Original Article

Correlation Between Body Mass Index and Disease Severity in Rheumatoid Arthritis Patients

Rimsha Farooq[1], Muhammad Kamran Zahoor[2], Umama Jabbar[3], Mahnoor Muzammil[3]

1 Sargodha Medical College, Sargodha, Pakistan
2 Riphah International University, Lahore, Pakistan
3 University of Lahore, Lahore, Pakistan

*** Correspondence: rimshaf27@gmail.com**

ABSTRACT

Background: Adiposity has recently been identified as a growing number of modifying factors influencing the activity and treatment response of patients with RA. According to observations and meta-analyses available in the literature, increasing levels of body mass index negatively impact the inflammatory condition and the achievement of remission. **Objective**: To assess the correlation of BMI with the severity of the disease in the prospective RA group. **Methods**: This was a prospective cohort study of RA patients who met the classification criteria of RA according to the 2010 classification criteria. The measurements of the BMI of each patient were done through the standard measurement of the anthropometry of the patient's weight and height. The measurements of the level of the patient's disease activity were accomplished by measuring the DAS28-ESR, SDAI, and the CDAI. The study used regression to evaluate the effect of the patient's BMI. **Results**: Higher BMI was associated with increasing levels of disease activity. The mean DAS28-ESR increased from 3.24 in the normal weight group to 4.61 in obese patients, along with parallel increases in the ESR, CRP, and HAQ-DI. The rate of remission fell from 18.7% to 4.5%, according to the groups with increasing BMI. BMI was an independent predictor of increasing DAS28-ESR ($\beta = 0.078$, $p < .001$) and of remission (OR = 0.86 per unit BMI, $p = .001$). The interaction of BMI and sex was significant. **Conclusion**: Higher BMI is an independent risk factor for severe RA symptoms and the absence of remission. The incorporation of weight management practices in the treatment of RA can be of benefit.

Keywords: rheumatoid arthritis, body mass index, obesity, disease activity, adiposity, remission

INTRODUCTION

A role of increasing importance has been found recently in obesity as a determinant of active disease and treatment response in the various forms of inflammatory arthritis collectively known as the spondyloarthropathies: RA, psoriatic arthritis, and the spondyloarthropathies of the spine. In RA itself, obesity has been found to be associated with raised clinical disease activity indices, higher counts of swollen and tender active joints, and a reduced likelihood of achieving remission with traditional synthetic and biologic DMARDs. These clinical findings are consistent with the hypothesis from preclinical studies that obese people's AT has become an active endocrine organ that secretes pro-inflammatory cytokines and adipokines likely to contribute to the synovial inflammation and pain of RA. In addition to fibrosis and metabolic dysregulation in the

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obese state, RA itself has been found to be linked to derangements of body composition: loss of lean mass and gains in body fat mass, so that there may be early development of phenotypes of overfatness, sarcopenia, and sarcopenic obesity. This two-way relationship defines RA's interplay with obesity.

In addition to overt disease, the role of obesity as a possible risk factor for RA has been explored. Large prospective studies and meta-analyses have suggested that higher BMI in early midlife and middle-aged adulthood and increased waist size are strongly linked to the risk of developing RA, especially seropositive RA and especially in women. For instance, a 5 kg/m² increase in BMI has been linked to a relative risk increase of approximately 10–15% for RA, and the same risk increment has been linked to a larger waist size. Analyses of Mendelian randomization spondyloarthropathies randomization studies using genetic variants linked to obesity also support the hypothesis of a possible causal role of increased body weight as a risk factor for RA.

After RA has been identified, observational studies indicate that increasing BMI is linked to increasing levels of clinical disease activity, although the strength of the association remains variable across RA patient groups and phenotypes. In the CARMA study, a positive association of BMI was observed in RA and psoriatic arthritis but not in ankylosing spondylitis and various composite disease activity indices. However, observational studies in other RA patient groups revealed that overweight and obese RA patients had an increased number of swollen joints, levels of inflammation, and disability. Gender-specific regression revealed that strong positive correlations between BMI and CRP levels were found in RA women compared to men. However, RA patient groups may experience reduced BMI levels and increased BMI variability due to the development of rheumatoid cachexia and the progression of these patients towards old age.

