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A Study of Microalbuminuria in Patients with Rheumatoid Arthritis

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Abstract

Background: Rheumatoid arthritis (RA) is a chronic inflammatory disorder with potential systemic complications including renal involvement. Microalbuminuria represents early endothelial dysfunction and may indicate disease severity and cardiovascular risk in RA patients. The aim of the study is to determine the prevalence of microalbuminuria in patients with rheumatoid arthritis and to examine its correlation with disease activity markers including rheumatoid factor (RF), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP). ***Methods:*** The study included 40 patients with established rheumatoid arthritis diagnosed according to the 2020 American College of Rheumatology-European Alliance of Associations for Rheumatology (ACR-EULAR) classification criteria. Patients underwent comprehensive clinical assessment including detailed history, physical examination with tender and swollen joint counts, and laboratory investigations. Microalbuminuria was assessed using spot morning urine samples with albumin-to-creatinine ratio (ACR) method, defining microalbuminuria as ACR more than or equal to 30 mg/g. Disease activity markers including ESR, CRP, and RF were measured using standard laboratory methods. Statistical analysis was performed using appropriate parametric and non-parametric tests, with p value less than 0.05 considered statistically significant. ***Results:*** Microalbuminuria was present in 8 out of 40 patients (20%). Patients with microalbuminuria demonstrated significantly elevated inflammatory markers compared to those without microalbuminuria. Mean ESR was 48.8±13.7 mm/hr in the microalbuminuria-positive group versus 31.6±12.2 mm/hr in the microalbuminuria- negative group (p=0.001). Mean CRP levels were 29.2±8.4 mg/L in microalbuminuria-positive patients compared to 20.06±12.1 mg/L in microalbuminuria-negative patients (p=0.05). Rheumatoid factor showed a strong association with microalbuminuria, with 100% (8/8) of microalbuminuria-positive patients being RF positive compared to 62.5% (20/32) of microalbuminuria-negative patients (p=0.038). ***Conclusion:*** Microalbuminuria was present in 20% of rheumatoid arthritis patients and showed significant correlations with disease activity markers including ESR, CRP, and RF. These findings establish microalbuminuria as a valuable biomarker of disease severity and systemic inflammation in RA. Routine microalbuminuria screening should be considered in RA patients, especially those with high disease activity or elevated inflammatory markers.

Keywords: Rheumatoid arthritis, Microalbuminuria, Kidney diseases, C-reactive protein.

INTRODUCTION

Rheumatoid Arthritis (RA) is a long-term systemic auto-immune inflammatory condition that is found to affect about 0.005 - 0.01% of the world's adult population with a female to male ratio of 3:1.[1] RA pathogenesis includes complex pathways of immune-mediated inflammatory processes that include the production of pro-inflammatory cytokines (such as TNF-alpha, IL-1 & IL-6) contributing to the joint destruction and also to the systemic inflammation of other organ systems (including the cardiac, pulmonary and renal systems).

Among modifiable risk factors, cigarette smoking has the strongest association with RA.[2] Diet and nutrition have been shown to play a significant role as environmental triggers for RA. The typical 'western' diet that is rich, high in caloric content, and low in fiber increases the risk of RA. Consumption of long-chain omega- 3 polyunsaturated fatty acids is associated with a reduced risk of RA.[3] Obesity is another well-established risk factor for RA. There is a 30% increase in the risk of RA for patients with a body mass index (BMI) of greater than

30 kg/m2 and a 15% increased risk for those with a BMI of 25 to 29.9 kg/m2.[4]

Renal involvement in RA has become a significant clinical complication that should be considered because of the high mortality and morbidity associated with it. The prevalence of kidney involvement in RA patients varies significantly in the literature with reports indicating that kidney involvement occurs in 20-50% of RA patients.[5] In the past, renal manifestations of RA were thought to be due primarily to the nephrotoxic effects of disease modifying antirheumatic drugs (DMARDs) such as gold salts and D-penicillamine and chronic non-steroidal anti-inflammatory drug (NSAID) therapy. However, current understanding recognizes that renal dysfunction in RA may be caused by several pathophysiologic mechanisms including direct disease related inflammation, secondary amyloidosis, glomerulonephritis, and drug induced nephrotoxicity.[6] The range of renal pathology seen in RA patients encompasses many different histologic patterns; however, the most commonly identified lesion in patients with established nephropathy is mesangial proliferative glomerulonephritis, occurring in 35-78% of RA patients with established nephropathy.

