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Changes in Blood Circulation and Pathomorphological Features of Ligament Tissues in Post-Traumatic Gonarthrosis

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Abstract: Post-traumatic gonarthrosis is a chronic pathological condition that develops after mechanical injuries of the knee joint and is characterized by degenerative and inflammatory changes in bone-cartilaginous structures, ligaments, and synovial tissues. Impairment of ligament tissue vascularization is considered one of the key pathogenetic mechanisms of this disorder. Circulatory insufficiency leads to fibrosis, necrosis, sclerosis, and trophic disturbances, which reduce the mechanical stability of the ligamentous apparatus and aggravate clinical manifestations. Pathomorphological analysis allows the identification of these alterations at the microscopic level and plays an essential role in understanding the pathogenesis of the disease from both scientific and clinical perspectives.

Keywords: post-traumatic gonarthrosis, ligament tissue, vascularization, pathomorphology, fibrosis, necrosis, trophic disturbances.

Relevance of the Problem

Post-traumatic gonarthrosis is a chronic condition that develops after various injuries to the knee joint, leading to disturbances in the integrity of joint tissues and the occurrence of specific pathomorphological changes within the ligaments. One of the primary pathogenetic mechanisms of this condition is impaired vascularization and microcirculation, which disrupts the delivery of essential nutrients to the tissues and the removal of metabolic by-products [1,2].

Vascularization disorders in post-traumatic gonarthrosis not only compromise overall joint function but also significantly hinder metabolic and reparative processes. Trauma-induced microvascular ruptures and progressive vasculitis can accelerate degenerative changes within the ligamentous structures, thereby aggravating the pathological process [3,4].

Pathomorphological manifestations of vascular dysfunction are often detected late or remain underestimated, contributing to the development of persistent or recurrent clinical syndromes. Furthermore, impaired vascularization in post-traumatic gonarthrosis predisposes to various complications, including functional weakening of the joint, persistent pain, swelling, and impaired mobility [5,6].

Timely recognition of these vascular and morphological alterations is essential for improving therapeutic strategies. Modern investigations emphasize that structural changes in ligament vascularization directly affect the progression of degenerative processes, the severity of clinical symptoms, and the efficacy of surgical and conservative treatments [7–9].

Therefore, the scientific study of the mechanisms underlying vascular impairment in ligamentous tissues during post-traumatic gonarthrosis holds high clinical and social relevance. It not only enhances understanding of disease pathogenesis but also contributes to optimizing treatment approaches and improving patient outcomes [10–14].

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Research Aim

To investigate the pathomorphological alterations associated with vascular and microcirculatory disturbances in ligament tissues in post-traumatic gonarthrosis using morphological and immunohistochemical methods, to evaluate the clinical significance of these changes, and to establish a scientific basis for the development of effective diagnostic and therapeutic strategies in the future.

Materials and Methods

This study was conducted to examine the pathomorphological state of ligament tissues in patients diagnosed with post-traumatic gonarthrosis who underwent surgical intervention. Biopsy specimens were obtained from 55 patients operated on at the Fergana Regional Traumatology Hospital between 2021 and 2024. In all cases, the diagnosis of post-traumatic gonarthrosis was established based on clinical and radiological findings.

All biopsy samples were collected intraoperatively and processed according to standard histopathological protocols. The tissues were fixed in 10% formalin solution, embedded in paraffin blocks, and sectioned into slices of 4–5 μm thickness. For morphological evaluation, hematoxylin and eosin, Van Gieson, and Mallory staining techniques were applied.

To assess vascular structures and endothelial integrity, immunohistochemical staining was performed using antibodies against CD31 and vascular endothelial growth factor (VEGF). The stained slides were examined under a light microscope (Olympus CX43) at magnifications of ×40 and ×100 to evaluate the microvascular network and endothelial condition.

Morphometric analysis was carried out using the ImageJ software. In five randomly selected fields from each slide, the number of capillaries, the percentage of the area occupied by them, and the extent of fibrotic tissue were quantitatively measured.

Results

According to the conducted examinations, a spectrum of pathomorphological and morphometric alterations was identified in the ligament tissues of patients with post-traumatic gonarthrosis. These changes were predominantly associated with impaired vascularization, intensification of fibrotic processes, and a noticeable reduction in microcirculatory elements.

Microscopic evaluation of hematoxylin and eosin-stained sections revealed pronounced structural disorganization within the ligamentous tissue. Clear dystrophic alterations were observed, accompanied by a decline in trophic activity and the presence of inflammatory infiltrates characteristic of vasculitis. Collagen fibrils exhibited disrupted parallel alignment, while hyalinized and homogenized regions were evident between the fibers, indicating progressive degeneration of the interstitial matrix.

Furthermore, focal areas of sclerosis and diffuse fibrotic remodeling were documented, leading to significant weakening of the structural framework of the ligaments. These pathological modifications contribute to the reduction of the connective and supportive functions of the tissue, thereby diminishing its ability to sustain normal biomechanical load and stability of the knee joint. Such alterations may underlie the clinical manifestations of pain, swelling, and reduced functional capacity frequently observed in post-traumatic gonarthrosis (see Fig. 1).

