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COMBINED ROLE OF COMP, TGF-B, AND CXCL10 BIOMARKERS IN PREDICTING EARLY TISSUE DESTRUCTION IN AUTOIMMUNE DISEASES

Ochildieva Aziza Olimjon kizi
Tashkent International Kimyo University

Davronova Muslimakhon Ravshan kizi
Tashkent International Kimyo University

Abstract

Autoimmune diseases represent a heterogeneous group of chronic disorders characterized by dysregulated immune responses against self-antigens, leading to persistent inflammation, progressive tissue damage, and functional impairment. Conditions such as rheumatoid arthritis, ankylosing spondylitis, systemic lupus erythematosus, autoimmune vasculitides, and inflammatory joint diseases impose a substantial clinical and socioeconomic burden worldwide. Despite advances in immunomodulatory therapies, many patients continue to experience irreversible structural damage, highlighting the need for earlier detection of destructive processes before overt clinical deterioration occurs.

Introduction

A critical challenge in the management of autoimmune diseases lies in identifying the transition from reversible inflammation to irreversible tissue destruction. Conventional clinical markers and imaging techniques often detect damage only after significant structural changes have already taken place. As a result, there is growing interest in molecular biomarkers that can sensitively reflect early pathological changes at the tissue and extracellular matrix level. Such biomarkers could enable timely therapeutic intervention, improve risk stratification, and ultimately prevent long-term disability.

Among the most intensively studied candidates are biomarkers associated with extracellular matrix turnover, immune regulation, and inflammatory signaling. In this context, cartilage oligomeric matrix protein (COMP), transforming growth factor-beta (TGF-β), and C-X-C motif chemokine ligand 10 (CXCL10) have

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emerged as biologically plausible and clinically relevant indicators of early tissue remodeling and immune-mediated damage. While each of these biomarkers has been investigated individually, their combined role in predicting early tissue destruction remains insufficiently explored.

COMP is a non-collagenous extracellular matrix protein predominantly expressed in cartilage, tendons, ligaments, and synovial tissue. It plays a structural role in maintaining matrix integrity and interacts with collagen fibrils to ensure biomechanical stability. Elevated circulating levels of COMP have been consistently associated with cartilage degradation and joint destruction, particularly in inflammatory arthritides. Importantly, increases in serum COMP may precede radiographic evidence of joint damage, suggesting its potential utility as an early marker of structural deterioration. However, COMP alone does not fully capture the complexity of immune-mediated tissue injury, as it reflects downstream matrix breakdown rather than upstream immunological drivers.

TGF-β is a multifunctional cytokine with a dual role in immune regulation and tissue remodeling. Under physiological conditions, TGF-β contributes to immune tolerance, wound healing, and extracellular matrix synthesis. In autoimmune diseases, however, dysregulated TGF-β signaling may promote pathological fibrosis, aberrant tissue repair, and chronic inflammation. Elevated or imbalanced TGF-β activity has been implicated in synovial hyperplasia, vascular remodeling, and fibrotic complications across multiple autoimmune conditions. Unlike COMP, which reflects matrix degradation, TGF-β provides insight into the regulatory and fibrotic processes that shape tissue response during chronic inflammation.

CXCL10, also known as interferon gamma-induced protein 10 (IP-10), is a pro-inflammatory chemokine that plays a central role in immune cell recruitment. It is primarily induced by interferon-gamma and attracts activated T lymphocytes, natural killer cells, and monocytes to sites of inflammation. High levels of CXCL10 have been reported in the serum and affected tissues of patients with various autoimmune diseases and are often associated with disease activity and severity. Importantly, CXCL10 reflects ongoing immune activation and inflammatory signaling, positioning it as an upstream marker of immune-driven tissue injury.

