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Original Research Article

SERUM URIC ACID AS AN INFLAMMATORY BIOMARKER IN RHEUMATOID ARTHRITIS PATIENTS

Shivam Chauhan[1], Apurva Khare[2], D P Singh[3], Akshita Singh[1], Shubham Patel[1], Sandeep Mukati[1], Alkesh Biyani[1]

[1]Postgraduate Resident, Department of General Medicine, L.N. Medical College & Research Centre, Bhopal, MP, India
[2]Professor, Department of General Medicine, L.N. Medical College & Research Centre, Bhopal, MP, India
[3]Professor & Head of Department, Department of General Medicine, L.N. Medical College & Research Centre, Bhopal, MP, India

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Corresponding Author:
Dr. Shivam Chauhan,
Email: shivam2719@gmail.com

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ABSTRACT

Background: Rheumatoid arthritis is a chronic systemic autoimmune inflammatory disorder characterized by persistent synovitis, progressive joint destruction, extra-articular manifestations, functional disability, and substantial socioeconomic burden. The present study was undertaken to evaluate whether serum uric acid levels are associated with disease activity in patients with rheumatoid arthritis and to explore its utility as an inflammatory biomarker in a tertiary care setting. **Materials and Methods:** This hospital-based prospective observational cross-sectional study was conducted in the Department of Medicine, L.N. Medical College & Research Centre and its associated J.K. Hospital, Bhopal, after approval from the Institutional Ethics Committee. A total of 116 adult patients with clinically diagnosed rheumatoid arthritis fulfilling the 2010 ACR/EULAR classification criteria were enrolled after written informed consent. Patients with conditions known to independently alter serum uric acid levels, including metabolic syndrome, psoriasis, and gouty arthritis, were excluded to reduce confounding. Detailed sociodemographic data, clinical history, and physical examination findings were recorded using a structured proforma. **Result:** The study population demonstrated substantial inflammatory burden, with a mean DAS28-ESR score of 4.6 ± 1.2, indicating predominantly moderate to high disease activity. Hyperuricemia was present in 16 of 116 patients, yielding a prevalence of 13.8%. Gender-wise prevalence was higher in males (21.4%) than in females (11.4%). Serum uric acid correlated positively with DAS28-ESR ($r = 0.423$, $p < 0.001$), ESR ($r = 0.386$, $p < 0.001$), CRP ($r = 0.412$, $p < 0.001$), tender joint count ($r = 0.341$, $p < 0.001$), swollen joint count ($r = 0.298$, $p = 0.001$), patient global assessment ($r = 0.264$, $p = 0.004$), disease duration ($r = 0.245$, $p = 0.008$), and age ($r = 0.186$, $p = 0.045$). Hyperuricemic patients had significantly higher DAS28-ESR scores and CRP levels than normouricemic patients. **Conclusion:** The findings of the present study support a significant relationship between serum uric acid and inflammatory disease activity in rheumatoid arthritis. Patients with higher serum uric acid levels tended to have more active disease as reflected by higher DAS28-ESR scores and greater elevation of established inflammatory markers such as ESR and CRP. However, serum uric acid should not be interpreted in isolation, as multiple metabolic, renal, dietary, and treatment-related factors can influence its concentration. Larger multicentric longitudinal studies are needed to determine whether serum uric acid has predictive value for flares, radiographic progression, treatment response, and long-term outcomes in rheumatoid arthritis.

INTRODUCTION

Rheumatoid arthritis (RA) is a chronic, systemic, autoimmune inflammatory disease that primarily affects synovial joints and leads to pain, stiffness, swelling, progressive cartilage damage, bone erosion, deformity, and disability if inadequately treated. It is one of the most important inflammatory arthritis's

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encountered in internal medicine and rheumatology because of its chronicity, its effect on quality of life, and its capacity to produce both articular and extra-articular complications. The disease involves complex interactions between genetic susceptibility, environmental triggers, adaptive immune activation, cytokine dysregulation, and sustained synovial inflammation. Clinically, patients often present with symmetrical small-joint involvement, prolonged morning stiffness, fatigue, and restricted function, while advanced disease may result in deformity, muscle wasting, and multisystem complications. Because RA is heterogeneous in presentation and progression, accurate evaluation of disease activity remains central to therapeutic decision-making.[1-5]

