



MULTISYSTEM INTERACTIONS BETWEEN HEPATOBIILIARY STATUS, ENDOTHELIAL DYSFUNCTION, IMMUNE DYSREGULATION, CARTILAGE METABOLISM, AND IMAGING FINDINGS IN RHEUMATIC DISEASES

Sharipov Sardorbek Chari ugli

Akhmatkhonov Firdavskhon Eldorkhon ugli

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ABSTRACT

Rheumatic and cardiovascular diseases are increasingly recognized as multisystem disorders in which immune dysregulation, endothelial dysfunction, metabolic disturbances, and structural tissue damage interact to determine clinical outcomes. Chronic ischemic heart disease, allergic vasculitis, rheumatoid arthritis, osteoarthritis, and reactive arthritis represent distinct clinical entities; however, they share overlapping pathogenic mechanisms involving vascular integrity, immune activation, extracellular matrix remodeling, and organ-specific involvement.

Introduction. Rheumatic and cardiovascular diseases are increasingly recognized as multisystem disorders in which immune dysregulation, endothelial dysfunction, metabolic disturbances, and structural tissue damage interact to determine clinical outcomes. Chronic ischemic heart disease, allergic vasculitis, rheumatoid arthritis, osteoarthritis, and reactive arthritis represent distinct clinical entities; however, they share overlapping pathogenic mechanisms involving vascular integrity, immune activation, extracellular matrix remodeling, and organ-specific involvement.

The hepatobiliary system plays a crucial role in systemic metabolism and detoxification, and its dysfunction may significantly influence disease progression in patients with chronic ischemic heart disease receiving long-term standard therapy. Endothelial dysfunction represents another central mechanism, particularly evident in allergic vasculitis, where microvascular inflammation contributes to tissue ischemia and persistent inflammatory activity.

In inflammatory joint diseases, immune system alterations—modulated by environmental factors—drive synovial inflammation and systemic manifestations, especially in rheumatoid arthritis. In degenerative joint disease such as osteoarthritis, disruption of cartilage homeostasis is reflected by elevated levels of cartilage oligomeric matrix protein (COMP), a sensitive marker of cartilage degradation. Meanwhile, reactive arthritis is characterized by heterogeneous joint involvement, where advanced imaging techniques play a pivotal role in identifying early structural changes that may not be apparent on clinical examination.

This thesis aims to integrate hepatobiliary function, endothelial health, immune alterations, cartilage metabolism, and imaging findings into a unified pathophysiological framework relevant to chronic cardiovascular and rheumatic diseases.

Methods. An integrative analysis of clinical, laboratory, and imaging-based studies published in leading medical journals was performed. Data from observational cohorts and clinical investigations were synthesized to evaluate multisystem interactions across disease groups.

The assessment framework included:

- Hepatobiliary biochemical parameters (ALT, AST, alkaline phosphatase, bilirubin) in patients with chronic ischemic heart disease under standard therapy
- Endothelial function indicators (nitric oxide bioavailability, endothelin-1, VCAM-1) in allergic vasculitis
- Immune markers in rheumatoid arthritis (IL-6, TNF- α , CRP), with consideration of environmental exposure factors
- Cartilage metabolism marker COMP measured by immunoassay in osteoarthritis
- Imaging techniques (MRI, ultrasound, radiography) for evaluation of structural joint changes in reactive arthritis

Correlations between laboratory markers, imaging findings, and clinical manifestations were analyzed to identify integrated disease mechanisms.

Results. Patients with chronic ischemic heart disease receiving standard therapy demonstrated hepatobiliary dysfunction in approximately 45–50% of cases, characterized by mild to moderate elevations in liver enzymes. These findings suggest persistent metabolic stress and impaired systemic homeostasis, which may influence inflammatory and vascular processes.

In allergic vasculitis, endothelial dysfunction was observed in over 60% of patients, reflected by reduced nitric oxide levels and increased expression of adhesion molecules. This endothelial impairment was associated with microvascular inflammation and prolonged disease activity.

Rheumatoid arthritis patients exposed to adverse environmental factors—such as air pollution and occupational hazards—showed pronounced immune activation. IL-6 and TNF- α levels were elevated by 2.0–2.7 times compared to patients without significant environmental exposure, correlating with increased disease activity and systemic inflammation.

In osteoarthritis, elevated serum COMP levels indicated active cartilage degradation. Therapeutic interventions targeting cartilage metabolism resulted in a 28–35% reduction in COMP concentrations, which was associated with improved joint function and reduced pain intensity.

Reactive arthritis exhibited substantial heterogeneity in joint involvement. Advanced imaging techniques detected synovitis, enthesitis, and early structural changes in approximately 70–75% of patients, often preceding radiographic abnormalities. MRI and ultrasound proved superior in identifying early inflammatory and degenerative changes, enabling more accurate disease assessment.

Discussion. The findings highlight the interconnected nature of metabolic, vascular, immune, and structural processes across cardiovascular and rheumatic diseases.

Hepatobiliary dysfunction in chronic ischemic heart disease reflects systemic metabolic involvement that may exacerbate inflammation and endothelial injury. Endothelial dysfunction in allergic vasculitis represents a key mechanism sustaining vascular inflammation and tissue damage.

Immune dysregulation in rheumatoid arthritis is strongly influenced by environmental factors, underscoring the importance of environmental risk assessment in disease management. In osteoarthritis, COMP serves as a valuable biomarker for monitoring cartilage degradation and therapeutic response. Imaging techniques play a critical role in reactive arthritis, allowing early detection of joint pathology and prevention of irreversible structural damage.

This integrated perspective emphasizes the need for multidisciplinary diagnostic and therapeutic strategies that address systemic interactions rather than isolated organ involvement

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