

Original Article

Body Mass Index Is Associated with Disease Activity, Functional Disability, and Remission in Patients With Rheumatoid Arthritis

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ABSTRACT

Background: Obesity and excess adiposity may influence rheumatoid arthritis disease activity, inflammation, functional limitation, and treatment-target achievement. Body mass index is a simple clinical measure that may help identify patients at higher risk of persistent disease burden. **Objective:** To assess the association between body mass index and rheumatoid arthritis disease severity, functional status, and remission in adult patients with established rheumatoid arthritis. **Methods:** This prospective cohort study included 140 adult patients with rheumatoid arthritis. Body mass index was measured using standard anthropometry and categorized as normal weight, overweight, or obese. Disease activity was assessed using DAS28-ESR, and functional status was assessed using HAQ-DI. DAS28-ESR remission was defined as a score below 2.6. Multivariable regression was used to assess the association of body mass index with disease activity and remission. **Results:** The mean age was 52.8 ± 11.4 years, and 76 patients were female. Mean body mass index was 28.7 ± 5.2 kg/m². DAS28-ESR increased from 3.24 ± 0.72 in normal-weight patients to 4.02 ± 0.83 in overweight patients and 4.61 ± 0.91 in obese patients. HAQ-DI increased from 0.86 ± 0.31 to 1.39 ± 0.45 across the same categories. Remission declined from 18.7% in normal-weight patients to 4.5% in obese patients. Body mass index remained associated with higher DAS28-ESR ($\beta = 0.078$, 95% CI 0.04–0.11, $p < 0.001$) and lower odds of remission (OR = 0.86, 95% CI 0.78–0.94, $p = 0.001$). **Conclusion:** Higher body mass index was associated with greater disease activity, poorer function, and lower remission rates in rheumatoid arthritis. **Keywords:** rheumatoid arthritis, body mass index, obesity, disease activity, DAS28-ESR, remission, functional disability.

EDITORIAL INFORMATION

Author Contributions: Concept: FB; Study Design: FB and JI; Data Collection: FB and KJ; Data Analysis: JI; Manuscript Drafting: FB; Critical Review and Revision: JI and KJ; Final Approval: FB, JI, and KJ.

Ethical Approval: PUMHSW, Pakistan.

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INTRODUCTION

Rheumatoid arthritis is a chronic systemic inflammatory disease characterized by persistent synovitis, progressive joint involvement, pain, functional limitation, and variable treatment response. Although pharmacological control of inflammation remains the central target of management, increasing attention has been directed toward modifiable clinical and metabolic factors that may influence disease activity, patient-reported disability, and achievement of remission. Among these factors, body mass index and adiposity are clinically important because excess adipose tissue is not only a marker of body weight but also an active endocrine and immunometabolic compartment capable of promoting low-grade systemic inflammation through adipokines, cytokines, and metabolic mediators that may interact with inflammatory pathways in rheumatoid arthritis (1).

Obesity has been associated with higher clinical disease activity, greater tender and swollen joint counts, poorer functional outcomes, and reduced likelihood of achieving remission in patients with rheumatoid

arthritis. Previous observational and registry-based studies have shown that overweight and obese patients may have less favorable responses to conventional synthetic and biologic disease-modifying antirheumatic drugs, particularly when remission or low disease activity is used as the treatment target (2, 3). These findings suggest that body composition and adiposity may contribute to both biological inflammation and patient-perceived disease burden, including pain, fatigue, physical limitation, and reduced quality of life (4, 5).

The relationship between obesity and rheumatoid arthritis is biologically plausible. Adipose tissue secretes inflammatory mediators such as leptin, resistin, visfatin, tumor necrosis factor- α , and interleukin-related pathways that may amplify systemic inflammation and contribute to synovial inflammatory activity (6, 7). In addition, increased body weight may impose biomechanical stress on already affected joints, particularly the feet, knees, and weight-bearing structures, thereby increasing pain and functional disability independent of inflammatory synovitis alone (8). Rheumatoid arthritis may also be associated with altered body composition, including loss of lean mass and relative increase in fat mass, making body mass index an imperfect but clinically accessible indicator of adiposity-related disease burden (9, 10).

Evidence also indicates that the association between body mass index and rheumatoid arthritis activity may vary by sex. Women with rheumatoid arthritis may demonstrate stronger associations between higher body mass index, inflammatory markers, pain perception, disability, and composite disease activity scores than men (11, 12). This may reflect differences in adipose tissue distribution, hormonal influences, inflammatory regulation, and pain sensitivity. Because rheumatoid arthritis is more common in women and because obesity is increasingly prevalent in many clinical populations, sex-specific assessment of the association between body mass index and disease activity has practical importance for individualized management.

