MedNEXT
Journal of Medical an Health Sciences

REVIEW ARTICLE

DOI: 10.54448/mdnt22305

Periodontal disease and muscle recovery: the concise systematic review

Beatriz Dourado Sant'Ana[1], Carolaine Naira Bogas[1], Patrícia Garani Fernandes[1,2]*

[1] UNORTE - University Center of Northern São Paulo - Dentistry department, Sao Jose do Rio Preto, Sao Paulo, Brazil.
[2] UNIPOS - Post graduate and continuing education, Dentistry department, Sao Jose do Rio Preto, Sao Paulo, Brazil.

*Corresponding author: Dr. Patrícia Garani Fernandes.
UNORTE/UNIPOS - Graduate and Postgraduate in Dentistry, Sao Jose do Rio Preto, Sao Paulo, Brazil.
E-mail: patriciagarani@gmail.com
DOI: https://doi.org/10.54448/mdnt22305
Received: 02-12-2022; Revised: 04-25-2022; Accepted: 06-18-2022; Published: 07-11-2022; MedNEXT-id: e22305

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.

Keywords: Periodontal disease. Inflammatory processes. Infectious processes. Cytokines. Muscle repair.

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. Inflammatory processes. 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

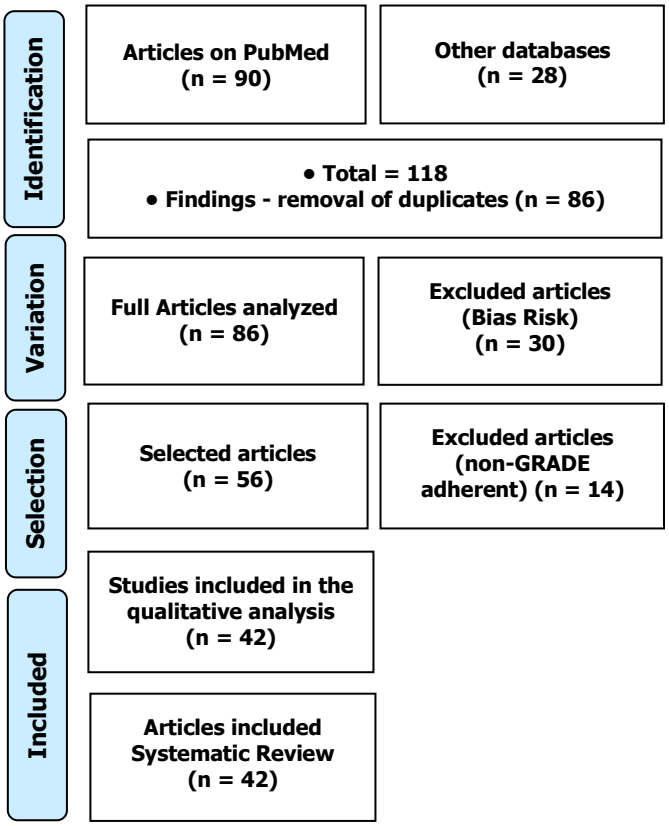
Two independent reviewers (1 and 2) performed research and study selection. Data extraction was performed by reviewer 1 and fully reviewed by reviewer 2. A third investigator decided some conflicting points and made the final decision to choose the articles. Only studies reported in Portuguese and English 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].

Figure 1. Flowchart showing the article selection process.



Furthermore, PD contributes to the maintenance of chronic inflation 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.

Acknowledgement

Not applicable.

Funding

Not applicable.

Ethics approval

Not applicable.

Informed consent

Not applicable.

Data sharing statement

No additional data are available.

Conflict of interest

The authors declare no conflict of interest.

Similarity check

It was applied by Ithenticate@.

About the License

© The authors (s) 2022. The text of this article is open access and licensed under a Creative Commons Attribution 4.0 International License.

References

1. Wong LB, Yap AU, Allen PF. Periodontal disease and quality of life: Umbrella review of systematic reviews. J Periodontal Res. 2021 Jan;56(1):1-17. doi: 10.1111/jre.12805. Epub 2020 Sep 23. PMID: 32965050.
2. Yang Y, Huang Y, Li W. Autophagy and its significance in periodontal disease. J Periodontal Res. 2021 Jan;56(1):18-26. doi: 10.1111/jre.12810. Epub 2020 Nov 27. PMID:

33247437.

