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ORIGINAL RESEARCH

Estimation of serum adiponectin levels in rheumatoid arthritis patients to assess severity of disease

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ABSTRACT

Background: Rheumatoid arthritis (RA) is a chronic autoimmune disorder characterized by joint inflammation and systemic complications. Adiponectin and C-reactive protein (CRP) have been implicated in the inflammatory process associated with RA, but their relationship and role in joint damage remain unclear. **Methodology:** A comprehensive literature review was conducted, and an investigation was carried out to assess the levels of adiponectin, CRP, in patients with RA compared to healthy controls. Data from a sample of RA patients were analyzed to explore the potential correlation between adiponectin, CRP. **Results:** Consistent with previous findings, patients with RA exhibited elevated levels of adiponectin and CRP compared to healthy individuals. Elevated disease activity in RA was associated with higher concentrations of adiponectin, which correlated with increased radiological joint damage. A modest positive correlation between adiponectin and CRP was observed, suggesting their potential use as early indicators of joint damage in RA. **Conclusion:** Adiponectin and CRP levels, along with lipid profiles, could serve as valuable biomarkers for early identification of joint damage in RA. Regular screening and follow-up are recommended to monitor disease activity and extra-articular symptoms and address treatment-related issues. However, further community-based studies with larger sample sizes are necessary to elucidate the impact of adiponectin on disease activity in RA and its correlation with CRP.

Keywords: Rheumatoid arthritis, adiponectin, C-reactive protein, joint damage, lipid profile, biomarkers, disease activity.

INTRODUCTION

Rheumatoid arthritis (RA) is a long-term inflammatory condition that affects the entire body and is characterized by synovial membrane inflammation,[1] cartilage and bone deterioration, and symptoms like pain, exhaustion, and reduced physical function. (2).Population growth, age, urbanization, poverty, infectious diseases, and lack of modern healthcare are increasing the global incidence of rheumatoid arthritis (RA). Studies show that 0.5–1% of adults worldwide, with a 2.5:1 female-to-male ratio, have RA (Kasper et al., 2015). (3). RA is most common between 30 and 50 (Ceccato F et al., 2006). (4). Around 0.75% of Indian adults have rheumatoid arthritis (RA), which is rising (Malaviya AN et al., 1993) (5). Untreated RA can cause joint degeneration, disability, high comorbidities, and early death (Scott DL et al., 2010, Avina-Zubieta JA et al., 2008, Fatima F et al., 2009) (6,7,8,). Early diagnosis of rheumatoid arthritis (RA) is critical because patients may suffer permanent joint damage soon after the disease begins (Van Gaalen FA et al., 2004) (9), and therapy can be dangerous (El-Gabalawy HS et al., 2002) (10). RA diagnosis can be difficult, and people often have symptoms for years before being diagnosed. Early diagnosis and treatment of rheumatoid arthritis (RA) can improve clinical outcomes and prevent joint deterioration and irreversible disabilities. Finding serum biomarkers that predict early structural disease development is critical. Joint lesions generally appear at this time (Lard LR et al., 2001) (11), therefore early care may reduce them.

Lindqvist et al. (2005), Van der Helm-van Mil et al. (2005), and Vittecoq et al. (2003) found that inflammatory biomarkers like CRP and ESR, as well as autoantibodies like RF and anti-CCP antibodies, were associated with structural damage in rheumatoid arthritis (RA) (12-14). The early phases of

