as possible within 6 months[1], but many patients have taken oral glucocorticoid for the long term in daily clinical practice. The frequency of use of glucocorticoid has gradually declined, and there are several reports on discontinuation of glucocorticoid due to the initiation of bDMARD[2, 3]. However, there is no report showing the relation between discontinuation of glucocorticoid and MTX dose.

Objectives: The aim of this study is to examine association of methotrexate (MTX) dose with discontinuation of glucocorticoid after one year since initiation of biological DMARD (bDMARD) as 1st bDMARD.

Methods: We established the large observational cohort, the Nagoya University orthopedic facility multicenter study (TBCR), and a total of 3119 patients used biological DMARD. 564 patients who used glucocorticoid and MTX when bDMARD was initiated as 1st bDMARD were enrolled. In the first study, we examined predictive factors of discontinuation of glucocorticoid after one year since initiation of bDMARD by using multivariate analysis in the two groups, which patients continued to use glucocorticoid and discontinued to use glucocorticoid. In the second study, we adjusted the background at the time of initiation of bDMARD by using propensity score matching (PS) in the two groups, MTX≤8mg (L group) and MTX>8mg (H group).

Results: 400 patients continued to use glucocorticoid and 164 patients discontinued to use glucocorticoid. In the multivariate analysis, age (Odds ratio (OR)0.98), MTX dose (OR1.09) and glucocorticoid dose (OR0.88) were independently predictive factors of discontinuation of glucocorticoid. When we adjusted age, disease duration, sex, disease activity, RF/ACPA, glucocorticoid dose by using PS matching, 105 pairs were extracted. There were obvious significant differences between 24 patients (22.9%) in the L group and 43 cases (41.0%) in the H group ($P = 0.007$), where glucocorticoid was discontinued at one year after the initiation of bDMARD.

Conclusion: This cohort study investigated the association with discontinuation of oral glucocorticoid and MTX dose in the patients treated with bDMARD. TBCR revealed that, in the clinical practice, glucocorticoid use was decreasing in the patients treated with bDMARD. MTX dose at the time of initiation of bDMARD was predictive factor of discontinuation of glucocorticoid. A higher dose of MTX associated with discontinuation of glucocorticoid in the patients treated with bDMARD.

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TNF ALPHA INHIBITORS INFLUENCE THE CARDIOVASCULAR RISK IN RHEUMATOID ARTHRITIS PATIENTS

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Background: Rheumatoid arthritis (RA) patients die because of cardiovascular diseases more often than the general population. Nowadays RA is considered as an independent risk factor for cardiovascular disease (CVD). A long-lasting inflammatory process, immunological dysregulation and endothelial dysfunction play an important role in the pathogenesis of progressive atherosclerosis in RA [1,2].

Objectives: The aim of the study was to assess the influence of biological treatment on cardiovascular risk factors in RA population

Methods: 38 RA patients with active disease (DAS28>5.1) and 22 age and sex matched healthy controls were enrolled in the study. All the patients were treated with TNF alpha inhibitors (n=20 etanercept, n=18 adalimumab), with the background methotrexate (10-25mg weekly). The clinical state was assessed at the beginning of the study and 6, 12 and 18 months after. All the patients underwent the following tests: DAS 28 assessment, basic laboratory measurements (including homocysteine levels, fibrinogen, lipid profile), electrocardiogram (ECG), flow-mediated dilation (FMD) and carotid intima-media thickness (cIMT) assessment and echocardiography. The local bioethics committee approved the study (KB-753).

Results: 27 woman and 11 man (71% and 29% respectively) with high disease activity despite long-term treatment with synthetic disease modifying antirheumatic drugs were enrolled in the study. Mean age was 52 years (SD ± 9.6), 95% of the patients were RF-positive, 63% anti-CCP-positive. Of them 24% were current smokers, 58% had arterial hypertension and 66% had concomitant hypercholesterolaemia. In comparison to the control group, there were statistically significantly higher values of cIMT in RA patients. Moreover there was a positive correlation between cIMT thickness and duration of RA. A reduction of cIMT (mainly during first 12 months) and an increase in FMD values were observed in the course of the treatment ($p<0.05$). The increase in FMD value (0.03 vs 0.37) correlated with decrease of the inflammatory markers, which may be a result of an improvement in endothelial dysfunction due to anti-TNF-alpha treatment. The decrease in total cholesterol ($p<0.05$), LDL cholesterol ($p=0.03$) and Castelli risk index ($p<0.05$) were also noted, no changes of the doses of cholesterol-lowering therapy were done during the follow up. There was a positive correlation between homocysteine and uric acid levels and there was a statistically significant decrease in both parameters in the course of the treatment (11.31 vs 8.91 [mol/l] and 5.92 vs 4.77 [mg/dl], respectively). Normal left ventricular geometry was observed after 12 months of biological therapy in patients with concentric remodeling of the left ventricle. Furthermore echocardiography revealed a significant decrease in LVEDD, IVSD, LA, FS, RVEDD after 18 months of the biologic treatment.

Conclusion: Our results confirm earlier data that TNF alpha inhibitors may favorably influence on endothelial dysfunction. Besides TNF inhibitors can protect against increase in relative wall thickness of left ventricle. The anti-TNF alpha therapy not only reduces the activity of the disease in affected joints but also has a positive impact on selected cardiovascular risk factors in RA population.

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