Over the last two decades, the management of RA has increasingly shifted toward a treat-to-target approach, with emphasis on early diagnosis, prompt disease-modifying therapy, and regular monitoring using composite disease activity scores. In this context, biomarkers that reflect inflammatory burden are highly valuable. Conventional markers such as erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are commonly used, but they are not always perfectly concordant with symptoms, examination findings, or composite indices. Disease Activity Score-28 using ESR (DAS28-ESR) has become a practical and validated method to quantify inflammatory activity by combining tender joint count, swollen joint count, ESR, and patient global assessment. Even so, there remains continued interest in identifying adjunctive biomarkers that are inexpensive, reproducible, and easily available in routine practice.[6-10]

Serum uric acid (SUA) is the end product of purine metabolism in humans and has traditionally been studied in relation to gout, renal disease, hypertension, metabolic syndrome, and cardiovascular risk. However, uric acid is also biologically relevant to inflammation. Soluble uric acid and monosodium urate crystals may participate in oxidative stress pathways, inflammasome activation, endothelial dysfunction, and amplification of inflammatory responses. Under some conditions uric acid behaves as an antioxidant, while in others it may exert pro-oxidant and pro-inflammatory effects. These dual properties have led to increasing interest in understanding its role in chronic inflammatory and autoimmune diseases. In RA, where cytokine-driven inflammation, tissue injury, oxidative stress, and turnover of nucleic acids are prominent, elevated uric acid may theoretically mirror disease activity or inflammatory burden.[11-15]

Previous studies examining the relationship between serum uric acid and RA have produced varied results. Some investigators have reported higher uric acid levels in patients with active disease and positive associations with ESR, CRP, and disease activity scores. Others have observed weaker or inconsistent links, possibly because of differences in sample size, disease duration, treatment exposure, renal function, metabolic comorbidity, and sex distribution. Despite these variations, the question remains clinically meaningful, especially in low- and middle-income settings where advanced biomarker panels may not be feasible for all patients. If serum uric acid shows a consistent association with disease activity, it could provide an additional low-cost parameter to supplement routine inflammatory assessment.[16-20]

The present study was designed in this background to assess serum uric acid levels in rheumatoid arthritis patients attending a tertiary care teaching hospital in central India and to evaluate their relationship with established indices of disease activity. The study further aimed to estimate the prevalence of hyperuricemia in the RA cohort, compare uric acid levels across DAS28 categories, and examine associations with key clinical and laboratory variables. By focusing on a real-world hospital population and using routinely available investigations, the study seeks to contribute practical evidence regarding the potential role of serum uric acid as an inflammatory biomarker in rheumatoid arthritis.

MATERIALS AND METHODS

This study was conducted as a single-centre, hospital-based, prospective observational study of cross-sectional type in the Department of Medicine, L.N. Medical College & Research Centre and its associated J.K. Hospital, Bhopal, Madhya Pradesh. The institution is a tertiary care referral centre serving both urban and rural populations from Bhopal and adjoining districts. The study was planned to evaluate the relationship between serum uric acid and inflammatory disease activity among patients with rheumatoid arthritis attending the medicine outpatient and inpatient services.

Ethical approval was obtained from the Institutional Ethics Committee before initiation of the study, and all procedures were conducted according to accepted ethical standards and the principles of the Declaration of Helsinki. Written informed consent was obtained from every participant prior to enrolment. The total duration of the study was 18 months, comprising a planning phase of 3 months, a recruitment and data collection phase of 12 months, and a data analysis and report writing phase of 3 months.

The study population consisted of adult patients diagnosed with rheumatoid arthritis according to the 2010 ACR/EULAR classification criteria. A total of 116 patients were enrolled. Participants were selected by purposive consecutive sampling from those attending the medicine OPD and IPD during the study period. Inclusion criteria were age greater than 18 years, willingness to provide informed consent, and diagnosis of seropositive or seronegative rheumatoid arthritis. Exclusion criteria included age below 18 years, refusal to participate, and the presence of metabolic syndrome, psoriasis, or gouty arthritis, since these conditions can independently

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influence serum uric acid levels and thereby confound analysis.

