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Obesity, C-Reactive Protein, and Calprotectin in Adalimumab-Treated Rheumatoid Arthritis Patients: Association with Clinical Response: A cross-sectional comparative study

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Abstract

Rheumatoid arthritis (RA) is a long-lasting inflammatory illness considered via synovial inflammation as well as joint destruction. Tumor necrosis factor-alpha and interleukin-6 are key mediators. Adalimumab is operative; a percentage of patients show inadequate response. C-reactive protein and erythrocyte sedimentation rate are generally run down inflammatory markers, but limited ability to predict treatment response. Calprotectin has been projected as a biomarker of illness activity. To assess serum CRP and calprotectin ranks in RA patients cured with adalimumab and to evaluate their connotation with BMI and medical response. A cross-sectional comparative study included 130 participants: 75 RA patients receiving adalimumab and 55 controls. Patients included 25 males and 50 females; mean age 47.57 ±10.68 (responders n = 35) and 46.40 ±8.76 (non-responders n = 40), categorized rendering to European Alliance of Associations for Rheumatology principles, using Disease Activity Score28 with Erythrocyte sedimentation rate. CRP and calprotectin were restrained using Enzyme-Linked Immunosorbent Assay**.** RA patients showed higher CRP and calprotectin ranks paralleled to controls ($p < 0.001$). Calprotectin did not diverge among responders and non-responders, while CRP was higher in non-responders ($p < 0.001$). BMI was positively associated with calprotectin ($r = 0.746$) and CRP ($r = 0.655$) ($p < 0.001$). CRP and calprotectin are elevated in RA patients treated with adalimumab; Only CRP is associated with treatment response. Calprotectin reflects inflammatory burden, while obesity is related with augmented inflammatory indicators and may negatively influence outcomes.

Keywords: Rheumatoid arthritis; Obesity; Calprotectin; CRP; Adalimumab

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Introduction

Rheumatoid arthritis (RA) is a long-lasting systemic autoimmune illness with synovial inflammation and joint destruction [1]. Tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) are vital drivers of this inflammatory state [2]. Adalimumab is a fully human monoclonal antibody that selectively impasses to TNF-α, neutralizing its pro-inflammatory effects and effectively halting synovial inflammation and joint obliteration in RA. Although Adalimumab improves outcomes, many patients fail to respond adequately[3]. CRP and ESR are classically used to identify and predict how well an individual will respond to therapy for rheumatoid diseases. However, due to the fact that these markers do not identify subclinical inflammation and their inconsistencies in predicting an individual's treatment response, many researchers have sought out new, more sensitive biomarkers [4]. Calprotectin, which is primarily found in the cytoplasm of neutrophils and monocytes, is a calcium-and zinc-binding protein heterodimer made up of S100 calcium-binding protein A8 (S100A8) and S100 calcium-binding protein A9 (S100A9) subunits. It is secreted extracellularly during active inflammation where it serves as a direct modulator of the innate immune response. Serum calprotectin, in contrast to C-reactive protein (CRP) , a systemic hepatic acute phase reactant, mirrors local leukocyte activation, and active inflammation of the microvascular endothelium in the synovial membrane, and may therefore have a clinical application as a highly sensitive biomarker to assess disease activity and treatment outcomes [5].

Obesity has been increasingly recognized as a potential barrier to biologic therapy [6]. Non-fatty tissue is now thought of as more than simply a location for storing fat; it has also been found to function as a hormone-releasing organ and can increase blood levels of cytokines (chemical messengers that stimulate immune system response) in the body. Adipose and IL-6 from adipose will trigger an inflammatory response in other parts of the body [7]. The pro-inflammatory condition related with overweightness may donate to elevated levels of inflammatory biomarkers, higher BMI has been linked to lower serum drug levels and reduced treatment efficacy of TNF inhibitors like adalimumab [8].