Despite increasing evidence linking obesity with poorer rheumatoid arthritis outcomes, important clinical gaps remain. Many available studies have focused on selected treatment groups, specific biologic therapies, or registry populations, while fewer studies have evaluated the graded association of body mass index with disease activity, inflammatory markers, functional disability, and remission status in routine clinical cohorts. In addition, evidence from local and regional clinical settings remains limited, although population-specific data are important because body composition, lifestyle patterns, access to care, medication exposure, and comorbidity profiles may differ across settings. A clearer understanding of the relationship between body mass index and rheumatoid arthritis severity may support more integrated care models in which weight assessment and weight management are incorporated alongside pharmacological treatment.

Therefore, this prospective cohort study was conducted to assess the association between body mass index and rheumatoid arthritis disease severity among adult patients with established rheumatoid arthritis. The study specifically evaluated whether increasing body mass index was associated with higher DAS28-ESR scores, greater inflammatory burden, poorer functional status, and lower likelihood of remission. The study further explored whether the relationship between body mass index and disease activity differed by sex.

MATERIAL AND METHODS

This prospective cohort study was conducted to evaluate the association between body mass index and rheumatoid arthritis disease severity among adult patients attending outpatient rheumatology clinics of a tertiary care center and its associated hospital network. Eligible participants were recruited consecutively during the study period to reduce selection bias and to reflect routine clinical practice. The study population consisted of adult patients aged 18 years or older with rheumatoid arthritis confirmed by a specialist rheumatologist according to the 2010 rheumatoid arthritis classification criteria. Patients were eligible if they had a disease duration of at least six months and had been receiving a stable regimen of conventional synthetic or biologic disease-modifying antirheumatic drugs for at least three months before enrollment. Patients were excluded if they were pregnant or lactating, had active malignancy or severe infection, had severe renal or hepatic impairment, had another systemic inflammatory rheumatic disease

in addition to rheumatoid arthritis, had undergone previous bariatric surgery, had recent extreme weight fluctuation unrelated to rheumatoid arthritis, or were unable to provide informed written consent.

Potential participants were identified from rheumatology clinic lists and screened systematically against the eligibility criteria. After confirming eligibility, informed written consent was obtained before study assessment. A total of 140 patients with rheumatoid arthritis were enrolled and assessed for body mass index, disease activity, inflammatory markers, functional status, and remission status. Consecutive recruitment was used to minimize sampling bias, and clinical assessments were performed using standardized procedures by trained personnel.

Anthropometric measurements were obtained at enrollment. Body weight was measured to the nearest 0.1 kg with participants wearing light clothing and no shoes, and height was measured to the nearest 0.5 cm using a standard stadiometer. Body mass index was calculated as weight in kilograms divided by height in meters squared and analyzed both as a continuous variable and as a categorical variable according to World Health Organization classification. Participants were classified as underweight if body mass index was below 18.5 kg/m², normal weight if 18.5–24.9 kg/m², overweight if 25.0–29.9 kg/m², and obese if 30.0 kg/m² or higher. Where applicable, obesity was further categorized into class I, class II, and class III obesity. Waist circumference was recorded when available using a standardized midpoint technique between the lower costal margin and the iliac crest.

Disease activity was assessed during the same visit using validated composite measures. The primary disease activity measure was the Disease Activity Score in 28 joints using erythrocyte sedimentation rate. Tender and swollen joint counts of 28 joints were performed by trained clinical or research personnel. Erythrocyte sedimentation rate and C-reactive protein were measured from venous blood samples obtained at baseline. Secondary disease activity measures included the Simplified Disease Activity Index and Clinical Disease Activity Index when all required components were available. Functional status was assessed using the Health Assessment Questionnaire Disability Index. Patient global assessment and pain intensity were recorded using 0–10 cm visual analogue scales.

Remission and low disease activity were defined using established DAS28-ESR thresholds. DAS28-ESR remission was defined as a score below 2.6, and low disease activity was defined as a score of 3.2 or lower. The main exposure variable was body mass index. The main outcome variable was DAS28-ESR score, while secondary outcomes included inflammatory markers, Health Assessment Questionnaire Disability Index score, remission status, and low disease activity status. Covariates recorded for confounding assessment included age, sex, disease duration, smoking status, serostatus, current disease-modifying antirheumatic drug therapy, biologic therapy exposure, glucocorticoid use, nonsteroidal anti-inflammatory drug use, and major comorbid conditions. Medication information was verified against clinical records and patient medication lists when available.

