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MICROSCOPIC CHANGES IN POST-TRAUMATIC GONARTHROSIS ACCORDING TO DISEASE STAGES

Yodgorov Nodirjon Abdumajidovich,
Fergana Medical Institute of Public Health.

Makhkamov Nosirjon Juraevich,
Andijan State Medical Institute, Doctor of Medical Sciences, Associate Professor

Abstract

Among all forms of osteoarthritis, gonarthrosis (knee joint osteoarthritis) remains one of the leading causes of disability worldwide. In particular, post-traumatic gonarthrosis is a specific form of secondary osteoarthritis that develops following acute or chronic mechanical injuries to the knee joint. This condition is most commonly observed in young and middle-aged individuals and is clinically characterized by persistent joint pain, restricted mobility, progressive joint deformity, and significant impairment in quality of life and working capacity.

The pathogenesis of post-traumatic gonarthrosis involves a complex sequence of degenerative and inflammatory changes in the articular cartilage (hyaline cartilage), synovial membrane, subchondral bone, and periarticular soft tissues. These changes initially begin at the microscopic level, well before the appearance of overt clinical symptoms. Therefore, detailed histopathological assessment of microscopic changes at various stages of the disease is essential for early diagnosis, appropriate staging, and the development of targeted therapeutic strategies.

Each stage of post-traumatic gonarthrosis—Stage I (early), Stage II (progressive), and Stage III (advanced)—is associated with distinct microscopic alterations. These include progressive chondrocyte degeneration, disorganization of the cartilage matrix, fibrotic and inflammatory transformations of the synovial membrane, and structural remodeling of subchondral bone with osteophyte formation. Understanding these stage-dependent histological changes is crucial for unraveling the morphogenetic mechanisms of the disease and correlating them with clinical severity.

This article presents a comprehensive analysis of the microscopic changes in post-traumatic gonarthrosis across different pathological stages, highlighting their significance in disease progression and clinical management.

Keywords: post-traumatic gonarthrosis, knee osteoarthritis, histopathology, microscopic changes, articular cartilage degeneration, synovial membrane, subchondral bone remodeling, chondrocyte apoptosis.

Introduction

Relevance of the Problem

Post-traumatic gonarthrosis is a progressive, non-inflammatory degenerative disease that develops after injury to the knee joint. This condition involves periodic morphological changes in the articular cartilage, synovial membrane, subchondral bone, and periarticular tissues. Post-traumatic gonarthrosis, in particular, frequently occurs among young and working-age populations, becoming a leading cause of pain, movement limitation, and disability [1, 2].

Injuries to the knee joint — such as meniscal tears, ligament ruptures, fresh trauma, or microtraumas — disrupt the mechanical balance of the joint and ultimately lead to the development of osteoarthritis. This process is characterized by impaired chondrocyte function, loss of collagen and proteoglycans in the cartilage matrix, and fibrosis and inflammatory reactions in the synovial membrane. These changes initially appear at the microscopic level, well before clinical symptoms manifest [3–5].

International studies clearly distinguish morphological stages in the progression of post-traumatic gonarthrosis — Stage I (early), Stage II (intermediate), and Stage III (advanced). Detailed scientific study of tissue changes at each stage allows for individualized treatment approaches, accurate clinical prognosis, and justified indications for surgical methods such as arthroplasty [6–8].

Furthermore, the results of pathomorphological analysis play an important role in the development of various biomarkers, updated classification systems, and novel tissue-based diagnostic methods. Therefore, microscopic analysis of changes in post-traumatic gonarthrosis according to disease stages holds significant importance not only from an academic perspective but also in clinical practice [9–11].

Research Objective

The primary objective of this study is to identify and perform a pathomorphological analysis of the microscopic changes in post-traumatic gonarthrosis at different stages of the disease (Stages I, II, and III), and to investigate the relationship of these changes with the clinical severity and progression of the condition. This aims to establish a foundation for early diagnosis and the development of individualized treatment strategies for post-traumatic gonarthrosis.

Materials and Methods

The study utilized knee joint tissue samples obtained from patients with post-traumatic gonarthrosis treated at trauma hospitals in the Fergana region during 2022–2024 for pathomorphological analysis. Patients were categorized according to different stages of the disease (Stages I, II, and III), with at least 10 samples collected for each stage. The tissues were fixed in formalin for histological preparation, embedded in paraffin blocks, and sectioned at a thickness of 5 µm.

