Periodontal disease and muscle recovery: the concise systematic review

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Abstract

Introduction: Periodontal Disease (PD) occurs with high frequency in the adult population, being an infectious and inflammatory disease of the soft and hard tissues around the teeth. PD can increase the levels of several pro-inflammatory cytokines. Objective: Was to conduct a concise systematic review of the relationship between periodontal disease and muscle repair.

Methods: The present study was followed by a systematic review model (PRISMA). The search strategy was performed in the PubMed, Scielo, Cochrane Library, Web of Science and Scopus, and Google Scholar databases, following the rules of the word PICOS (Patient; Intervention; Control; Outcomes; Study Design). The Cochrane Instrument was used to assess the risk of bias of the included studies. Results and Conclusion: The results showed that it is possible that changes in serum levels of inflammatory markers, triggered by different chronic inflammatory situations such as periodontal disease, may indirectly influence the muscle repair process. It was observed that periodontal disease was able to modify the leukocyte count and the levels of IL-6, IL-10, and TNF-α. When related to physical exercise, periodontal disease negatively influenced the muscle repair process.


Introduction

In the context of periodontal diseases (PD), this problem occurs with a high frequency in the adult population, being an infectious and inflammatory disease of the soft and hard tissues around the teeth [1]. In this aspect, the gingiva and periodontium around the teeth can develop an inflammatory response due to the chronic accumulation of oral bacteria, which can modify several local and systemic inflammatory mediators [2-4].

In this scenario, PD can increase the levels of several pro-inflammatory cytokines [5], being a risk factor for several diseases, and can modify the response of skeletal muscle cells [6-8]. In this sense, the increase in mechanical or chemical stress on skeletal muscle cells leads to a decrease in the repair of damaged fibers [9-12]. Eccentric exercises with moderate intensities can cause muscle damage affecting muscle structure and performance [13].

Thus, some substances stimulated by exercises, such as free radicals and inflammatory mediators, can induce serious alterations in the structure of crucial molecules responsible for maintaining cell homeostasis, resulting in a possible loss of cell function or vitality [14-19]. The tissue repair process involves the synchronized action of leukocytes and cytokines in the injured areas to restore homeostasis and tissue structure [20-22].

In this context, the interruption of this process can compromise the efficiency of the inflammatory response and result in reduced tissue repair [23]. Furthermore, the structural micro-injuries that occur during muscle activity can cause a decrease in the capacity to produce force, which alters the maximum force produced by the tissue, in addition to increasing the tension of the muscle's passive structures [24]. This process increases the number of proteins in the blood, generating additional inflammatory responses linked to microlesions [24,25]. Therefore, it is possible that changes in serum levels of inflammatory markers, triggered by different chronic inflammatory situations such as PD, may indirectly influence the muscle repair process.

Thus, the present study aimed to carry out a...
concise systematic review of the relationship between periodontal disease and muscle repair.

Methods

Study Design

The present study followed a systematic review model, following the rules of systematic review - PRISMA (Transparent reporting of systematic review and meta-analysis, access available in: http://www.prisma-statement.org/).

Data Sources And MeSH Terms

The search strategy was performed in the PubMed, Scielo, Cochrane Library, Web of Science and Scopus, and Google Scholar databases, using scientific articles from 2002 to 2021. The main MeSH Terms used were “Periodontal disease, Infectious processes, Cytokines, Muscle repair”. For greater specification, the description “Periodontal disease and Inflammatory processes” for refinement was added during the searches, following the rules of the word PICOS (Patient; Intervention; Control; Outcomes; Study Design).

Selection Of Studies And Risk Of Bias In Each Study

Two independent reviewers (1 and 2) performed research and study selection. Data extraction was performed by reviewer 1 and fully reviewed by reviewer 2. A third investigator decided some conflicting points and made the final decision to choose the articles. Only studies reported in Portuguese and English were evaluated. The Cochrane Instrument was used to assess the risk of bias of the included studies.

Results

A total of 118 articles were found involving periodontal disease and inflammatory processes. Initially, the duplication of articles was excluded. After this process, the abstracts were evaluated and a new exclusion was performed, based on the elimination of articles with biases that could compromise the reliability of the results, according to the rules of the Cochrane instrument, as well as articles that presented low quality in their methodologies, according to the GRADE classification. A total of 56 articles were fully evaluated and 42 were included in this study (Figure 1).