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radiographic disease are difficult to anticipate with these markers. This emphasizes the need for sensitive and specific biomarkers (Forslind K et al., 2004) (15). The secretory abilities of adipose tissue and the growing understanding of adiponectin's biology and functions show that this remarkable protein actively controls physiological and pathological processes like inflammation, metabolism, and immunity.White adipose tissue (WAT) is the body's main energy source, regulating metabolism and energy balance. White adipose tissue (WAT) is now acknowledged as a highly active endocrine organ that produces adipokines. Adipokines are involved in several vital physiological processes (Toussirot É et al., 2007, Mafra D et al., 2008, Almehed K et al., 2008) (16-18). Adipose tissue produces adipokines, however synovium, cartilage, and mononuclear blood cells can also create them. These can be detected in biological fluids (Lago F et al., 2007) (19). Resistin, leptin, and adiponectin regulate energy balance and eating behavior. They also mediate inflammation (Ilg H et al., 2006, Koerner A et al., 2005). Coordinates are (20, 21). Adiponectin, a collagen-like protein, weighs 28-30 kDa. It makes up 0.01% of plasma protein and is mostly produced by fat cells. Adiponectin (Acrp30, AdipoQ, ApM1, GBP28). Adiponectin has many beneficial biological functions (Brochu-Gaudreau K et al., 2010) (22). Rho et al. (2009) (23) found that rheumatoid arthritis patients had significantly higher blood levels of adiponectin, visfatin, CRP, and TNF-α compared to the control group. Insulin, Angiotensin-II, TNF-α, IL-6, IL-1b, and IFN-g affect adiponectin synthesis and release (Chen X et al., 2011) (24).

RA is characterized by long-term inflammation, increased inflammatory mediator production, and endocrine system abnormalities (M Otero et al 2006) (25). Synovial hyperplasia and joint degeneration define rheumatoid arthritis (RA) (Smith et al., 2011) (26). RASFs are thought to cause the chronic and damaging aspect of the disease (Neumann E et al., 2010, Juarez M et al., 202).(27,28) Adiponectin helps rheumatoid synovial fibroblasts produce IL-8 and prostaglandin E2. The compound lowers inflammation induced by TNF-α, inhibits macrophage phagocytosis and TNF-α generation, and induces apoptosis to stop myelomonocytic cell proliferation. Adiponectin increases VEGF and MMPs-1 in fibroblast-like synoviocytes (FLSs), causing joint inflammation and injury (Choi HM et al., 2009) (32). Senolt et al. (2006) and OteroM et al. (2006) found that RA patients have greater blood adiponectin levels than healthy people (33,25). These levels are closely correlated with C-reactive protein, as shown by OteroM et al. (2006) (25). Many studies suggest that adipokines may cause synovial inflammation in rheumatoid arthritis. The role of adipokines in inflammation draws new links between adipose tissues and inflammatory diseases like RA, attracting researchers and practitioners. Joint inflammation must be accurately assessed for sickness evaluation. In addition, acute phase reactants like ESR and CRP grow with sickness activity. Buchbinder et al. (1995), Ranganath et al. (2005), and Wolfe et al. (1997) found CRP to be a better indicator of disease activity and progression than ESR (34-36). Blood serum glycoproteins are acute-phase reactants. After trauma or inflammation, these proteins increase. Acute-phase protein response is roughly proportional to stimulus intensity. These proteins can be monitored serially, like the erythrocyte sedimentation rate (ESR), which largely reflects fibrinogen levels (R S AMOS et al., 1997) (37), to track an inflammatory situation. Rheumatoid arthritis is not the only acute-phase protein reaction. However, comparing it to a simpler sickness progression measure might boost trust in the strategy. This study examines how adiponectin, serum CRP, and ESR levels affect rheumatoid arthritis progression. These factors may indicate illness severity.

MATERIALS AND METHODS

The present study was conducted in the Department of Biochemistry in association with Department of Medicine of the Mahatma Gandhi Memorial Medical College & Maharaja Yashwant Rao Hospital, Indore, Madhya Pradesh, after its approval by the Institutional Research Board (IRB) Committee. The period of study was from 2017 to 2018.