The sections were stained with hematoxylin and eosin, and special stains such as Safranin O and Azur B were used to better visualize the condition of chondrocytes and changes in the extracellular matrix. Histological preparations were examined under a light microscope, during which

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microscopic changes in the joint tissues—including chondrocyte apoptosis, matrix degradation, subchondral bone remodeling, and synovial membrane inflammation—were described.

The study results focused on comparing the characteristics and extent of pathomorphological changes observed at each stage of the disease. The collected data were analyzed using specialized statistical software.

Results and Discussion

During the pathomorphological evaluation of the knee joint tissue samples obtained in this study, morphological changes at various disease stages were identified. Although the samples were fixed in formalin, embedded in paraffin blocks, and cut into 5 µm sections, their structural alterations were determined using hematoxylin-eosin–stained slides and supplementary photomicrographs. Throughout the analysis, the number of chondrocytes, changes in their cytoplasmic and nuclear morphology, reduction in collagen and proteoglycan content within the cartilage matrix, subchondral bone sclerosis and cystic processes, as well as the degree of synovial membrane inflammation and fibrosis, were all recorded.

In Stage I, the number of chondrocytes remained relatively stable; however, slight cytoplasmic swelling and focal nuclear degradation were observed. A decrease in proteoglycan content and disorganization of collagen fibers within the cartilage matrix became apparent for the first time. In the subchondral bone region, early sclerosis and small cystic foci were detected at a subclinical level. The synovial membrane exhibited mild signs of fibrosis and hyperplasia, often accompanied by minimal lymphoid infiltration and increased vascularization (see Figure 1).

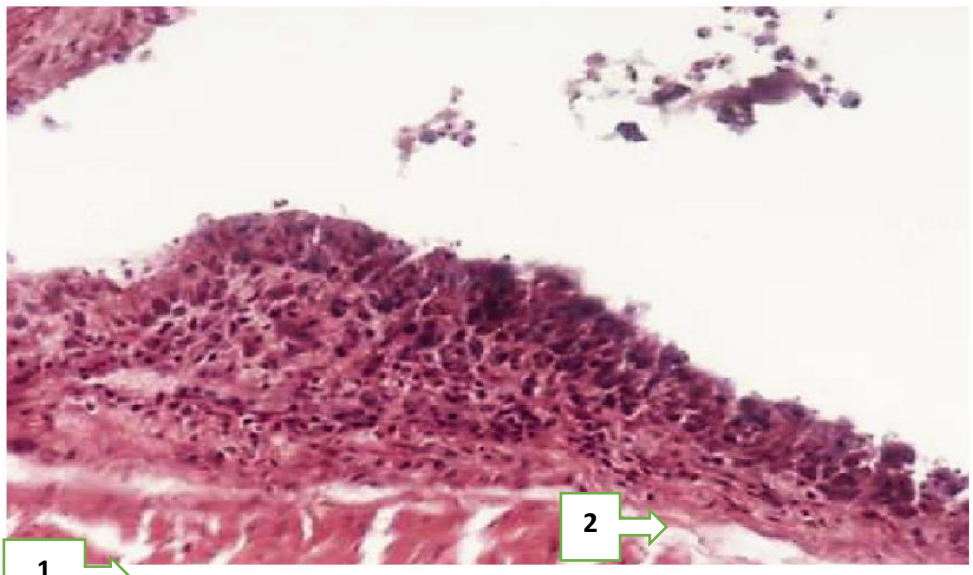

Figure 1. Synovial membrane hyperplasia (1). Lymphoid infiltration and vascularization (2).

In Stage II, a significant decrease in chondrocyte numbers was observed, with early signs of apoptosis and cell necrosis detected. Cytoplasmic pigmentation and extensive nuclear degradation were noted. Cartilage matrix degradation intensified, characterized by a marked reduction in proteoglycan levels and disorganization of collagen fibers. In the subchondral bone, the sclerosis process progressed, small cystic areas expanded, and the bone architecture entered a stage of remodeling. Signs of increased inflammation in the synovial membrane were recorded, including

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heightened hyperplasia, additional lymphoid infiltration, and expanded vascularization (see Figure 2).

