmed.ncbi.nlm.nih.gov/30289440/>
- [39] Jepsen K, Jepsen S. Antibiotics/antimicrobials: systemic and local administration in the therapy of mild to moderately advanced periodontitis. *Periodontol 2000.* junio de 2016;71(1):82-112.
- [40] Pérez-Barrero BR, Ortiz-Moncada C, Zambrano-Rivero Y, Garbey-Bonne Z, González-Rodríguez W del C. Effectiveness of antimicrobials in the treatment of scaling and root planing in the elderly. *Rev Inf Científica.* 2020;99(2):124-33.
- [41] Farina R, Simonelli A, Baraldi A, Pramstraller M, Minenna L, Toselli L, et al. Tooth loss in complying and non-complying periodontitis patients with different periodontal risk levels during supportive periodontal care. *Clin Oral Investig.* 1 de octubre de 2021;25(10):5897-906.
- [42] Periodontal disease prevention. Mechanical Dentobacterial Plate Control Methods [Internet]. [citado 5 de enero de 2023]. Disponible en: http://scielo.sld.cu/scielo.php?script=sci_arttext&pid=S1028-48182019000200386&lng=es.
- [43] Badanian A, Bueno L, Papone V, Badanian A, Bueno L, Papone V. Comparative bacterial analysis of chronic and aggressive periodontitis pictures in a sample population of Uruguay. *Odontoestomatología.* junio de 2019;21(33):5-13.
- [44] Zhang Y, Kang N, Xue F, Duan J, Chen F, Cai Y, et al. Survival of nonsurgically splinted mandibular anterior teeth during supportive maintenance care in periodontitis patients. *J Dent Sci.* 1 de enero de 2023;18(1):229-36.
- [45] Ultrasound subgingival scaling combined with manual root planing for treatment of chronic periodontitis in elderly patients [Internet]. [citado 4 de enero de 2023]. Disponible en: <https://www.j-smu.com/CN/10.12122/j.issn.1673-4254.2020.05.18>