Conclusion: Our study confirmed that the percentage of patients showing low adherence to therapy is relevant. Moreover, the association of lower adherence to treatments with higher values of the subjective clinimetric indexes suggests to pay attention to the apparent ineffectiveness or loss of efficacy of therapy.

References:

[1] Morisky DE et al. J Clin Hypertens 2008; 10:348–354.

Disclosure of Interests: None declared

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AB0185 PROSPECTIVE PROFILE OF URINE METABOLOME IN RHEUMATOID ARTHRITIS

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Background: Rheumatoid arthritis (RA) is a chronic inflammatory disease characterized by increased mortality and associated with metabolic disorders. Since the metabolomic profile is known to vary in response to different inflammatory conditions, metabolome analysis could substantially improve diagnosis and prognosis of RA.

Objectives: To analyze the urine metabolome profile in RA patients and correlate it with disease activity changes over 12 months

Methods: Seventy-nine RA patients, according to ACR/EULAR 2010 classification criteria, between 40 and 70 years old, were recruited and followed for 12 months. Metabolome analysis was performed by Nuclear Magnetic Resonance spectroscopy (NMR), resulting in the identification of 93 metabolites in urine collected at the baseline and after 12 months. Frequency analysis, Pearson Correlation and Multivariate data analysis with orthogonal projections to latent structures (OPLS) method were performed and a statistical significance was considered as $p < 0.05$.

Results: The study population was characterized by the majority of women (86.7%), mean age of 56 years old, around 80% with positive anti-CCP or Rheumatoid Factor. During the one year of follow-up, there was no substantial variation in the DAS28 measurement (baseline: 3.8, after 12 months: 4.0). There was no significant correlation between the metabolome pattern and DAS28 score ($p > 0.05$) over time. However, multivariate analysis (OPLS-DA) demonstrated an adequate differentiation of the population with 0.92 of accuracy (Q2: 0.72 and R2: 0.89). There was a significant increase of L-cysteine, choline, L-Phenylalanine, creatine, L-histidine, oxalacetic acid and xanthine, and a decrease of L-threonine, taurine, butyric and gluconic acid ($p < 0.05$) during the follow-up, metabolites that are involved in the skeletal muscle metabolism.

Figure 9: OPLS-DA score plot of all metabolite features.

Figure 32: Significant features identified by Random Forest. The features are ranked by the mean decrease in classification accuracy when they are permuted.

Conclusion: The observed biomarkers indicate, as expected, that the RA metabolic profile is associated with inflammation injury and skeletal muscle amino acid metabolism. Correlations with disease activity changes was compromised by the stable disease status during the 12 months. More studies evaluating correlations with skeletal muscle function and mass are underway.

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AB0186 NO PREDICTIVE VALUE OF ADALIMUMAB SERUM LEVELS AND ANTI-ADALIMUMAB ANTIBODIES AT TIME OF ADALIMUMAB FAILURE FOR PREDICTION OF RESPONSE TO THE NEXT bDMARD

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Background: After adalimumab treatment failure, TNFi and non-TNFi bDMARDs are equally viable as subsequent treatment in RA. However, preliminary data suggest that anti-drug antibodies (ADA) and adalimumab serum levels (ADL) predict response to a subsequent TNFi [1].

Objectives: To assess the association of presence of ADA and/or low ADL with response to a subsequent TNFi bDMARD or non-TNFi bDMARD.

Methods: A retrospective cohort study to assess the predictive value of ADA and ADL for response to a subsequent TNFi or non-TNFi bDMARD in RA patients. All RA patients who received adalimumab (standard dose, ≥ 3 months) and subsequently switched to another TNFi or a non-TNFi (rituximab, tocilizumab, abatacept) in the Sint Maartenskliniek or Radboud University Medical Centre between January 2012 and January 2018 were considered for inclusion in the current study. Further inclusion criteria were the availability of (random timed) serum samples between ≥ 8 weeks after start, and ≤ 2 weeks (for ADL) or ≤ 12 weeks (for ADA) after discontinuation of adalimumab, and clinical outcome measurements (DAS28-CRP/BSE) between 3-6 months after treatment switch. Serum samples were derived from a period of biobanking at every visit of RA patients and an observational cohort study including consecutive bDMARD starters.

The primary outcome of this study was the association between ADL or ADA and EULAR good response (DAS28-CRP/ESR based) to the subsequent bDMARD. When DAS28-based response was unreliable due to glucocorticoid use, or low baseline DAS28 (if switching due to adverse effects), judgement of the rheumatologist was used.

A drug-tolerant competitive enzyme-linked immunosorbent assay (Sanquin, the Netherlands) was used to quantify ADA, and thereafter, ADL was determined via an ELISA. Reference values were ≥ 5 $\mu\text{g/ml}$ for ADL and < 12 AU/ml for ADA [2,3]. Treatment was blinded for ADL and ADA levels.