



INTEGRATED CLINICAL, IMMUNOLOGICAL, AND ENDOTHELIAL CONSEQUENCES OF POST-COVID-19 CONDITIONS IN INFLAMMATORY AND DEGENERATIVE RHEUMATIC DISEASES

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The COVID-19 pandemic has fundamentally altered the clinical course of chronic inflammatory and degenerative rheumatic diseases

ABSTRACT

These effects are particularly pronounced in patients with ankylosing spondylitis, rheumatoid arthritis, reactive arthritis, osteoarthritis, systemic sclerosis, allergic vasculitis, and ischemic heart disease with comorbid metabolic involvement

Introduction. The COVID-19 pandemic has fundamentally altered the clinical course of chronic inflammatory and degenerative rheumatic diseases. Beyond the acute viral infection, post-COVID-19 sequelae have emerged as a major modifier of immune regulation, endothelial integrity, and connective tissue metabolism. According to recent cohort studies, persistent symptoms lasting more than 12 weeks occur in 30–45% of recovered COVID-19 patients, with musculoskeletal manifestations reported in up to 27% of cases. These effects are particularly pronounced in patients with ankylosing spondylitis, rheumatoid arthritis, reactive arthritis, osteoarthritis, systemic sclerosis, allergic vasculitis, and ischemic heart disease with comorbid metabolic involvement.

SARS-CoV-2 induces widespread endothelial dysfunction through direct viral endothelial invasion and cytokine-mediated injury. This mechanism overlaps with the pathophysiology of inflammatory rheumatic diseases, where endothelial activation, immune dysregulation, and fibrotic signaling (TGF- β , LOX, CXCL10) play a central role. Additionally, biomarkers such as anti-CD74 antibodies and cartilage oligomeric matrix protein (COMP) have gained diagnostic and prognostic relevance, particularly in ankylosing spondylitis and osteoarthritis.

Genetic predisposition, environmental exposure, and advanced imaging findings further modulate disease heterogeneity, especially in reactive arthritis. Therefore, a comprehensive integrative approach is required to understand post-COVID-19 impacts across different rheumatic disease spectra.

The aim of this thesis is to analyze the combined effects of post-COVID-19 sequelae on immune status, endothelial function, cartilage metabolism, genetic susceptibility, and structural joint changes across inflammatory and degenerative rheumatic disorders.

Methods. This integrative analysis is based on a systematic review of publications indexed in Scopus, PubMed, and Web of Science (2020–2024), combined with regional clinical data from Central Asia, including Uzbek patient cohorts. A total of over 140 peer-reviewed studies were evaluated.

Clinical and laboratory assessments included:

- Endothelial function markers: nitric oxide (NO), endothelin-1, VCAM-1, ICAM-1
- Immune mediators: IL-6, TNF- α , CXCL10, TGF- β
- Autoimmune markers: anti-CD74 antibodies
- Cartilage metabolism marker: COMP (ELISA-based quantification)
- Hepatobiliary biochemical parameters (ALT, AST, ALP, bilirubin)
- Imaging modalities: MRI, ultrasound, radiography
- Genetic polymorphism analysis associated with reactive arthritis

Comparative analyses were conducted between post-COVID and non-COVID rheumatic patient groups.

Results. Post-COVID patients with ankylosing spondylitis demonstrated a 1.5–1.8-fold increase in disease activity scores (BASDAI), accompanied by elevated anti-CD74 antibody titers in 42–48% of cases, confirming their diagnostic significance in the Uzbek population.

Endothelial dysfunction was observed in 58–65% of patients with allergic vasculitis and inflammatory arthritis following COVID-19, characterized by reduced NO bioavailability and increased VCAM-1 levels. These changes were strongly associated with persistent microvascular inflammation.

Patients with rheumatoid arthritis exposed to adverse environmental factors (air pollution, occupational toxins) showed amplified immune activation, with IL-6 and TNF- α levels increased by 2.1–2.6 times compared to baseline. Genetic susceptibility further influenced disease severity in reactive arthritis, explaining variations in joint syndrome presentation.

Imaging studies revealed early structural joint changes in reactive arthritis, with MRI detecting synovitis and enthesitis in over 70% of cases before radiographic abnormalities became evident.

In osteoarthritis, therapeutic interventions targeting cartilage metabolism resulted in a 25–35% reduction in serum COMP levels, indicating slowed cartilage degradation. Similar improvements were noted in targeted therapies aimed at optimizing COMP dynamics.

Patients with systemic sclerosis exhibited significantly elevated TGF- β , LOX, and CXCL10 levels, correlating with progressive skin and visceral fibrosis (correlation coefficient $r = 0.69–0.74$).

Among patients with chronic ischemic heart disease receiving standard therapy, hepatobiliary dysfunction was detected in 45–50%, reflecting systemic metabolic and inflammatory burden exacerbated by post-COVID conditions.

Discussion. The findings confirm that post-COVID-19 sequelae act as a systemic amplifier of pathological processes common to rheumatic diseases. Endothelial dysfunction represents a unifying mechanism linking vascular injury, immune activation, and tissue remodeling. Immune dysregulation driven by cytokines and chemokines accelerates joint damage, fibrosis, and extra-articular manifestations.

Biomarkers such as anti-CD74 and COMP offer valuable tools for early diagnosis, monitoring disease activity, and evaluating therapeutic efficacy. Imaging techniques significantly enhance early detection of joint pathology, while genetic factors explain inter-individual variability in reactive arthritis progression.

An integrated multidisciplinary approach is essential for personalized management of rheumatic patients in the post-COVID era..

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