htd
hrvatsko torakalno društvo

TORAKS 2019

9. Kongres Hrvatskog torakalnog društva
9th Congress of Croatian Thoracic Society

Hotel Westin Zagreb
10.–13. 4. 2019.



LIPID PROFILE (BASIC AND CALCULATED) IN PATIENTS WITH STABLE CHRONIC OBSTRUCTIVE PULMONARY DISEASE

MARKELIĆ I.[1], Vukić Dugac A.[1, 2], Popović-Grle S.[1, 2], Samaržija M.[1, 2], Rogić D.[3, 4], Rako I.[4], Rumora L.[3]

[1] University Hospital Centre Zagreb, Zagreb, Croatia
Clinical department for respiratory diseases Jordanovac

[2] University of Zagreb, Zagreb, Croatia
School of medicine

[3] University of Zagreb, Zagreb, Croatia
Faculty of Pharmacy and Biochemistry, Department of Medical Biochemistry and Hematology

[4] University Hospital Centre Zagreb, Zagreb, Croatia
Clinical Institute of Laboratory Diagnostics

Objective: Introduction: There is a limited number of studies investigating lipid profile in patients with chronic obstructive pulmonary disease (COPD). Therefore, the role of dyslipidaemia in COPD pathogenesis remains unclear. This study aimed to investigate differences in complete lipid profile (basic and calculated) and inflammatory parameters between healthy individuals and patients with stable COPD.

Material and methods: There were 232 subjects included in the present study - 137 with stable COPD and 95 healthy volunteers. Blood samples were collected, and the analysis of lipid and inflammatory parameters were determined.

Results: We observed increased levels of high-density lipoprotein cholesterol (HDL-C, $P < 0.038$) and atherogenic



index of plasma (AIP = log (TG/HDL-C), P = 0.003) in COPD patients compared to healthy individuals. Total cholesterol (TC, P < 0.001), low-density lipoprotein cholesterol (LDL-C, P < 0.001), triglycerides (TG, P= 0.008), atherogenic coefficient (AC = (TC-HDL-C)/HDL-C, P < 0.001), Castelli's risk index I (CRI I = TC/HDL-C, P < 0.001), Castelli's risk index II (CRI II = LDL-C/HDL-C, P < 0.001), non HDL-cholesterol (NHC, P < 0.001), and TG/HDL-C ratio (P = 0.002) were significantly decreased in patients with COPD. In addition, increased concentrations of C-reactive protein (CRP) and fibrinogen, as well as increased number of total leukocytes and monocytes, (P = 0.001, P = 0.002, P = 0.001, P < 0.001, respectively) were observed in COPD.
Monocyte to HDL-C ratio (MHR) combines inflammatory and lipid profile parameters. It was increased in COPD group of patients (P = 0.005).
Positive correlation between fibrinogen (r = 0.275, P = 0.007) and total leukocytes (r = 0.549, P < 0.001) with MHR was observed.
Moreover, AC (r = 0.508, P < 0.001), AIP (r = 0.632, P < 0.001), CRI I (r = 0.508, P < 0.001), CRI II (r = 0.439, P < 0.001), TG (r = 0.437, P < 0.001), and TG/HDL-C ratio (r = 0.632, P < 0.001) positively correlated with MHR.
Finally, all of the parameters, that showed statistically significant results in univariate logistic regression, were selected for multivariate logistic regression analysis. CRI II, MHR, CRP and fibrinogen gave the best combination for COPD prediction with an AUC of 0.815 (95% CI = 0.759 - 0.863, P < 0.001) and correctly classified 72% of COPD cases.
Conclusions: According to our results, dyslipidaemia could play an important role in systemic inflammation underlying COPD. A more comprehensive approach is suggested for better assessment of COPD. We suggest that MHR could be a helpful diagnostic tool in COPD.