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Research Article

CLINICAL FEATURES AND IMMUNOLOGICAL MECHANISMS OF DEVELOPMENT OF OSTEOARTHRITIS OF THE FEMORAL HIP JOINT

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ABSTRACT

Osteoarthritis (OA) is one of the most common chronic degenerative joint diseases, significantly affecting the quality of life of patients. The COVID-19 pandemic has revealed the long-term effects of the SARS-CoV-2 virus, including the development of OA. The object of this study is the impact of COVID-19 on the clinical, immunological, and genetic features of OA. The article discusses the mechanisms leading to inflammatory processes in the joints, as well as genetic aspects that may be associated with cartilage regeneration. Patients after COVID-19 have more pronounced OA symptoms, which confirms the need for further study of this relationship to develop effective approaches to treatment and rehabilitation.

KEYWORDS

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Osteoarthritis (OA), COVID-19, SARS-CoV-2 virus, femoral hip joint, interleukin-6 (IL-6) and interleukin-8 (IL-8), tumor necrosis factor-alpha (TNF-α), non-steroidal anti-inflammatory drugs (NSAIDs).

INTRODUCTION

Osteoarthritis (OA) is one of the most common chronic degenerative joint pathologies, significantly affecting patients' quality of life and ability to work. The COVID-19 pandemic caused by the SARS-CoV-2 virus has shown that the infection can have long-lasting effects on various body systems. Recent studies show the impact of COVID-19 on the clinical, immunological, and genetic features of OA development in patients with this infection. This literature review aims to deepen our understanding of the pathogenesis of the disease and to search for possible mechanisms of COVID-19's influence on osteoarthritis.

Impact of viral infections on the immune system: Recent studies have shown that COVID-19 can cause various immune system disturbances, including activation of inflammatory processes. SARS-CoV-2 causes an enhanced immune response accompanied by increased levels of cytokines and inflammatory markers. This inflammatory response may contribute to the development and progression of OA in COVID-19 patients.

Clinical manifestations of OA after COVID-19: Observations show that COVID-19 patients present with osteoarthritis with some peculiarities compared to normal OA. These patients have more severe pain, limitation of mobility and joint inflammation. This may be due to the effect of the virus on cartilage tissues as well as surrounding structures and joints.

Genetic aspects of OA after COVID-19: There is evidence of abnormalities in genetic mechanisms related to cartilage regeneration and metabolic processes in joints in patients with OA after COVID-19. Some genes associated with cartilage formation and destruction may play a role in the pathogenesis of OA after infection. Genetic predisposition studies help to identify risk groups and understand the molecular mechanisms involved in the pathogenesis of OA after COVID-19.

Despite the relative novelty of the problem, existing studies point to possible clinical, immunological, and genetic features of OA in COVID-19 patients. Continued research in this area is important to better understand the molecular mechanisms and to develop tailored treatment and rehabilitation strategies to prevent or delay the development of OA after COVID-19.

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Risk factors and metabolic disorders: To understand the etiopathogenesis of the disease, it is important to consider risk factors such as age, obesity, diabetes mellitus, hypertension, and dyslipidemia. One mechanism explaining the association between metabolic disorders and OA is chronic inflammation associated with metabolic diseases. Inflammatory cytokines such as interleukin-1β (IL-1β) and tumor necrosis factor-alpha (TNF-α) activate destructive processes in cartilage and promote its degeneration.

Traumatic osteoarthritis: Mechanisms associated with the development of traumatic osteoarthritis include damage to joint structures, cartilage, and synovial membrane, inflammation, and immune cell activation. Traumatic injury causes inflammatory processes that lead to the release of cytokines such as interleukin-6 (IL-6) and interleukin-8 (IL-8) that activate destructive processes in cartilage and promote cartilage degeneration.

Effect of drugs: Drugs play an important role in the treatment and management of osteoarthritis. Different classes of drugs have different effects on the pathogenesis and symptoms of osteoarthritis. For example, non-steroidal anti-inflammatory drugs (NSAIDs) are widely used to relieve pain and inflammation in OA, but their long-term use can cause serious side effects. Glucosamine and chondroitin sulfate may have a positive effect on cartilage tissue and reduce OA symptoms, but their effectiveness requires further research.