3. Fi C, Wo W. Periodontal disease and systemic diseases: an overview on recent progresses. J Biol Regul Homeost Agents. 2021 Jan-Feb;35(1 Suppl. 1):1-9. PMID: 33463138.
4. Bostanci N, Bao K, Greenwood D, Silbereisen A, Belibasakis GN. Periodontal disease: From the lenses of light microscopy to the specs of proteomics and next-generation sequencing. Adv Clin Chem. 2019;93:263-290. doi: 10.1016/bs.acc.2019.07.006. Epub 2019 Aug 30. PMID: 31655732.
5. Kajiya M, Kurihara H. Molecular Mechanisms of Periodontal Disease. Int J Mol Sci. 2021 Jan 19;22(2):930. doi: 10.3390/ijms22020930. PMID: 33477754; PMCID: PMC7832304.
6. Lasserre, J.F., M.C. Brecx, and S. Toma. 2018. Oral Microbes,Biofilms and Their Role in Periodontal and Peri-implant Diseases.Materials (Basel)11: E1802.
7. Irfan, U.M., D.V. Dawson, and N. Bissada. 2001. Epidemiology ofperiodontal disease: a review and clinical perspectives.Journal ofthe International Academy of Periodontology3: 14–21.
8. Teles, R.P., V. Likhari, S.S. Socransky, and A.D. Haffajee. 2009.Salivary Cytokine Levels in Chronic Periodontitis and PeriodontallyHealthy Subjects. A cross-sectional Study.Journal of PeriodontalResearch44: 411–417.
9. Keelan, J.A., P.M. Wong, P.S. Bird, and M.D. 2010. Mitchell. Innateinflammatory responses of human decidual cells to periodontopathicbacteria.American Journal of Obstetrics and Gynecology202: 1–11.389Periodical Disease Impairs Muscle Recovery.
10. Eming, S.A., M. Hammerschmidt, T. Krieg, and U. Roers. 2009.Interrelation of immunity and tissue repair or regeneration.Seminarsin Cell & Developmental Biology20: 517–527.
11. Panezai, J., A. Ghaffar, M. Altamash, K.G. Sundqvist, P.E.Engström, and A. Larsson. 2017. Correlation of serum cytokines,chemokines, growth factors and enzymes with periodontal diseaseparameters.PLoS One12: e0188945.
12. Kampits, C., M.M. Montenegro, I.W. Ribeiro, M.V. Furtado, C.A.Polanczyk, C.K. Rösing, and A.N. Haas. 2016. Periodontal diseaseand inflammatory blood cytokines in patients with stable coronaryartery disease.Journal of Applied Oral Science24: 352–358.
13. Seymour, G.J., P.J. Ford, M.P. Cullinan, S. Leishman, and K.Yamazaki. 2007. Relationship between periodontal infections andsystemic disease.Clinical Microbiology and Infection13: 3–10.
14. Leite, M.A., T.M. de Mattia, C.M.M. Kakihata, B.M. Bortolini, P.H.de Carli Rodrigues, G.R.F. Bertolini, R.M.C. Brancalhão, L.F.C.Ribeiro, C.A. Nassar, and P.O. Nassar. 2017. Experimental Periodontitis in the Potentialization of the Effects of Immobilism in theSkeletal Striated Muscle.Inflammation40: 2000–2011.
15. Souza, B.C., M.E. Ribas, A.R. Oliveira, J.B. Burzlaff, and A.N.Haas. 2012. Impact of periodontal inflammation on changes of amarker of muscle injury in young soccer players during training.Revista Odonto Ciencia27: 294–299.
16. Souza, B.C., M.E. Ribas, A.L. Lopes, B.C. Teixeira, and M.L.Lamers. 2017. Periodontal disease influences the recovery processesin the muscles in trained mice.Journal of Physical Education andSport17: 572–581.
17. Lieber, R.L., S. Shah, and J. Fridén. 2002. Cytoskeletal disruptionafter eccentric contraction-induced muscle injury.Clinical Ortho-paedics403: S90–S99.
18. Paschalis, V., Y. Koutedakis, A.Z. Jamurtas, V. Mougios, and V.Baltzopoulos. 2005. Equal volumes of high and low intensity ofeccentric exercise in relation to muscle damage and performance.Journal of Strength and Conditioning Research19: 121–125.
19. Paulsen, G., U.R. Mikkelsen, T. Raastad, and J.M. Peake. 2012.Leucocytes, cytokines and satellite cells: what role do they play inmuscle damage and regeneration following eccentric exercise?Exercise Immunology Review18: 42–97.
20. Soehnlein, O., and L. Lindbom. 2010. Phagocyte partnership duringthe onset and resolution of inflammation.Nature Reviews. Immu-nology10: 427–439.
21. Malm, C. 2001. Exercise-induced muscle damage and inflamma-tion:factorfiction?Acta Physiologica Scandinavica171: 233–239.
22. Tidball, J.G. 2005. Mechanical signal transduction in skeletal mus-cle growth and adaptation.Journal of Applied Physiology98: 1900–1908.
23. Clarkson, P.M., and M.J. Hubal. 2002. Exercise-induced MuscleDamage in Humans.Am J Phys Rehabil81: S52–S69.
24. Proske, U., and D.L. Morgan. 2001. Muscle damage from eccentricexercise: mechanism, mechanical signs, adaptation and clinical applications.The Journal of Physiology537: 333–345.
25. Cheung, K., P. Hume, and L. Maxwell. 2003. Delayed onset musclesoreness—treatment

strategies and performance factors.SportsMedicine33: 145–164.