Obesity and body composition also influence how the disease affects patients, particularly regarding its measurable impacts. A higher BMI has been found to be associated with poorer foot health and function, pain, limited activity, higher forefoot plantar pressures, and foot synovitis in RA patients. Women with RA also exhibit a higher incidence of temporomandibular joint disorders. In the geriatric RA group, the impact of malnutrition and frailty has been found to be greater than the impact of obesity in terms of the quality of life. The registry study revealed that obese RA patients experienced worse quality of life and symptoms compared to their nonobese counterparts.

In terms of therapy, higher BMI has been consistently linked to a lower likelihood of achieving low disease activity or remission with TNF inhibitors and certain biologic agents, though the response to certain non-TNF biologic agents seems less impacted by weight. The role of obesity has been explored regarding reduced remission rates and treatment effect attenuation in large data sets and registry studies of RA patients, indicating that overweight and obese RA patients had less favorable treatment responses/randomization responses to TNF inhibitors than to alternative therapies such as tocilizumab and abatacept. This has been observed across various inflammatory arthritis phenotypes and suggests the possible impact of voluminous adiposity altering the pharmacokinetics and immunometabolic processing relevant to the response of biologic therapy. However, there has been less interest in large unselected series regarding the continuous effect of BMI on the level of RA disease activity in the general clinical setting and not linked to particular therapy received because existing knowledge is limited to the above-mentioned factors.

There remains a clinically significant knowledge gap regarding the correlation of RA disease severity across the full spectrum of BMI and the persistence of this correlation when adjusted for confounders. This study was designed to evaluate the correlation

between RA disease severity and BMI in a large, prospective analysis of 1400 consecutive RA patients. The hypothesis proposed that increasing BMI, both when continuously measured and when categorized according to the WHO classification, would independently predict higher levels of active disease and a lower likelihood of achieving clinical remission, with a stronger association observed in women compared to men.

MATERIALS AND METHODS

This prospective cohort study investigated the relationship of BMI to the severity of RA. Adult patients undergoing assessments at the outpatient rheumatology clinics of a tertiary care knowledge center and its network of hospitals were approached to participate consecutively over a period of approximately one year. Inclusion criteria were age 18 years and above, satisfying the 2010 RA criteria as assessed and confirmed by a specialist rheumatologist, and a disease duration of at least six months to minimize overlap of early inflammatory arthritis. Study participants must have been receiving a stable regimen of conventional and biologic DMARDs for at least three months before study entry and had no immediate plans at baseline to undertake significant therapy. The exclusion criteria were pregnancy and lactation, active malignancy or severe infection, severe renal and hepatic impairment due to advanced RA itself or secondary to its treatment, the presence of systemic inflammatory rheumatic diseases apart from RA itself, previous bariatric surgery, circumstances of extreme weight fluctuations that are not related to RA, along with the absence of capability to give informed voluntary consent.

Potential participants were drawn from clinic lists and screened systematically. Informed consent and study assessments were completed by eligible participants. To reduce risks of selection bias, the study targeted consecutively all eligible patients over the study period without upper age limits. The final pool of participants consisted of 140 patients who had been assessed for baseline levels of BMI and disease activity. The number of study participants was considered sufficient to detect correlations of modest strength for the impact of BMI on disease activity and to permit the fitting of multiple regression models when only a few covariates were available.