Despite the high prevalence of obesity among RA patients, limited data are accessible concerning its influence on inflammatory biomarkers and treatment response in the Mid-Euphrates region, particularly the role of calprotectin as a predictive marker. Thus, the goal of this investigation was to evaluate serum CRP and calprotectin levels in RA sick cured with adalimumab and to investigate their association with BMI and clinical response.

Materials and Methods

Study design and participants

A cross-sectional comparative study included 130 participants: 75 RA patients sustaining the 2010 ACR/EULAR principles (25 males and 50 females, with a mean age of 47.57 ±10.68 years for responders and 46.40 ±8.76 years for non-responders) and 55 well controls. All RA sick stood treated with adalimumab monotherapy (40 mg

subcutaneously every 2 weeks) for at least 6 months.

Patients were classified into:

- Responders (n = 35): DAS28 < 2.6 or improvement ≥ 1.2
- Non-responders (n = 40): DAS28 ≥ 2.6 and improvement < 1.2

Disease Activity Score (DAS28-ESR)

The DAS utilizing 28 joints (DAS28) is a commonly employed composite metric for evaluating illness action in individuals with RA. This score combines clinical assessment of joint involvement with laboratory indicators of inflammation and the patient's overall health evaluation.
The DAS28 is computed using the count of tender joints (TJC28) and swollen joints (SJC28) from a total of 28 joints, along with an inflammating indicator, The (ESR) and the patient's global health assessment (GH).

DAS28 Formula Utilizing ESR

$$\mathrm{DAS28} = 0.56\sqrt{\mathrm{TJC28}} + 0.28\sqrt{}$$

SJC28 : the number of swelling joints (from a total of 28).

TJC28: number of joints that are tender (out of 28).

GH = patient's global health evaluation quantified on a visual analogue scale (VAS) from 0 to 100.

For categorizing the DAS28 was used according to the EULAR criteria. Patients with a DAS28 < 2.6 or an improvement delta of ≥ 1.2 from baseline were considered as responders. Patients with a persistent DAS28 ≥ 2.6 and an improvement delta of < 1.2 were labeled as non-responders.

Exclusion Criteria

Active infections, immunosuppression, pregnancy/lactation, other autoimmune/inflammatory diseases, including any changes to DMARDs in the last 3 months.

The ethical deliberation

Ethical approval has stood acquired by the approved committee of ethical supervision at Al-Qadisiyah University College of Medicine.

Clinical/Demographic Data

Age, body mass index (BMI), sex, length of illness, DAS28 score, concurrent use of corticosteroids or non-steroidal anti-inflammatory drugs (NSAIDs), and patient adherence, including missing adalimumab doses, were among the clinical and demographic information gathered.

BMI Classification

The formula for scheming (BMI) is a person's weight separated by their height squared in meters. As defined by the World Health Organization (WHO), an individual is considered overweight if they have a BMI larger than or equal to 25 kg/m², and obese if their BMI is greater than or equal to 30 kg/m²[9].

Laboratory Measurements

Blood samples were taken from a vein to collect 5 mL of blood, then spun on a centrifuge for 10 minutes at 3000 rpm before serum was frozen at -80 degrees Celsius until it could be checked. CRP levels had been determined via a human CRP ELISA kit (catalog no. ELK1040). Calprotectin levels had been measured using a human calprotectin ELISA kit (catalog no. ELK10596).

Statistical investigation

Data were examined by means of SPSS version 23 (IBM Corp., Armonk, NY, USA). Incessant variables stood articulated as mean ± standard deviation (SD). Assessments among two groups were achieved by independent Student's t-test, and assessments among three groups stood achieved by one-way analysis of variance (ANOVA) with post-hoc Tukey examination. Pearson association coefficient was run down to evaluate bivariate relations. Multivariable linear reversion examination stood achieved to recognize independent analysts of calprotectin and CRP levels, with modification for sex,age, and illness duration. A p- value < 0.05 stayed deliberated statistically important.

