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ESTABLISHED AND EMERGING BIOMARKERS OF INFLAMMATION IN RHEUMATOID ARTHRITIS: A LITERATURE REVIEW

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ABSTRACT

Background and Objective: Rheumatoid arthritis (RA) is a chronic systemic autoimmune disease characterized by persistent inflammation, progressive joint damage, and systemic consequences contributing to increased morbidity and mortality. Although conventional biomarkers are fundamental for diagnosis and disease activity assessment, their prognostic accuracy remains limited. This review aimed to summarize current evidence on emerging inflammatory biomarkers in RA, with emphasis on their prognostic value and translational relevance for personalized disease management.
Methods: A structured literature search was conducted in major scientific databases, covering English-language publications from 2014 to March 2025. Studies were selected according to predefined inclusion criteria to identify clinically relevant data on novel inflammatory biomarkers in RA.
Results: Emerging biomarkers, including novel autoantibodies, 14-3-3η protein, calprotectin, selected cytokines, adipokines, circulating microRNAs, and hematological inflammatory indices, provide complementary insights into immune dysregulation and systemic inflammation. Several markers are associated with radiographic progression, extra-articular involvement, and treatment response. Multibiomarker disease activity scores represent an integrative approach linking molecular pathways with clinical assessment and risk stratification.
Conclusions: Integration of conventional and emerging biomarkers may enhance prognostic precision and support precision medicine strategies in RA. However, robust prospective validation and cost-effectiveness analyses are required before routine clinical implementation.

KEYWORDS

Rheumatoid Arthritis, Inflammatory Biomarkers, Prognostic Markers, microRNA, Adipokines, Autoantibody

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Introduction

Chronic inflammatory diseases represent a major challenge for modern healthcare systems due to their long-term course, complex pathophysiology, and significant socioeconomic burden. Rheumatoid arthritis (RA) is one of the most prevalent chronic autoimmune disorders, characterized by persistent inflammation, progressive joint damage, reduced quality of life, and increased cardiovascular risk and pulmonary complications, contributing to elevated morbidity and mortality rates. Its prevalence in the population across different regions of the world is estimated to be approximately 0.5–1%. (Abdelhafiz, Kilborn, & Bukhari 2021)

Inflammation is a key mechanism underlying the development and progression of rheumatoid arthritis. The disease is driven by dysregulated immune responses involving inflammatory cells, soluble mediators, and molecular signaling pathways that contribute to both local joint destruction and systemic manifestations. Traditionally, the assessment of inflammatory activity in RA has relied on established laboratory markers such as C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), rheumatoid factor (RF), and anti-citrullinated protein antibodies (ACPAs). While these biomarkers are widely used in clinical practice, their ability to predict long-term disease progression, structural damage, and treatment outcomes remains limited (Sahin et al., 2025).

In recent years, growing attention has been directed toward the identification of emerging inflammatory biomarkers that may improve prognostic assessment in rheumatoid arthritis. Advances in biomedical research and analytical technologies have enabled the investigation of novel biomarkers reflecting different aspects of inflammatory activity, including cytokines, adipokines, hematological indices and microRNAs. These biomarkers offer new opportunities for a more precise evaluation of disease severity and future outcomes, potentially supporting earlier intervention and individualized therapeutic strategies (Karimov et al., 2025).

Emerging evidence indicates that key inflammatory mediators, including interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α), play a central role in the pathogenesis and systemic manifestations of rheumatoid arthritis, contributing not only to disease activity but also to long-term structural damage and extra-articular complications (Smolen et al., 2016; Mena-Vázquez et al., 2023). Additionally, novel molecular markers, including circulating microRNAs and adipokines, have been linked to immune regulation and chronic inflammation, highlighting their potential role as innovative prognostic tools (Machaj et al., 2025; Bilski et al., 2023). From a broader perspective, easily accessible biomarkers derived from routine laboratory tests, such as hematological inflammatory indices, may represent cost-effective solutions applicable in diverse healthcare settings.

This review aims to summarize current evidence on emerging inflammatory biomarkers in rheumatoid arthritis, with a particular focus on their potential prognostic significance and their relevance for innovative approaches to disease management.