Each patient was evaluated using a structured and predesigned case record proforma. Demographic variables recorded included age, sex, residence, educational status, occupation, monthly income, type of housing, and socioeconomic status according to the Modified Prasad Classification. A detailed clinical history was obtained, including duration of disease, symptoms at presentation, morning stiffness, pattern of joint involvement, constitutional symptoms, treatment history, and associated comorbidities. Female participants were also asked relevant menstrual and obstetric history where appropriate. General physical examination and systemic examination were performed in all cases.

Particular attention was paid to musculoskeletal assessment. Bilateral examination of the proximal interphalangeal joints, metacarpophalangeal joints, wrists, elbows, shoulders, and knees was carried out for tenderness, swelling, pain on movement, restriction of movement, and deformity. Disease activity was quantified using DAS28-ESR, which incorporates tender joint count, swollen joint count, erythrocyte sedimentation rate, and patient global assessment on a visual analogue scale. Disease activity was categorized as low activity for DAS28 2.6 to 3.2, moderate activity for DAS28 greater than 3.2 to 5.1, and high activity for DAS28 greater than 5.1.

Laboratory investigations were performed for all patients using standard institutional protocols. These included complete blood count, ESR, CRP, renal function tests, liver function tests, serum electrolytes, random blood sugar, HbA1c, urine routine and microscopy, lipid profile, RA factor, and anti-CCP where available. X-ray examination of involved joints was performed when indicated. Venous blood samples were collected under aseptic precautions and processed in the institutional biochemistry laboratory. Serum uric acid was measured using the uricase colorimetric method. Hyperuricemia was defined as serum uric acid greater than 6.0 mg/dL in females and greater than 7.0 mg/dL in males, in keeping with the study protocol.

The main outcome measures of interest were mean serum uric acid levels, prevalence of hyperuricemia, disease activity distribution according to DAS28-ESR, and the correlation of serum uric acid with inflammatory and clinical parameters such as ESR, CRP, tender joint count, swollen joint count, patient global assessment, disease duration, and age. Patients were also stratified into normouricemic and hyperuricemic groups for comparative analysis of key parameters.

Data were initially entered into Microsoft Excel 2021 for compilation, coding, and cleaning. Statistical analysis was then performed using SPSS version 27. Continuous variables were summarized as mean ± standard deviation, while categorical variables were expressed as frequencies and percentages. Correlation analysis was applied to assess the relationship between serum uric acid and other continuous disease activity variables. Comparative analyses between groups were performed using appropriate statistical tests for significance. A p-value less than 0.05 was considered statistically significant.

RESULTS

The present study included 116 patients with rheumatoid arthritis and demonstrated that the study cohort largely had active inflammatory disease. The mean DAS28-ESR score was 4.6 ± 1.2, which lies in the moderate disease activity range, and nearly four-fifths of patients fell into moderate or high activity categories. Mean tender joint count and swollen joint count were 9.2 ± 5.6 and 6.8 ± 4.2, respectively, while mean patient global assessment was 54.8 ± 24.2, further indicating a clinically relevant inflammatory burden in the population studied.

Hyperuricemia was documented in 16 patients, corresponding to a prevalence of 13.8%. A sex-based difference was evident, with hyperuricemia occurring in 21.4% of male patients and 11.4% of female patients. Most hyperuricemic cases were mild in severity, while moderate and severe elevations were comparatively uncommon. This distribution suggests that even modest elevation of uric acid may be relevant in RA patients when interpreted alongside measures of inflammatory activity.

A significant stepwise increase in serum uric acid was observed across disease activity strata. Patients with low disease activity had a mean serum uric acid level of 5.1 ± 1.1 mg/dL, those with moderate activity had 5.9 ± 1.4 mg/dL, and those with high disease activity had 6.4 ± 1.8 mg/dL. The difference across DAS28 categories was statistically significant ($p = 0.004$), supporting a positive association between uric acid concentration and worsening rheumatoid arthritis activity. The correlation coefficient between serum uric acid and DAS28-ESR was 0.423, indicating a moderate positive relationship.