Results

General appearances compared among study groups

General characteristics compared among study groups are revealed in **table 1**. Comparison of mean age revealed no significant difference (p = 0.603). There was also no significant alteration in proportions of males and females between groups (p = 0.870). BMI was lower considerably in response group in comparison with failure group, 24.28 ±2.94 kg/m2 versus 32.66 ±4.56 kg/m2, separately (p <0.001). Comparison of duration of disease between study groups revealed no significant difference, 0.75 (0.5) years versus 0.75 (1.56) years, respectively (p = 0.816). Mean disease activity score was lower significantly in response group in comparison with failure group, 3.35 ±0.78 versus 5.65 ±0.39, respectively (p <0.001). Use of steroids or non-steroids was upper in failure group parallelled to response group, 27.5 % versus 11.4 %, respectively; however, the alteration was not important (p = 0.083).

Table 1: General appearances compared among study groups

Characteristic	Response group *n* = 35	Failure group *n* = 40	*P*
Age (years)			
	47.57 ±10.68	46.40 ±8.76	0.603 N
	27 -65	29 -60	
Gender			
Male, *n* (%)	12 (34.3 %)	13 (32.5 %)	0.870 N
Female, *n* (%)	23 (65.7 %)	27 (67.5 %)	
BMI (kg/m^2)			
Mean± SD	24.28 ±2.94	32.66 ±4.56	**<0.001 S**
Range	19.5 -35.3	22.5 -39	
Duration (years)			
Median (IQR)	0.75 (0.5)	0.75 (1.56)	0.816 N
Range	0.33 -10	0.25 -3	
DAS-28			
Mean± SD	3.35 ±0.78	5.65 ±0.39	<0.001 S
Range	0.97 -4.8	5 -6.3	
NSAID or steroid			
Yes	4 (11.4 %)	11 (27.5 %)	0.083 N
No	31 (88.6 %)	29 (72.5 %)	

SD: standard deviation; **DAS-28**: disease activity score-28; **NSAID**: non-steroidal anti-inflammatory drugs **S**: significant; **N**: not significant; **P-value:** Probability value

Characteristics of enrolled patients

Patient's appearances are revealed in **Table 2.** Missing dose was limited to failure group, 27.5 % versus 0 %, respectively (p = 0.001).

Comparison of CRP mean levels among study groups

Comparison of C-reactive protein (CRP) mean levels among study groups is shown in **table 3.** This comparison revealed significant difference (p < 0.001). The level stayed significantly highest in failure group (4910.2 ±123.85) followed by response group (3989.80 ±294.68) then by control group (3666.1 ±660.67).

Table 2: Patients' characteristics

Characteristic	Response group *n* = 35	Failure group *n* = 40	*p*
Miss dose			
Yes, *n* (%)	0 (0.0 %)	11 (27.5 %)	**0.001 S**
No, *n* (%)	35 (100.0 %)	29 (72.5 %)	

Table 3: Comparison of CRP mean levels among study groups

Characteristic	Response group *n* =35	Failure group *n* = 40	Control group *n* = 55	*P*
CRP (ng/ml)				
Mean ±SD	3989.80 ±294.68 b	4910.2 ±123.85 a	3666.1 ±660.67 C	<0.001 S
Range	3007.39 -4514.39	4706.12 -5124.9	37.58 -4182.74	

CRP: C-reactive protein; **SD**: standard deviation; **S**: significant

Comparison of calprotectin mean levels among study groups

Comparison of calprotectin mean levels among study groups is shown in **table 4**. This comparison revealed significant difference ($p < 0.001$). The level was significantly highest in response and failure groups in comparison to control group.

Correlation and regression analysis

Immune markers correlations to BMI are demonstrated in **table 5.** All markers showed significant and positive correlations to BMI.