Methods

The review followed a predefined search and selection strategy to enhance transparency and methodological rigor. A comprehensive literature search was conducted in PubMed, Google Scholar, and the MDPI database. The search covered publications from January 2014 to June 2026 and was limited to English-language articles with full-text availability. Earlier landmark publications and internationally recognized classification criteria (e.g., ACR/EULAR 2010 criteria) were included when necessary to provide appropriate clinical context. Search terms included combinations of the following keywords: “rheumatoid arthritis,” “inflammatory biomarkers,” “prognostic markers,” “microRNA”, “adipokines,”.„IL-6,” „TNF-α,”. Additionally, reference lists of eligible articles were manually screened to identify further relevant studies. Data collection involved a two-stage screening process. First, titles and abstracts were reviewed to assess relevance. Second, full texts of potentially eligible studies were examined in detail according to predefined inclusion and exclusion criteria.

Results

1. Autoantibodies

Autoantibodies play a fundamental role in the diagnosis of rheumatoid arthritis (RA), with rheumatoid factor (RF) and anti-citrullinated protein antibodies (ACPA) being the primary markers. Their presence is included among the key classification criteria for RA according to the 2010 ACR/EULAR guidelines (Aletaha et al., 2010). The sensitivity of RF ranges from 60% to 80%, with a specificity of approximately 70–85%, whereas anti-CCP tests demonstrate a comparable sensitivity of 60–75% but higher specificity, reaching 95–99% (Soyfoo & Sarrand, 2026). Moreover, the titers at which these antibodies occur are clinically significant, as higher titers correlate with more aggressive disease forms (Soyfoo & Sarrand, 2026). Anti-CCP antibodies have been associated with increased radiographic joint damage, while RF is linked to bone erosions detectable by ultrasound but without a clear association with joint destruction (Abdelhafiz et al., 2023). Importantly, 20–30% of patients may lack these antibodies, presenting with seronegative RA, which can delay diagnosis and management (Soyfoo & Sarrand, 2026).

Emerging autoantibodies offer promise as diagnostic markers, particularly in patients with seronegative rheumatoid arthritis (RA). Anti-Carbamylated Protein antibodies (Anti-CarP) can be detected in 16–30% of ACPA-negative patients, and their presence increases the risk of radiographic progression, potentially preceding disease onset by several years (Soyfoo & Sarrand, 2026). Anti-Peptidylarginine Deiminase antibodies (Anti-PAD) are potential markers in ACPA-positive patients, with Anti-PAD3/4 associated with radiographic joint damage (Sahin et al., 2025). Anti-Acetylated Protein and anti-malondialdehyde-acetaldehyde antibodies (anti-MAA) may also be useful in seronegative patients, with positive results in approximately 15–20% of cases (Soyfoo & Sarrand, 2026). Currently, these antibodies are not employed in routine clinical diagnostics (Sahin et al., 2025; Soyfoo & Sarrand, 2026).

2. Acute phase reactants

Acute-phase indicators, commonly measured as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), are also included among the key classification criteria for rheumatoid arthritis (RA) according to the 2010 ACR/EULAR guidelines. (Aletaha et al., 2010) They are components of disease activity assessment tools such as DAS28, and CRP is additionally incorporated into SDAI and CDAI scores, however, these

markers are not specific to joint inflammation, as their levels can increase in various clinical situations (Sahin et al., 2025; Soyfoo & Sarrand, 2026).

CRP does not always correlate with disease activity, in nearly half of patients with normal CRP levels, joint inflammation was still present (Sahin et al., 2025). Nevertheless, CRP levels should be considered when selecting treatment strategies for RA patients (Sahin et al., 2025).

ESR can be influenced by multiple factors, including age, sex, physical activity, stress, and pregnancy. Elevated ESR is associated with poorer prognosis in RA patients and has been linked to a reduced response to both conventional synthetic DMARDs (csDMARDs) and biological DMARDs (bDMARDs) (Sahin et al., 2025).