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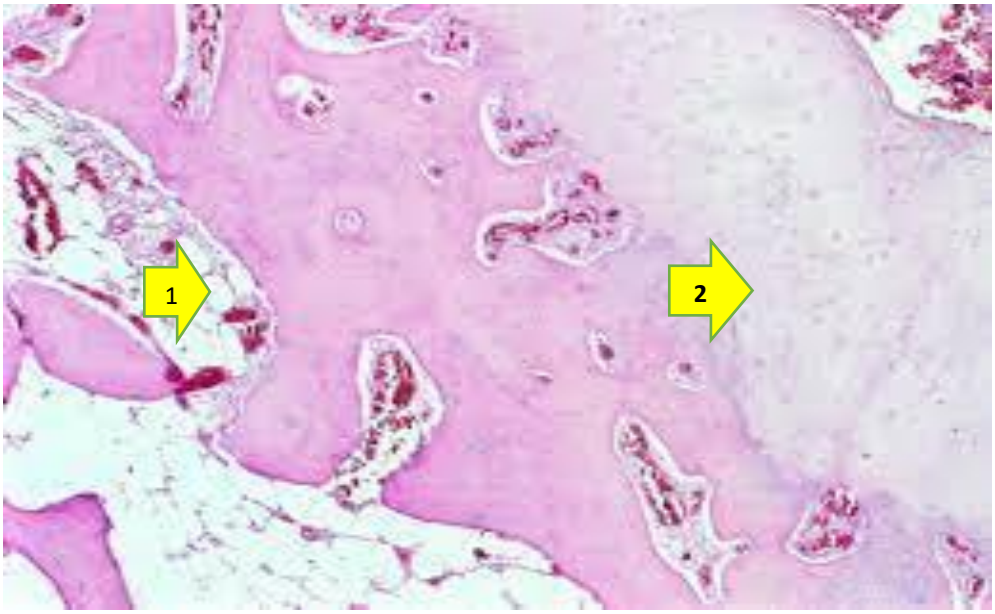

Figure 1. Dystrophic alterations in the interstitial tissue (1) and structural components of collagen fibrils (2). Staining: H&E. Magnification: 40x10. The image demonstrates disrupted collagen organization and degenerative changes compromising the structural integrity of the ligament.

Vascular alterations played a particularly significant role, with disturbances in microcirculation emerging as a key pathogenetic factor. Histological analysis revealed a marked reduction in the number of capillaries within the ligament tissue, accompanied by endothelial desquamation along the vascular walls. Pericapillary edema and inflammatory cell infiltrates were clearly observed, reflecting ongoing degenerative and inflammatory processes. Moreover, narrowing of vascular lumina was evident, while in several cases, stasis and early thrombotic changes were documented, indicating impaired blood flow and compromised tissue perfusion (see Fig. 2).

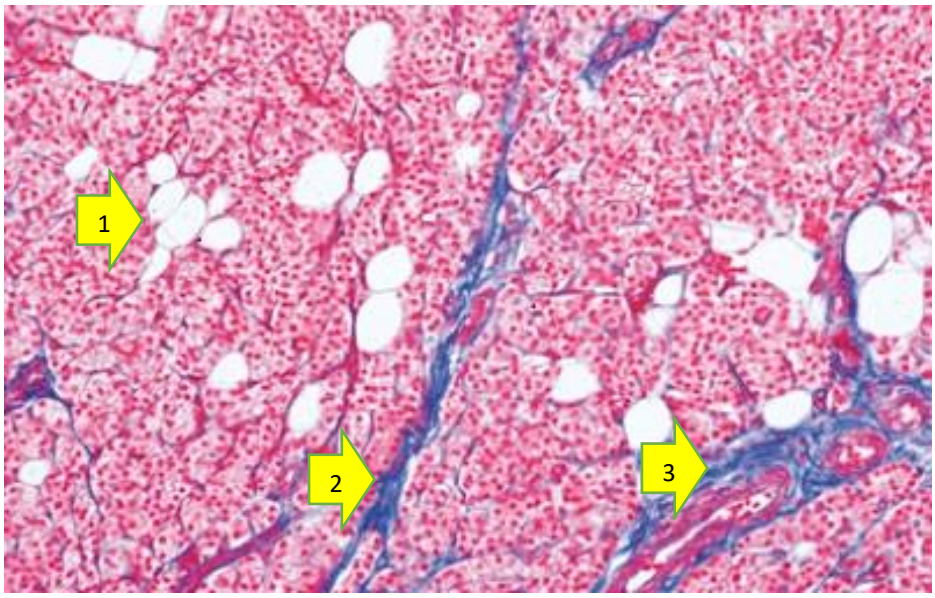

Figure 2. Thickened blood vessel wall (1), inflammatory infiltration in the perivascular region (2), and intraluminal thrombotic formations (3). Staining: H&E. Magnification: 40x10. The image highlights vascular wall remodeling, perivascular inflammatory response, and evidence of thrombosis, reflecting impaired microcirculation and ongoing pathological vascular changes.