26. Villalta, S.A., C. Rinaldi, B. Deng, G. Liu, B. Fedor, and J.G.Tidball. 2011. Interleukin-10 reduces the pathology of mdx muscu-lar dystrophy by deactivating M1 macrophages and modulatingmacrophage phenotype.Human Molecular Genetics20: 790–805.
27. Tidball, J.G., and S.A. Villalta. 2010. Regulatory interactions be-tween muscle and the immune system during muscle regeneration.American Journal of Physiology. Regulatory, Integrative and Com-parative Physiology298: 1173–1187.
28. Noh, M.K., M. Jung, S.H. Kim, S.R. Lee, K.H. Park, D.H. Kim,H.H. Kim, and Y.G. Park. 2013. Assessment of IL-6, IL-8 and TNF-αlevels in the gingival tissue of patients with periodontitis.Exper-imental and Therapeutic Medicine6: 847–851.
29. Lomba, K.S.B., T.F.C. de Souza Breves Beiler, M.R.C. Sete, F.R.Pires, and C.M. da Silva Figueredo. 2015. Use of minimally invasivegingival biopsies in the study of inflammatory mediators expressionand their correlation with gingival fluid in patients with chronicperiodontitis.Indian Journal of Dental Research26: 126–130.
30. Hoare, A., C. Soto, V. Rojas-Celis, and D. Bravo. 2019. ChronicInflammation as a Link between Periodontitis and Carcinogenesis.Mediators of Inflammation1029857.
31. Dias, I.H.K., I.L.C. Chapple, M. Milward, M.M. Grant, E. Hill, J.Brown, and H.R. Griffiths. 2013. Sulforaphane restores cellularglutathione levels and reduces chronic periodontitis neutrophilshyperactivityin vitro.PLoS One8: e66407.
32. Chapple, L.C., and J.B. Matthews. 2007. The role of reactive oxygenand antioxidant species in periodontal tissue destruction.Periodontol 2000(43): 160–232.
33. Matthews, J.B., H.J. Wright, A. Roberts, P.R. Cooper, and I.L.Chapple. 2007. Hyperactivity and reactivity of peripheral blood390de Souza, Matte, Lopes, Teixeira, and Lamers neutrophils in chronic periodontitis.Clinical and Experimental Im-munology147: 255–264.
34. Jin, R.M., J. Warunek, and E.A. Wohlfert. 2018. Therapeutic ad-ministration of IL-10 and amphiregulin alleviates chronic skeletalmuscle inflammation and damage induced by infection.Immunohorizons2: 142–154.
35. Lockhart, N.C., and S.V. Brooks. 2008. Neutrophil accumulationfollowing passive stretches contributes to adaptations that reducecontraction-induced skeletal muscle injury in mice.Journal of Ap-plied Physiology104: 1109–1115.
36. Pizza, F.X., J.M. Peterson, J.H. Baas, and T.J. Koh. 2005. Neutro-phils contribute to muscle injury and impair its resolution afterlengthening contractions in mice.The Journal of Physiology562:899–913.
37. Gordon, S., and P.R. Taylor. 2005. Monocyte and macrophageheterogeneity.Nature Reviews. Immunology5: 953–964.
38. Lagrota-Candido, J., I. Canella, D.F. Pinheiro, L.P. Santos-Silva,R.S. Ferreira, F.J. Guimarães-Joca, J. Lannes-Vieira, and T.Quirico-Santos. 2010. Characteristic pattern of skeletal muscle re-modelling in different mouse strains.International Journal of Ex-perimental Pathology91: 522–529.
39. Perdiguero, E., Y. Kharraz, A.L. Serrano, and P. Munoz-Canoves.2012. MKP-1 coordinates ordered macrophage-phenotype transi-tions essential for stem cell-dependent tissue repair.Cell Cycle11:877–886.
40. Cantini, M., E. Giurisato, C. Radu, S. Tiozzo, F. Pampinella, D.Senigaglia, L. Zaniolo, F. Mazzoleni, and L. Vitiello. 2002.Macrophage-secreted myogenic factors: a promising tool for greatlyenhancing the proliferative capacity of myoblastsin vitroandin vivo.Neurological Sciences23: 189–194.
41. de Souza BC, Matte BF, Lopes AL, Teixeira BC, Lamers ML. Periodontal Disease Impairs Muscle Recovery by Modulating the Recruitment of Leukocytes. Inflammation. 2020 Feb;43(1):382-391. doi: 10.1007/s10753-019-01128-5. Erratum in: Inflammation. 2020 Dec;43(6):2392. PMID: 31760525.
42. Nakajima K, Yamaguchi T, Maki K. Surgical orthodontic treatment for a patient with advanced periodontal disease: evaluation with electromyography and 3-dimensional cone-beam computed tomography. Am J Orthod Dentofacial Orthop. 2009 Sep;136(3):450-9. doi: 10.1016/j.ajodo.2007.03.042. PMID: 19732680.

FACERES SCHOOL OF MEDICINE
https://faceres.com.br/

MetaScience Press The Best Science
https://zotarellifilhoscientificworks.com/