After a thorough analysis of the studies selected in this review, it was found that several infectious and inflammatory diseases, such as PD, can modulate the degree of a systemic immune response, regulating the profile of circulating leukocytes and the serum levels of interleukins, which can influence the balance of regeneration/repair [26,27]. Thus, in periodontal tissues, the stimulation and release of inflammatory mediators and immune system cells, cytokines such as IL-1β, IL-6, IL8, and TNF-α, modulate the immune response [28,29].

Furthermore, PD contributes to the maintenance of chronic inflammation through the activation of specific intracellular pathways [30]. It has been shown that PD induces an increase in pro-inflammatory mediators that is associated with the repair of injured striated muscles. Furthermore, PD is responsible for increases in lymphocytes and neutrophils. Also, physical exercise associated with PD can result in higher levels of eosinophils. Thus, physical exercise can trigger different levels of muscle damage, and the muscle repair process involves the activation of satellite cells, which are myogenic precursor cells [27]. Activation of these cells is influenced by local or systemic levels of inflammatory mediators such as IL-6 [31-33].

Furthermore, the release of cytokines such as IL-6 and TNF-α in muscle tissue due to chronic inflammatory processes has shown a high capacity to compromise the homeostasis of local metabolic pathways, since the presence of inflammatory macrophages in the injured area of skeletal muscle damages repaired tissue [34].
Patients with PD showed a significant difference in the increase in neutrophils, which can delay the healing process, impairing tissue repair.

In addition, reactive oxygen species (ROS) and proteases are released, as well as by phagocytosis and monocyte recruitment by cytokine release [35,36]. Although leukocytes are required for proper tissue repair through the secretion of growth factors and cytokines that coordinate myogenesis factors, dysregulation of inflammatory cytokine expression resulting from increased recruitment of these cells or prolonged accumulation in the injured area may lead to a delay in the healing process [37-40].

In addition, leukocyte results also indicate a significant increase in eosinophils in physically active PD patients. The inflammatory process also increased the activation of eosinophils, which contribute to muscle lysis by lymphocytes and an increase in the production of major basic protein-1 (major basic protein-1, MBP-1). In addition, eosinophils can also increase muscle fibrosis with MBP-dependent processes and negatively regulate the cellular immune response to injured muscle [27].

Besides, monocyte-macrophage precursor levels are reduced in patients with PD and increased in patients who are physically active and free from the disease [40]. Thus, PD slows down the muscle repair process. Furthermore, the association of PD with physical exercise can have an additive effect, being responsible for the prolongation of local inflammatory events. The results obtained in the analysis of serum levels of inflammatory mediators indicate that PD can alter the levels of IL-6, TNF-α, and IL-10, with a cumulative effect with exercise. The increase in cytokines such as IL-6 and TNF-α, after muscle injury, indicates that these substances can contribute to the regeneration process.

Also, PD may have an additive effect on the inflammatory response, inducing greater IL-10 recruitment [31]. Analyzes of serum levels of inflammatory mediators in PD indicate altered levels of IL-6, TNF-α, and IL-10 and an additive effect when combined with physical exercise for IL-6 and IL-10. The increase in cytokines such as IL6 and TNF-α after muscle injury indicates that these substances can contribute to the regenerative process, stimulating inflammatory cells to start the tissue repair process [26,41].

Finally, a study showed the case of a woman with Class III malocclusion and advanced PD who was treated with surgical orthodontic correction. Functional recovery after orthodontic treatment is often monitored by serial electromyography of masticatory muscles. Outcomes for this patient included stable occlusion and improved facial aesthetics. This case report illustrates the benefits of setting acceptable treatment goals for the patient, based on an accurate three-dimensional assessment of the dentoalveolar bone and using the muscle activity of chewing to monitor occlusion stability [42].

**Conclusion**

The results showed that it is possible that changes in serum levels of inflammatory markers, triggered by different chronic inflammatory situations such as periodontal disease, can indirectly influence the muscle repair process. It was observed that periodontal disease was able to modify the leukocyte count and the levels of IL-6, IL-10, and TNF-α. When related to physical exercise, periodontal disease negatively influenced the muscle repair process.

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No additional data are available.

**Conflict of interest**

The authors declare no conflict of interest.

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