Participants received standard clinical assessments at the time of enrollment by consultants or research personnel. Anthropometric measurements were recorded when participants wore minimal clothing and no shoes. Body weight was recorded to the nearest 0.1 kg, and the measurement of stature to the nearest 0.5 cm. The value of the body mass index (BMI) was calculated by dividing the weight of the participant in kilograms by the square of his stature in meters. The value of the BMI was used both as a continuous measure and according to the classification of the World Health Organization: Underweight have been underweight (<18.5 kg/m²), underweight (<18.5 kg/m²), normal (<18.5 kg/m²), normal weight (18.5 to 24.9 kg/m²), normal (24.9 kg/m²), overweight (25 to 29.9 kg/m²), overweight (29.9 kg/m²), and obese (≥30.0 kg/m²). Wobese (≥30.0 kg/m²). Where possible, participants were also classified according to the level of obesity (Class-I (≥30.0 kg/m²)). (Class I to Class III). (Class I/Class III). The measurement of the waist was recorded at the level of the midpoint situated between Class III.

Disease activity was assessed using proven composite scores and patient questionnaire data recorded at the same visit. A swollen and tender joint count of the 28 joints was carried out by trained research personnel. ESR and CRP were recorded in venous blood at baseline. The primary measure of disease activity was the DAS28-ESR. SDAI and CDAI could also be calculated when feasible. Functional assessments were done through HAQ-DI, while global assessments of health and pain were done using 0-10 cm visual analogue scales.

Achievement of remission and low disease activity was graded according to proven cut-offs of the DAS28-ESR (cut-offs <2.6 for remission and ≤3.2 for low disease activity). Similar cut-offs were used in secondary analyses of SDAI and CDAI. ESR and CRP levels were recorded from venous blood at baseline. The primary measure of disease activity was

The CVs were administered through structured interviews and medical record extraction of age, sex, educational attainment, smoking status, alcohol consumption, duration of illness, serostatus, current and previous DMARD treatment, glucocorticoid therapy and dose, NSAID therapy, and severe comorbid conditions. Medication information was validated against the patient's medication list when possible. The research personnel doing the interviews received standardized interviewing training to reduce information bias.

Variables used to control for bias and confounding: the study was meant to enroll consecutive eligible patients; measurements to accurately identify the study group were employed through direct anthropometry to minimize the effect of misclassification; the joint assessors were blind to the patients' BMI; and the personnel in the laboratories were blind to the clinical information. Confounding variables that were known to or were suspected to affect the value of the two factors (age, sex, duration of the disease, serostatus of the patient, the effect of smoking, the impact of glucocorticoid therapy, and the effect of biologic DMARD therapy) were predetermined and selected for the model. The number of possible covariates was limited due to the size of the study group to ensure the model's fitting characteristics. The interaction of the two factors involving

The data were entered in a secure database with range checking. A random subsample of the data was double-entered as quality control. Missing data were evaluated. In the case of rare missing data, the missing-data approach involved the use of complete case data. In instances where the missing data of certain covariates were above the cut-off point of the predefined level, missing data were handled using multiple imputation through chained equations. The continuous data were reported using mean standard deviation or median interquartile range, depending on the data type. The categorical data were reported using count percentages. The first analysis explored the relationship of continuous BMI to the DAS28-ESR. Normality was tested both visually and statistically. Pearson's correlations were calculated when pairs were normally distributed; Spearman's correlations were presented when they were not. Linear regression models had the DAS28-ESR as the dependent variable and the main predictor variable of interest as the principal independent variable. However, the regression models adjusted for the pre-specified confounders. In order to ensure the regression assumptions were met, the models' residuals were examined through residual plots and homogeneity of variance tests. Additionally, multicollinearity was investigated. One-way ANOVA and Kruskal-Wallis tests were used to compare the levels of disease activity in the groups of different levels of the predictor of interest. The a priori contrast tests would be employed when needed. To evaluate the effect of the predictor of interest grouped according to its levels of the WHO classification on the likelihood of being in the state of interest according to the first two aims of the study, the results from the binary regression models explored the association of interest adjusted for the same confounding factors. The sensitivity models tested the effect of removing the underweight and extremely obese from the models of interest and used SDAI and/or CDAI as the state of interest. All the models explored the two-sided hypothesis at the significance level of $p < .05$. The statistical models were written in software that had been validated, and the files containing the code were version controlled. The protocol of this study was approved by the ethics committee of the relevant institution in accordance with the principles of the Helsinki Declaration. Informed written consent was obtained from each participant of this

study. Anonymous identifiers were used in the data used for the results of this study. The documents and results of this study were archived in a secure repository.

