MORPHOLOGICAL CHANGES IN THE TISSUES OF THE HIP JOINT IN 45-DAY EXPERIMENTAL CHRONIC COLITIS

Author: ***Nortayeva Nilufar Abdiraximovna***

Independent Researcher, Department of Human Anatomy and OHTA, Tashkent State Medical UniversityScientific Consultant: DSc, Professor Z.Sh. Sadiqova; Tashkent State Medical university, Faculcy of General medicine No.1, Group, 212-B, Student ***Xushvaqtova O'g'iloy Shodibek qizi.***

Abstract*: Chronic inflammatory diseases of the colon may be accompanied by systemic structural and functional changes in organs and tissues outside the gastrointestinal tract. The musculoskeletal system, including the hip joint, can be affected by persistent inflammatory, vascular, metabolic, and immune-mediated mechanisms. The aim of this study was to characterize the morphological changes occurring in the tissues of the hip joint during a 45-day experimental model of chronic colitis. The study focused on the articular cartilage, synovial membrane, subchondral bone, periarticular connective tissue, and microcirculatory structures. Histomorphological examination demonstrated signs of tissue remodeling, microcirculatory disturbances, inflammatory cellular infiltration, changes in the organization of connective tissue, and degenerative alterations of articular structures. The findings indicate that prolonged colonic inflammation may be associated with secondary structural changes in the hip joint and provide morphological evidence for the systemic nature of chronic inflammatory processes.*

Keywords*: chronic colitis; experimental model; hip joint; articular cartilage; synovial membrane; connective tissue; microcirculation; inflammation; morphology; tissue remodeling.*

Introduction

Chronic inflammatory disorders of the large intestine are characterized not only by persistent local injury of the intestinal mucosa but also by systemic immune, vascular, and metabolic responses. Long-lasting inflammation can influence distant

tissues through circulating inflammatory mediators, immune-cell activation, endothelial dysfunction, and disturbances of connective-tissue metabolism. The musculoskeletal system is particularly important in this context because connective tissues, cartilage, synovial structures, and bone are sensitive to prolonged inflammatory and microcirculatory changes.

The hip joint is a large synovial joint whose normal structure depends on the integrity of articular cartilage, synovial membrane, subchondral bone, capsule, ligaments, and periarticular connective tissue. Alterations in any of these components may influence joint nutrition, mechanical stability, and tissue regeneration. However, the morphological characteristics of the hip joint under prolonged experimental colonic inflammation remain insufficiently described. Therefore, experimental assessment of these changes may help clarify the relationship between chronic intestinal inflammation and secondary musculoskeletal remodeling.

Aim of the study

To investigate and characterize the morphological changes in the tissues of the hip joint after 45 days of experimentally induced chronic inflammation of the large intestine.

Materials and Methods

The study was designed as an experimental morphological investigation of animals with a 45-day model of chronic colitis. Experimental inflammation of the large intestine was maintained for 45 days, after which the animals were examined according to the approved experimental protocol. A control group of intact animals was used for comparison.

At the end of the experimental period, the hip joints were obtained together with the surrounding soft tissues. Tissue samples were fixed in an appropriate fixative, processed using standard histological techniques, embedded in paraffin, sectioned, and stained with routine histological stains. Microscopic examination was performed to assess the structural organization of the articular cartilage,

synovial membrane, subchondral bone, joint capsule, periarticular connective tissue, and microvascular structures. Particular attention was paid to cellular composition, tissue integrity, vascular congestion, edema, inflammatory infiltration, and signs of degenerative or reparative remodeling.

Morphological findings were compared qualitatively between the experimental and control groups. The interpretation was based on the degree of structural preservation and the presence of pathological changes in the examined joint tissues.

Results and Discussion

After 45 days of experimental chronic colitis, morphological examination of the hip joint demonstrated a complex pattern of structural alterations. The changes were most clearly expressed in the synovial membrane, articular cartilage, periarticular connective tissue, and microcirculatory bed.

In the synovial membrane, vascular congestion and moderate edema were observed. The microcirculatory vessels showed signs of altered blood flow, including dilation and uneven filling. In some areas, the connective tissue stroma appeared loosened, with an increase in intercellular substance. These findings may reflect persistent microcirculatory disturbances and the influence of systemic inflammatory mediators.

The articular cartilage demonstrated changes in the regular organization of chondrocytes. In comparison with the control morphology, some areas showed uneven distribution of chondrocytes, alteration of the extracellular matrix, and focal reduction in the normal compact organization of the cartilage. Such changes can be interpreted as manifestations of impaired trophic support and altered cartilage metabolism under conditions of prolonged systemic inflammation.

The subchondral bone also demonstrated signs of remodeling. Variations in the organization of bone trabeculae and changes in the cellular components of the bone tissue were noted. These alterations may be associated with disturbed interactions between cartilage and subchondral bone and with changes in local

vascular supply.

The periarticular connective tissue showed edema, altered fiber arrangement, and focal cellular infiltration. Inflammatory cells were observed predominantly around small vessels and within areas of altered connective-tissue organization. The presence of these changes suggests that chronic intestinal inflammation can be accompanied by a systemic inflammatory response capable of affecting distant connective-tissue structures.

The observed morphological alterations can be explained by several interconnected mechanisms. Persistent intestinal inflammation promotes the release of cytokines and other inflammatory mediators into the systemic circulation. These mediators may influence vascular permeability, endothelial function, immune-cell activity, and extracellular-matrix metabolism. Disturbance of microcirculation can additionally reduce tissue oxygenation and impair the nutritional support of cartilage and connective tissue. Consequently, prolonged inflammation may create conditions for degenerative and reparative remodeling of joint structures.

The results support the concept that chronic inflammation of the large intestine is not restricted to the gastrointestinal tract. Structural changes in the hip joint may represent one of the morphological manifestations of systemic involvement. The findings are also relevant for understanding the pathogenesis of musculoskeletal complications associated with chronic inflammatory intestinal diseases.

Conclusion

A 45-day experimental model of chronic colitis is associated with pronounced morphological remodeling of the hip joint tissues. The principal changes include microcirculatory disturbances, edema, inflammatory cellular infiltration, alterations in synovial connective tissue, changes in chondrocyte organization and cartilage matrix, and remodeling of subchondral bone. These findings suggest that prolonged colonic inflammation may exert a systemic effect on the musculoskeletal system and contribute to structural changes in synovial joint

tissues. Further quantitative morphometric and immunohistochemical studies are warranted to clarify the mechanisms and severity of these alterations.

References

1. Danese S., Fiocchi C. Ulcerative colitis. New England Journal of Medicine. 2011;365:1713–1725.

2. Ordás I., Eckmann L., Talamini M., Baumgart D.C., Sandborn W.J. Ulcerative colitis. The Lancet. 2012;380:1606–1619.

3. Firestein G.S., McInnes I.B. Immunopathogenesis of rheumatoid arthritis. Immunity. 2017;46:183–196.

4. Goldring S.R., Goldring M.B. Changes in the osteochondral unit during osteoarthritis. Journal of Musculoskeletal and Neuronal Interactions. 2007;7:316–319.

5. Kumar V., Abbas A.K., Aster J.C. Robbins and Cotran Pathologic Basis of Disease. Elsevier.

6. Ross M.H., Pawlina W. Histology: A Text and Atlas. Wolters Kluwer.