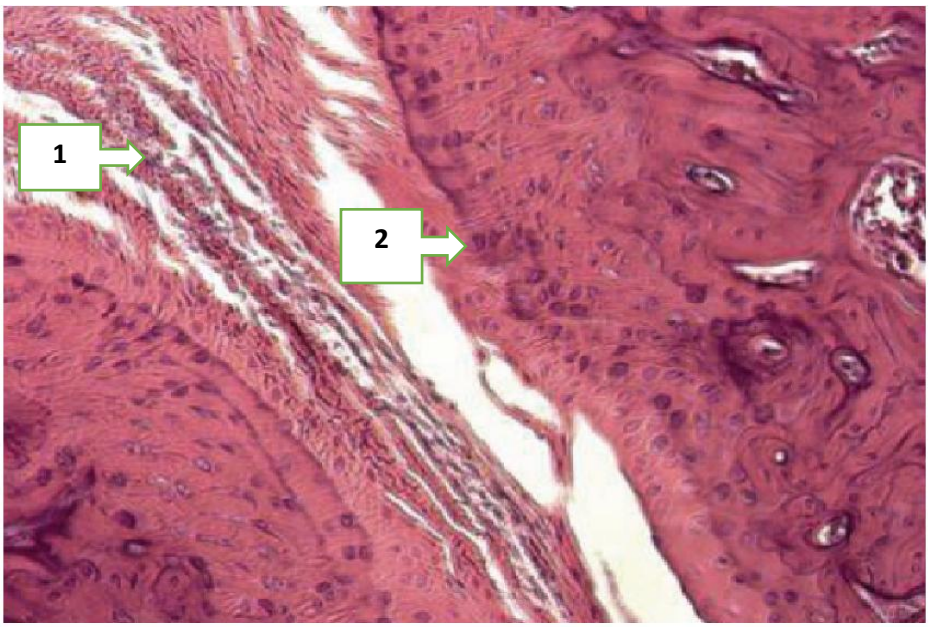

Figure 2. Subchondral bone sclerosis (1). Disorganization of collagen fibers (2).

In Stage III, chondrocytes were nearly absent; those remaining were markedly reduced in size and exhibited advanced nuclear degradation. Cartilage matrix degradation extended into deeper layers, resulting in extensive necrotic regions. Subchondral bone sclerosis intensified, large cystic formations were observed, and early microfracture signs were noted. The synovial membrane displayed pronounced fibrosis and hyperplasia, accompanied by abundant vascular and lymphoid elements. Osteophyte formation reached its highest degree, manifesting as confluent outgrowths and granular ossicle-like projections along the bone margin (see Figure 2).

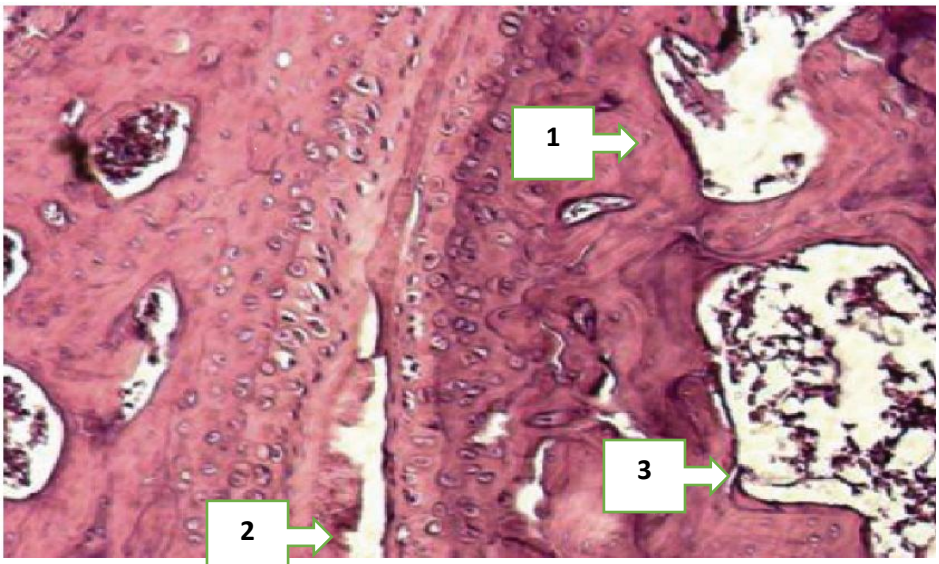

Figure 3. Osteophyte formation (1). Extensive cystic formation (2). Synovial membrane fibrosis (3). Stained with H&E. Magnification 40×10.