Table 4: Comparison of calprotectin mean levels among study groups

Characteristic	Response group *n* =35	Failure group *n* = 40	Controlling group *n* = 55	*P*
Calprotectin (ng/ml)				
Mean ±SD	617.82 ±16.08 a	611.91 ±5.94 a	535.41 ±34.81 B	<0.001 S
Range	511.23 -785.27	600 -625.12	401.4 -602.11	

SD: standard deviation; **S**: significant; ; small letters (a, b and c) were used to show the results of post-hoc LSD multiple comparison test so that letter a takes the maximum value surveyed via letter b then letter c; like letters indicated no important variation; whereas, different letters indicated important variance

Table 5: Immune marker correlations to BMI

Characteristics	BMI	
	r	***P***
C-reactive protein	0.655	<0.001 S
Calprotectin	0.746	<0.001 S

S: significant; **P-value**: Probability value; r: Correlation coefficient

Multivariable linear regression analysis demonstrated that BMI remained an independent predictor of both CRP and calprotectin ranks next adjustment for age and sex ($p < 0.05$).

Discussion

In this study, the mean BMI was higher in a significant manner in those patients who failed to respond to adalimumab treatment when comparison was made with response group, indicating that BMI may have a important influence on patients' outcome. Similarly, Kaeley and his colleagues, [10] reported that obese RA patients showed less well improvement when treated with adalimumab upon comparison with non-obese patients.

According to previous studies, obese RA sick tend to have inferior medical results paralleled with patients of normal weight [11]. Our findings are consistent with these outcomes and propose that increased BMI might be related with reduced improvement in disease activity.

Excess adiposity has dual negative effects in patients with RA who fail anti-TNF therapy: Fat tissue is an active endocrine organ that hypersecretes major pro-inflammatory cytokines, such as TNF-α and IL-6 [12, 13]. These cytokines induce a cascade that propagates joint pathology and directly promotes hepatic synthesis of CRP which is consistently elevated in patients with higher BMI [14] but also hastens leukocyte recruitment into the synovium, which then stimulates a robust local relief of calprotectin from activated neutrophils and macrophages. Furthermore, calprotectin has been proposed as a marker of residual inflammation in patients treated with TNF inhibitors[15] Pharmacokinetically, an increased BMI significantly alters the biodistribution and clearance of monoclonal antibodies. The ongoing inflammatory burden may counteract fixed doses of adalimumab and poses a serious biological resistance barrier [16].

In this study, mean CRP levels stood meaningfully upper in the failure group parallelled with the controlling group. This supports the concept that obesity contribute to persistent systemic inflammation, which may worsen RA pathogenesis and reduce the effectiveness of anti-TNF therapy. Preceding investigations have exposed that anti-TNF agents decrease CRP levels in RA patients [17,18, 19, 20]. The higher CRP levels in the failure group may also be partly related to reduced treatment adherence and missed doses compared with the response group.

The present study demonstrated that missed doses were significantly associated with poor treatment response. This suggests that

inadequate adherence to adalimumab therapy may contribute to treatment failure. Reduced drug exposure due to missed doses may also explain the higher CRP levels observed in non-responders.

Calprotectin (MRP8/14) has been proposed as a more sensitive biomarker of disease activity in RA compared with traditional inflammatory markers such as CRP [5]. In our study, calprotectin levels showed a significant difference among groups, being higher in both response and failure groups parallelled with the controlling group, whereas no important alteration was detected between the response and failure groups.

Earlier trainings have stated contradictory outcomes concerning the predictive value of calprotectin. Choi et al., [21] found that serum MRP8/14 ranks were related with medical symptoms and could potentially predict and monitor treatment response. They also reported that adalimumab improved outcomes in responders but not in non-responders.

However, other studies, such as Tweehuysen et al., [22] found no significant differences in calprotectin levels between response groups and reported limited value in predicting treatment response during therapy tapering. Similarly, Anink et al., [23] noted a lack of additional predictive utility of calprotectin after initiation of TNF inhibitors.

Calprotectin is not specific to RA activity alone, as its ranks may likewise be predisposed by comorbid circumstances such as obesity and cardiovascular disease, which may interfere with its interpretability [24].Therefore, its role as a reliable predictor of treatment response remains controversial, and additional investigations are still needed to clarify its medical efficacy [25].

The positive correlation between BMI and inflammatory markers indicates that obesity contributes to increased systemic inflammation in RA patients. This may negatively affect disease control and response to biologic therapy.