Several procedures were used to reduce bias and improve measurement reliability. Consecutive recruitment was used to reduce selection bias. Direct measurement of weight and height was used instead of self-reported anthropometry to reduce exposure misclassification. Joint assessment was performed using a standardized 28-joint count approach. Laboratory personnel were blinded to clinical disease activity data where feasible. Confounders were selected a priori based on clinical relevance and prior evidence, with particular attention to age, sex, disease duration, serostatus, smoking, glucocorticoid therapy, and biologic disease-modifying antirheumatic drug use. The interaction between body mass index and sex was assessed because previous evidence suggests that the association between adiposity and rheumatoid arthritis activity may differ between women and men.

Data were entered into a secure database with range checks to reduce entry errors. A random subset of records was reviewed for quality control. Continuous variables were summarized as mean and standard deviation when approximately normally distributed and as median with interquartile range when non-normally distributed. Categorical variables were summarized as frequencies and percentages. The association between continuous body mass index and DAS28-ESR was assessed using correlation analysis and linear regression. Pearson correlation was used for normally distributed variables, while Spearman

correlation was used when distributional assumptions were not met. One-way analysis of variance or the Kruskal–Wallis test was used to compare disease activity and inflammatory markers across body mass index categories, depending on data distribution. Multivariable linear regression was used to assess whether body mass index independently predicted DAS28-ESR after adjustment for prespecified confounders. Binary logistic regression was used to evaluate the association between body mass index and remission status. Model assumptions were assessed using residual plots, homogeneity of variance checks, and multicollinearity diagnostics. Sensitivity analyses were performed using alternative disease activity measures when available and by assessing the influence of extreme body mass index categories.

Missing data were examined before analysis. Complete-case analysis was used when missing values were minimal. If missingness exceeded an acceptable threshold for key covariates, multiple imputation by chained equations was planned. All statistical tests were two-sided, and a p-value below 0.05 was considered statistically significant. Ethical approval was obtained from the Institutional Review Board of Sargodha Medical College, and the study was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants before enrollment. Patient data were anonymized before analysis and stored securely to maintain confidentiality and reproducibility.

RESULTS

A total of 140 patients with rheumatoid arthritis were included in the analysis. The mean age of the participants was 52.8 ± 11.4 years, and 76 participants were female, representing 54.3% of the study population. The median disease duration was 6.2 years, with an interquartile range of 3.8–10.5 years. The mean body mass index was $28.7 \pm 5.2 \text{ kg/m}^2$.

Table 1. Baseline Characteristics of the Study Population

Variable	Overall
Sample size, n	140
Age, years, Mean $\pm$ SD	52.8 ± 11.4
Female sex, n (%)	76 (54.3)
Disease duration, years, Median (IQR)	6.2 (3.8–10.5)
Body mass index, kg/m^2 , Mean $\pm$ SD	28.7 ± 5.2

Abbreviations: IQR, interquartile range; SD, standard deviation.

The study population consisted of middle-aged adults with established rheumatoid arthritis and a median disease duration of more than six years. The mean body mass index of $28.7 \pm 5.2 \text{ kg/m}^2$ indicates that the overall cohort was, on average, within the overweight range. Female participants represented slightly more than half of the sample. Disease activity and functional impairment increased across higher body mass index categories. The mean DAS28-ESR score increased from 3.24 ± 0.72 in normal-weight patients to 4.02 ± 0.83 in overweight patients and 4.61 ± 0.91 in obese patients. Functional disability showed a similar gradient, with HAQ-DI increasing from 0.86 ± 0.31 in normal-weight patients to 1.12 ± 0.38 in overweight patients and 1.39 ± 0.45 in obese patients.

Table 2. Disease Activity and Functional Status Across Body Mass Index Categories

Outcome	Normal Weight	Overweight	Obese
DAS28-ESR, Mean $\pm$ SD	3.24 ± 0.72	4.02 ± 0.83	4.61 ± 0.91
HAQ-DI, Mean $\pm$ SD	0.86 ± 0.31	1.12 ± 0.38	1.39 ± 0.45

Abbreviations: DAS28-ESR, Disease Activity Score in 28 joints using erythrocyte sedimentation rate; HAQ-DI, Health Assessment Questionnaire Disability Index; SD, standard deviation.