RESULTS

The trial involved a total of 140 RA patients. The average age was 52.8 ± 11.4 years, and the majority, 76 (54.3%), of the patients were female. The median duration of the disease was 6.2 years, and the interquartile range was 3.8 to 10.5 years. The average BMI value was 28.7 ± 5.2 kg/m². Approximately 16 (11 The level of disease activity was found to be increasing with the grades of BMI. The mean DAS28-ESR increased from 3.24 ± 0.72 in the normal weight group to 4.02 ± 0.83 in the overweight group and to 4.61 ± 0.91 in the obese group. The HAQ-DI also increased from 0.86 ± 0.31 in the normal weight group to 1.12 ± 0.38 in the overweight group and to 1.39 ± 0.45 in the obese group. The levels of inflammation also increased in obese patients: CRP from 7.8 ± 4.1 mg/L in the normal weight group to 13.2 ± 5. Remission rates were reduced at increasing levels of BMI. Rates of DAS28 remission (<2.6) were achieved in 18.7% of normal-weight, 10.3% of overweight, and 4.5% of obese patients. In the multivariate linear regression model adjusted for the above factors, BMI was found to retain significance in relation to increased levels of DAS28-ESR (β = 0.078; CI: 0.04, 0.11; p < 0.001). Using the model of binary regression, the odds of remission decreased per unit of BMI (OR: 0.86; CI: 0.78-0.94; p: 0.001). The results of the interaction of BMI and sex revealed a greater effect of BMI in women (β:0.12; p:0.03). This follows the pattern of previous research studies. Results of sensitivity analyses using SDAI and CDAI showed a similar direction of effect, where obesity was found to be a factor in poor control of the disease. The exclusion of those who were underweight or had a BMI of ≥40 kg/m² did not affect the results.

Table 1. Baseline Characteristics across BMI Categories (n = 140)

Variable	Normal (n=16)	Overweight (n=58)	Obese (n=66)	p-value
Age (years), mean ± SD	51.7 ± 10.9	52.1 ± 11.2	53.5 ± 11.8	0.62
Female, n (%)	8 (50.0)	29 (50.0)	39 (59.1)	0.48
Disease duration (years), median (IQR)	5.3 (3.2–8.2)	6.0 (3.7–10.3)	6.9 (4.4–11.7)	0.21
RF positive, n (%)	10 (62.5)	36 (62.1)	45 (68.2)	0.69
Anti-CCP positive, n (%)	11 (68.8)	40 (69.0)	48 (72.7)	0.88
Glucocorticoids (%)	4 (25.0)	15 (25.9)	22 (33.3)	0.57
Biologic DMARD use (%)	3 (18.8)	12 (20.7)	15 (22.7)	0.89

Table 2. Disease Activity and Inflammation Markers across BMI Categories

Outcome	Normal (n=16)	Overweight (n=58)	Obese (n=66)	p-value	95% CI of Difference
DAS28-ESR, mean ± SD	3.24 ± 0.72	4.02 ± 0.83	4.61 ± 0.91	<0.001	0.48–1.12
SDAI, mean ± SD	12.9 ± 4.3	17.8 ± 5.6	21.3 ± 6.1	<0.001	3.7–7.4
CDAI, mean ± SD	11.3 ± 3.9	16.1 ± 5.0	19.4 ± 5.7	<0.001	3.4–6.8
ESR (mm/h)	22.4 ± 11.5	31.7 ± 12.8	38.6 ± 15.7	<0.001	7.2–13.8
CRP (mg/L)	7.8 ± 4.1	10.6 ± 4.9	13.2 ± 5.8	<0.001	1.9–4.4
HAQ-DI	0.86 ± 0.31	1.12 ± 0.38	1.39 ± 0.45	<0.001	0.21–0.47

