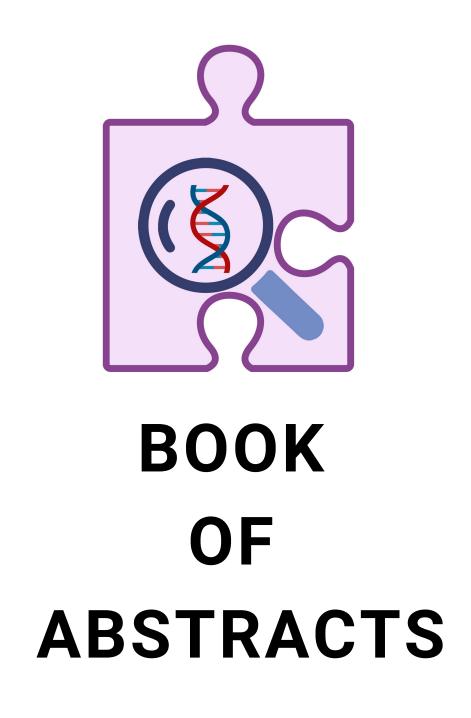
1ST INTERNATIONAL CONFERENCE FOR YOUNG SCIENTISTS

BIOMARKERS OF CIVILIZATION DISEASES

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Pro- and anti-inflammatory biomarkers in non-alcoholic fatty liver disease and hypertension patients

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Non-alcoholic fatty liver disease (NAFLD) affects up to 50% of patients with hypertension (HT). The objective of our study was to assess the changes in antiinflammatory systems (kallistatin, IL-10) and pro-inflammatory activity (IL-1ß and highsensitivity CRP (hsCRP)) in patients with NAFLD under the influence of concomitant HT. 63 patients with NAFLD and HT and 52 patients with isolated NAFLD were examined. Plasma kallistatin, IL-10, IL-18 and hsCRP levels were evaluate using ELISA. Kallistatin levels in patients with NAFLD and HT were 65.03 ng/ml (95% CI 61.38; 68.68), which was significantly lower than in the isolated NAFLD group (83.42) ng/ml (95% CI 81.89; 84.94), p<0.001) and control results (111.70 ng/ml (95% CI 106.14; 113.22), p<0.001). The level of IL-10 in the group of NAFLD and HT also reached minimal values (12.69 pg/ml (95% CI 11.93; 12.95) against 14.34 pg/ml (95% CI 13.27; 14,34) in the group with isolated NAFLD (p<0.001) and 16.19 pg/ml (95% CI 15.15; 17.74) in the control group (p<0.001)). The opposite results were observed in the study of IL-1 β content, which was increased in the NAFLD and HT group (17.55 pg/ml (95% CI 17.06; 19.73) versus 15.72 pg/ml (95% CI 15.25; 17.44) in the isolated NAFLD group (p<0.001) and 8.26 (95% CI 7.79; 8.46) in the control group (p<0.001)). Patients with NAFLD and HT had an increase in hsCRP (7.90 mg/l (95% CI 7.96; 8.75) versus 6.55 mg/l (95% CI 6.47; 7.57) in the group with isolated NAFLD (p<0.001) and 2.07 mg/l (95% CI 1.83; 2.85 mg/l) in the control group (p<0.001)). It has been shown that with HT progressing in NAFLD patients, the kallistatin level substantially reduces (p<0.001, p=0.011 for HT severity and BP grade) along with IL-10 levels decrease (p<0.001) and IL-1 β (p<0.001) and CRP levels (p<0.001) increase. Thus, patients with NAFLD and HT are likely to experience changes in biomarker status toward a proinflammatory state and deepening of these deviations with the progression of concomitant hypertension.