Microalbuminuria, defined as urinary albumin excretion between 30-300 mg/24 hours or an albumin to creatinine ratio of 3-30 mg/mmol, has become a useful biomarker for identifying incipient renal dysfunction in many different clinical contexts. Initially used to predict diabetic nephropathy, microalbuminuria has since been validated as an early indicator of renal injury in hypertension, cardiovascular disease and in various systemic inflammatory conditions.[7] The pathophysiology underlying microalbuminuria involves increased glomerular permeability to albumin, potentially resulting from endothelial dysfunction, inflammatory damage to the glomerular filtration barrier, or altered renal hemodynamics.

Multiple studies have investigated the relationship between microalbuminuria and disease activity parameters in RA. Each of these studies has demonstrated significant positive correlations between urinary albumin excretion and acute phase reactants (e.g., CRP and ESR), providing evidence that microalbuminuria may represent an indicator of systemic inflammation.[8] Additional correlations were noted between microalbuminuria and RA disease duration, number of involved joints, and other composite disease activity scores; these correlations are supportive of the idea that microalbuminuria in RA could arise due to systemic inflammatory processes as opposed to drug-related nephrotoxicity or simply coincidental renal disease. Studies showing elevated inflammatory mediator levels in RA patients with microalbuminuria compared to those without it, also support the inflammatory hypothesis.

Microalbuminuria in RA patients has implications for cardiovascular morbidity and mortality beyond serving as a marker of subclinical renal dysfunction. Longitudinal studies have shown that RA patients who develop microalbuminuria, experience increased cardiovascular morbidity and mortality. Therefore, it is reasonable to conclude that there is a complex interplay among inflammation, endothelial dysfunction and rapid atherosclerosis in this patient group. Recently, a large prospective study conducted by Chen et al., found that RA patients who developed elevated albumin-to-creatinine ratios, experienced significantly increased all-cause mortality and cardiovascular related mortality, compared to RA patients who did not exhibit this abnormality.[9] These findings highlight the importance of incorporating microalbuminuria screening into the overall RA management plan and in stratifying risk.

Modern analytic techniques such as immunoturbidimetry and immunonephelometry have improved the reliability and practicality of measuring microalbuminuria in RA patients, typically using spot urine albumin-to-creatinine ratios, although results remain influenced by collection, storage, and interfering factors.[10] Ongoing research continues to explore the mechanisms, long-term implications, and potential role of microalbuminuria as a therapeutic target. Routine screening may enable earlier detection of subclinical renal dysfunction and improved cardiovascular risk stratification, supporting better patient management and outcomes.

MATERIALS AND METHODS

The study was conducted in OPD and IPD in Department of General Medicine, Geriatrics and Orthopedics. The duration of the study was for 18 months (March 2024 to December 2025). 40 participants were selected based on the inclusion and exclusion criteria. Ethical clearance was obtained prior to the commencement of the study.

Inclusion Criteria

Patients aged 18 years and above, who met the ACR-EULAR criteria (2020) for rheumatoid arthritis, and willing to provide informed consent.

Exclusion Criteria

Patients with essential hypertension, diabetes mellitus, acute kidney injury and chronic kidney disease, patients on angiotensin converting enzyme inhibitors and angiotensin receptor blockers.

All eligible patients were approached for participation after explaining the study objectives and obtaining written informed consent in their preferred language. A specially designed structured questionnaire was administered to collect comprehensive patient information. Each participant underwent detailed history taking and comprehensive clinical examination following the standardized proforma. Demographic details were recorded. Disease-specific parameters were systematically recorded, including duration of morning

stiffness, list of affected arthritic joints, presence of tenderness, swelling, deformity, and range of movements. Constitutional symptoms such as fever, anorexia, and tiredness were documented, along with involvement of other joints in upper limbs, lower limbs, or both.

The diagnosis of rheumatoid arthritis was confirmed using the 2020 ACR- EULAR classification criteria. Disease activity was assessed using standardized inflammatory markers and clinical parameters. Past medical history, personal history including tobacco chewing, smoking, alcoholism, dietary preferences, and family history of rheumatoid arthritis, hypertension, or diabetes were systematically recorded.

