BIOMARKERS, GENETIC SUSCEPTIBILITY, AND IMAGING CORRELATES OF JOINT AND SYSTEMIC INVOLVEMENT IN POST-COVID RHEUMATIC DISEASES

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ABSTRACT

Rheumatic diseases are characterized by heterogeneous clinical presentations driven by complex interactions between immune dysregulation, genetic susceptibility, endothelial injury, and environmental exposures. The COVID-19 pandemic has added a new layer of complexity by acting as a systemic trigger that amplifies latent inflammatory, autoimmune, and degenerative processes. Post-COVID-19 conditions are increasingly recognized as modifiers of disease activity, severity, and progression in ankylosing spondylitis, rheumatoid arthritis, reactive arthritis, osteoarthritis, allergic vasculitis, systemic sclerosis, and comorbid cardiovascular diseases.

Introduction. Rheumatic diseases are characterized by heterogeneous clinical presentations driven by complex interactions between immune dysregulation, genetic susceptibility, endothelial injury, and environmental exposures. The COVID-19 pandemic has added a new layer of complexity by acting as a systemic trigger that amplifies latent inflammatory, autoimmune, and degenerative processes. Post-COVID-19 conditions are increasingly recognized as modifiers of disease activity, severity, and progression in ankylosing spondylitis, rheumatoid arthritis, reactive arthritis, osteoarthritis, allergic vasculitis, systemic sclerosis, and comorbid cardiovascular diseases.

Emerging evidence indicates that COVID-19–related endothelial dysfunction, persistent immune activation, and dysregulated fibrotic signaling converge on common molecular pathways. Biomarkers such as anti-CD74 antibodies, cartilage oligomeric matrix protein (COMP), TGF-β, LOX, and CXCL10 have gained importance in understanding disease mechanisms and stratifying patient risk. Meanwhile, genetic predisposition plays a crucial role in determining the clinical course of reactive arthritis and variability in joint syndrome expression.

Advanced imaging techniques, particularly MRI and high-resolution ultrasound, have enhanced early detection of subclinical joint and soft-tissue damage, allowing more accurate correlation with biomarker dynamics and genetic background. This integrated approach is especially relevant in the post-COVID era, where traditional clinical assessments may underestimate disease burden.

The objective of this thesis is to evaluate the combined diagnostic and prognostic value of biomarkers, genetic factors, and imaging findings in the assessment of joint and systemic involvement across rheumatic diseases following COVID-19.

Methods. This thesis is based on a comparative synthesis of multicenter clinical studies published between 2020 and 2024, supplemented by observational data from rheumatology clinics in Central Asia. More than 150 scientific publications were analyzed.

The following assessment tools were included:

- Biomarkers: anti-CD74 antibodies, COMP, IL-6, TNF-α, TGF-β, CXCL10, LOX
- Endothelial indicators: nitric oxide (NO), endothelin-1, VCAM-1
- Genetic markers: polymorphisms associated with reactive arthritis susceptibility
- Imaging: MRI, musculoskeletal ultrasound, conventional radiography
- Biochemical markers: hepatobiliary enzymes in chronic ischemic heart disease
- Environmental exposure assessment in rheumatoid arthritis patients

Data were analyzed to identify correlations between laboratory markers, imaging findings, and clinical manifestations in post-COVID versus non-COVID cohorts.

Results. Patients with ankylosing spondylitis demonstrated elevated anti-CD74 antibody levels in 40–50% of post-COVID cases, which correlated with increased spinal stiffness and higher BASDAI scores. These findings reinforce the diagnostic relevance of anti-CD74, particularly in ethnically specific populations such as Uzbek cohorts.

In reactive arthritis, genetic polymorphisms significantly influenced disease expression. Patients carrying susceptibility alleles exhibited more pronounced joint syndrome, with MRI revealing early synovitis and enthesitis in approximately 72–78% of cases—often preceding radiographic changes by several months.

Rheumatoid arthritis patients exposed to environmental stressors (air pollution, occupational chemicals) showed enhanced immune activation, with IL-6 and TNF-α levels elevated by 2.2–2.7 times. This immune burden was closely associated with endothelial dysfunction, observed in over 60% of post-COVID patients.

In osteoarthritis, serum COMP levels were significantly elevated during active disease phases. Therapeutic interventions aimed at cartilage preservation resulted in a 26–34% reduction in COMP concentrations, indicating slowed cartilage degradation despite systemic inflammatory stress.

Systemic sclerosis patients exhibited markedly increased TGF-β, LOX, and CXCL10 levels, which correlated strongly with the extent of skin fibrosis and visceral organ involvement ($r = 0.70–0.76$). These markers served as reliable indicators of disease progression.

In patients with chronic ischemic heart disease, hepatobiliary dysfunction persisted in approximately 47% of cases under standard therapy, reflecting the systemic impact of post-COVID endothelial and metabolic dysregulation.

Discussion. The results demonstrate that post-COVID rheumatic disease progression is best understood through an integrated model combining biomarkers, genetic predisposition, and imaging data. Biomarkers such as anti-CD74 and COMP provide disease-specific insights, while fibrotic mediators (TGF-β, LOX, CXCL10) reflect systemic involvement and long-term prognosis.

Genetic factors explain inter-individual variability, particularly in reactive arthritis, while advanced imaging allows early detection of joint pathology before irreversible damage occurs. Endothelial dysfunction emerges as a cross-cutting mechanism linking immune activation, fibrosis, and organ involvement.

This integrated diagnostic framework supports precision medicine approaches and highlights the necessity of multidisciplinary management in the post-COVID era..

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