The increase in DAS28-ESR across body mass index categories indicates a graded pattern of higher rheumatoid arthritis disease activity among patients with higher body mass index. Mean DAS28-ESR was 0.78 points higher in overweight patients and 1.37 points higher in obese patients compared with normal-weight patients. Functional impairment followed the same direction, with mean HAQ-DI increasing by 0.26 points in overweight patients and 0.53 points in obese patients compared with normal-weight patients.

Remission rates decreased across increasing body mass index categories. DAS28-ESR remission was reported in 18.7% of normal-weight patients, 10.3% of overweight patients, and 4.5% of obese patients.

Table 3. DAS28-ESR Remission Across Body Mass Index Categories

Outcome	Normal Weight	Overweight	Obese
DAS28-ESR remission, %	18.7	10.3	4.5

Abbreviation: DAS28-ESR, Disease Activity Score in 28 joints using erythrocyte sedimentation rate. Remission was defined as DAS28-ESR <2.6.

The proportion of patients achieving DAS28-ESR remission decreased progressively with increasing body mass index. Remission was 8.4 percentage points lower in overweight patients and 14.2 percentage points lower in obese patients compared with normal-weight patients. The remission rate in obese patients was less than one-fourth of the remission rate observed in normal-weight patients. In multivariable linear regression for prespecified clinical confounders, body mass index remained independently associated with higher DAS28-ESR. Each one-unit increase in body mass index was associated with a 0.078-point increase in DAS28-ESR. In binary logistic regression, each one-unit increase in body mass index was associated with lower odds of DAS28-ESR remission, with an odds ratio of 0.86. The interaction between body mass index and sex indicated a stronger association between body mass index and DAS28-ESR among women.

Table 4. Multivariable Association of Body Mass Index With Disease Activity and Remission

Model	Outcome	Predictor	Estimate	95% CI	p-value
Linear regression	DAS28-ESR	Body mass index, kg/m ²	$\beta = 0.078$	0.04 to 0.11	<0.001
Logistic regression	DAS28-ESR remission	Body mass index, kg/m ²	OR = 0.86	0.78 to 0.94	0.001
Interaction model	DAS28-ESR	Body mass index $\times$ sex	$\beta = 0.12$	—	0.03

The regression findings support a consistent association between higher body mass index and worse rheumatoid arthritis disease control. The linear model showed that disease activity increased with each additional kg/m² of body mass index, while the logistic model showed that the likelihood of remission decreased by 14% for each one-unit increase in body mass index. The sex interaction suggests that the association between body mass index and disease activity may be stronger among female patients, although the confidence interval for this interaction was not reported in the manuscript.