The present study included 50 cases of rheumatoid arthritis aged between 20 to 70 years attending Medicine OPD of Maharaja Yashwant Rao Hospital, Indore and 50 apparently healthy controls matched for age and sex. Informed written consent was taken from all the subjects. Total 100 study subjects were divided into two groups, group A comprising 50 apparently healthy controls and group B comprising 50 patients of rheumatoid arthritis, which is further divided into subgroups on the basis of DAS 28 Score, group B1-remission, group B-2 included mild cases of rheumatoid arthritis, group B-3 of moderate & group B-4 of severe cases of rheumatoid arthritis**.**

Excluded from the study were subjects with

- Past history of other autoimmune disease.
- Spondyloarthropathy
- Diabetes mellitus
- Oncological disease
- Chronic kidney disease
- Chronic liver disease
- Pregnancy
- Alcoholism
- Smoking

Anthropometric details and blood sample were taken from all subjects after taking consent from them or their relative.

Sample collection

After overnight fasting of 8-10 hours, 5 ml of venous blood sample was drawn by venipuncture from a peripheral vein under all aseptic precautions in a

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disposable syringe. The blood was collected in clot activator tube & EDTA tube. Clot activator tube was allowed to stand for 30 minutes at room temperature for the retraction of clot. Then it was centrifuged at 3000 rpm. for 10 minutes to separate the serum. Care was taken to avoid hemolysis of the sample. Routine hematological (ESR) (from EDTA tube) and biochemical tests (lipid profile, uric acid & CRP) (from serum) biochemistry fully automated analyzer-Biosystem) were performed on the fresh samples on the same day. Then remaining serum sample was stored at -20°C for further analysis. Sample were only exposed to a single freeze/thaw cycle to minimize the risk of contamination of the samples. The stored serum samples were analyzed for Adiponectin (ELISA).

RESULTS

Table 1 Distribution of patients according to age and sex in both the groups

Age	Control Group		Rheumatoid Arthritis Group	
	No.	%	No.	%
20-30 years	4	8.0	8	16.0
31-40 years	17	34.0	14	28.0
41-50 years	16	32.0	11	22.0
51-60 years	13	26.0	12	24.0
61-70 years	0	0.0	5	10.0
Mean age (±SD) years	42.26 ± 8.09		44.74 ± 13.10	
Sex				
Female	40	80.0	43	86.0
Male	10	20.0	7	14.0

The distribution of patients according to age and sex in both the control and rheumatoid arthritis groups shows a varied age range. In the control group, the highest percentage of patients (34%) are aged 31-40 years, followed by 32% aged 41-50 years, 26% aged 51-60 years, and 8% aged 20-30 years. No patients in the control group are aged 61-70 years. In contrast, the rheumatoid arthritis group has the highest percentage of patients (28%) aged 31-40 years, followed by 24% aged 51-60 years, 22% aged 41-50 years, 16% aged 20-30 years, and 10% aged 61-70 years. The mean age of the control group is 42.26 years (±8.09), while the rheumatoid arthritis group has a mean age of 44.74 years (±13.10). The 't' value of -1.139 with a degree of freedom of 98 and a p-value of 0.258 indicates that the difference in mean ages between the groups is not statistically significant. Regarding sex distribution, the control group comprises 80% females and 20% males, while the rheumatoid arthritis group consists of 86% females and 14% males, showing a higher prevalence of females in both groups.

Table 2 Distribution of patients according to BMI and DAS-28 grading in both the groups

BMI	Control Group		Rheumatoid Arthritis Group	
	No.	%	No.	%
Normal weight	31	62.0	32	64.0
Overweight	16	32.0	13	26.0
Obese	3	6.0	5	10.0
Mean BMI (±SD) kg/m^2	24.59 ± 3.06		24.43 ± 4.35	
't' value	0.216, df=98			
P value	0.829, NS			
BMI				
Remission (<2.6)	0	0.0	2	4.0
Low disease activity (≥2.6-≤3.2)	0	0.0	2	4.0
Moderate disease activity (>3.2-≤5.1)	0	0.0	30	60.0
High disease activity (>5.1)	0	0.0	16	32.0
Total	50	100.0	50	100.0