All participants underwent a comprehensive laboratory evaluation, including complete blood count, erythrocyte sedimentation rate (ESR), blood urea, serum creatinine, blood glucose estimation, rheumatoid factor, anti-CCP antibody, and C-reactive protein (CRP). Urinary albumin was measured using 24-hour urine collection in inpatients and urine albumin-to-creatinine ratio (ACR) in both inpatients and outpatients.

Microalbuminuria was assessed using the immunoturbidimetric method and defined according to American Diabetes Association criteria as a urine albumin excretion of 30–299 mg/24 hours or an ACR of 30–299 µg albumin/mg creatinine.
Blood and urine samples were collected on the day of admission for inpatients or during outpatient visits for ambulatory patients. Random urine samples were obtained in sterile containers for microalbumin estimation. All samples were processed according to standard laboratory protocols and analyzed using appropriate validated analytical methods, including immunoturbidimetry for microalbumin assessment. Data was recorded in Microsoft Excel and analyzed using SPSS version 20. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Age Distribution (n=40)

The majority of patients (50.0%) were in the (41-60) years age group, with 40.0% were in the (20-40) years age group and 10.0% above the age of 60 years. The average age of research participants was 45.60 ± 11.18 years.

Table 1: Age Distribution (n=40)

Age Category	Frequency (n)	Percentage (%)
20-40 years	16	40.0
41-60 years	20	50.0
>60 years	4	10.0
Mean ± SD	**45.60 ± 11.18 years**	

Gender Distribution

Out of the 40 study participants, 28 (70 %) were females while males comprised 12 (30%), demonstrating a female predominance in this rheumatoid arthritis cohort.

Rheumatoid Factor (RA Factor)

Out of 40 patients, 28 (70.0%) tested positive for RA Factor, while 12 (30.0%) tested negative, indicating that the vast majority of RA patients were seropositive for this immunological marker.

ESR (Erythrocyte Sedimentation Rate)

The ESR values were reasonably distributed throughout several ranges, with 35.0% having an ESR of less than 20 mm at one hour, 32.5% in the 20- 40 range, 17.5% in the 41-60 range, and 15.0% above 60. The cohort had a mean ESR of 36.90 ± 30.52 mm at 1 hour, indicating moderate disease activity.

Prevalence of microalbuminuria (n=40)

Out of 40 patients, 32 (80.0%) had ACR values less than 30 mg/g (negative for microalbuminuria), while 8 (20.0%) had ACR values greater than 30 mg/g (positive for microalbuminuria). The average ACR was 23.23 ± 32.19 mg/g, demonstrating microalbuminuria in one-fifth of the rheumatoid arthritis patients surveyed.

Table 2: Based on Albumin-Creatinine Ratio

ACR Category	Frequency (n)	Percentage (%)
<30 mg/g (Negative)	32	80.0
≥30 mg/g (Positive)	8	20.0
Mean ± SD	**23.23 ± 32.19 mg/g**	

ACR: Urine albumin to creatinine ratio

Comparison of inflammatory markers between microalbuminuria groups

Patients with positive microalbuminuria (ACR ≥30 mg/g) exhibited significantly higher ESR values (48.8 ± 13.7 mm/hr) compared to those without microalbuminuria (31.6 ± 12.2 mm/hr) (p-value = 0.001). The microalbuminuria group had higher CRP levels (29.2 ± 8.4 mg/L) than the non- microalbuminuria group (20.06 ± 12.1 mg/L) (p-value = 0.05). Both relationships were statistically significant, demonstrating that a larger inflammatory burden is linked to microalbuminuria.

Table 3: Comparison of inflammatory markers between microalbuminuria groups

Parameter (Mean ± SD)	ACR <30 mg/g (n=32)	ACR ≥30 mg/g (n=8)	p-value
ESR (mm at 1hr)	31.6 ± 12.2	48.8 ± 13.7	**0.001***
CRP (mg/L)	20.06 ± 12.1	29.2 ± 8.4	**0.05***

ACR: Urine albumin to creatinine ratio; ESR: Erythrocyte sedimentation ratio; CRP: C-reactive Protein

Association between RA factor and microalbuminuria

All 8 patients (100%) with microalbuminuria tested positive for RA Factor, while those without microalbuminuria tested 62.5% positive and 37.5% negative. This connection was statistically significant (p=0.038), indicating that RA Factor positive is closely linked to the prevalence of microalbuminuria.

