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DEFORMATIVE OSTEOARTHRITIS

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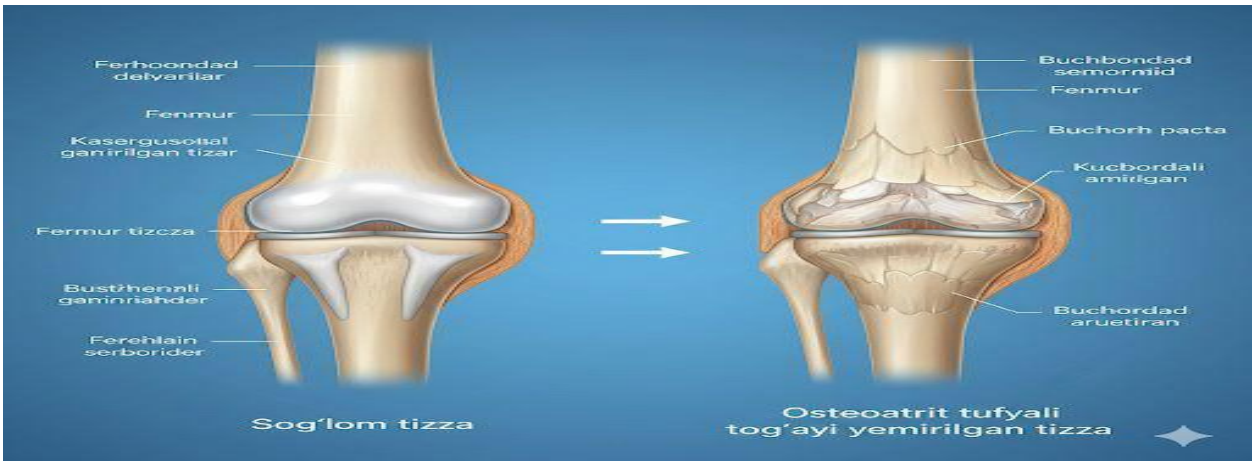
Abstract: The pathogenesis of osteoarthritis of the knee (OAH) is interpreted in modern rheumatology as a complex cascade process resulting from the activation of pro-inflammatory mediators (IL-1β, TNF-α) and matrix metalloproteinases (MMP-13). This study analyzes the limitations of traditional drug therapy in restoring joint homeostasis and the therapeutic efficacy of regenerative medicine methods, in particular autologous plasma (PRP) and stromal vascular fraction (SVF) at the molecular level. The results obtained provide scientific justification for the possibility of modifying the pathological process at early stages and inhibiting chondrocyte apoptosis.

Keywords: Osteoarthritis, chondrosenescence, MMP-13, Kellgren-Lawrence classification, PRP therapy, SVF technology, chondromalacia, subchondral remodeling.

I. INTRODUCTION

Deforming osteoarthritis is a chronic progressive pathology of the musculoskeletal system, which involves all components of the joint (chondral bone, subchondral bone, synovial membrane). In modern international protocols (OARSI, ESCEO), DOA is considered not just a "sign of aging", but as a low-grade systemic inflammation caused by biochemical and mechanical imbalance. The increase in disability rates worldwide requires the introduction of structural-modifying (DMOADs) approaches to the treatment of this pathology.

Comparative image of a healthy knee and a knee with cartilage erosion due to osteoarthritis.



II. MOLECULAR-BIOLOGY ANALYSIS OF ETIOPATHOGENESIS

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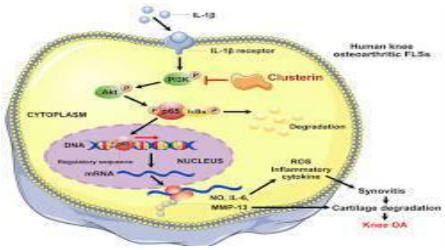
The theory of the "molecular cascade" plays a central role in the development of the disease. Chondrocytes (articular cartilage cells) change their phenotype as a result of mechanical loading and metabolic stress:

Chondrocyte senescence: Cells lose their regenerative potential and sharply increase the synthesis of matrix-degrading enzymes - metalloproteinases (MMP-1, MMP-13) and ADAMTS.

Subchondral osteosclerosis: As a result of impaired microcirculation of the bone under the cartilage, the activity of osteoblasts changes, which leads to pathological densification of bone tissue and loss of joint cushioning.

Molecular level degradation scheme of cartilage tissue (involvement of enzymes and



cytokines)).

III. RADIOLOGICAL AND BIOCHEMICAL CRITERIA IN DIAGNOSTICS

The Kellgren-Lawrence (K-L) classification is an international standard for determining the degree of DOA. Joint space narrowing and osteophyte formation are the main radiological markers of the disease:

Stage I-II: Minimal joint space narrowing, subchondral cysts and initial osteophytes.

Stage III-IV: Valgus or varus deformation of the joint axis, complete obliteration of the interosseous space.

Radiographic stages of knee osteoarthritis (Kellgren-Lawrence classification).

IV. REGENERATIVE THERAPY: AN INNOVATIVE PARADIGM

The strategic goal of treatment is to move from temporary symptom relief to cartilage structure regeneration:

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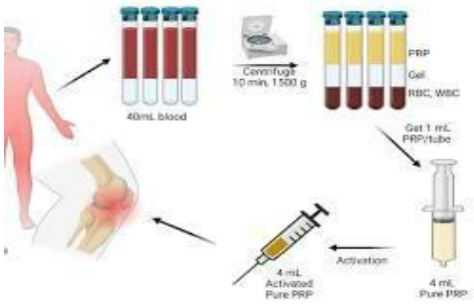
PRP therapy (Platelet-Rich Plasma): Growth factors (PDGF, TGF-β, VEGF) contained in autologous platelets stimulate chondrocyte proliferation and block synovial inflammation.

SVF (Stromal Vascular Fraction) technology: Mesenchymal cells derived from adipose tissue exert a paracrine effect in the joint, allowing for the restoration of the matrix undergoing degradation.

Viscosupplementation: High-molecular hyaluronic acid preparations act as lubrication and cushioning in the joint.

The process of injecting regenerative drugs (PRP/SVF) into the joint.



V. RESULTS AND DISCUSSION

The results of multicenter clinical studies show that patients treated with combined regenerative therapy achieve a functional improvement of 65-70% according to the WOMAC (Western Ontario and McMaster Universities Osteoarthritis Index). The use of nonsteroidal anti-inflammatory drugs (NSAIDs) alone cannot stop the progression of the disease.

Conclusion: In the treatment of deforming osteoarthritis, personalized regenerative methodology and therapeutic modification at the molecular level are the only effective way to save the patient from disability.

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