Overall, obesity appears to be an important factor influencing management reaction in RA patients cured with adalimumab, while CRP may be more useful than calprotectin in predicting response.

Study Limitations

Limitations include the cross-sectional (no causal conclusions), lack of pre-treatment baseline measurements, small sample size (n=75). Therefore, longitudinal studies with baseline measurements are needed.

Conclusion

Obesity appears to be an important factor influencing disease activity and treatment response in RA patients preserved with adalimumab. CRP may help as a more beneficial biomarker for predicting treatment response compared with calprotectin, which reflects overall inflammatory burden rather than therapeutic efficacy. These results focus the significance of considering body weight in the administration of RA patients receipt biologic treatment.

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ملخص

الخلفية العلمية: التهاب المفاصل الروماتويدي مرض التهابي مزمن يتميز بالتهاب الغشاء الزلالي وتلف المفاصل. يُعد عامل نخر الورم ألفا والإنترلوكين-6 من الوسائط الرئيسية. على الرغم من فعالية أداليموماب، إلا أن نسبة من المرضى لا يستجيبون له بشكل كافٍ. يُستخدم كل من البروتين التفاعلي (CRP) وسرعة ترسب الدم ESRعلى نطاق واسع كمؤشرات للالتهاب، لكن قدرتهما على التنبؤ بالاستجابة للعلاج محدودة. وقد تم اقتراح الكالبروتكتين كمؤشر حيوي لنشاط المرض. **الاهداف:** تقييم مستويات بروتين سي التفاعلي والكالبروتكتين في مصل مرضى التهاب المفاصل الروماتويدي الذين تم شفاؤهم باستخدام أداليموماب، وتقييم ارتباطها بمؤشر كتلة الجسم والاستجابة للعلاج **المنهجية**: شملت دراسة مقطعية مقارنة130 مشاركًا: 75 مريضًا بالتهاب المفاصل الروماتويدي يتلقون أداليموماب، و55 شخصًا كمجموعة ضابطة. شمل المرضى 25 ذكرًا و50 أنثى، بمتوسط عمر 47.57 ± 10.68 عامًا للمستجيبين (ن = 35) و46.40 ± 8.76 عامًا لغير المستجيبين (ن = 40) مصنفين وفقًا لمعايير التحالف الأوروبي لجمعيات أمراض الروماتيزم، باستخدام مؤشر نشاط المرض 28 مع سرعة ترسب الكريات الحمراء.وقُيس مستوى البروتين سي التفاعلي والكالبروتكتين باستخدام مقايسة الامتصاص المناعي المرتبط بالإنزيم **النتائج**: أظهر مرضى التهاب المفاصل الروماتويدي مستويات أعلى من البروتين سي التفاعلي والكالبروتكتين مقارنةً بالمجموعة الضابطة قيمة $p < 0.001$لم تختلف مستويات الكالبروتكتين بين المستجيبين وغير المستجيبين، بينما كانت مستويات البروتين سي التفاعلي أعلى لدى غير المستجيبين قيمة $p < 0.001$وارتبط مؤشر كتلة الجسم ارتباطًا إيجابيًا بالكالبروتكتين معامل الارتباط $r = 0.746$ والبروتين سي التفاعلي معامل الارتباط $r = 0.655$قيمة $p < 0.001$ **الاستنتاج:** ترتفع مستويات البروتين السي التفاعلي والكالبروتكتين لدى مرضى التهاب المفاصل الروماتويدي الذين عولجوا بالأداليموماب؛ البروتين سي التفاعلي فقط هو المرتبط بالاستجابة للعلاج. يعكس الكالبروتكتين العبء الالتهابي، بينما ترتبط السمنة بارتفاع مؤشرات الالتهاب، وقد تؤثر سلبًا على النتائج.

الكلمات المفتاحية: التهاب المفاصل الروماتويدي؛ السمنة؛ الكالبروتكتين؛ بروتين سي التفاعلي (CRP)؛ أداليموماب.