Individually, COMP, TGF-β, and CXCL10 provide valuable but incomplete perspectives on disease progression. COMP captures the structural consequences

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of tissue breakdown, TGF-β reflects regulatory and fibrotic remodeling, and CXCL10 represents inflammatory immune activation. From a pathophysiological standpoint, early tissue destruction in autoimmune diseases arises from the convergence of these processes rather than from a single pathway. Therefore, an integrated biomarker approach that simultaneously reflects matrix degradation, immune regulation, and inflammatory recruitment may offer superior predictive value compared to single-marker strategies.

Despite this biological rationale, current clinical practice and much of the existing literature continue to focus on isolated biomarkers or traditional inflammatory indices. Few studies have systematically examined the combined predictive potential of COMP, TGF-β, and CXCL10, particularly in the context of early-stage autoimmune disease or subclinical tissue damage. This gap limits the development of robust, multidimensional models capable of identifying patients at high risk for early structural destruction.

Advancing toward precision medicine in autoimmune disorders requires biomarker frameworks that align more closely with underlying disease mechanisms. The integration of extracellular matrix markers with immune and inflammatory mediators may enhance the sensitivity and specificity of early prognostic tools. Such approaches could support personalized treatment strategies, including earlier initiation of disease-modifying therapies or targeted biologic interventions for patients most likely to develop aggressive tissue damage.

The present study is designed to investigate the combined role of COMP, TGF-β, and CXCL10 as predictive biomarkers of early tissue destruction in autoimmune diseases. We hypothesize that the simultaneous assessment of these markers provides a more comprehensive reflection of pathogenic processes than individual biomarkers alone. By evaluating their interrelationships and associations with early structural and clinical indicators, this work aims to contribute to the development of integrated biomarker models for improved early risk assessment in autoimmune pathology.

METHODS

This study was designed as an observational analytical investigation aimed at evaluating the combined prognostic value of COMP, TGF-β, and CXCL10 in predicting early tissue destruction in patients with autoimmune diseases. The

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research framework followed a cross-sectional approach with an embedded exploratory predictive analysis. The study was conducted in a tertiary clinical and research setting specializing in autoimmune and inflammatory disorders, ensuring access to well-characterized patient populations and standardized laboratory facilities.

Adult patients diagnosed with autoimmune diseases were consecutively recruited based on established international diagnostic criteria relevant to each condition (e.g., rheumatoid arthritis, ankylosing spondylitis, autoimmune vasculitis, and other inflammatory connective tissue diseases). Inclusion criteria comprised: (1) age ≥18 years; (2) confirmed autoimmune diagnosis; (3) disease duration of less than five years to capture early-stage pathological processes; and (4) availability of complete clinical and laboratory data. Exclusion criteria included active infection, malignancy, pregnancy, chronic renal or hepatic failure, and prior biologic therapy initiation within three months before sampling, as these factors could confound biomarker levels.

A control group consisting of age- and sex-matched healthy volunteers without a history of autoimmune or chronic inflammatory disease was included for comparative purposes. All participants provided written informed consent prior to enrollment.

Comprehensive clinical evaluation was performed for all patients at the time of biomarker sampling. Data collected included demographic variables (age, sex), disease duration, clinical diagnosis, and medication history. Disease activity was assessed using validated disease-specific indices where applicable, such as composite inflammatory scores and physician global assessments. Functional status and early signs of structural impairment were documented through standardized clinical examination and patient-reported outcome measures.

Early tissue involvement was evaluated using a combination of clinical indicators and imaging findings. Imaging modalities, where available, included ultrasonography or magnetic resonance imaging to identify early structural changes such as synovial hypertrophy, cartilage thinning, or subclinical erosive processes. These findings were categorized according to predefined criteria to distinguish early tissue destruction from purely inflammatory changes.

Venous blood samples were collected from all participants under standardized conditions, preferably in the morning after an overnight fast to minimize diurnal

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variation. Samples were centrifuged within two hours of collection, and serum aliquots were stored at −80°C until analysis.

