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Original Research Article

TO FIND CORRELATION BETWEEN ADIPONECTIN AND CRP LEVELS IN PATIENTS OF RHEUMATOID ARTHRITIS.

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Abstract:
Background & Method: The aim of the study is find correlation between adiponectin and CRP levels in patients of rheumatoid arthritis. 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.
Result: The correlation between adiponectin and CRP level in the rheumatoid arthritis group. There was a weak positive, statistically non-significant correlation seen between adiponectin level and CRP level in the rheumatoid arthritis group ($p>0.05$), showing that there is no significant correlation seen between adiponectin level and CRP level in the patients with rheumatoid arthritis.
Conclusion: It can be concluded that patients of rheumatoid arthritis have raised level of adiponectin as compared to normal healthy control group. Patients of RA also have higher concentration of CRP. Patients with high disease activity also have higher concentration of adiponectin and have increased radiological damage to joints. In our study we observed that there is a weak positive correlation between adiponectin and CRP. Since adiponectin & CRP both are involved in inflammatory process, our study explores the possible association of these parameters as marker for early detection of joint destruction in RA.

Keywords: correlation, adiponectin, CRP & rheumatoid arthritis.

Study Designed: Observational Study.

1. Introduction

Rheumatoid arthritis (RA) is an autoimmune inflammatory disease characterized by joint damage and disability. Genetics, female gender, and smoking are the risk factors of RA. Recently, studies showed that obesity also play a role in RA.
Fatty tissue cans secret many proteins called adipokines. Adiponectin (APN), a collagen-like protein called Acrp30, adipoQ, ApM1, and GBP28, is a kind of adipokines. APN exists as globular APN and fulllength APN (f-APN). f-APN has 3 main isoforms, including low-molecular-weight APN, middle-molecular-weight APN, and high-molecular-weight APN

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(HMW-APN). Furthermore, in both healthy populations and patients with RA, APN is negatively correlated with body mass index (BMI). In metabolic diseases, APN has both antiatherogenic and anti-inflammatory functions and can increase insulin sensitivity, and the circulating concentration of HMW-APN is mostly related to type 2 diabetes mellitus and atherosclerosis. However, in autoimmune diseases, the effect of APN has not been clearly demonstrated.

In previous studies, APN was found to be controversial influences on inflammation in different studies. Some scholars thought that in patients with RA, APN can aggravate inflammation levels and the degree of joint damage, and it has a positive correlation with C-reactive protein (CRP) levels as well as disease activity score, erythrocyte sedimentation rate (ESR) and rheumatoid factor (RF) levels. However, in collagen-induced arthritis mice, APN can decrease the severity of arthritis and inflammatory cytokine levels such as tumor necrosis factor-a (TNF-a), interleukin-1b (IL-b) and matrix metalloproteinase-3 (MMP-3) in joint tissues. In addition, in different in vitro experiments with fibroblast-like synoviocytes (FLSs), chondrocytes or monocytes, APN has both proinflammatory and anti-inflammatory functions.

2. Material & Method

The present study was conducted in the Department of Biochemistry Index Medical College Hospital & Research Centre, Indore, M.P., for 01 Year. The present study included 50 cases of rheumatoid arthritis aged between 20 to 70 years 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 criteria:

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

3. Results

Table No. 1: Gender Distribution

Gender	Control		Rheumatoid Arthritis	
	No.	%	No.	%
Female	40	80	43	86
Male	10	20	07	14

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Table No. 2: BMI Distribution

BMI	Control		Rheumatoid Arthritis		P Value
	No.	%	No.	%	
Normal Weight	31	62	32	64	0.639
Over Weight	16	32	13	26	
Obese	03	06	05	10	
Total	50	100	50	100	
Mean	24.59±3.06		24.43±4.35		

Table No. 3: Distribution of patients to DAS-28 grading in RA

BMI	Control		Rheumatoid Arthritis	
	No.	%	No.	%
Remission (<2.6)	0	0	02	04
Low disease activity (≥2.6-≤3.2)	0	0	02	04
Moderate disease activity (>3.2-≤5.1)	0	0	30	60
High disease activity (>5.1)	0	0	16	32
Total	0	0	50	100

Table No. 4: Correlation between adiponectin level and CRP in RA

Pair	r value	p value
Adiponectin level and CRP	0.244	0.421

The correlation between adiponectin and CRP level in the rheumatoid arthritis group. There was a weak positive, statistically non-significant correlation seen between adiponectin level and CRP level in the rheumatoid arthritis group ($p>0.05$), showing that there is no significant correlation seen between adiponectin level and CRP level in the patients with rheumatoid arthritis.

4. Discussion

In our study, we focused on one of the adipokines known as APN and found that the levels of total APN and HMWAPN were lower in patients with RA than those in the healthy population. Moreover, we also found that in the serum of patients with RA, total APN and HMW-APN were both positively associated with CRP levels. HMW-APN levels were significantly higher in patients with high disease activities, both total APN and HMW-APN levels were significantly higher in patients with severe synovial thickening.

The most likely reason was that APN had anti-inflammatory functions similar to the results in some other studies. Thus, in the patients with higher disease activities and inflammation levels, APN may be protectively elevated by the negative feedback regulation. Moreover, APN was found to be negatively correlated with BMI in many studies. It has been found that obesity can decrease APN receptor expression levels and thereby reduce the sensitivity of APN. Besides, both in RA and obesity populations, there are higher inflammatory cytokine

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levels in the in vivo microenvironment than levels in healthy subjects. In previous studies, we also found that APN can be suppressed by inflammatory cytokines such as TNF-a and IL-6 in CD4+ T cells as well as 3T3-L1 adipocytes. In addition, the suppression of APN might be partly mediated through p44/42 MAP kinase and the suppression may cause the lower levels of APN in patients with RA compared with healthy donors.
However, there were still some previous studies found that APN can exacerbate inflammation. In RA synovial fibroblasts, APN can also stimulate the production of some inflammatory cytokines, such as IL-6, IL-8, IL-11, monocyte chemotactic protein-1, and prostaglandin E2 (PGE-2). To verify the biological function of APN, more in vivo and in vitro studies need to be carried out initially.

5. Conclusion

It can be concluded that patients of rheumatoid arthritis have raised level of adiponectin as compared to normal healthy control group. Patients of RA also have higher concentration of CRP. Patients with high disease activity also have higher concentration of adiponectin and have increased radiological damage to joints. In our study we observed that there is a weak positive correlation between adiponectin and CRP. Since adiponectin & CRP both are involved in inflammatory process, our study explores the possible association of these parameters as marker for early detection of joint destruction in RA.

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