AB0197 INCREASED CIRCULATING ADIPONECTIN IS AN INDEPENDENT DISEASE ACTIVITY MARKER IN PATIENTS WITH RHEUMATOID ARTHRITIS: A CROSS-SECTIONAL STUDY USING THE KURAMA DATABASE

M. Katsushima1, M. Hashimoto2, M. Shirakashi1, T. Yoshida3, W. Yamamoto4, K. Murakami1, K. Murata5, K. Nishitani6, M. Tanaka1, H. Ito7, S. Matsuda8
1Kyoto University, Department of Rheumatology and Clinical Immunology, Kyoto, Japan; 2Kyoto University, Department of Advanced Medicine for Rheumatic Diseases, Kyoto, Japan; 3Kyoto Prefectoral University of Medicine, Department of Nursing, Kyoto, Japan; 4Kurashiki Sweet Hospital, Department of Health Information Management, Kurashiki, Japan; 5Kyoto University, Department of Orthopedic Surgery, Kyoto, Japan

Background: Adiponectin is a major adipokine with pleiotropic effects on inflammatory conditions including rheumatoid arthritis (RA). Adiponectin generally has anti-atherogenic effects, and its serum level inversely correlates with body mass index (BMI) and visceral fat area (VFA). On the other hand, several studies have indicated a deleterious role of adiponectin in RA progression [1]. Recently, both low BMI and increased serum adiponectin have been reported as poor prognostic factors of RA [2, 3]. However, large-scale surveys have not been done focusing on both BMI and serum adiponectin, and it is unclear which factor provides further contribution to RA disease activity. In addition, the effects of biological disease-modifying antirheumatic drugs (bDMARDs) and Janus kinase (JAK) inhibitors on serum adiponectin are largely unknown.

Objectives: To clarify the relationship among serum adiponectin, body composition, current disease activity and therapeutic agents of RA.

Methods: We conducted a cross-sectional study in RA patients under treatment with agents including bDMARDs and JAK inhibitors. A total of 351 subjects from the Kyoto University RA Management Alliance cohort (KURAMA) were enrolled. We classified the participants into five body composition groups (overweight with or without visceral adiposity, normal with or without visceral adiposity, and underweight) according to the cut-off points for obesity and visceral fat used in Japan: BMI, 18.5-22.9 kg/m² for underweight and 23.0-24.9 kg/m² for overweight and 25.0-29.9 kg/m² for obesity, and VFA, 100 cm² for visceral adiposity. Differences of continuous variables among the five groups were assessed by the Steel-Dwass test or one-way analysis of variance (ANOVA). We adopted a multiple standardized linear regression model to analyze effects of serum adiponectin level on DAS28-ESR.

Results: Serum adiponectin levels (20.9±12.5 vs. 14.7±8.4 µg/ml, p = 0.001) and DAS28-ESR (3.04±1.0 vs. 2.63±0.9, p = 0.017) in the underweight group were significantly higher than those in the others. In multiple regression analysis, serum adiponectin level, but not BMI, was positively correlated with DAS28-ESR (estimate = 0.017, p = 0.0258). Subanalysis also showed that the use of bDMARD or JAK inhibitor did not have an obvious influence on circulating adiponectin.

Conclusion: In the multiple regression analysis we revealed a positive and independent correlation between serum adiponectin and DAS28-ESR in Japanese RA patients. Thus, serum adiponectin is an potential marker reflecting high disease activity of RA regardless of current medications.

References: