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PATHOPHYSIOLOGICAL MECHANISMS OF THE INFLUENCE OF RENAL FAILURE ON INFLAMMATORY PROCESSES IN PERIODONTAL TISSUES

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Annotation: Chronic renal failure (CRF) is a systemic disease characterized by accumulation of uremic toxins, impaired microcirculation, and activation of systemic inflammation. These processes have a significant impact on periodontal tissues, enhancing inflammatory responses and leading to destruction of connective and bone tissue. This article reviews the pathophysiological mechanisms linking CRF with inflammatory periodontal diseases, including the role of uremic toxins, calcium-phosphorus imbalance, and oxidative stress. Promising therapeutic approaches, including the use of biomarkers , new-generation antioxidants, and regenerative technologies, are also discussed. This work highlights the need for an interdisciplinary approach to the diagnosis and treatment of patients with CRF to minimize negative consequences on the periodontium.

Keywords: chronic renal failure, inflammatory periodontal diseases, uremic toxins, oxidative stress, biomarkers , regenerative technologies, systemic inflammation.

Introduction

The relevance of studying the pathophysiological mechanisms underlying the relationship between renal failure and inflammatory processes in periodontal tissues is due to the high prevalence of chronic renal failure (CRF), which, according to statistics, affects about 10% of the adult population worldwide, as well as its systemic impact on the body. CRF is accompanied by metabolic disorders, activation of systemic inflammation, and accumulation of uremic toxins, which can significantly affect the condition of the periodontium. Periodontal tissues, being highly sensitive to changes in microcirculation and inflammatory processes, are subject to significant destructive changes under the influence of chronic pathologies.

The aim of this review article is to summarize current data on the mechanisms linking renal failure with inflammatory periodontal diseases, as well as to identify key pathogenetic links that can serve as a basis for the development of new therapeutic approaches.

The main mechanisms of renal failure

Chronic renal failure (CRF) is a progressive impairment of renal function resulting in accumulation of metabolites, electrolyte imbalance, and systemic changes in the body. The main pathogenetic mechanisms are uremic intoxication, including accumulation of toxins such as p- cresyl sulfate and indoxyl sulfate , activation of the renin-angiotensin- aldosterone system (RAAS) accompanied by hypertension and vascular damage, chronic inflammation, and increased oxidative stress, which disrupts normal endothelial and tissue functions. Uremic toxins such as p- cresyl sulfate , indoylacetic acid, and guanidinosuccinate have pronounced proinflammatory properties, increasing oxidative stress and damaging the vascular endothelium.

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Activation of RAAS in CRF leads to vasoconstriction , increased systemic arterial pressure and vascular hypertrophy, which in turn disrupts microcirculation and promotes the progression of inflammatory processes. Chronic inflammation is one of the key links in the pathogenesis of CRF and is accompanied by increased expression of proinflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α) and C-reactive protein (CRP). These mediators enhance destructive changes in tissues, including periodontal structures.

The role of inflammatory processes in the pathogenesis of periodontal diseases

Periodontal diseases, including gingivitis and periodontitis, are inflammatory pathologies that arise as a result of dysbiosis of the oral microbiota and a decrease in the body's defense mechanisms. The main pathogenetic mechanisms include activation of innate and adaptive immunity in response to lipopolysaccharides (LPS) of pathogenic bacteria, such as *Porphyromonas gingivalis* , *Tannerella forsythia* and *treponema denticola* . These microorganisms stimulate the production of cytokines such as IL-1β, IL-6, IL-8 and TNF-α, which promote collagen degradation, vascular permeability impairment and osteoclast activation. The interaction of these cytokines activates immune cells such as macrophages and neutrophils, which release reactive oxygen species and proteolytic enzymes that enhance the inflammatory process. Increased levels of TNF-α stimulate the expression of adhesion molecules on endothelial cells, promoting leukocyte migration into the tissue, which increases local inflammation. IL-1β and IL-6, in turn, promote the production of prostaglandins, which enhance the destruction of connective tissue and bone resorption.