Table 4: Association between RA factor and microalbuminuria

RA Factor	ACR <30 mg/g	ACR ≥30 mg/g	p-value
Negative	12 (37.5%)	0	**0.038***
Positive	20 (62.5%)	8 (100%)	
Total	**32 (100%)**	**8 (100%)**	

ACR: Urine albumin to creatinine ratio

Correlation between microalbuminuria parameters and inflammatory markers

ACR levels showed a positive connection with CRP (r=0.482, p=0.002) and ESR (r=0.527, p<0.001). Both relationships were statistically significant, indicating that as inflammatory indicators increase, so does the degree of microalbuminuria in rheumatoid arthritis patients.

Table 5: Correlation between microalbuminuria parameters and inflammatory markers

ACR (mg/g)	Pearson's correlation (r)	p-value
CRP (mg/L)	0.482	**0.002***
ESR (mm/hr)	0.527	**<0.001***

ESR: Erythrocyte sedimentation ratio; CRP: C-reactive Protein

DISCUSSION

The study aimed to determine the prevalence of microalbuminuria in patients with rheumatoid arthritis, as well as to investigate the relationship between microalbuminuria and disease activity markers such as rheumatoid factor (RF), erythrocyte sedimentation rate (ESR) and c-reactive protein (CRP). The assessment of this relationship can be critical for complete care of the patient, identification of potential subclinical renal involvement, and to appropriately risk stratify patients with RA. The objective of this section is to discuss the results within the context of the current literature and to explore clinical implications for the management of RA.

In the above study, microalbuminuria was detected in 20% (8/40) of patients with rheumatoid arthritis (RA) using the albumin–creatinine ratio (ACR). This prevalence aligns closely with previously published studies, where rates ranged from 20% to 31.7%, including reports by Saiduzzaman et al.[11] (30%), Gupta et al.[12] (30%), Verma et al.[13] (26%). These consistent findings across diverse populations and time periods suggest that microalbuminuria represents a common and intrinsic manifestation of RA rather than a complication limited to specific treatment regimens or healthcare settings.

Notably, studies that included control groups demonstrated a three- to four-fold higher prevalence of microalbuminuria in RA patients compared to healthy controls, supporting the concept that chronic systemic inflammation in RA contributes to renal endothelial dysfunction and increased glomerular permeability.

A significant association was observed between microalbuminuria and inflammatory markers in our study. Patients with microalbuminuria had significantly higher ESR levels (48.8 ± 13.7 mm/hr) compared to those without microalbuminuria (31.6 ± 12.2 mm/hr; $p = 0.001$). This finding is consistent with previous studies, including Saiduzzaman et al.[11], Gupta et al.[12], and Ganesan et al.[14], which also reported substantially higher ESR values in microalbuminuria-positive patients.

Similarly, CRP levels were significantly elevated in the microalbuminuria-positive group (29.2 ± 8.4 mg/L) compared to the microalbuminuria-negative group (20.06 ± 12.1 mg/L; $p = 0.05$). This association parallels findings reported by Saiduzzaman et al. [11], Gupta et al. [12] Ganesan et al. [14], and earlier correlation-based studies by Verma et al.[13] all of which demonstrated a strong relationship between elevated CRP and microalbuminuria. These findings reinforce the hypothesis that systemic inflammation, mediated through cytokines such as IL-6, TNF-α, and IL-1, plays a central role in renal endothelial injury and increased urinary albumin excretion in RA[15].

A strong association was observed between rheumatoid factor (RF) positivity and microalbuminuria. In our study, all patients with microalbuminuria (100%) were RF positive, compared to 62.5% of patients without microalbuminuria ($p = 0.038$). Similar associations have been reported by Gupta et al. [12] and Ganesan et al. [14], who demonstrated significantly higher RF positivity or RF titers in microalbuminuria-positive patients. These findings suggest that seropositive RA may be associated with more severe systemic involvement, including subclinical renal damage.