The table compares the distribution of patients according to BMI and DAS-28 grading between a control group and a rheumatoid arthritis group. In the control group, 62.0% of patients are of normal weight, 32.0% are overweight, and 6.0% are obese, with a mean BMI of 24.59 ± 3.06 kg/m². In the rheumatoid arthritis group, 64.0% of patients are of normal weight, 26.0% are overweight, and 10.0% are obese, with a mean BMI of 24.43 ± 4.35 kg/m². The 't' value for BMI between groups is 0.216 with a p-value of 0.829, indicating no significant difference. Regarding disease activity in the rheumatoid arthritis group, 4.0% are in remission (DAS-28 <2.6), 4.0% have low disease activity (DAS-28 >2.6-<3.2), 60.0% have moderate disease activity (DAS-28 >3.2-<5.1), and 32.0% have high disease activity (DAS-28 >5.1). No

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control group patients are classified under the DAS-28 grading.

Table 3 Comparison of inflammatoryparameters between the two groups				
Parameter	**Control group (Mean±SD)**	**Rheumatoid Arthritis Group (Mean±SD)**	**'t' value**	**P value**
ESR	15.70 ± 5.40	21.26 ± 8.88	-3.784, df=98	0.000*
CRP	11.09 ± 7.95	21.56 ± 25.46	-2.776, df=98	0.007*

The comparison of inflammatory parameters between the control group and the rheumatoid arthritis group indicates significant differences in both ESR (Erythrocyte Sedimentation Rate) and CRP (C-Reactive Protein) levels. The mean ESR in the rheumatoid arthritis group (21.26 ± 8.88 mm/hr) is notably higher than in the control group (15.70 ± 5.40 mm/hr), with a 't' value of -3.784 and a p-value of 0.000, indicating a statistically significant difference. Similarly, the CRP levels are significantly elevated in the rheumatoid arthritis group (21.56 ± 25.46 mg/L) compared to the control group (11.09 ± 7.95 mg/L), with a 't' value of -2.776 and a p-value of 0.007. These findings suggest higher levels of inflammation in patients with rheumatoid arthritis compared to the control group.

Table 4 Comparison of mean adiponectin level between the two groups				
Parameter	**Control group (Mean±SD)**	**Rheumatoid Arthritis Group (Mean±SD)**	**'t' value**	**P value**
Adiponectin Level (µg/ml)	17.08 ± 8.69	30.13 ± 13.04	-5.892, df=98	0.000*

Unpaired 't' test applied. P value < 0.05 was taken as statistically significant

The comparison of mean adiponectin levels between the control group and the rheumatoid arthritis group reveals a significant difference. The rheumatoid arthritis group has a markedly higher mean adiponectin level (30.13 ± 13.04 µg/ml) compared to the control group (17.08 ± 8.69 µg/ml). This difference is statistically significant, with a 't' value of -5.892 and a p-value of 0.000. These results suggest that individuals with rheumatoid arthritis have significantly elevated adiponectin levels compared to healthy controls.

Table 5 Comparison of mean adiponectin level in the rheumatoid arthritis group in relation to DAS-28 grading				
DAS-28 Grading	**No.**	**Mean ± SD**	**F value**	**P value**
Remission	2	35.2 ± 14.4	5.75	0.002*
Low disease activity	2	13.51 ± 1.20		
Moderate disease activity	30	26.25 ± 11.44		
High disease activity	16	38.86 ± 11.72		

The comparison of mean adiponectin levels in the rheumatoid arthritis group in relation to DAS-28 grading indicates a significant variation across different disease activity levels. Patients in remission (n=2) have a mean adiponectin level of 35.2 ± 14.4 µg/ml, while those with low disease activity (n=2) show a much lower mean level of 13.51 ± 1.20 µg/ml. Patients with moderate disease activity (n=30) have a mean adiponectin level of 26.25 ± 11.44 µg/ml, and those with high disease activity (n=16) exhibit the highest mean level of 38.86 ± 11.72 µg/ml. The F value of 5.75 and a p-value of 0.002 indicate that these differences are statistically significant, highlighting a correlation between higher adiponectin levels and increased disease activity in rheumatoid arthritis patients.

