Methods: 50 consecutive Spanish patients undergoing periodic treatment with TCZ who fulfilled the 2010 classification criteria for RA [5] were recruited. Adiponectin serum levels were assessed in samples obtained immediately prior to (pre-infusion) and 60 minutes after the end of a TCZ intravenous infusion (post-infusion) by a commercial Enzyme-Linked ImmunoSorbent Assay (ELISA) kit.

Results: Similar serum levels of adiponectin were found following the TCZ infusion (mean ± standard deviation: 23.01 ± 18.58 µg/mL versus 22.35 ± 17.84 µg/mL, pre- and post-infusion, respectively, p=0.69). Additionally, there was a negative correlation between adiponectin basal levels and body mass index (r=-0.45, p<0.01), insulin resistance index (r=-0.50, p<0.01), insulinogenic index (r=-0.32, p=0.03) and C-peptide levels (r=-0.32, p=0.03) of RA patients, whereas a strong positive correlation was observed with HDL-cholesterol levels (r=0.51, p<0.01). Moreover, basal levels of adiponectin were significantly lower in obese patients when compared to those with normal weight (p=0.03).

Conclusion: Our study suggests that anti-IL-6 therapy has no short-term effect on adiponectin serum levels in patients with RA. Furthermore, our results support that low adiponectin levels are associated with MetS features and, therefore, with CV disease in RA.

REFERENCES:

Methods: This cohort study was based on data from the Swedish Rheumatology Quality register (SRQ). All patients with RA who started treatment with abatacept between 2006 and 2017 were included. Clinical data from the SRQ included demographics, disease characteristics and antirheumatic treatment. Disease activity was measured according to DAS28-ESR (Disease Activity Score 28 of joints based on erythrocyte sedimentation rate) at inclusion and at follow-up visits at 6 and 12 months from start of treatment with abatacept. Baseline predictors of change in disease activity were investigated using linear regression models bivariately and adjusted for baseline values of DAS28. Covariates with a p-value of <0.1 were retained for the final multivariate model. In case of covariates with major collinearity, the one with the stronger association with change of DAS28 was selected.

Results: In total, 2176 patients, 872 had data on change in DAS28 at 12 months. Among these, most patients were women (79.6%) and the mean age at start of abatacept was 58.4 years (SD 13.6). The majority of patients had established RA, with a mean disease duration of 13.5 years (SD 11.1). Most patients had severe, active disease, with substantial pain and disability, despite extensive treatment. The mean number of bDMARDs that a patient had been exposed to was 1.90 (SD 1.32), DAS28 decreased significantly over the first year (mean 1.22: 95 % CI 1.12, 1.32). The greatest decrease in DAS28 (mean 1.09) occurred during the first 6 months from start of abatacept. The multivariate regression model identified male sex and limited previous bDMARD exposure as independent predictors of change in DAS28 at 12 months from start of abatacept – adjusted for baseline DAS28, RA duration and current treatment with methotrexate or prednisolone (Table 1).

Conclusion: In this national register study, male sex and limited previous exposure to bDMARDs were independent predictors of reduction of disease activity one year after start of treatment with abatacept for RA, possibly reflecting a better prognosis overall in patients.

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