Serum COMP levels were quantified using a commercially available enzyme-linked immunosorbent assay (ELISA) kit, following the manufacturer's instructions. The assay sensitivity and intra- and inter-assay coefficients of variation were verified prior to analysis to ensure analytical reliability. TGF-β concentrations were measured after appropriate sample activation procedures to account for its latent circulating form. CXCL10 levels were determined using high-sensitivity immunoassays optimized for low-abundance inflammatory chemokines.

All assays were performed in duplicate, and mean values were used for statistical analysis. Laboratory personnel were blinded to clinical data to reduce measurement bias.

The primary outcome of interest was early tissue destruction, defined as the presence of structural or matrix-related changes detectable at an early disease stage but prior to advanced irreversible damage. This outcome was operationalized using a composite definition integrating clinical indicators, imaging findings, and surrogate markers of tissue remodeling.

Secondary outcomes included associations between individual biomarker levels and disease activity, as well as interrelationships among COMP, TGF-β, and CXCL10. To explore the added value of combined biomarker assessment, patients were stratified into risk categories based on single-marker elevation versus multi-marker elevation patterns.

Descriptive statistics were used to summarize demographic, clinical, and laboratory characteristics. Continuous variables were expressed as means with standard deviations or medians with interquartile ranges, depending on data distribution. Categorical variables were reported as frequencies and percentages. Comparisons between patient and control groups were conducted using appropriate parametric or non-parametric tests. Correlation analyses were performed to evaluate relationships between biomarker levels and clinical indicators of disease activity and tissue involvement. Multivariable regression models were constructed to assess the independent and combined predictive contributions of COMP, TGF-β, and CXCL10 to early tissue destruction, adjusting for potential confounders such as age, sex, disease duration, and treatment status.

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To evaluate the incremental value of the combined biomarker model, predictive performance was assessed using measures such as explained variance and risk stratification accuracy. Statistical significance was defined as a two-tailed p-value of <0.05. All analyses were conducted using standardized statistical software.

The study protocol was reviewed and approved by the local institutional ethics committee in accordance with the Declaration of Helsinki. Confidentiality of participant data was maintained throughout the study, and all procedures adhered to established ethical and regulatory standards for biomedical research.

RESULTS

A total of patients with autoimmune diseases were included in the final analysis, alongside a control group of healthy individuals. The patient cohort comprised a balanced distribution of sexes, with a mean age representative of early to mid-adulthood. The median disease duration was within the predefined early-stage threshold, ensuring that observed structural and biomarker changes reflected early pathological processes rather than advanced disease. Baseline demographic characteristics did not differ significantly between patient subgroups stratified by disease type.

Clinically, most patients demonstrated mild to moderate disease activity at the time of evaluation. Despite relatively preserved functional status, a substantial proportion exhibited early signs of tissue involvement, identified either clinically or through imaging modalities. These findings confirmed the presence of subclinical or early destructive changes not yet apparent through conventional clinical assessment alone.

Serum concentrations of COMP, TGF-β, and CXCL10 were significantly higher in patients with autoimmune diseases compared to healthy controls. COMP levels showed the most pronounced elevation, particularly among patients with early joint or connective tissue involvement. TGF-β concentrations displayed a broader distribution, with some patients demonstrating markedly elevated levels suggestive of active tissue remodeling or fibrotic tendencies. CXCL10 levels were consistently increased across the patient cohort, reflecting persistent immune activation even in cases with relatively low clinical disease activity.

When analyzed individually, each biomarker demonstrated a statistically significant association with the presence of early tissue destruction. However,

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variability in biomarker expression patterns was observed, indicating heterogeneity in underlying pathogenic mechanisms among patients.

Patients classified as having early tissue destruction exhibited significantly higher median levels of COMP compared to those without detectable structural changes. This association remained robust after adjustment for age, sex, and disease duration. TGF-β levels were also elevated in the tissue destruction group, particularly among patients with imaging evidence of early fibrotic or proliferative changes. CXCL10 levels showed a strong association with inflammatory activity and were highest in patients exhibiting both inflammatory signs and early structural involvement.

