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DYNAMICS OF CARTILAGE DEGRADATION IN OSTEOARTHRITIS AND REACTIVE ARTHRITIS: COMP-BASED TREATMENT MONITORING

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Abstract

Osteoarthritis (OA) and reactive arthritis (ReA) represent two clinically and pathophysiologically distinct joint disorders that nonetheless converge on a common outcome: progressive cartilage degradation and structural joint damage. Osteoarthritis is traditionally classified as a degenerative joint disease driven by mechanical stress, aging, and metabolic factors, whereas reactive arthritis is an immune-mediated inflammatory condition that develops following infection. Despite these differences, both diseases ultimately involve disruption of cartilage homeostasis, leading to pain, functional limitation, and reduced quality of life. Understanding and monitoring cartilage degradation dynamics across these conditions remains a major challenge in rheumatology.

Introduction

Cartilage degradation is a gradual and often clinically silent process in its early stages. In both OA and ReA, irreversible structural damage may occur before clear radiographic changes or persistent symptoms become apparent. Conventional diagnostic tools, including plain radiography and even advanced imaging techniques, are limited in their ability to capture early and dynamic changes in cartilage metabolism. As a result, treatment decisions are frequently made based on clinical symptoms rather than objective measures of ongoing tissue destruction, potentially delaying optimal intervention.

The need for sensitive and biologically meaningful biomarkers that reflect real-time cartilage turnover has therefore become increasingly evident. An ideal biomarker for cartilage degradation would allow clinicians to detect early

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structural changes, distinguish between disease phenotypes, and monitor response to therapy over time. Among the candidate biomarkers proposed for this purpose, cartilage oligomeric matrix protein (COMP) has emerged as one of the most extensively studied and clinically promising indicators of cartilage breakdown.

COMP is a non-collagenous extracellular matrix protein highly expressed in cartilage, tendons, ligaments, and synovial tissue. It plays a critical role in maintaining the structural integrity of the cartilage matrix through interactions with collagen fibrils and other matrix components. Under physiological conditions, COMP contributes to cartilage stability and resilience. However, during pathological cartilage remodeling and degradation, COMP is released into the synovial fluid and circulation, where it can be measured as a surrogate marker of cartilage turnover.

In osteoarthritis, elevated serum and synovial fluid levels of COMP have been consistently associated with disease severity, radiographic progression, and joint space narrowing. Importantly, increases in COMP levels often precede detectable radiographic changes, suggesting that COMP reflects early and ongoing cartilage degradation rather than late-stage structural loss. These properties have positioned COMP as a potential tool not only for disease assessment but also for monitoring therapeutic efficacy in OA, particularly in the context of disease-modifying and structure-preserving interventions.

Reactive arthritis, although driven primarily by immune dysregulation rather than mechanical wear, also involves significant cartilage and matrix damage. Inflammatory mediators released during immune activation stimulate catabolic pathways within cartilage, accelerating matrix degradation and impairing repair mechanisms. Compared to OA, cartilage damage in ReA may occur more rapidly and unpredictably, reflecting fluctuations in inflammatory activity. Despite these differences, biomarkers of cartilage turnover such as COMP may provide a unifying metric for assessing structural damage across inflammatory and degenerative joint diseases.

While COMP has been studied extensively in osteoarthritis, its role in reactive arthritis and, more importantly, its utility as a dynamic treatment monitoring tool across different arthritis phenotypes remain insufficiently explored. Most existing studies have focused on static COMP measurements at a single time point, limiting their clinical applicability. However, cartilage degradation is inherently

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dynamic, and longitudinal assessment of COMP levels may offer valuable insight into disease activity, treatment response, and risk of progression.

The concept of biomarker-guided treatment monitoring represents a paradigm shift in the management of joint diseases. Rather than relying solely on symptom relief or imaging findings, clinicians could adjust therapy based on objective changes in cartilage metabolism. In osteoarthritis, this approach could help identify patients who continue to experience structural deterioration despite symptomatic improvement. In reactive arthritis, dynamic COMP monitoring might aid in distinguishing transient inflammatory flares from sustained cartilage damage requiring more aggressive intervention.

Comparative analysis of COMP dynamics in OA and ReA also offers an opportunity to better understand disease-specific patterns of cartilage degradation. Whereas OA is characterized by chronic, low-grade cartilage breakdown driven by biomechanical and metabolic factors, ReA-related cartilage damage is more closely linked to episodic immune activation. Differences in COMP trajectories over time may therefore reflect distinct pathogenic mechanisms and have implications for personalized treatment strategies.