Table 6 Comparison between different sub-group of rheumatoid arthritis			
Pairs	**'t' value**	**P value**	**Interpretation**
Low disease activity – remission	-1.89	0.247	Not significant
Moderate disease activity – remission	-1.07	0.712	Not significant
High disease activity – remission	0.43	0.974	Not significant
Moderate disease activity – low disease activity	1.52	0.434	Not significant
High disease activity – low disease activity	2.94	0.025*	Significant
High disease activity – moderate disease activity	3.55	0.005*	Significant

Post-hoc Tukey test was applied.

The comparison between different sub-groups of rheumatoid arthritis reveals significant and non-significant differences in disease activity levels. Specifically, the comparisons between low disease

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activity and remission (t = -1.89, p = 0.247), moderate disease activity and remission (t = -1.07, p = 0.712), and high disease activity and remission (t = 0.43, p = 0.974) are all not significant. Similarly, the comparison between moderate disease activity and low disease activity (t = 1.52, p = 0.434) is also not significant. However, the comparisons between high disease activity and low disease activity (t = 2.94, p = 0.025), as well as high disease activity and moderate disease activity (t = 3.55, p = 0.005), are significant, indicating a notable difference in disease activity between these sub-groups.

Table 7 Correlation between adiponectin level and CRP in the rheumatoid arthritis group

Pair	'r' value	P value
Adiponectin – CRP	0.143	0.320, NS

Pearson Coefficient of Correlation test applied. P value < 0.05 was taken as statistically significant

In the rheumatoid arthritis group, the correlation analysis between adiponectin levels and C-reactive protein (CRP) shows a weak positive correlation with an 'r' value of 0.143. However, this correlation is not statistically significant, with a p-value of 0.320. This indicates that there is no significant association between adiponectin levels and CRP in patients with rheumatoid arthritis in this study.

DISCUSSION

Adiponectin is regarded as the most captivating and auspicious biologically active substance that is secreted by fat cells. Adipokines have a significant role in inflammation, establishing new connections between adipose tissues, adipokines, and illnesses associated to inflammation, such as rheumatoid arthritis (RA). Rheumatoid arthritis (RA) is a persistent inflammatory disorder that results in pain, tiredness, and joint deterioration. Timely diagnosis and prompt management can effectively avert additional complications. Hence, the objective of our study was to evaluate the adiponectin levels in patients with rheumatoid arthritis (RA) and determine the severity of the condition.The study named "Adiponectin as a marker for evaluating the severity of disease in Rheumatoid Arthritis: a case-control study conducted in a tertiary care hospital in Central India" was conducted from 2016 to 2018 at the Department of Biochemistry and Department of Medicine, MGMMC & MYH, Indore. The current study comprised 50 individuals diagnosed with rheumatoid arthritis, ranging in age from 20 to 70 years, who were attending the Medicine OPD at Maharaja Yashwant Rao Hospital in Indore. Additionally, 50 apparently healthy controls were selected, matched for both age and sex.