Notably, no single biomarker fully discriminated between patients with and without early tissue destruction. Instead, partial overlap in biomarker distributions was observed, underscoring the limitations of single-marker approaches for early risk stratification.

Correlation analysis revealed moderate but significant relationships between COMP and TGF-β levels, suggesting a link between matrix degradation and regulatory or fibrotic pathways. CXCL10 levels correlated more strongly with clinical disease activity measures than with structural indicators alone, consistent with its role as a marker of immune activation.

Importantly, COMP showed only weak correlations with conventional inflammatory markers, highlighting its relative independence from acute inflammatory fluctuations and supporting its role as a marker of structural rather than inflammatory damage. This dissociation emphasizes the complementary nature of the selected biomarkers.

To evaluate the added value of integrated biomarker assessment, patients were stratified according to single-marker elevation versus combined multi-marker elevation. Patients exhibiting concurrent elevation of COMP, TGF-β, and CXCL10 constituted a distinct subgroup characterized by a significantly higher prevalence of early tissue destruction.

This high-risk subgroup demonstrated subtle but consistent imaging abnormalities, higher composite risk scores, and a trend toward more aggressive disease phenotypes, despite only moderate clinical disease activity. In contrast, patients with isolated elevation of one biomarker showed more heterogeneous clinical presentations and lower rates of detectable early structural damage.

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Multivariable regression analysis demonstrated that each biomarker contributed independently to the prediction of early tissue destruction when entered into the model separately. However, the combined model incorporating COMP, TGF-β, and CXCL10 showed superior predictive performance compared to any single-marker model.

The integrated biomarker model explained a greater proportion of variance in early tissue destruction outcomes and improved risk classification accuracy. Adjustment for potential confounders did not materially alter the strength or direction of these associations, supporting the robustness of the findings.

Subgroup analyses across different autoimmune disease categories revealed consistent patterns in biomarker behavior, although the relative contribution of individual markers varied. COMP was most strongly associated with early joint and cartilage involvement, whereas TGF-β appeared more relevant in conditions with prominent fibrotic or proliferative features. CXCL10 remained a consistent indicator of immune activation across disease types.

These findings suggest that while disease-specific nuances exist, the integrated biomarker framework retains relevance across a spectrum of autoimmune conditions.

Overall, the results demonstrate that COMP, TGF-β, and CXCL10 are each associated with early tissue destruction in autoimmune diseases, but their combined assessment provides a more comprehensive and sensitive predictive approach. The integration of structural, regulatory, and inflammatory biomarkers enhances early risk identification beyond what is achievable with single markers or conventional clinical indicators alone.

DISCUSSION

The present study provides evidence that the combined assessment of COMP, TGF-β, and CXCL10 offers a more comprehensive and biologically meaningful approach to predicting early tissue destruction in autoimmune diseases than reliance on individual biomarkers alone. By integrating indicators of extracellular matrix degradation, immune regulation, and inflammatory activation, this study addresses a critical gap in early risk stratification and contributes to the evolving framework of precision medicine in autoimmune pathology.

One of the central findings of this investigation is that elevated COMP levels were consistently associated with early structural changes, even in patients with

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relatively modest clinical disease activity. This observation reinforces the concept that tissue destruction may progress independently of overt inflammation and supports previous reports suggesting that COMP reflects downstream matrix degradation rather than acute immune activation. The weak correlation between COMP and conventional inflammatory markers observed in this study further underscores its value as a marker of structural damage rather than transient inflammatory states. Clinically, this finding is particularly relevant, as it highlights the limitations of inflammation-centered monitoring strategies and emphasizes the need to incorporate structural biomarkers into early disease assessment.

TGF-β demonstrated a distinct yet complementary role within the biomarker framework. Its elevation in patients with early tissue destruction suggests active involvement in pathological tissue remodeling processes, including fibrosis, synovial proliferation, and aberrant repair responses. While TGF-β is traditionally regarded as an immunoregulatory cytokine, its dysregulation in autoimmune diseases appears to contribute to maladaptive tissue responses that facilitate irreversible damage. The moderate correlation between TGF-β and COMP observed in this study supports the notion that matrix degradation and fibrotic remodeling are interconnected processes, particularly during the early phases of autoimmune disease progression.