Genetic disorders: Genetic factors play an important role in the development of osteoarthritis. Several genes related to the metabolism of cartilage components, collagen structure, and proteoglycans are of potential importance in the development of the disease. For example, polymorphisms in the COL2A1 gene encoding type II collagen may contribute to impaired cartilage structure and increased risk of osteoarthritis. Genes encoding matrix metalloproteinases (MMPs) and tissue inhibitors of metalloproteinases (TIMPs), as well as cytokines such as IL-1β and TNF-α, may also play a role in the pathogenesis of osteoarthritis. Alterations in these genes may affect the activity of cartilage-related enzymes and joint inflammation.

Thus, the etiopathogenesis of osteoarthritis is a complex and multifaceted process involving various risk factors, metabolic disorders, traumatic injuries, drug effects, and genetic factors. Age, gender, obesity and occupational factors significantly influence the development of the disease. Metabolic disorders, especially those associated with metabolic syndrome, also play an important role in the pathogenesis of osteoarthritis. Traumatic osteoarthritis may result from joint injuries leading to inflammatory and degenerative processes. Medications such as NSAIDs, glucosamine and chondroitin sulfate may have a

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positive effect on osteoarthritis symptoms but require further research. Genetic factors, including polymorphisms of genes related to cartilage metabolism and inflammation, also play an important role in the development of osteoarthritis.

Etiopathogenesis of COVID-19 and its musculoskeletal complications: COVID-19, caused by the SARS-CoV-2 coronavirus, has become one of the most serious public health problems in world history. Since the start of the pandemic in late 2019, the disease continues to spread and affect various body systems. The main symptoms of COVID-19 are related to the respiratory system, but a growing body of research points to possible complications in other systems, including the musculoskeletal system.

Etiopathogenesis of COVID-19: The SARS-CoV-2 coronavirus enters body cells via angiotensin-converting enzyme 2 (ACE2) receptors widely distributed in various tissues, including lungs, heart, kidneys, blood vessels, and musculoskeletal tissues. Infection causes systemic inflammation and activation of the immune system, leading to a complex set of molecular and cellular responses.

Complications of COVID-19 in the musculoskeletal system are:

1. Myalgia and arthralgia: One of the most common symptoms of COVID-19 is muscle pain (myalgia) and joint pain (arthralgia). These symptoms may be caused by systemic exposure to the virus or the body's response to a viral infection. Studies show that myalgia occurs in 45% of patients with COVID-19. These symptoms can cause significant discomfort and limit mobility.

2. Joint inflammation: In some patients, COVID-19 may cause joint inflammation, leading to arthritis-like symptoms. Research suggests that the virus may cause inflammatory processes in the joints, leading to rheumatic symptoms. This may cause pain, swelling, and limitation of joint motion.

3. Myopathies: COVID-19 may cause musculoskeletal disorders such as myopathies. These disorders may be caused by the direct effect of the virus on the muscles or by inflammatory processes caused by the body's immune response. Patients may experience muscle weakness, pain, and limitation of motion.

4. Thrombosis and embolism: COVID-19 is associated with an increased risk of thrombotic complications such as vascular thrombosis and pulmonary embolism. These complications may be due to hypercoagulability, inflammatory changes, and damage to the vascular wall.

CONCLUSION

Thus, studies show that osteoarthritis (OA) may be exacerbated in patients after COVID-19 due to immune system disorders, clinical manifestations, and genetic

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factors. The SARS-CoV-2 virus activates inflammatory processes, which may accelerate the development of OA. In this case, specific clinical symptoms are observed, such as increased pain and limited joint mobility. Genetic mechanisms associated with cartilage regeneration may also play an important role in the pathogenesis of OA in patients after COVID-19. It is important to consider risk factors such as age, comorbid diseases, and metabolic disorders, which can aggravate the course of the disease. This emphasizes the need for further research to develop specific therapeutic strategies and rehabilitation measures.

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