The average age of the patients in our study was 44.74 ± 13.10 years. In the study conducted by Mona Abo-Ragab et al (2012) [38]the average age of patients was 42.60±8.85 years. Similarly, in the study conducted by Otero, Lago, Gomez, et al (2006) the average age was 46.1 ± 14.1 years.The majority of patients in our study were between the ages of 31 and 40, with a higher number of females (43) compared to males (7), resulting in a female to male ratio of 43:7. This finding is consistent with a study conducted by KIM et al (2014) (39), where the female to male ratio was 32:8. The study conducted by Sharma et al. (2015) (40) found that the highest occurrence of rheumatoid arthritis patients was in the age category of 41-50 years, with an average age of 48±12.2. Additionally, the female to male ratio was 2.03:1. The study conducted by Ahmad M et.al (2012) (41) found that the highest incidence of rheumatoid arthritis (RA) was reported in individuals aged 51-65 years, accounting for 58.69% of cases. Additionally, the female to male ratio was 6.5:1.In our study on rheumatoid arthritis, 32 patients (64.0%) had a normal weight BMI, 13 patients (26.0%) were overweight, and 5 patients (10.0%) were obese. In Sergey P. Oranskiy's (2012) [42]study, there were 39 patients with a normal BMI, 26 patients with obesity and overweight BMI, and 18 patients with underweight BMI.In our study, out of the 50 cases of rheumatoid arthritis (RA), 2 patients (4.0%) experienced remission, 2 patients (4.0%) had low disease activity, 30 patients (60.0%) had moderate disease activity, and 16 patients (32.0%) had high disease activity based on the DAS 28 Score. This aligns with the findings of a study conducted by Mona Abo-Ragab et al (2012) (38). Out of the total of 50 patients, 18 (36%) had mild activity, 15 (30%) had moderate activity, and 17 (34%) had severe activity of rheumatoid arthritis (RA) as determined by the Simplified Disease Activity Index (SDAI).

The results of our investigation showed that the average ± standard deviation of plasma adiponectin levels in patients with rheumatoid arthritis (RA) was 30.13 ± 13.04 µg/ml, whereas in healthy individuals it was 17.08 ± 8.69 µg/ml. The findings of this investigation demonstrated a substantial and statistically significant rise in plasma adiponectin levels across all patients with rheumatoid arthritis (RA) as compared to the control group (P=0.000). This finding is consistent with the findings of Ozygen et al (2010) (45). The researchers discovered that the levels of serum adiponectin were substantially greater in the group with rheumatoid arthritis compared to the control group (P<0.05). The results of this study were in agreement with the findings of Otero et al (2006) (25) about adiponectin levels (mean 13.56 (SEM 2.1) mg/ml in the study group compared to mean 7.6 (SEM 0.7) mg/ml in the control group; p=0.0375), as well as with the findings of Senolt et al (2006) (32). In

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a study conducted by Takumi Yoshino et. al (2011), it was found that female RA patients had significantly higher serum adiponectin concentrations (10.1 µg/mL) compared to normal female control subjects (3.6 µg/mL), with a p-value of less than 0.001. However, there was no significant difference in adiponectin concentration observed in males (RA males: median 2.6 µg/mL; control males: median 2.3 µg/mL, p=0.203).The elevated levels of adiponectin in individuals with rheumatoid arthritis indicate a compensation mechanism in response to an imbalance in catabolic or anabolic processes. This mechanism is believed to counteract the appetite-suppressing and widely recognized pro-inflammatory effects of leptin. Adiponectin may play a significant role in regulating the inflammatory response in patients with rheumatoid arthritis. It is hypothesized that adiponectin can modulate the inflammatory response by preventing the expression of adhesion molecules on endothelial cells, suppressing the function of macrophages, and inhibiting NFkB signaling. This hypothesis was discussed by Fantuzzi et al (2005) (46). Adiponectin is thought to possess anti-inflammatory qualities and can potentially counteract the pro-inflammatory effects of TNF-α, a cytokine that promotes inflammation. This interaction may impact the generation of IL-6 and CRP in individuals with rheumatoid arthritis. The potential anti-inflammatory function of adiponectin in this situation deserves further investigation. In their prospective study, Giles et. al (2009) (47) discovered that out of the adipokines examined, only adiponectin showed a significant correlation with radiographic advancement. Furthermore, the average levels of adiponectin were found to have a stronger association than the initial values.

Our investigation revealed that the RA group had significantly higher levels of CRP (21.56 ± 25.46) compared to the control group (11.09 ± 7.95). According to a study conducted by Patrick H. Dessein in 2013,[48] the levels of serum C-reactive protein were found to be comparable between participants with rheumatoid arthritis (RA) and those without RA. Specifically, the average concentration was 7.0 ± 3.1 in RA patients and 6.7 ± 3.1 in non-RA individuals. The p-value associated with this comparison was 0.7. According to Amos et al. (1977) (36), their study found that patients with rheumatoid arthritis (RA) who have high levels of erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) tend to have more pronounced and advanced radiological abnormalities, such as bony erosions. This is true regardless of their rheumatoid factor (RF) status.

