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CHANGES IN THE HIP JOINT AFTER COVID-19

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ABSTRACT

This article examines the morphological and functional changes occurring in the hip joint, particularly in the femoral head, in patients following COVID-19 infection. Special attention is given to the development of aseptic necrosis as one of the serious complications associated with post-COVID syndrome. Clinical, radiological, and histological findings demonstrate that impaired blood circulation, vascular thrombosis, and endothelial dysfunction contribute significantly to the pathogenesis of bone tissue damage. MRI studies reveal characteristic changes in signal intensity depending on the stage and severity of necrosis, while morphological analysis confirms necrobiotic, dystrophic, and destructive alterations in bone trabeculae, osteoid structures, and vascular components. The progression of the disease leads to fragmentation of bone tissue and sequestrum formation. Early diagnosis and timely treatment of post-COVID musculoskeletal complications are essential to prevent disability and improve patient outcomes. The findings highlight the importance of understanding the pathogenesis, morphological features, and clinical implications of aseptic necrosis of the femoral head in post-COVID patients.

Keywords: COVID-19; post-COVID syndrome; femoral head; hip joint; aseptic necrosis; osteonecrosis; MRI; bone tissue; vascular thrombosis; morphological changes; histopathology; rehabilitation.

INTRODUCTION

At the end of 2019, the world was affected by the coronavirus infection caused by SARS-CoV-2. From the first days, international medical communities began studying the acute phase of this infection. However, it later became clear that the disease also has delayed consequences with multisystem manifestations and diverse clinical forms. In the autumn of 2021, following the proposal of the World Health Organization, the term “post-COVID syndrome,” its risk factors, pathogenesis, and clinical and morphological course were formally discussed and defined. Since post-COVID syndrome has a multisystem nature, one of its manifestations includes aseptic necrosis of bones, particularly aseptic necrosis of the femoral head, which leads to the development of necrotic and degenerative diseases (1, 2). In addition, the presence of multiple comorbidities significantly worsens the clinical course of SARS-CoV-2 infection and often increases mortality risk. Among individuals over 60 years of age, the following comorbid conditions have been reported: arterial hypertension (55.4%), diabetes mellitus (17.5%), obesity (35.5%), coronary heart disease (21.6%), and degenerative diseases of the femoral head (18.4%). Several factors play an important role in the development of post-COVID syndrome, including immunodeficiency, persistence of residual tissue damage, exacerbation of comorbidities, viral reactivation, catabolic syndrome, persistent viremia, reinfection, endothelial dysfunction, and vascular thrombosis. Osteonecrosis, which has a polyetiological origin and is characterized by damage to both bone marrow and trabecular bone structures, is one of the serious complications observed in post-COVID syndrome. Globally, osteonecrosis affects approximately 20,000 people annually (1, 4). It should be emphasized that aseptic necrosis may occur in multiple bones in a single patient. In approximately 13% of cases, it affects the femoral head and adjacent cartilage structures. In aseptic necrosis, the death of osteocytes and bone marrow cells occurs as a result of bone infarction. This process is primarily associated with

limited collateral blood supply in the arteries that nourish the bone and the development of thrombosis as a complication of COVID-19 infection. Currently, there is limited scientific information regarding the morphogenesis and pathohistological changes associated with aseptic necrosis of hip bone tissue following COVID-19. Therefore, studying the pathogenesis, morphogenesis, and histopathological changes of aseptic necrosis in bone tissue related to post-COVID syndrome remains an urgent and clinically significant scientific problem.

MATERIALS AND METHODS

The study material consisted of clinical-anamnestic data and surgically obtained tissue samples from 19 patients who were treated for dystrophic-degenerative diseases of the femoral head in 2021 at the Surkhandarya Regional Multidisciplinary Hospital. The patients ranged in age from 25 to 65 years, with a mean age of 42.6 years. All 19 patients were diagnosed with varying degrees of aseptic necrosis of the femoral head. Surgical intervention was performed in 11 cases. During surgery, the necrotic area of the femoral head, the periosteum, the articular cartilage, and part of the femoral neck were removed simultaneously. The excised tissue specimens were examined macroscopically. From each sample, fragments measuring approximately 1.5 × 1.5 cm were obtained and fixed in 10% phosphate-buffered formalin solution for 72 hours. The osseous portions of the specimens were decalcified in 10% nitric acid. After decalcification, the samples were washed in running water for 3–4 hours, dehydrated through ascending concentrations of alcohol, and embedded in paraffin wax to prepare tissue blocks. Histological sections 5–7 μm thick were cut from the paraffin blocks. The sections were deparaffinized and stained with hematoxylin and eosin (H&E). The prepared histological slides were examined under a light microscope, and microphotographs were taken of representative areas for further analysis.