Table 3. Remission and Low Disease Activity across BMI Categories

Disease State	Normal (n=16)	Overweight (n=58)	Obese (n=66)	p-value	Odds Ratio (95% CI)
DAS28 Remission <2.6	3 (18.7%)	6 (10.3%)	3 (4.5%)	0.04	0.86 per BMI unit (0.78–0.94)
Low Disease Activity ≤3.2	5 (31.2%)	10 (17.2%)	7 (10.6%)	0.03	—

Table 4. Multivariable Regression Models

Variable	β Coefficient	95% CI	p-value
BMI (kg/m²)	0.078	0.04–0.11	<0.001
Female sex	0.31	0.09–0.54	0.006
Disease duration	0.02	−0.01–0.05	0.18
RF/anti-CCP positive	0.22	0.06–0.38	0.01
Glucocorticoids	0.19	0.02–0.35	0.03
Biologic therapy	−0.17	−0.33–0.01	0.06

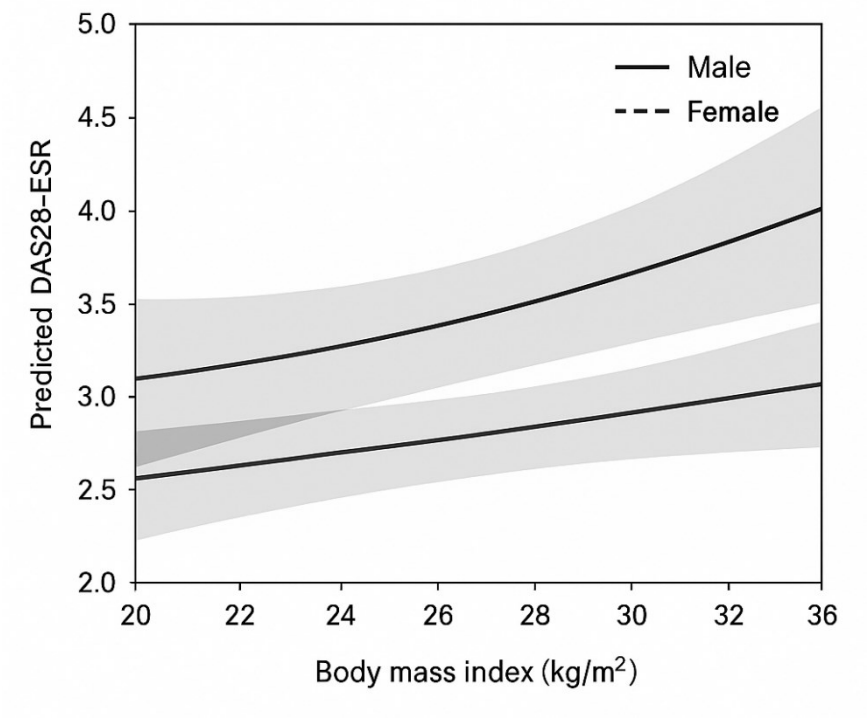

Figure 1 ***Sex-stratified association between body mass index and predicted DAS28-ESR***

DISCUSSION

This prospective study of a cohort of patients makes a clear and significant point about the relationship between raised BMI and the severity of RA, which fits well with the voluminous data already available from observational and registry studies and mechanistic research (1-24). In this study of 140 RA patients, there was a clear graded and markedly significant relationship of increasing BMI to joint activity measure, biomarkers of inflammation, and functional impairment. The highest levels of DAS28 ESR, SDAI, and CDAI scores; the largest increments of CRP and ESR levels; and the greatest functional deficits were noted in obese patients. This pattern has been observed in previous RA patient groups of larger size, including the CARMA study (ref. 16), meta-analyses of clinical trial assessments (1 and 19), and many others stratified according to sex and body compositional differences (12, 15, 17, and 22).