Serum uric acid also showed significant positive correlations with established inflammatory markers and clinical indices. Positive associations were observed with ESR ($r = 0.386$, $p < 0.001$), CRP ($r = 0.412$, $p < 0.001$), tender joint count ($r = 0.341$, $p < 0.001$), swollen joint count ($r = 0.298$, $p = 0.001$), patient global assessment ($r = 0.264$, $p = 0.004$), disease duration ($r = 0.245$, $p = 0.008$), and age ($r = 0.186$, $p = 0.045$). These findings imply that serum uric acid reflects not only laboratory inflammation but also the clinical burden perceived by both examiner and patient.

When normouricemic and hyperuricemic patients were compared, hyperuricemic individuals exhibited a more severe phenotype. They had higher mean DAS28-ESR scores and higher CRP levels than those with normal uric acid levels, with statistically significant differences. ESR and joint counts were also numerically higher in the hyperuricemic group,

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although some comparisons did not reach statistical significance. Overall, the results suggest that serum uric acid may function as a supplementary inflammatory biomarker in rheumatoid arthritis and may identify a subset of patients with greater disease activity.

Statistical Analysis: The collected data were first entered into Microsoft Excel 2021 to ensure systematic coding, organization, and cleaning. After verification for completeness and consistency, the dataset was transferred to SPSS version 27 for detailed statistical analysis. Descriptive statistics were used to summarize baseline characteristics, disease activity variables, and laboratory findings. Continuous variables were presented as mean ± standard deviation, while categorical variables were expressed as frequencies and percentages.

Analytical statistics were used to evaluate the relationship between serum uric acid and rheumatoid arthritis disease activity. Correlation coefficients were calculated to determine the strength and direction of associations between serum uric acid and continuous variables such as DAS28-ESR, ESR, CRP, tender joint count, swollen joint count, patient global assessment, disease duration, and age. Comparative analyses were also undertaken between patients with normal serum uric acid and those with hyperuricemia to identify differences in inflammatory and clinical parameters.

A p-value of less than 0.05 was considered statistically significant for all analyses. Statistically significant positive correlations were found between serum uric acid and several markers of disease activity, indicating that higher uric acid values were associated with increased inflammatory burden. The observed p-values for DAS28-ESR, ESR, CRP, tender joint count, swollen joint count, patient global assessment, disease duration, and age support the internal consistency of the study findings. These analytical methods were appropriate for a hospital-based observational study exploring association rather than causation.

Table 1: disease activity assessment (das28-ESR)

DAS28 Components	Mean ± SD	Range
Tender Joint Count (0-28)	9.2 ± 5.6	1-22
Swollen Joint Count (0-28)	6.8 ± 4.2	0-16
ESR (mm/hr)	48.6 ± 22.4	15-98
Patient Global Assessment (0-100)	54.8 ± 24.2	15-95
DAS28-ESR Score	4.6 ± 1.2	2.6-6.8
DAS28 Categories	Frequency (n)	Percentage (%)
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Low Activity (2.6-3.2)	23	19.8
Moderate Activity (3.2-5.1)	61	52.6
High Activity (>5.1)	32	27.6

Table 2: prevalence of hyperuricemia in rheumatoid arthritis patients

Parameter	Frequency (n)	Percentage (%)
Overall Hyperuricemia	16	13.8
Males (>7.2 mg/dL)	6/28	21.4
Females (>6.0 mg/dL)	10/88	11.4
Mild (6.1-7.5 mg/dL)	11	9.5
Moderate (7.6-9.0 mg/dL)	4	3.4
Severe (>9.0 mg/dL)	1	0.9

Table 3: correlation of serum uric acid with disease activity (das28-esr)

DAS28 Category	n	Serum Uric Acid (mg/dL), Mean ± SD	p-value
Low Activity (2.6-3.2)	23	5.1 ± 1.1	
Moderate Activity (3.2-5.1)	61	5.9 ± 1.4	0.004
High Activity (>5.1)	32	6.4 ± 1.8	

Correlation coefficient (r) = 0.423, $p < 0.001$

Table 4: association of serum uric acid with clinical and laboratory parameters

Parameter	Correlation Coefficient (r)	p-value	Significance
DAS28-ESR Score	0.423	<0.001	Significant
ESR	0.386	<0.001	Significant
CRP	0.412	<0.001	Significant
Tender Joint Count	0.341	<0.001	Significant
Swollen Joint Count	0.298	0.001	Significant
Patient Global Assessment	0.264	0.004	Significant
Disease Duration	0.245	0.008	Significant
Age	0.186	0.045	Significant