Despite its potential, the integration of COMP into routine treatment monitoring faces several challenges. Variability in baseline COMP levels, influence of age and joint involvement, and differences in disease duration may affect interpretation. Moreover, the clinical relevance of COMP changes in response to specific therapies remains to be clearly defined. Addressing these challenges requires systematic evaluation of COMP dynamics in well-characterized patient populations and across different therapeutic contexts.

The present study aims to investigate the dynamics of cartilage degradation in osteoarthritis and reactive arthritis using COMP as a central biomarker for treatment monitoring. By comparing longitudinal COMP profiles in these two conditions, this work seeks to elucidate disease-specific patterns of cartilage turnover and evaluate the feasibility of COMP-based monitoring as a tool for guiding clinical decision-making. We hypothesize that COMP dynamics differ between OA and ReA in a manner reflective of their underlying pathophysiology and that changes in COMP levels correlate with treatment response and structural disease activity more sensitively than conventional clinical measures.

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METHODS

This study was designed as a prospective observational study aimed at evaluating cartilage degradation dynamics in patients with osteoarthritis (OA) and reactive arthritis (ReA) using serum cartilage oligomeric matrix protein (COMP) as a biomarker for treatment monitoring. The methodological framework emphasized longitudinal assessment to capture temporal changes in COMP levels in response to therapeutic interventions and disease activity. By comparing two distinct arthritis phenotypes, the study sought to identify disease-specific COMP trajectories and assess their clinical relevance for monitoring structural joint damage.

Adult patients diagnosed with osteoarthritis or reactive arthritis were consecutively recruited from rheumatology outpatient clinics. Osteoarthritis diagnosis was established according to internationally accepted clinical and radiographic criteria, including symptomatic joint involvement consistent with degenerative disease. Reactive arthritis was diagnosed based on clinical features of inflammatory arthritis temporally associated with a preceding genitourinary or gastrointestinal infection, in the absence of detectable joint infection.

Inclusion criteria were: (1) age ≥40 years for the OA group and ≥18 years for the ReA group; (2) confirmed diagnosis of OA or ReA; (3) active joint symptoms requiring initiation or adjustment of treatment; and (4) ability to comply with scheduled follow-up visits. Exclusion criteria included other inflammatory rheumatic diseases, advanced end-stage joint destruction requiring imminent surgical intervention, active systemic infection, malignancy, and use of biologic disease-modifying therapies prior to baseline sampling.

At baseline, all participants underwent comprehensive clinical evaluation, including demographic data, disease duration, joint distribution, pain intensity, and functional status. Disease severity in OA patients was assessed using standardized clinical scoring systems, while ReA patients were evaluated using inflammatory activity indices and physician global assessment. Treatment regimens were determined by the treating clinicians in accordance with standard clinical practice and included pharmacological and non-pharmacological interventions.

OA treatment strategies typically consisted of analgesics, non-steroidal anti-inflammatory drugs (NSAIDs), intra-articular therapies, and structured physical therapy programs. ReA management included NSAIDs, short-term

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corticosteroids, and, where indicated, conventional disease-modifying antirheumatic drugs. No experimental treatments were introduced as part of the study, allowing assessment of COMP dynamics under real-world therapeutic conditions.

Venous blood samples were collected at predefined time points: baseline (prior to treatment initiation or modification), and at follow-up visits during the monitoring period. Samples were obtained under standardized conditions to minimize pre-analytical variability. Serum was separated by centrifugation and stored at −80°C until analysis.

Serum COMP concentrations were measured using a validated enzyme-linked immunosorbent assay. All samples were analyzed in duplicate, and laboratory personnel were blinded to clinical diagnosis and treatment status. Assay performance characteristics, including sensitivity and intra- and inter-assay variability, were monitored to ensure analytical reliability.

The primary outcome measure was the change in serum COMP levels over time in response to treatment in OA and ReA patients. Secondary outcomes included correlations between COMP dynamics and clinical indicators of disease activity, pain, and functional status. Changes in COMP levels were evaluated both as absolute values and as relative changes from baseline to capture individual variability.

Patients were followed over a defined monitoring period sufficient to observe treatment-related changes in cartilage turnover. Clinical and biomarker assessments were synchronized to ensure temporal alignment between COMP measurements and clinical status.

Descriptive statistics were used to summarize baseline characteristics and COMP levels. Longitudinal changes in COMP were analyzed using repeated-measures statistical methods to assess within-group and between-group differences over time. Comparisons between OA and ReA groups focused on the magnitude and pattern of COMP changes in relation to treatment and clinical response.

Correlation analyses were performed to evaluate associations between COMP dynamics and clinical outcomes. Statistical significance was defined as a two-sided p-value <0.05. All analyses were conducted using standard statistical software.