RESULTS

Radiological and MRI examinations conducted in patients presenting with post-COVID syndrome revealed that aseptic necrosis in the femoral head region is characterized by the presence of necrotic foci with varying magnetic resonance signal intensities. These lesions were surrounded by crescent-shaped lines visible on T1–T2 coronal projections, as well as a characteristic double-line sign on T2–T1 coronal projections, consisting of an inner high-intensity line and an outer low-intensity line (see Figure 1). The MRI signal characteristics were found to vary depending on the degree of bone tissue necrosis. In cases where hemorrhage occurred within the necrotic focus, high-intensity MRI signals were observed in both projections. When the necrotic area was infiltrated with tissue fluid, the MRI signal intensity appeared reduced. Similarly, in cases where fibrosis and osteosclerosis had developed, the MRI signals were also characterized by low intensity. Morphological examination demonstrated that in the early stage of aseptic necrosis, pronounced edema developed in the soft tissues and periosteal structures surrounding the proximal femur. Cellular and fibrous structures appeared disorganized and fragmented, and vacuolated spaces were observed in the intercellular matrix. Significant morphological changes were particularly evident in the blood vessels within these tissues. Arterial vessels showed dystrophic and disorganized cellular and fibrous structures in their walls. As a result of edema and dilation, thickening of the vascular walls was observed (see Figure 2). Endothelial cells lining the inner surface of the arteries were enlarged and protruded into the vascular lumen. In contrast, venous vessels were dilated with thinned walls, and their lumens contained highly concentrated plasma proteins and blood cells. These vascular alterations indicate impaired microcirculation and confirm that vascular damage and thrombosis play a key role in the pathogenesis of aseptic necrosis associated with post-COVID syndrome.

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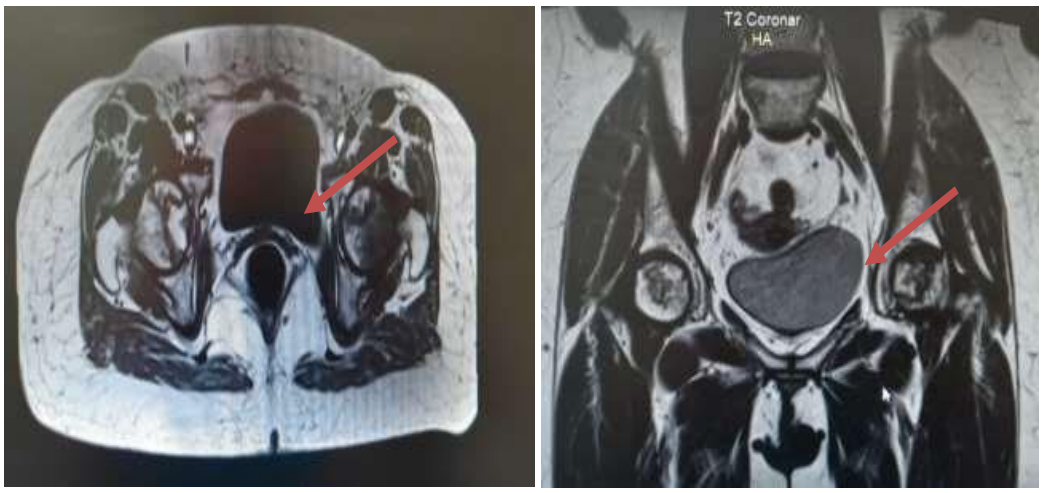

Figure 1. Aseptic necrosis of the femoral head after COVID-19: low-intensity MRI signals in T1-weighted projections and high-intensity signals in T2-weighted projections.

DISCUSSION

In the early stage of aseptic necrosis of the femoral head, circulatory, dystrophic, and necrobiotic processes develop within the bone tissue. The outer hyaline layer of the femoral head becomes slightly thickened compared to normal, and the connective tissue cells within it enlarge in response to ischemia. These cells undergo dystrophy and disorganization, exhibiting increased hematoxylin staining and appearing diffusely dark purple. The fibrous structures of the articular cartilage also undergo mesenchymal dystrophy, resulting in altered staining properties and increased volume. Necrobiotic processes in the peripheral compact bone columns cause swelling of the osteoid matrix and fragmentation of fibrous structures, which become hyperchromatic and disorganized. Osteocytes within the bone columns exhibit dystrophic changes, with nuclei becoming rounded and smaller, and the cytoplasm swelling and vacuolated. Internal trabecular structures show variable thickness, often deformed, and osteocytes within them take on a chondrocyte-like appearance due to dystrophy and edema. In the inner central regions of the femoral head, necrosis of the osteoid matrix leads to the formation of vacuolated spaces of varying sizes, some of which contain necrobiotic clusters of osteoblasts, osteoclasts, and fibroblasts. In some cases of femoral head aseptic necrosis, when blood enters the necrotic bone tissue, MRI shows high-intensity signals in both T1- and T2-weighted projections. If the necrotic focus is infiltrated with tissue fluid or plasma, the MRI signals appear of low intensity. Morphological analysis also revealed that vascular disruption in the developing necrotic bone tissue leads to massive hemorrhages. The extravasated blood accumulates in the intertrabecular spaces and necrobiotic matrix, containing erythrocytes, plasma proteins, and hemoglobin pigments. In the third stage of aseptic necrosis, when necrotic foci are fully formed, dense bone trabeculae fragment and break down, producing sequestra of various sizes. Meanwhile, the intertrabecular osteoid matrix transforms into unstructured, dendritic-like masses.

CONCLUSION

Aseptic necrotic foci in the femoral head are characterized by variable MRI signal intensities. Hemorrhage into the necrotic area increases signal intensity, while tissue fluid infiltration or osteosclerosis reduces it. In the early stage of femoral head necrosis, vascular constriction and thrombosis in the surrounding periosteum and soft tissues lead to necrobiotic changes. Initial necrosis of the intertrabecular osteoid matrix produces spaces containing destroyed osteoblasts, osteoclasts, and fibroblasts. Subsequently, dystrophic and destructive changes progress in the solid trabeculae, deepening necrobiotic processes. In the third stage, true necrotic foci form, with sequestra developing from compact bone trabeculae and unstructured dendritic masses from the osteoid matrix. In

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summary, early and accurate diagnosis of these pathological changes is essential to improve treatment outcomes. Timely intervention helps maintain patient health and contributes to the social and economic development of society.

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