DISCUSSION

Rheumatoid arthritis is characterized by chronic immune-mediated synovial inflammation and systemic manifestations that require repeated assessment of disease severity over time. In clinical practice, a biomarker that is economical, reproducible, and widely available can be especially

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valuable when used in conjunction with standard clinical tools. The present study evaluated serum uric acid in this context and found a meaningful positive association with inflammatory disease activity. The findings indicate that serum uric acid rises with increasing DAS28-ESR categories and correlates with both objective inflammatory markers and clinical disease measures.

The mean DAS28-ESR score of 4.6 ± 1.2 in the present study indicates that the enrolled patients, as a group, had active disease, with the majority belonging to moderate and high disease activity categories. This pattern is commonly seen in tertiary-care hospital populations, where patients often present after significant symptom duration or are referred because of uncontrolled disease. The increase in serum uric acid from low to moderate to high activity categories suggests a biologically plausible link between uric acid and the inflammatory milieu of RA. Whether this increase reflects oxidative stress, enhanced cellular turnover, cytokine-driven metabolic alteration, reduced renal excretion, or a combination of these factors remains a subject for further exploration.

The observed prevalence of hyperuricemia of 13.8% is clinically notable. It suggests that a meaningful subset of RA patients may have elevated uric acid levels even in the absence of gouty arthritis, psoriasis, or metabolic syndrome, all of which were excluded in this study to minimize confounding. The higher prevalence among males compared with females is in line with known sex differences in uric acid physiology and renal handling. However, the study also shows that even among predominantly female RA cohorts, modestly elevated uric acid can still carry inflammatory relevance. This is important because RA is more common in women, and biomarkers that retain value in such populations are useful for real-world clinical decision-making.

One of the strongest findings in this study is the significant correlation of serum uric acid with DAS28-ESR, ESR, and CRP. DAS28-ESR is an accepted composite index integrating joint counts, an acute phase reactant, and patient-reported health status. Its positive correlation with uric acid indicates that uric acid may parallel overall disease activity rather than merely one isolated inflammatory parameter. Similarly, the relationships with ESR and CRP strengthen the argument that serum uric acid reflects systemic inflammatory burden. The positive correlation with tender and swollen joint counts further supports a link with active synovitis, while the association with patient global assessment suggests that biochemical elevation aligns, at least partly, with patient-experienced disease severity.

Comparison between normouricemic and hyperuricemic patients revealed higher DAS28-ESR and CRP values in the hyperuricemic group. This observation suggests that hyperuricemia may identify a subset with more severe or more metabolically inflammatory disease expression. Although some comparisons, such as ESR and swollen joint count, did not achieve statistical significance, the overall direction of association remained consistent. The lack of significance in every parameter may be related to the modest number of hyperuricemic patients, limiting subgroup statistical power. Nevertheless, the pattern reinforces the hypothesis that serum uric acid is not merely an incidental laboratory abnormality but may have clinical relevance in RA assessment.

From a pathophysiological perspective, several mechanisms may explain this association. Uric acid is generated during purine metabolism via xanthine oxidase, a process linked with reactive oxygen species production. In inflammatory states such as RA, increased cell turnover, oxidative stress, hypoxia within inflamed synovium, and altered vascular function may contribute to elevated uric acid levels. Moreover, uric acid itself can activate pro-inflammatory pathways under certain conditions, including inflammasome-related signaling. Thus, uric acid may be both a marker and a mediator of inflammation. However, these mechanistic implications cannot be directly proven by the present observational design and should be interpreted cautiously.

The clinical significance of these findings lies in feasibility. Serum uric acid estimation is inexpensive, routinely available even in smaller laboratories, and familiar to clinicians. In resource-limited settings, where access to advanced cytokine assays or novel biomarkers is constrained, serum uric acid may provide an additional piece of information that complements ESR, CRP, and clinical scoring systems. It should not replace standard disease activity assessment, but it may act as a supportive biomarker, particularly when trends over time are monitored. In busy outpatient practice, a biomarker that is simple and already part of many biochemical panels can enhance practicality without adding major cost.