Correlation analysis in the present study demonstrated moderate to strong positive correlations between ACR and ESR ($r = 0.527$, $p < 0.001$) and CRP ($r = 0.482$, $p = 0.002$), indicating that renal albumin excretion increases proportionately with systemic inflammatory burden. Comparable findings have been reported by Verma et al.[13] and Pedersen et al.[16], who documented significant correlations between inflammatory markers and microalbuminuria.

Furthermore, multiple studies have demonstrated higher disease activity scores among microalbuminuria-positive patients. Saiduzzaman et al. [11] and Ganesan et al. [14] reported significantly higher DAS/DAS28 scores, swollen joint counts, and tender joint counts in patients with microalbuminuria compared to those without. Verma et al.[13] also reported a positive correlation between joint involvement and microalbuminuria. Collectively, these findings indicate that microalbuminuria reflects overall disease severity rather than isolated renal pathology.

The present findings are consistent with earlier evidence suggesting that microalbuminuria is more closely related to cumulative inflammatory burden than to acute disease activity alone. Studies by Verma et al. [13], Ganesan et

al. [14] and Pedersen et al. [15] demonstrated longer disease duration in microalbuminuria-positive patients. Saiduzzaman et al. [11] and Ganesan et al. [14] further reported higher frequencies of prolonged morning stiffness and constitutional symptoms among patients with microalbuminuria. These observations support the hypothesis that chronic, persistent inflammation contributes to progressive microvascular damage, including renal endothelial dysfunction.

The consistent association of microalbuminuria with higher inflammatory markers, RF positivity, greater disease activity, and longer disease duration underscores its value as a clinically meaningful biomarker in RA [17]. Microalbuminuria may serve as an indicator of subclinical renal involvement, a marker of disease severity, and a predictor of increased cardiovascular risk.

This study used standard RA diagnostic criteria and validated laboratory methods, ensuring accurate assessment. The inclusion of multiple disease activity markers (RF, ESR, CRP) allowed comprehensive evaluation of the relationship between systemic inflammation and renal involvement. Consistency with previous studies supports the reliability and reproducibility of our findings.

However, the small number of microalbuminuria-positive patients (n = 8) limited precision, though statistically significant associations were still observed. The cross-sectional design prevents causal inference and temporal assessment. The absence of a healthy control group, lack of detailed medication history, and unmeasured confounders such as hypertension, diabetes, obesity, and smoking may have influenced results. Additionally, renal biopsies were not performed, limiting pathological correlation. Larger, longitudinal studies incorporating controls and comorbidity assessment are needed to better define these relationships.

LIMITATIONS

The study is limited by a small sample size (n=40) and is a cross-sectional design. The lack of a healthy control group makes it difficult to compare the results. The important confounding variables like smoking and the use of long-term medication were not described in detail. Also, renal biopsy was not done to confirm histopathology. The measurement of microalbuminuria was done by a spot urine ACR, which can be affected by transient changes. Future studies should include larger samples, longitudinal follow-up, control groups, and histopathological correlation.

CONCLUSION

20% of rheumatoid arthritis patients had microalbuminuria, which was significantly correlated with higher ESR, CRP, and rheumatoid factor positive. Since the urine albumin–creatinine ratio and inflammatory indicators are positively correlated, microalbuminuria may indicate systemic inflammation rather than limited renal involvement. Microalbuminuria is a valuable biomarker of subclinical renal involvement and overall disease severity in RA, according to these data. It could also be a sign of underlying endothelial dysfunction, which raises cardiovascular risk. To facilitate early detection and prompt intervention, RA patients should be routinely screened for microalbuminuria with spot urine ACR, especially if they have high disease activity, higher inflammatory markers, seropositive illness, or a prolonged disease duration.

Author contributions

BG was responsible for data collection, methodology, formal analysis, investigation, data curation, original draft preparation, and visualization. RMH supervised the study, administered the project, and reviewed and edited the manuscript. PMP contributed to methodology, validation, resources, and writing, review, and editing. All authors have read and agreed to the final version of the manuscript.

Data availability statement

The datasets generated and analyzed during the current study are not publicly available due to patient privacy and confidentiality agreements, but are available from the corresponding author upon reasonable request.

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Conflicts of interest

There are no conflicts of interest.

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