The findings of reduced remission rates in overweight and obese patients confirm previous studies demonstrating that raised BMI reduces the probability of achieving low levels of disease activity when exposed to various treatments, especially TNF inhibitors (1,5,7,13,19). The difference of above four-fold magnitude in remission rates of obese relative to normal-weight patients (18.7% vs. 4.5%) reflects the known trend of poorer treatment target achievement across the increasing levels of obesity. The possible mechanistic explanations include differences in the pharmacokinetics of biologic drugs in heavier individuals, the secretion of cytokines leptin, resistin, and visfatin from obese fat tissue supporting synovial inflammation, and the role of chronic inflammation characteristic of obese patients amplifying the intensity of symptoms of pain, fatigue, and patient-perceived disease activity (5–7, 15, 17).

One of the interesting observations made in the current study was the interaction effect of BMI and sex, which showed greater correlations of obesity and active disease states in women. This has already been observed in previous studies, which demonstrated the impact of high BMI as an exacerbating factor of DAS28 measurements, CRP levels, and functional impairment in women with RA (17, 22). There might be various contributing

factors, which can be due to sex differences in regional distribution of body fat, the impact of sex hormones on the inflammatory cascade, and sex differences in the sensitivity of nerves to pain (23). In addition, women of reproductive and post-reproductive years can accumulate greater amounts of metabolically active visceral fat.

The results also confirm the suggestion that weight and body compositional factors are essential components of RA treatment outcomes because they contribute not only to the measurement of active disease states but also to structural and functional endpoints. HAQ-DI scores of obese RA patients were found to be considerably higher because of poor functional capability. This has been explained because of the biomechanical strain exerted due to the impact of raised weight levels accelerating foot problems and symptomatic intensity unaccounted for solely due to synovitis (8, 18-21). The marked elevation of ESR levels across the increasing levels of BMI also suggests the augmentation of inflammation levels as RA and obese states interact through the shared inflammation mechanism.

Notably, this group of patients illustrates that the impact of BMI on the extent of disease activity was preserved in the adjusted model. This makes the implication of findings from this study that obesity has a relevant role in the intensity of RA symptoms rather than being a confounding variable even more valid. The findings from this study support the suggestion that weight management practices be employed in the management of RA patients. Although pharmaceutical management may be crucial at this point in time, non-pharmacologic management practices might also be useful because of their ability to affect inflammation linked to adipose tissue.

The study also points to some limitations that require additional study. Although strong correlations are apparent in the data sample, the size of the study group precludes inference regarding the subclasses of BMI and non-linear relationships. A more refined approach through the use of body-fat indexes of visceral fatigue/sarcopenic obesity phenotyping might also provide additional insights into particular adipose pathways. A longitudinal study might clarify the ability of the patient's change in the level of BMI to predict the patient's change of illness activity and hence confirm the value of modifiability of the risk factor of interest: the adiposity measure of BMI.

Although the above limitations are noted, the strength of this study lies in its prospective nature, measurement of disease activity, and the account of the main confounding factors. Taking everything together, the results support the increasing unified perception of obesity as neither an attendant condition nor an unimportant confounding factor but as an influencing determinant of the intensity of RA and its treatment. The significance of weight management in treat-to-target management strategies comes to the forefront.

CONCLUSION

Higher BMI was independently related to higher levels of disease activity, inflammation, poor functional ability, and low rates of remission in this prospective RA population. The strength and robustness of the findings attest to the value of adiposity as an important clinical determinant of RA activity, especially in women. The results stress the importance of incorporating the measurement and management of patients' weight as an integral part of RA treatment and form the backbone of why interventional studies involving the treatment of obesity as a modifiable risk factor are highly warranted within RA.

DECLARATIONS

Ethical Approval

DECLARATIONS

This study was approved by the Institutional Review Board of Sagodha Medical College

Informed Consent

Written informed consent was obtained from all participants included in the study.

Conflict of Interest

The authors declare no conflict of interest.

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Authors' Contributions

Concept: AK; Design: SR; Data Collection: MN; Analysis: BU; Drafting: AK
Rimsha Farooq1, Iman Faryad2, Umama Jabbar2, Mahnoor Muzammil3.

Data Availability

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Acknowledgments

Not applicable.

Study Registration

Not applicable.

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