3. Cytokines

Cytokines such as IL-6, IL-1, IL-8, IL-18, IL-23, and tumor necrosis factor-alpha (TNF-α) play a key role in the pathogenesis of rheumatoid arthritis (RA), and their dysregulated levels can be detected even in the preclinical phase of the disease (Sahin et al., 2025). In patients with active RA, serum TNF-α concentrations are elevated, which has led to the development of biologic therapies targeting this proinflammatory mediator—TNF-α inhibitors (Sahin et al., 2025). IL-6 is a central cytokine in RA with effector functions similar to TNF-α. It stimulates the synthesis of acute-phase proteins, including CRP, thereby amplifying systemic inflammation, and its elevated levels correlate with higher disease activity. IL-6 inhibitors, such as tocilizumab and sarilumab, are used therapeutically (Sahin et al., 2025). Measurement of individual cytokines does not provide sufficient specificity or reproducibility to guide personalized clinical decisions. Both TNF-α and IL-6 are not routinely employed in the diagnosis or monitoring of RA, as their elevated levels are also observed in other inflammatory diseases (Sahin et al., 2025; Soyfoo & Sarrand, 2026). Selected cytokines, including MCP-1/CCL2 and SDF-1α, have been shown to correlate with the diagnosis of interstitial lung disease associated with RA (RA-ILD) (Mena-Vázquez et al., 2023). Furthermore, elevated IL-18 levels were associated not only with the presence of RA-ILD but also with more advanced and rapidly progressing pulmonary changes (Mena-Vázquez et al., 2023).

4. Hematological inflammatory indices

Hematological markers of systemic inflammation such as the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and lymphocyte-to-monocyte ratio (LMR) are considered novel, sensitive indicators of inflammatory response, complementing traditional markers such as ESR, CRP, and DAS28 (Targońska-Stępniak et al., 2020). Recent studies have shown that NLR and PLR are significantly elevated in RA patients compared to healthy controls and positively correlate with disease activity, inflammatory markers, and treatment response, while LMR has also been associated with disease activity in RA (Targońska-Stępniak et al., 2020). NLR may serve as a useful marker for monitoring disease activity, as six months of rituximab treatment significantly reduced NLR levels, and RA patients with higher baseline NLR receiving TNF inhibitors exhibited a poorer EULAR response at 12 weeks (Choe, Lee, & Kim, 2023). Hematological indices, including NLR, PLR, LMR, and the Systemic Immune-Inflammation Index (SII), did not correlate with RF or anti-CCP antibody status, suggesting that these markers primarily reflect inflammatory activity rather than serological status in RA (Choe et al., 2023). In addition, because these hematological indices are derived from routine complete blood count tests, they are inexpensive, widely accessible, and easily implementable in everyday clinical practice. However, they remain nonspecific markers of systemic inflammation (Zhang et al., 2026).

5. Adipokines

In patients with rheumatoid arthritis (RA), adipokines including adiponectin, leptin, resistin, and visfatin are elevated. Adipokines are produced not only by adipose tissue but also by chondrocytes and synoviocytes (Bilski et al., 2023). Primarily secreted by visceral adipose tissue, adipokines promote inflammation in RA, stimulating fibroblast-like synoviocytes and macrophages to produce proinflammatory cytokines and MMPs, which contribute to cartilage and matrix degradation, as well as upregulating RANKL to enhance osteoclast activity and bone erosion (Bilski et al., 2023).

Adiponectin plays a particularly important role: although it exerts anti-inflammatory effects in metabolic diseases, in RA it demonstrates proinflammatory activity. It increases IL-6, IL-8, and MMP production in synoviocytes and chondrocytes, contributing to cartilage degradation and progression of destructive joint changes (Szumilas et al., 2020). A meta-analysis showed that circulating adiponectin levels are significantly higher in RA patients than in controls, while circulating visfatin levels are also elevated, suggesting a positive correlation between visfatin levels and RA activity (Lee & Bae, 2018). Bilski et al. (2023) reported a positive

correlation between visfatin levels, anti-CCP antibodies, RA progression, disease activity, and radiographic joint damage.

Another meta-analysis found no significant correlation between leptin levels and RA activity, although leptin concentrations were higher in RA patients compared to controls (Lee & Bae, 2016).

Resistin is elevated in the serum and synovial fluid of RA patients, correlates with inflammatory markers (CRP, ESR), and stimulates the production of proinflammatory mediators and VEGF in joint cells (Toussirot, 2023). Anti-TNFα therapy reduces serum resistin levels in RA patients, indicating a strong association with inflammatory markers (Bilski et al., 2023). Early in active RA, resistin measurement may serve as a valuable biomarker for identifying individuals at high risk of developing erosive disease (Bilski et al., 2023).