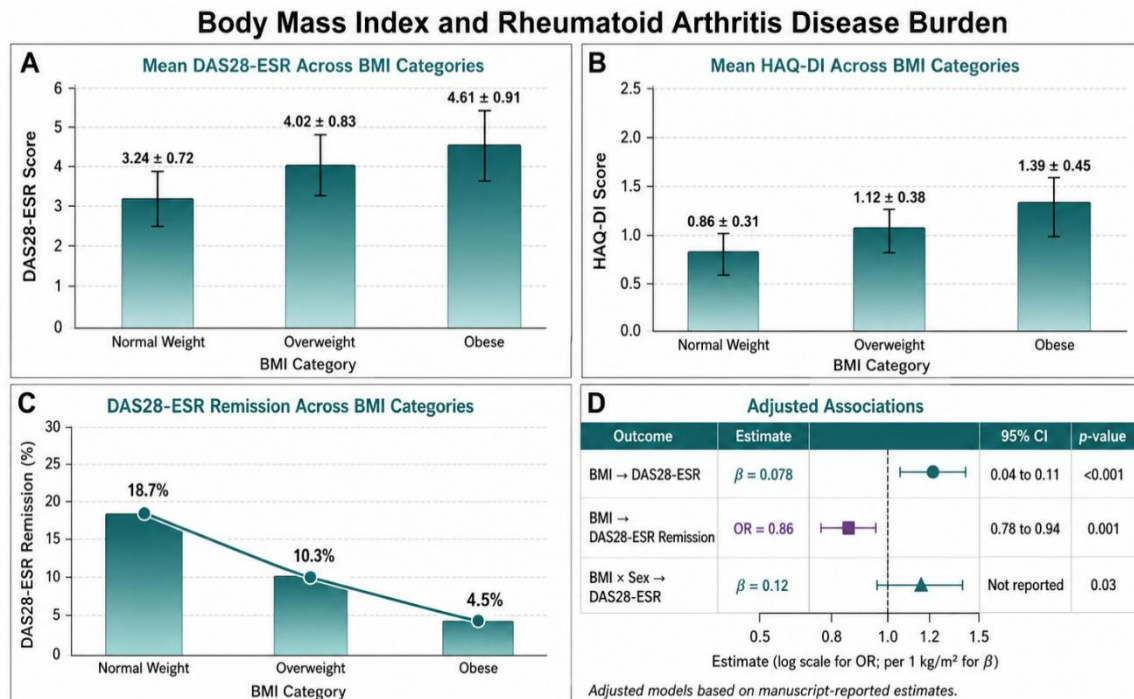


Figure 1 This multipanel figure illustrates the relationship between body mass index and rheumatoid arthritis disease burden. Panel A shows a progressive increase in mean DAS28-ESR scores across BMI categories, rising from 3.24 ± 0.72 in normal-weight patients to 4.02 ± 0.83 in overweight patients and 4.61 ± 0.91 in obese patients. Panel B demonstrates a similar upward trend in functional disability, with HAQ-DI increasing from 0.86 ± 0.31 in normal-weight

patients to 1.12 ± 0.38 in overweight patients and 1.39 ± 0.45 in obese patients. Panel C shows a marked decline in DAS28-ESR remission rates as BMI increases, decreasing from 18.7% in normal-weight patients to 10.3% in overweight patients and 4.5% in obese patients. Panel D summarizes the regression findings, indicating that higher BMI was associated with increased DAS28-ESR scores ($\beta = 0.078$, 95% CI 0.04–0.11, $p < 0.001$) and reduced odds of DAS28-ESR remission (OR = 0.86, 95% CI 0.78–0.94, $p = 0.001$). The reported BMI-by-sex interaction also suggested a stronger BMI–disease activity association among women ($\beta = 0.12$, $p = 0.03$). Overall, the figure demonstrates a consistent gradient in which increasing BMI is associated with higher disease activity, greater functional disability, and lower remission rates in rheumatoid arthritis.

Sensitivity analyses using SDAI and CDAI were reported to show the same direction of association, with obesity related to poorer disease control. Exclusion of underweight patients and patients with body mass index $\geq 40 \text{ kg/m}^2$ did not materially change the observed association between body mass index and disease activity. The available results show a coherent gradient in which higher body mass index is associated with higher disease activity, poorer functional status, and lower remission rates in patients with rheumatoid arthritis. The direction of association remained consistent across descriptive comparisons, regression models, and reported sensitivity analyses.

DISCUSSION

This prospective cohort study showed a consistent graded association between increasing body mass index and greater rheumatoid arthritis disease burden. Patients with higher body mass index had higher DAS28-ESR scores, greater functional disability measured by HAQ-DI, and lower remission rates. The mean DAS28-ESR increased from 3.24 ± 0.72 in normal-weight patients to 4.02 ± 0.83 in overweight patients and 4.61 ± 0.91 in obese patients, while HAQ-DI increased from 0.86 ± 0.31 to 1.39 ± 0.45 across the same categories. Remission declined from 18.7% among normal-weight patients to 4.5% among obese patients. These findings support the clinical relevance of body mass index as an accessible marker associated with inflammatory activity, functional limitation, and treatment-target achievement in patients with rheumatoid arthritis.

The observed relationship between higher body mass index and greater disease activity is consistent with previous evidence suggesting that obesity is associated with higher composite disease activity scores and reduced likelihood of remission in rheumatoid arthritis (1, 5, 6, 13, 19). Although the present study cannot establish causality, the persistence of the association in regression models suggests that body mass index is not merely a background demographic variable but may be an important clinical correlate of disease severity. Each 1 kg/m^2 increase in body mass index was associated with a 0.078-point increase in DAS28-ESR, and the odds of remission decreased by 14% for each 1 kg/m^2 increase in body mass index. These estimates indicate that even incremental increases in body mass index may have measurable relevance when considered at the population level and across the full body mass index spectrum.

