Figure 1. Differences in values of nephropathy markers in the patients with early RA, depending on TNF- α level.

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SAT0136 THE INFLUENCE OF HDL-CHOLESTEROL AND CRP ON INCREASED INSULIN RESISTANCE AND IMPAIRED BETA-CELL FUNCTION IN PATIENTS WITH RHEUMATOID ARTHRITIS

G. G. Ristić¹, V. Subota², D. Stanisavljević³, B. Glišić¹, M. Petronijević¹, D. Stefanović¹. ¹Department of Rheumatology and Clinical Immunology; ²Institute of Medical Biochemistry, Military Medical Academy; ³Institute of Medical Statistics, Faculty of Medicine, University of Belgrade, Belgrade, Serbia

Background: Increased CRP in RApts is associated with lower levels HDL cholesterol. HDL cholesterol may enhance insulin secretion and stimulate glucose uptake into skeletal muscle, adipose tissue, and liver.

Objectives: To investigate whether low HDL, alone or in combination with CRP, could explain increased insulin resistance (IR) and impaired β -cell function in RA pts in comparison to healthy controls.

Methods: The study population included 127 nondiabetic subjects (90 RA pts and 37 matched controls). We determined body mass index, waist circumference (WC), and presence of metabolic syndrome (MetS). All pts were on disease modifying antirheumatic drugs, 65.6% on steroids (none on steroids >10 mg/day), and 27.8% on biologic therapy. Laboratory analyses included hsCRP, glucose, insulin, and C-peptide (as a marker of insulin secretion). Insulin resistance (IR) was calculated using the updated Homeostasis Model Assessment (HOMA2-IR), based on fasting plasma glucose and specific insulin concentrations. The output of the HOMA2 model was calibrated to give IR of 1 as normal. HOMA2-B indicates the potential of β -cells to compensate increased IR. Lack of compensatory rise of HOMA2-B implied impaired β -cell function.

Results: IR was detected in 74.4% of RA pts and in 54.2% controls, $p=0.025$. RA pts had significantly higher concentration of specific insulin, C peptide, and HOMA2-IR than controls, while HOMA2-B was not statistically different. Both groups were comparable regarding all other factors known to affect glucose metabolism (age, WC, presence of MetS). We found significant differences in inflammation markers between RA pts and controls: ESR 29.5 (14–44) vs. 16.0 (10.0–20.0); hsCRP 5.5 (2.8–15) vs. 3.0 (1.8–3.9); $p<0.000$ for both, as well as in lipids, especially HDL concentration 1.5 ± 0.4 vs 1.60 ± 0.3 , $p=0.027$ and a number of the pts with low HDL (37.8 vs 13.5%, $p=0.007$). Univariate regression analysis revealed significant positive effect of HDL on logHOMA2-B (β 0.099, 95%CI 0.029–0.169, $p=0.006$) and negative, but not significant effect on HOMA2-IR. In the logistic regression, after adjustment for HDL concentration, significant differences for HOMA2-IR and insulin became less important but still persisted (Table), while significance for C peptide became more prominent. When we added the influence of inflammation, this favourable effect of HDL-cholesterol on C-peptide disappeared.

Conclusions: RA pts had higher IR and impaired β -cell function in comparison to healthy controls. The augmentation of statistical significance for C peptide, as a marker of insulin secretion, after adjustment for low HDL-cholesterol and significant effect of HDL on logHOMA2-B implicate its important role in disturbances of glucose metabolism in RA.

Disclosure of Interest: None declared

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SAT0136 THE INFLUENCE OF HDL-CHOLESTEROL AND CRP ON INCREASED INSULIN RESISTANCE AND IMPAIRED BETA-CELL FUNCTION IN PATIENTS WITH RHEUMATOID ARTHRITIS

G. G. Ristić¹, V. Subota², D. Stanisavljević³, B. Glišić¹, M. Petronijević¹, D. Stefanović¹. ¹Department of Rheumatology and Clinical Immunology; ²Institute of Medical Biochemistry, Military Medical Academy; ³Institute of Medical Statistics, Faculty of Medicine, University of Belgrade, Belgrade, Serbia

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Laboratory parameters	RA pts (N=90)	Controls (N=37)	P	Adjusted for HDL	Adjusted for CRP	Adjusted for HDL+CRP
Glucose (mmol/l)	4.8 $\pm$ 0.6	4.7 $\pm$ 0.8	ns	/		
Insulin (pmol/L)	68.5 (50.2–102.7)	55.3 (36.0–69.1)	0.008	0.018	0.048	0.054
C peptide (pmol/L)	785 (520–1010)	600 (450–880)	0.046	0.025	0.078	0.066
HOMA2-IR	1.4 (1.0–2.3)	1.2 (0.8–1.4)	0.008	0.019	0.046	0.053
HOMA2-B	148 (116–190)	141 (114–158)	0.186	0.136	0.258	0.165
HOMA2-IS	70 (46–100)	84 (71.0–132)	0.010	0.017	0.074	0.089

Conclusions: RA pts had higher IR and impaired β -cell function in comparison to healthy controls. The augmentation of statistical significance for C peptide, as a marker of insulin secretion, after adjustment for low HDL-cholesterol and significant effect of HDL on logHOMA2-B implicate its important role in disturbances of glucose metabolism in RA.

Disclosure of Interest: None declared

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