Overall, adipokines play a significant role in RA pathogenesis and may serve as potential biomarkers; however, further research is needed to explore their utility and potential for targeted therapies in the future (Bilski et al., 2023).

6. 14-3-3η Protein

The 14-3-3 protein family comprises seven isoforms present in the circulation, whose biological activity is associated with the regulation of inflammatory processes. The 14-3-3η isoform has been shown to be elevated in both the serum and synovial fluid of patients with rheumatoid arthritis (Abdelhafiz, Kilborn, & Bukhari, 2021). Elevated levels of this marker are observed in 60–75% of RA patients, with a specificity of 78–93% (Soyfoo & Sarrand, 2026).

This protein family exhibits significant additive value—simultaneous measurement of RF, ACPA, and 14-3-3η increases sensitivity to over 80% for early RA detection. Importantly, elevated 14-3-3η levels are also observed in 45–58% of seronegative patients, substantially improving detection in this diagnostically challenging group (Soyfoo & Sarrand, 2026). Moreover, 14-3-3η levels have been associated with radiographic progression, particularly in early RA, compared to patients with more advanced disease already under treatment (Sahin et al., 2025).

14-3-3η may represent a promising diagnostic and prognostic biomarker in RA; however, despite growing evidence supporting its utility, it has not yet been incorporated into current disease classification criteria (Sahin et al., 2025; Soyfoo & Sarrand, 2026).

7. Calprotectin (S100A8/A9)

Calprotectin, a heterodimer composed of S100A8 and S100A9 belonging to the S100 protein family, plays a critical role in initiating and regulating the innate immune response, promoting chemotaxis and migration of phagocytes, and modulating macrophages and neutrophils (Inciarte-Mundo, Frade-Sosa, & Sanmartí, 2022). Elevated calprotectin levels have been observed in both the serum and synovial fluid of patients with rheumatoid arthritis (RA) (Inciarte-Mundo et al., 2022). Studies have demonstrated a significant association between serum calprotectin concentrations and RA activity markers, including DAS28-CRP, as well as synovial membrane inflammation detected by ultrasound (Sahin et al., 2025; Soyfoo & Sarrand, 2026).

Increased calprotectin levels in patients in clinical remission may serve as a prognostic factor for subsequent disease relapse. Calprotectin has also been shown to be useful as a predictive marker of relapse in patients undergoing gradual tapering of biologic therapy (Inciarte-Mundo et al., 2022; Soyfoo & Sarrand, 2026). However, the role of calprotectin as a predictive biomarker for RA relapse has not been definitively established, as targeted randomized clinical trials assessing its practical prognostic value are still lacking (Inciarte-Mundo et al., 2022).

8. MicroRNAs (miRNA)

MicroRNAs (miRNAs) are a class of small, single-stranded, non-coding RNA molecules approximately 21–23 nucleotides in length that regulate gene expression at the post-transcriptional level. miRNAs modulate numerous biological processes, ranging from cell development and differentiation to homeostasis and disease pathogenesis (Hynes & Kakumani, 2024). The miRNA expression profile may be particularly relevant in patients with seronegative rheumatoid arthritis (RA); however, their clinical utility is currently limited (Machaj et al., 2025).

To enhance diagnostic precision, panels of miRNAs have been analyzed to identify combinations with the highest sensitivity and specificity. Simultaneous upregulation of miR-24, miR-26a, and miR-125a has been shown to provide the best diagnostic performance in detecting RA, regardless of serological status (Machaj et al., 2025). Nevertheless, interactions between miRNAs and other epigenetic mechanisms, as well as their combined influence on RA pathogenesis, remain insufficiently understood. Circulating miRNAs, however, exhibit considerable potential as biomarkers for disease diagnosis, assessment of progression, and monitoring therapeutic responses (Machaj et al., 2025).

9. Multi-Biomarker Disease Activity Assessment

The Multibiomarker Disease Activity (MBDA) system was developed to assess disease activity in rheumatoid arthritis (RA), which is critical for achieving remission or low disease activity. It is based on the analysis of 12 serum biomarkers, including IL-6, TNFR1, VCAM-1, EGF, VEGF-A, YKL-40, MMP-1, MMP-3, CRP, SAA, leptin, and resistin (Sahin et al., 2025).