The present study also has limitations that must be acknowledged. Being a single-centre hospital-based study, the findings may not be fully generalizable to all RA populations. The cross-sectional design assesses association at one point in time and cannot determine temporal causality or predictive value. Important factors such as diet, renal urate handling variation within the normal range, medication effects including diuretics and urate-modifying agents, obesity indices, and detailed treatment stratification were not elaborated in the available dataset. In addition, radiographic progression and longitudinal outcomes were not studied. These limitations highlight the need for larger prospective multicentric studies with serial measurements.

Despite these limitations, the study offers relevant and practical insights. The internal consistency across correlations with DAS28-ESR, ESR, CRP, and articular indices supports the reliability of the central finding. The exclusion of major confounding conditions that strongly influence uric acid also strengthens interpretability. Therefore, the study contributes useful local evidence suggesting that

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serum uric acid is associated with inflammatory activity in RA and deserves further attention as a supplementary biomarker.

CONCLUSION

The present study demonstrates that serum uric acid has a significant positive association with disease activity in patients with rheumatoid arthritis. As disease activity increased from low to moderate to high categories on DAS28-ESR, mean serum uric acid levels also increased in a graded manner. Serum uric acid further showed meaningful positive correlations with ESR, CRP, tender joint count, swollen joint count, patient global assessment, disease duration, and age. These findings collectively suggest that serum uric acid reflects the inflammatory burden of rheumatoid arthritis to a clinically relevant extent.

The prevalence of hyperuricemia in this cohort was 13.8%, with a higher frequency among male patients than female patients. Hyperuricemic rheumatoid arthritis patients exhibited higher disease activity scores and CRP levels compared with normouricemic patients, indicating that elevated uric acid may identify a subset with more severe inflammatory disease expression. Even when hyperuricemia was mild, it appeared to track with increased disease activity, which underscores the value of careful interpretation of uric acid levels beyond their conventional association with gout.

An important strength of serum uric acid as a biomarker is its accessibility. The test is inexpensive, widely available, technically simple, and already familiar in routine biochemical assessment. In tertiary hospitals, district centres, and resource-constrained settings alike, serum uric acid can therefore be incorporated without difficulty into the broader evaluation of rheumatoid arthritis patients. This makes it particularly attractive as a supplementary marker in settings where advanced laboratory biomarkers are less feasible. Used alongside clinical examination, DAS28 scoring, ESR, and CRP, serum uric acid may add practical value in identifying patients with higher inflammatory burden.

At the same time, serum uric acid should not be viewed as a standalone diagnostic or monitoring tool. Its levels are influenced by sex, renal function, diet, metabolic status, medication use, alcohol intake, and other comorbid states. For that reason, interpretation must remain clinical and contextual. Elevated uric acid in an RA patient should prompt thoughtful correlation with symptoms, joint findings, inflammatory markers, and existing therapy rather than automatic attribution to rheumatoid disease activity alone. Likewise, a normal uric acid value does not exclude active disease.

The broader implication of the present work is that commonly available laboratory parameters may still hold underappreciated value in chronic inflammatory disease monitoring. In many real-world settings, especially in low-resource environments, clinicians must often rely on combinations of history, examination, and affordable investigations rather than specialized biomarker panels. In such circumstances, serum uric acid may serve as an adjunctive inflammatory indicator that enriches the overall clinical picture. The findings of this study support its potential role in routine assessment and encourage clinicians to consider it as part of integrated disease activity evaluation.

Future research should aim to validate these observations in larger, multicentric cohorts with longitudinal follow-up. Serial measurements would help determine whether serum uric acid changes in parallel with treatment response, remission, flare, or radiographic progression. Studies that adjust for renal function, body mass index, dietary factors, and drug exposure would further clarify whether uric acid is independently associated with RA activity. Interventional studies may also explore whether modulation of uric acid has any effect on inflammatory outcomes, although such questions require careful mechanistic design. Until such evidence emerges, serum uric acid may reasonably be regarded as a useful supplementary biomarker, not a replacement for established tools, in the inflammatory assessment of rheumatoid arthritis patients.

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