Several biological and clinical mechanisms may explain the association between higher body mass index and increased rheumatoid arthritis activity. Adipose tissue functions as an immunometabolic organ that secretes adipokines and pro-inflammatory mediators, including leptin, resistin, visfatin, and cytokine-related pathways, which may contribute to systemic inflammation and synovial activity (6, 7, 11). Obesity may also influence inflammatory biomarkers and patient-reported components of disease activity, particularly pain, fatigue, and global health assessment. In addition, greater body weight increases mechanical loading on joints and may worsen pain and disability, especially in patients with established inflammatory joint disease. This may partly explain why functional limitation increased across body mass index categories in the present cohort, with the highest HAQ-DI score observed among obese patients.

The reduced remission rate among overweight and obese patients is clinically important. Treat-to-target strategies in rheumatoid arthritis depend on achieving remission or low disease activity, and factors that reduce the probability of remission may require attention alongside pharmacological management. Previous studies have reported that obesity may attenuate treatment response to biologic and targeted synthetic disease-modifying antirheumatic drugs, particularly tumor necrosis factor inhibitors, although the magnitude of this effect may vary by drug class and patient phenotype (5, 7, 13, 19). The present findings are aligned with this broader evidence base by showing lower remission rates among patients with higher body mass index. However, because this study was not designed to compare medication-specific

treatment response, the results should be interpreted as an association between body mass index and disease control rather than evidence of differential drug efficacy.

The interaction between body mass index and sex suggested that the association between body mass index and DAS28-ESR may be stronger among women. This finding is consistent with previous reports describing sex-specific effects of body mass index on inflammatory markers, disease activity, pain, and functional impairment in rheumatoid arthritis (12, 17, 22). Possible explanations include sex differences in fat distribution, hormonal regulation of immune pathways, pain sensitivity, and patient-reported disease activity components. Since women represented more than half of the present cohort, this interaction may have practical implications for risk stratification and individualized care. Nevertheless, the confidence interval for the interaction estimate was not reported, and future studies should evaluate sex-specific associations with adequate power and complete model reporting.

The findings also reinforce the importance of integrating weight assessment into routine rheumatoid arthritis care. Body mass index alone does not fully capture adiposity, visceral fat, sarcopenic obesity, or inflammatory body composition changes, but it remains a simple and widely available clinical measure. In routine clinical settings, identifying patients with overweight or obesity may help clinicians recognize individuals who are less likely to achieve remission and who may require more comprehensive management. Weight management, nutritional counseling, supervised exercise, and functional rehabilitation may be considered as adjunctive strategies within multidisciplinary rheumatoid arthritis care, particularly when excess body weight coexists with persistent disease activity, functional limitation, or poor quality of life. These approaches should complement, not replace, disease-modifying pharmacological therapy.

This study has several limitations. First, the sample size was modest, which limits precision for subgroup analyses, particularly sex-stratified models and extreme body mass index categories. Second, the study used body mass index rather than direct measures of body composition, such as waist-to-hip ratio, visceral adiposity, lean mass, or fat mass, which may have provided a more refined understanding of adiposity-related disease burden. Third, although adjustment was performed for prespecified confounders, residual confounding may remain due to medication heterogeneity, comorbidities, physical activity, dietary factors, socioeconomic status, and disease chronicity. Fourth, complete numerical data were not available for all inflammatory markers and secondary disease activity indices in the reported manuscript, limiting the depth of interpretation for ESR, CRP, SDAI, and CDAI. Finally, although the study was prospective in recruitment and assessment, the reported analyses primarily describe associations at baseline; therefore, causal conclusions about body mass index and subsequent disease progression should be avoided.

Despite these limitations, the study provides clinically relevant evidence that higher body mass index is associated with greater rheumatoid arthritis disease activity, poorer function, and lower remission rates in a routine clinical cohort. The findings support the inclusion of body mass index assessment in rheumatoid arthritis evaluation and suggest that adiposity-related factors may contribute meaningfully to disease burden. Future longitudinal studies should assess whether intentional weight reduction, body composition improvement, and multidisciplinary lifestyle intervention can improve inflammatory disease activity, functional outcomes, and remission achievement in patients with rheumatoid arthritis.

CONCLUSION

Higher body mass index was associated with greater rheumatoid arthritis disease activity, poorer functional status, and lower DAS28-ESR remission rates in this prospective cohort. Disease activity and HAQ-DI scores increased progressively across normal-weight, overweight, and obese categories, while remission declined markedly with increasing body mass index. regression findings further supported body mass index as an independent clinical correlate of DAS28-ESR and remission status, with a stronger reported association among women. These findings highlight the importance of routinely assessing body mass index in rheumatoid arthritis care and support the integration of weight management, lifestyle counseling, and functional rehabilitation as adjunctive components of treat-to-target management.

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