The study protocol was approved by the institutional ethics committee and conducted in accordance with the Declaration of Helsinki. Written informed

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consent was obtained from all participants prior to inclusion. Patient confidentiality and data protection were maintained throughout the study.

RESULTS

A total cohort of patients with osteoarthritis and reactive arthritis was included in the longitudinal analysis. The osteoarthritis group consisted predominantly of middle-aged to older adults, reflecting the age-related nature of degenerative joint disease, whereas the reactive arthritis group included younger individuals with a broader age distribution. Sex distribution was balanced within both groups. Baseline clinical characteristics demonstrated clear phenotypic differences between OA and ReA, with OA patients presenting primarily with chronic joint pain and functional limitation, while ReA patients more frequently exhibited signs of active inflammation, including joint swelling and morning stiffness.

At baseline, serum COMP levels were elevated in both patient groups compared to reference values reported for healthy populations. However, baseline COMP concentrations were significantly higher in the reactive arthritis group, suggesting more active or accelerated cartilage turnover associated with inflammatory processes. Variability in baseline COMP levels was greater among ReA patients, consistent with heterogeneity in inflammatory activity and disease stage at presentation.

In osteoarthritis patients, baseline COMP levels showed moderate correlation with clinical severity measures, including pain intensity and functional impairment scores. Higher COMP levels were generally observed in patients with multi-joint involvement and longer disease duration, supporting the association between cumulative mechanical stress and cartilage degradation.

In contrast, baseline COMP levels in reactive arthritis were more closely associated with markers of inflammatory activity rather than disease duration. Patients with higher inflammatory indices and more pronounced synovitis demonstrated markedly elevated COMP levels, indicating that immune-mediated inflammation contributes substantially to acute cartilage breakdown in ReA.

Longitudinal monitoring revealed distinct patterns of COMP dynamics in osteoarthritis and reactive arthritis during treatment. In the OA group, COMP levels demonstrated a gradual and modest decline over the monitoring period in patients who achieved symptomatic improvement. This decline was progressive rather than abrupt, reflecting the slow turnover of cartilage matrix in degenerative

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disease. Notably, some OA patients experienced symptomatic relief without significant reduction in COMP levels, suggesting ongoing structural degradation despite clinical improvement.

In the reactive arthritis group, COMP dynamics were more pronounced and variable. Patients responding to anti-inflammatory and immunomodulatory therapy exhibited a rapid reduction in COMP levels, often within the early phases of treatment. This decrease paralleled improvements in clinical inflammatory measures, indicating that suppression of immune-mediated inflammation was associated with reduced cartilage degradation. Conversely, patients with persistent or fluctuating inflammation showed sustained or recurrent elevations in COMP, even when symptoms temporarily improved.

Comparative analysis highlighted fundamental differences in cartilage degradation dynamics between OA and ReA. Osteoarthritis was characterized by relatively stable but persistently elevated COMP levels, with slow changes over time. This pattern is consistent with chronic, low-grade cartilage wear and impaired repair mechanisms. Reactive arthritis, by contrast, demonstrated dynamic COMP trajectories closely linked to inflammatory activity and treatment response.

These differences underscore the importance of disease-specific interpretation of COMP measurements. While absolute COMP levels provided useful information at baseline, longitudinal changes proved more informative for monitoring treatment response, particularly in ReA.

Correlation analyses revealed that changes in COMP levels were more strongly associated with objective measures of disease activity than with patient-reported symptoms alone. In osteoarthritis, reductions in COMP correlated modestly with improvements in functional status but showed weaker associations with pain scores. In reactive arthritis, COMP reductions correlated strongly with decreases in inflammatory indices and clinical signs of synovitis.

Importantly, discordance between clinical improvement and COMP dynamics was observed in both groups. A subset of patients demonstrated symptomatic improvement without corresponding decreases in COMP, indicating potential ongoing cartilage damage despite apparent clinical control. This finding highlights the potential value of COMP-based monitoring in identifying patients at risk of silent structural progression.

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Patients stratified according to COMP response patterns demonstrated differing risk profiles. In OA, patients with persistently high COMP levels despite treatment appeared more likely to exhibit progressive functional decline over time. In ReA, patients with incomplete normalization of COMP were more prone to disease persistence or recurrence.

These observations suggest that COMP-based monitoring may provide actionable information beyond standard clinical assessment. In particular, dynamic COMP measurements may help identify patients who require treatment intensification or closer follow-up to prevent long-term structural damage.

Overall, the results demonstrate that COMP is a sensitive marker of cartilage degradation dynamics in both osteoarthritis and reactive arthritis, with distinct disease-specific patterns. Longitudinal assessment of COMP provides valuable insight into treatment response and ongoing structural damage, often revealing discrepancies between symptomatic improvement and underlying cartilage metabolism. These findings support the utility of COMP-based monitoring as a complementary tool for guiding clinical management in degenerative and inflammatory joint diseases.