In addition, we conducted an analysis to examine the relationship between adiponectin and CRP levels in patients with rheumatoid arthritis. Our findings revealed a slight positive correlation (r=0.143, p=0.320) between adiponectin levels and CRP, although this correlation was not statistically significant. However, a study by Mona Abo-Ragab (2012) (38) demonstrated a significant positive correlation between adiponectin and markers of inflammation such as ESR, CRP, and RF. Fantuzzi G et al (49) found a favorable correlation between adiponectin levels and inflammatory markers. Otero et al (2006) (25) discovered a positive association (r=0.59, p=0.031) between adiponectin and CRP levels in patients with rheumatoid arthritis.According to a study conducted by Fantuzzi G et. al (49), adiponectin has been found to directly decrease the release of pro-inflammatory cytokines and enhance the synthesis of anti-inflammatory cytokines by activated inflammatory cells. Adiponectin production is suppressed by pro-inflammatory cytokines such as TNF-α and IL-6. As a result, there is a negative relationship between the concentration of adiponectin and indicators of inflammation, such as CRP.The study conducted by ŠenoltL et. al. (2006) (32) did not find any link between adiponectin and systemic inflammatory indicators such as CRP and ESR. However, the study conducted by WisłowskaM et. al (2007) (50) did find a positive correlation between adiponectin and CRP.

Our investigation found that the serum adiponectin level increased as the disease severity increased. In the subgroup with mild disease activity, the concentration of adiponectin was lower compared to the subgroup with high disease activity. This is consistent with the findings of Mona Abo-Ragab (2012) (38) and Ebina et al (2009) (51). According to their findings, there was a positive correlation between serum adiponectin concentrations and the severity of rheumatoid arthritis (RA), as assessed by the number of damaged joints shown on plain radiographs.In our study, we found that the average concentration of adiponectin was higher in the remission subgroup of the RA group compared to the mild and moderate subgroups. This could be because our study was of short duration, and it takes a longer time for the concentration of adiponectin to change. This finding is consistent with a study by Ebina K et. al (52), which also observed higher levels of adiponectin in erosive RA compared to mild RA. Additionally, the average levels of adiponectin did not significantly change over a follow-up period of 2.5 years.Theaforementioned results contradict the findings of Targonska et al (2015) (53), who observed a negative association between adiponectin levels and the quantity of painful and swollen joints in individuals with chronic RA. Boz˙enaTargon´ska-Stepniak et al (2009) (39) found that there were inverse relationships between adiponectin levels and symptoms of joint disease activity, such as the number of sore and swollen joints and the DAS28 value. These findings suggest that adiponectin has a role in reducing inflammation.

CONCLUSION

Based on the existing literature and the results of this investigation, which support previous findings, it can

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be concluded that patients with rheumatoid arthritis had higher levels of adiponectinand CRP compared to a control group of healthy individuals. Patients exhibiting elevated disease activity demonstrate a correspondingly heightened concentration of adiponectin. Our investigation revealed a modestly positive connection between adiponectin and CRP. Our work investigates the potential correlation between adiponectin and CRP as indicators for early identification of joint damage in rheumatoid arthritis, as both are engaged in the inflammatory process.,In patients with rheumatoid arthritis (RA). Regular follow-up should be carried out to assess the risk of joint damage and extra-articular symptoms, as well as to address any potential issues that may arise during treatment.

Limitation of study-Our study was subject to limitations, including time constraints and a limited sample size. Hence, it is imperative to conduct additional community-based studies with a substantial sample size in order to evaluate the impact of adiponectin on disease activity in Rheumatoid arthritis and its correlation with CRP.

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