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The morphometric analysis of ligament tissues obtained from patients with post-traumatic gonarthrosis demonstrated pronounced structural alterations within the vascular component. Specifically, the mean number of capillaries per 1 mm² was found to be 6.3 ± 1.2, which represents a statistically significant reduction compared to the control group ($p < 0.05$). In addition to the decreased density of microvessels, the total percentage of tissue area occupied by capillaries was reduced to an average of 8.7 ± 2.1%, further confirming impaired microcirculation and insufficient trophic support to the ligament structures ($p < 0.01$).

Moreover, the proportion of fibrotic areas within the examined specimens was notably elevated, averaging 42.5 ± 4.6%. Such an increase in fibrosis likely reflects ongoing tissue remodeling and maladaptive reparative processes, which may compromise the functional resilience of ligament structures ($p < 0.01$). Hyalinized zones were also identified, with a mean of 3.1 ± 0.8 per microscopic field, providing histological evidence of chronic degenerative changes ($p < 0.05$).

Assessment of endothelial dysfunction using a semi-quantitative scoring system revealed an average score of 2.7 ± 0.5, indicating distinct morphological damage to the vascular endothelium ($p < 0.05$). The presence of endothelial desquamation, narrowing of vascular lumina, and perivascular edema further corroborated these findings.

Collectively, these parameters strongly suggest that post-traumatic gonarthrosis is accompanied by microcirculatory impairment, progressive fibrosis, hyalinization, and endothelial injury within the ligament tissues. Such pathological alterations delay the processes of structural and functional recovery and highlight the necessity of tailoring therapeutic approaches to the specific morphological and vascular status of each patient (see Table 1).

Table 1. Morphometric parameters of ligamentous tissues in patients with post-traumatic gonarthrosis (compared to the control group)

Morphometric Indicators	Mean ± SD	Min.–Max.	p-value (vs. control)
Number of capillaries (per 1 mm² field)	6.3 ± 1.2	3 – 9	< 0.05
Capillary area (%)	8.7 ± 2.1	5.1 – 13.4	< 0.01
Proportion of fibrotic tissue (%)	42.5 ± 4.6	35.2 – 51.7	< 0.01
Number of hyalinized areas (per histological slide)	3.1 ± 0.8	2 – 5	< 0.05
Endothelial dysfunction score (semi-quantitative)	2.7 ± 0.5	2 – 4	< 0.05

Note: The data presented in Table 1 reflect significant vascular and structural changes in ligament tissues of patients with post-traumatic gonarthrosis. The marked reduction in capillary density and area indicates compromised microcirculatory support, while the increased proportion of fibrotic and hyalinized regions illustrates maladaptive remodeling. Elevated endothelial dysfunction scores further confirm the presence of vascular injury, underscoring the close interplay between microvascular pathology and connective tissue alterations.

Conclusion

The comprehensive analysis of ligamentous tissues in patients with post-traumatic gonarthrosis demonstrates that this chronic condition is characterized by profound structural and functional alterations, predominantly driven by vascular and microcirculatory impairment. Morphological examination revealed significant disruption of capillary networks, including a reduction in capillary density and lumen narrowing, accompanied by endothelial desquamation, perivascular edema, and thrombotic phenomena. These vascular changes compromise nutrient delivery and waste removal, thereby creating an environment conducive to tissue hypoxia, degeneration, and delayed reparative processes.

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Concurrently, the morphometric assessment highlighted a substantial increase in fibrotic tissue proportion and the presence of hyalinized zones, indicating ongoing maladaptive remodeling within ligament structures. The semi-quantitative scoring of endothelial dysfunction further confirmed the extent of vascular injury, reflecting a direct correlation between microvascular pathology and connective tissue deterioration. Collectively, these pathological changes contribute to the loss of biomechanical integrity of the ligamentous apparatus, leading to diminished joint stability, chronic pain, and functional impairment observed clinically.

The findings underscore the pivotal role of microcirculatory disturbances in the pathogenesis of post-traumatic gonarthrosis and highlight the necessity of individualized diagnostic and therapeutic strategies. Targeted interventions aimed at restoring vascular integrity, mitigating fibrosis, and enhancing tissue regeneration may improve structural recovery and functional outcomes. Moreover, integrating histomorphological and immunohistochemical assessments into routine clinical evaluation could facilitate the early identification of patients at higher risk of progressive joint degeneration, allowing for timely and personalized management.

In conclusion, post-traumatic gonarthrosis is not solely a mechanical joint disorder but a complex pathophysiological condition in which vascular compromise, fibrosis, hyalinization, and endothelial dysfunction interplay to drive chronic ligamentous degeneration. Future research should focus on therapeutic modalities that address these microstructural and vascular abnormalities to optimize long-term outcomes and preserve joint function.

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