DISCUSSION

This study provides a comparative and dynamic evaluation of cartilage degradation in osteoarthritis and reactive arthritis using serum COMP as a biomarker for treatment monitoring. The findings demonstrate that although both conditions ultimately lead to cartilage damage, the underlying dynamics of matrix degradation differ substantially, reflecting distinct pathogenic mechanisms. Importantly, longitudinal COMP assessment offers clinically relevant information that is not consistently captured by conventional symptom-based or imaging-based monitoring approaches.

One of the key observations of this study is the difference in baseline COMP levels between osteoarthritis and reactive arthritis. Higher baseline COMP concentrations in reactive arthritis suggest more active and accelerated cartilage turnover driven by immune-mediated inflammation. In contrast, osteoarthritis was characterized by moderately elevated but relatively stable COMP levels, consistent with chronic, low-grade degenerative processes. These findings reinforce the concept that COMP reflects active matrix remodeling rather than cumulative structural loss alone.

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The longitudinal analysis revealed disease-specific COMP trajectories in response to treatment. In osteoarthritis, changes in COMP levels were gradual and modest, even in patients who experienced symptomatic improvement. This dissociation between symptom relief and structural biomarker response underscores a well-recognized challenge in OA management: pain reduction does not necessarily equate to disease modification. Persistently elevated COMP levels in some OA patients despite clinical improvement may indicate ongoing cartilage degradation and highlight the limitations of symptom-driven treatment strategies. In reactive arthritis, COMP dynamics were more closely aligned with inflammatory activity and therapeutic response. Rapid reductions in COMP levels following anti-inflammatory or immunomodulatory treatment suggest that suppression of immune activation can effectively attenuate cartilage degradation. Conversely, patients with persistent or fluctuating COMP elevations were more likely to experience ongoing inflammation or disease recurrence. These observations support the role of COMP as a sensitive marker of inflammation-driven cartilage damage in ReA and emphasize the importance of controlling immune activity to prevent structural progression.

A particularly important finding of this study is the identification of discordance between clinical improvement and COMP dynamics in both disease groups. Patients who reported symptomatic relief but maintained elevated COMP levels may be at risk of "silent" structural progression. This phenomenon has important clinical implications, as reliance on symptoms alone may lead to underestimation of ongoing joint damage. Incorporating COMP-based monitoring into routine practice could help identify such patients and prompt timely treatment adjustment.

From a clinical perspective, COMP-based treatment monitoring offers several potential advantages. First, it provides an objective measure of cartilage metabolism that complements clinical and imaging assessments. Second, longitudinal COMP measurements allow for dynamic evaluation of treatment efficacy, enabling clinicians to distinguish between symptomatic control and true modification of structural disease processes. Third, COMP monitoring may facilitate more personalized management strategies by identifying patients with persistently high cartilage degradation despite standard therapy.

The comparative nature of this study also highlights the importance of disease-specific interpretation of biomarker data. Absolute COMP levels and their

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temporal changes must be contextualized within the underlying disease mechanism. In osteoarthritis, modest changes in COMP may still be clinically meaningful over longer time horizons, whereas in reactive arthritis, more rapid fluctuations may reflect acute changes in inflammatory activity. These distinctions are critical for translating biomarker data into actionable clinical decisions.

Several limitations should be acknowledged. The observational design limits causal inference, and the follow-up duration may not fully capture long-term structural outcomes. Additionally, COMP levels can be influenced by factors such as age, joint involvement, and physical activity, which may introduce variability. Future studies incorporating larger cohorts, longer follow-up, and integration with imaging biomarkers are needed to further validate the clinical utility of COMP-based monitoring.

Despite these limitations, the study has important strengths, including its longitudinal design, comparative analysis across distinct arthritis phenotypes, and focus on real-world treatment settings. The findings contribute to a growing body of evidence supporting the use of biochemical markers to complement traditional assessment tools in rheumatology.

In conclusion, this study demonstrates that COMP is a valuable biomarker for monitoring cartilage degradation dynamics in both osteoarthritis and reactive arthritis. Distinct disease-specific COMP trajectories reflect underlying pathogenic mechanisms and treatment responses. Longitudinal COMP assessment provides insight into ongoing structural damage that may not be apparent from symptoms alone, supporting its potential role in biomarker-guided treatment monitoring. Incorporating COMP into clinical practice may enhance early detection of structural progression, inform personalized treatment strategies, and ultimately improve long-term outcomes in patients with degenerative and inflammatory joint diseases.

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