An important role in the development of inflammatory processes is played by oxidative stress, which leads to damage to periodontal cells, a decrease in their regenerative potential and increased apoptosis. This, together with microcirculatory disorders, creates conditions for chronic inflammation and progressive tissue destruction.

The relationship between chronic renal failure and periodontal disease

Numerous studies confirm that patients with CRF have an increased risk of developing and progressing periodontal diseases. Uremic toxins circulating in the blood penetrate into periodontal tissues, where they enhance inflammatory reactions. Chronic systemic inflammation in CRF aggravates local inflammatory processes by increasing the level of IL-6, TNF-α and other mediators. In addition, microcirculation disorders and endothelial dysfunction characteristic of CRF prevent normal regeneration of periodontal tissues.

Of particular interest is the finding that hemodialysis patients have a significant deterioration in periodontal health associated with high levels of proinflammatory cytokines and calcium-phosphorus imbalance. Hormonal imbalances such as decreased vitamin D levels also contribute to alveolar bone loss and connective tissue deterioration.

Discussion

The obtained data on the pathophysiological mechanisms of the influence of chronic renal failure on inflammatory processes in periodontal tissues emphasize the relationship between systemic inflammation and local pathological changes in the oral cavity. Uremic toxins

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circulating in the body during CRF initiate chronic inflammation in periodontal tissues due to the activation of proinflammatory cytokines and increased oxidative stress. At the same time, there is a pronounced violation of microcirculation in periodontal tissues, which aggravates the inflammatory process and contributes to the progressive destruction of bone and connective tissue.

Systemic changes associated with CRF, including activation of the renin-angiotensin - aldosterone system and imbalance of calcium-phosphorus metabolism, have a significant impact on the periodontium . Increased levels of interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) promote osteoclast activation, which leads to accelerated alveolar bone resorption. Moreover, vitamin D deficiency, typical for patients with CRF, reduces the regenerative potential of tissues, impairing their ability to recover from inflammatory damage.

It is interesting that treatment of patients with CRF, including control of uremic toxins and proinflammatory markers, improves the condition of the periodontium. Clinical studies show that regular hemodialysis reduces the concentration of toxins, and vitamin D therapy reduces the severity of inflammatory reactions. The introduction of new antioxidants, such as N-acetylcysteine, and anti-inflammatory drugs has demonstrated a significant decrease in inflammation markers in periodontal tissues. The most effective control methods are hemodialysis, which reduces the concentration of uremic toxins, and correction of vitamin D levels aimed at restoring mineral balance and reducing inflammatory reactions. In addition, therapy with anti-inflammatory drugs and antioxidants, such as tocopherol and N-acetylcysteine, has demonstrated a positive effect on reducing oxidative stress in periodontal tissues. This confirms the need for an integrated approach, including both systemic and local treatment methods for this category of patients.

Conclusion

Chronic renal failure and inflammatory periodontal diseases mutually reinforce each other through complex pathophysiological mechanisms including systemic and local inflammation, uremic intoxication, oxidative stress and microcirculation disorders. These interactions highlight the need for a multidisciplinary approach to the treatment of patients with CRF, including monitoring and therapy of inflammatory periodontal diseases. Future research should be aimed at developing therapeutic strategies that take into account the systemic nature of these diseases and their impact on periodontal tissues. It is important to study the use of biomarkers such as IL-6 and TNF-α levels to predict the risk of inflammation, as well as the development of innovative therapies, including the use of stem cells to restore damaged periodontal tissues and nanomaterials for local drug delivery. Promising areas include the use of biomarkers for early diagnostics of inflammatory processes in the periodontium in patients with chronic renal failure, as well as the introduction of innovative therapies, such as the use of anti-inflammatory biopreparations, new-generation antioxidants, and regenerative techniques using stem cells. These approaches can significantly improve clinical outcomes and minimize the negative impact of systemic diseases on the periodontium.

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