The MBDA score (ranging from 1 to 100) classifies RA activity as low (<30), moderate (30–44), or high (>44), correlates with DAS28-CRP ($r \approx 0.5$), and predicts radiographic progression independently of clinical assessments (Soyfoo & Sarrand, 2026). Limitations of widespread MBDA use include obesity and other inflammatory conditions, high costs, and variable effectiveness depending on therapy—particularly IL-6 and JAK inhibitors, which directly affect the measured biomarkers. Moreover, major EULAR guidelines do not currently recommend its routine use (Soyfoo & Sarrand, 2026).

Despite these limitations, MBDA may have significant value in RA management, serving both to assess disease activity and to predict the progression of radiographic changes (Meznerics et al., 2023).

Discussion

This review underscores the complementary roles of conventional and emerging biomarkers in rheumatoid arthritis (RA). Established markers such as CRP and ESR remain fundamental for assessing inflammatory activity and are incorporated into composite indices including DAS28, as reflected in current classification and management frameworks (Aletaha et al., 2010; Sahin et al., 2025). However, they do not fully capture disease heterogeneity or reliably predict structural progression.

Recent research has expanded the biomarker spectrum to include novel autoantibodies, 14-3-3η protein, cytokines, adipokines, calprotectin, and microRNAs. The 14-3-3η protein has shown additive diagnostic value, particularly in early RA (Abdelhafiz et al., 2021). Pro-inflammatory cytokines, particularly TNF-α and IL-6, constitute central mediators in the inflammatory network underlying rheumatoid arthritis and have significantly influenced the development of targeted biologic therapies. However, despite their well-established pathogenic relevance, the routine clinical measurement of individual cytokines remains limited due to issues related to specificity, standardization, and cost-effectiveness (Smolen et al., 2016; Sahin et al., 2025). Adipokines are increasingly recognized as mediators linking metabolic and inflammatory pathways with joint damage (Bilski et al., 2023; Toussirot, 2023). Calprotectin (S100A8/S100A9) correlates with synovial inflammation and may reflect subclinical disease activity (Inciarte-Mundo et al., 2022). Additionally, circulating microRNAs represent promising diagnostic and therapeutic targets, although further validation is required (Machaj et al., 2025).

Hematological indices derived from complete blood count such as NLR, PLR, LMR, and SII have emerged as accessible markers associated with disease activity (Targońska-Stępniak et al., 2020; Choe et al., 2023; Zhang et al., 2026). NLR, in particular, has been linked to treatment response in patients receiving biologic therapy (Choe et al., 2023). These indices are inexpensive, widely available, and easily implemented in routine practice; however, they remain nonspecific markers of systemic inflammation (Zhang et al., 2026).

Multibiomarker approaches, including the MBDA score, attempt to integrate various inflammatory pathways into a single quantitative tool and have shown correlation with disease activity and radiographic progression (Meznerics et al., 2023). Nevertheless, cost and clinical standardization remain challenges.

Despite the growing number of candidate biomarkers, several critical barriers limit their routine clinical implementation. The absence of standardized integration algorithms, heterogeneity of assay methodologies, variability in cut-off values, and differences in study populations substantially affect reproducibility and comparability across studies. Moreover, many proposed biomarkers lack validation in large, prospective, multicenter cohorts, and their incremental value beyond established composite indices remains uncertain. These methodological and translational gaps currently hinder the development of unified, evidence-based biomarker-driven decision models in RA management.

Taken together, current evidence supports the complementary use of conventional and emerging biomarkers to improve disease assessment in RA. Their combined application may enhance clinical decision-making, particularly in heterogeneous or treatment-resistant cases.

Conclusions

Rheumatoid arthritis requires a multidimensional approach to inflammatory assessment and prognostic evaluation. While conventional biomarkers remain essential in routine practice, they incompletely reflect disease heterogeneity. Emerging molecular and hematological markers offer promising complementary information and may enhance disease monitoring and therapeutic stratification.

However, no single biomarker adequately captures the complexity of RA. Future research should focus on validating novel markers in large, prospective cohorts and integrating them into standardized, clinically applicable models. Further studies are required to establish their reproducibility, specificity, and cost-effectiveness before widespread implementation in routine clinical practice.

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