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Statistical analysis demonstrated a significant stepwise decrease in chondrocyte numbers from Stage I to Stage III ($p < 0.01$). Changes associated with matrix degradation were widespread in the advanced stages, with statistically significant differences observed between progression stages ($p < 0.01$). Subchondral bone remodeling (sclerosis and cystic changes) and synovial membrane inflammation markedly increased in later stages of the disease ($p < 0.05$). The Pearson correlation coefficient between chondrocyte number and pain intensity was $r = –0.72$ ($p < 0.01$), indicating that the reduction in chondrocyte count was associated with increasing pain severity. Furthermore, a strong positive correlation was identified between the decrease in matrix proteoglycan levels and the functional limitation index ($r = 0.68$, $p < 0.01$).

These pathomorphological findings provide crucial scientific insights into stage-based disease progression. Detection of mild early-stage morphological changes aids in discussing pain and functional impairment, whereas advanced-stage pathological alterations guide clinicians in selecting appropriate surgical interventions. The developed pathomorphological scoring scale thus lays the groundwork for implementing novel approaches to assess and manage post-traumatic gonarthrosis.

Conclusion

This study has demonstrated that post-traumatic gonarthrosis is characterized by distinct, stage-dependent histopathological changes in the knee joint. In Stage I, chondrocyte counts remain relatively stable, though early signs of cytoplasmic swelling and focal nuclear degradation become apparent alongside initial matrix changes, including reduced proteoglycan content and disorganized collagen fibers. Subchondral bone exhibits subtle sclerosis and small cystic foci without overt clinical manifestations, while the synovial membrane shows mild fibrosis and hyperplasia with minimal lymphoid infiltration and vascular proliferation (Figure 1).

By Stage II, chondrocyte numbers decline significantly, with clear evidence of apoptosis and necrosis. Cartilage matrix degradation intensifies, marked by a sharp drop in proteoglycan levels and further disruption of collagen architecture. Subchondral bone remodeling progresses, with expanding sclerosis and cystic areas, and bone architecture beginning to reorganize. Concurrently, synovial inflammation becomes more pronounced, evidenced by increased hyperplasia, lymphoid infiltration, and vascular proliferation, reflecting a transition toward more aggressive tissue degeneration.

In Stage III, chondrocytes are nearly absent; those that remain exhibit marked shrinkage and advanced nuclear degradation. Cartilage matrix breakdown extends into deep layers, producing extensive necrotic regions. Subchondral bone demonstrates intense sclerosis, large cystic formations, and early microfracture signs (Figure 2). The synovial membrane undergoes pronounced fibrosis and hyperplasia, enriched with abundant vascular and lymphoid elements. Osteophyte formation reaches its maximum expression, manifesting as confluent outgrowths and granular ossicle-like projections along the bone margin (Figure 3).

Statistical analyses reveal a significant, stepwise decrease in chondrocyte counts from Stage I to Stage III ($p < 0.01$), alongside increasingly widespread matrix degradation ($p < 0.01$). Subchondral bone remodeling (sclerosis and cystic changes) and synovial inflammation also rise significantly in later stages ($p < 0.05$). Importantly, a strong negative correlation exists between chondrocyte

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number and pain intensity ($r = –0.72$, $p < 0.01$), indicating that chondrocyte loss is closely associated with worsening pain. Furthermore, the decline in matrix proteoglycan levels correlates positively with functional limitation ($r = 0.68$, $p < 0.01$), highlighting the clinical relevance of histological degeneration.

Taken together, these findings underscore the critical role of stage-specific histopathological assessment in understanding post-traumatic gonarthrosis progression. Early identification of subtle morphological changes may facilitate prompt, targeted interventions to mitigate pain and preserve function, whereas recognition of advanced pathological features can inform surgical decision-making. The pathomorphological scoring scale developed in this study provides a robust framework for standardized evaluation and management of post-traumatic gonarthrosis, paving the way for future research into novel diagnostic biomarkers and therapeutic strategies.

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