ROLE OF ADIPOSE TISSUE IN RHEUMATOID ARTHRITIS

Student: **Khakimova Omina**

Scientific advisor: **Zaraqov Nabijon**

Andijan State Medical Institute, Uzbekistan

Abstract: The study included 47 patients with early RA, meeting the ACR/EULAR criteria (2023), who did not receive basic anti-inflammatory drugs and glucocorticoids. The average age of patients was 57 [47, 62] years, disease duration - 7 [4, 8] months, median DAS28 - 5.9 [5.3, 6.9] points. The control group consisted of 30 healthy donors without rheumatic diseases, comparable in age to the main group. In both groups, the presence of obesity was assessed according to the criteria of metabolic syndrome, NCEP/ATPIII, GFCF, WHO, and the concentrations of leptin and adiponectin (by ELISA) and their ratio (L/A) were determined.

Keywords: Rheumatoid arthritis, metabolic syndrome, obesity, insulin resistance, adiponectin, leptin.

INTRODUCTION

One of the most common autoimmune rheumatic diseases, rheumatoid arthritis (RA), is characterized by chronic erosive arthritis (synovitis) and systemic inflammatory damage to internal organs [1]. Along with the study of pathogenesis, the search for new possibilities for diagnosis and pharmacotherapy of RA, an increasing number of studies are currently devoted to the study of cardiovascular diseases (CVD), the risk of development of which in RA is similar to that in diabetes mellitus [2].

MATERIALS AND METHODS

In recent years, among the classical components of MS, obesity has attracted much attention from rheumatologists. There are several basic prerequisites for this. Firstly, the idea of a typical RA patient as a person with underweight has changed. According to the Norfolk Register [3], among patients with early RA (damage to two or more joints for 4 weeks or more), 25% of patients were already obese. Secondly, the concept of rheumatoid cachexia appeared - a condition in which a decrease in body weight is caused by hypo- and atrophy of muscle mass against the background of a stable or even increased fat content [4]. In this case, the body mass index (BMI) may not change or deviate slightly from the norm in any direction. Finally, adipose tissue, previously perceived only as an energy depot and a shock absorber for internal organs, began to be positioned as an important endocrine organ capable of producing not only a huge number of different pro- and anti-inflammatory cytokines, but also more than 50 hormonal factors, also called adipocytokines (ACC) [5]. The influence of two of them - adiponectin and leptin - has been relatively well studied in patients with cardiovascular diseases, type 2 diabetes mellitus and obesity, but information about these ACCs in RA is extremely limited and contradictory.

The study included 47 patients (35 women and 12 men) with early RA.

RESULTS AND DISCUSSION

Patients with RA did not differ from healthy donors in terms of the average BMI value (26.4 [22.8; 31.5] and 27.9 [25; 30.3] kg/m2, p = 0.6); in terms of the frequency of excess - body

weight (31.9 and 46.7%, p = 0.2) and obesity (29.8 and 30%, p = 0.9), determined by this anthropometric indicator. At the same time, WC and WC/WC ratio were greater in patients with RA than in healthy donors (102 [97; 111] and 88.6 [83; 96] cm, p = 0.003; 0.9 [0.8 ; 1.0] and 0.84 [0.8; 0.9], p = 0.04, respectively.

Abdominal obesity according to NCEP/ATPIII criteria was diagnosed in 30 (63.8%) patients with RA and only in 12 (40%) healthy donors (p = 0.04), according to WHO criteria - in 31 (65.9%) and 12 (40%), respectively (p = 0.02). According to the GFCF criteria, no statistically significant differences in the incidence of obesity were detected between the groups (p = 0.9).

When assessing the effect of obesity on RA activity, it turned out that the WC indicator correlated with the ESR (r = 0.36, p = 0.02) and CRP (r = 0.31, p = 0.04) indicators.

An increase in BMI was associated not only with an increase in laboratory parameters: ESR (r = 0.46, p = 0.001), CRP (r = 0.38, p = 0.007), but also the DAS28 index (r = 0.40, p = 0.005).

The level of adiponectin in patients with RA was higher (p = 0.04), and the level of leptin and the L/A ratio were lower (p = 0.02 and 0.003, respectively) than in healthy donors. In RA, there was a dependence of leptin concentration and L/A ratio on BMI (r = 0.60, p = 0.0001 and r = 0.56, p = 0.0001) and WC (r = 0.51, p = 0.0003 and r = 0.44, p = 0.002, respectively). Anthropometric parameters did not affect the level of adiponectin. There was a direct correlation of ESR with leptin concentration (r = 0.37, p = 0.01), L/A ratio (r = 0.4, p = 0.005), and an inverse correlation with the level of adiponectin (r = -0.30, p = 0.04).

Our results correspond to a situation opposite to the classical picture of obesity: in patients with RA, the concentration of adiponectin was higher and leptin was lower than in healthy donors. At the same time, anthropometric indicators characterizing obesity did not affect the level of adiponectin. At the same time, as in the general population, BMI and WC indicators correlated with leptin and insulin resistance, represented by the L/A ratio.

Insulin resistance is a decrease in the response of usually insulin-sensitive tissues (muscle, adipose tissue, hepatocytes) to insulin at sufficient concentrations, leading to chronic compensatory basal hyperinsulinemia.

On the one hand, the present study has a small sample size. At the same time, all patients with rheumatoid arthritis had high disease activity and did not take glucocorticoids and basic anti-inflammatory drugs, which excludes the influence of these drugs on the data obtained.

CONCLUSION

Further study of this problem requires prospective observations, primarily regarding the association of indicators of insulin resistance, obesity with the progression of rheumatoid arthritis, the relationship with the destruction and functional disorders of the joints, as well as with the influence of antirheumatic therapy.

REFERENCES

1. Nasonov E.L. ed. Rheumatology: Clinical guidelines. 2nd ed. M.: GEOTAR-Media; 2010.

2. van Halm V.P., Peters M.J., Voskuyl A.E., Boers M., Lems W.F., Visser M. et al. Rheumatoid arthritis versus diabetes as a risk factor for cardiovascular disease: a cross-sectional study, the CARRE Investigation. Ann. Rheum. Dis. 2019; 68: 1395–400.

3. Humphreys J.H., Verstappen S.M., Mirjafari H., Bunn D., Lunt M., Bruce I.T. et al. Association of morbid obesity with disability in early inflammatory polyarthritis: Results from the Norfolk Arthritis Register. Arthr. Care Res. 2013; 65 (1): 122–6.

4. Anikin S.G., Toroptsova N.V. Modern view on the problem of cachexia and sarcopenia in rheumatoid arthritis. Scientific and practical rheumatology. 2012; 53 (4): 73-9.

5. Galic S., Oakhill J.S., Steinberg G.R. Adipose tissue as an endocrine organ. Mol. Cell. Endocrinol. 2010; 316 (2): 129–39.