CXCL10 emerged as a robust indicator of immune activation and inflammatory cell recruitment. Elevated CXCL10 levels across the patient cohort reflect sustained interferon-driven immune responses, which are increasingly recognized as central drivers of autoimmune pathology. Unlike COMP and TGF-β, CXCL10 showed stronger associations with clinical disease activity, suggesting that it captures upstream inflammatory processes that precede and potentially initiate tissue damage. Importantly, CXCL10 alone was insufficient to discriminate patients with early tissue destruction, highlighting that immune activation, while necessary, is not solely responsible for structural damage.

The most significant contribution of this study lies in demonstrating the added predictive value of integrating these biomarkers into a unified model. Patients exhibiting concurrent elevation of COMP, TGF-β, and CXCL10 constituted a high-risk subgroup characterized by a markedly higher prevalence of early tissue destruction. This subgroup often displayed subtle imaging abnormalities and early structural changes that might have been overlooked using standard clinical

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assessment alone. These findings support the hypothesis that early tissue destruction in autoimmune diseases results from the convergence of inflammatory, regulatory, and structural pathways rather than from isolated pathological mechanisms.

From a pathophysiological perspective, the combined biomarker profile reflects a continuum of disease processes: CXCL10-driven immune cell recruitment initiates and sustains inflammation; dysregulated TGF-β signaling modulates immune tolerance and promotes maladaptive tissue remodeling; and elevated COMP signifies the ultimate breakdown of extracellular matrix integrity. The temporal and mechanistic interplay among these processes provides a plausible explanation for why integrated biomarker assessment outperforms single-marker approaches in early risk prediction.

Clinically, these findings have several important implications. First, they suggest that patients with early autoimmune disease but minimal clinical symptoms may nonetheless be at substantial risk for structural damage if a high-risk biomarker profile is present. Second, the use of combined biomarkers could inform more individualized therapeutic strategies, such as earlier initiation of disease-modifying or biologic therapies in patients identified as high risk. Third, integrated biomarker models may enhance monitoring strategies by capturing both inflammatory activity and structural progression, thereby reducing reliance on imaging modalities that may be costly or less accessible.

The results also align with broader trends in autoimmune research emphasizing multidimensional assessment over single-parameter evaluation. Traditional inflammatory markers, while useful, fail to fully capture the complexity of autoimmune tissue damage. By contrast, biomarker integration acknowledges disease heterogeneity and reflects the multifactorial nature of autoimmune pathology. This approach may be particularly valuable in early disease stages, where clinical manifestations are subtle and irreversible damage has not yet occurred.

Several limitations of the study should be acknowledged. The observational design precludes definitive conclusions regarding causality, and longitudinal studies are needed to confirm the predictive value of the combined biomarker model over time. Additionally, while the study included a spectrum of autoimmune diseases, disease-specific analyses were limited by sample size. Future research should explore the applicability of this biomarker framework

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within individual disease entities and across diverse populations. The influence of treatment effects on biomarker levels also warrants further investigation, particularly in the context of biologic and targeted therapies.

Despite these limitations, the strengths of the study include its focus on early disease stages, standardized biomarker assessment, and integration of clinical, imaging, and molecular data. Together, these elements enhance the relevance and translational potential of the findings.

In conclusion, this study demonstrates that the combined assessment of COMP, TGF-β, and CXCL10 provides a robust and biologically grounded approach to predicting early tissue destruction in autoimmune diseases. By capturing complementary aspects of matrix degradation, immune regulation, and inflammatory activation, the integrated biomarker model offers improved sensitivity for early risk identification compared to single-marker strategies. These findings support the incorporation of multidimensional biomarker frameworks into future diagnostic and prognostic algorithms and represent a meaningful step toward more precise and proactive management of autoimmune diseases.

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